THE PILL

Addressing the Scientific and Ethical Questions of the Abortifacient Issue

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INTRODUCTION
INTRODUCTION

THE ABORTIFACIENT QUESTION IS BUT ONE AMONG MANY:
OTHER CONSIDERATIONS REGARDING PILL USE

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The present collection of articles is designed to assist those struggling with the question of whether the birth control pill is sometimes an abortifacient—i.e., whether or not it sometimes causes abortions. Answers to this question should be well-examined rather than arrived at lightly or hastily, and the Center has therefore prepared this booklet to help people reach a scientifically-informed, ethically sound, and God-honoring conclusion on this matter.

However, CBHD does not wish to convey to the reader that the abortifacient question is the only question that couples should consider when making the decision whether or not to use the birth control pill. Other considerations are also worthy of attention, such as God’s design for marriage and the family, the purpose(s) of sexuality, attitudes about child-bearing and child-rearing, the proper response to economic and social problems, the relationship between God’s original design of nature and the current operations of the “natural” world, and the proper way to give expression and shape to biblical concepts like God’s sovereignty, human responsibility/stewardship, and the blessing of children. The Center has therefore included this introduction as a means of encouraging the reader to ponder prayerfully some of these broader issues surrounding the decision whether or not to use (or recommend use of) the birth control pill.

Even within the Christian community, it is often assumed that apart from abortifacient concerns, there are no genuinely moral questions to grapple with when considering whether to go on the pill. This assumption often silently governs the character of discussions that emerge from the pulpits, small-group Bible studies, and pre-marital counseling sessions in our local congregations. CBHD does not share this assumption, since the decision to use a drug related to reproduction often has both medical and moral aspects that need to be carefully weighed by those who take and prescribe it.

This introduction is also designed to heighten the reader’s awareness that he or she may be predisposed to resonate more with some of the articles than others as a result of his or her assumptions and/or convictions regarding these broader issues. Those reading the materials in this booklet should be sensitive to how their views on these broader issues may inform their judgment of the various positions being articulated. The same thing can be said about the reader’s beliefs about the pill itself. For example, if a person is already committed to using, recommending, or prescribing the pill, it may be more difficult for him or her to fairly appraise arguments suggesting that the pill sometimes causes abortions. This is also true in the other direction: if a person has become convinced that the pill sometimes causes the death of unborn human beings, it may be more difficult for him or her to fairly appraise arguments suggesting that the pill does not have an abortifacient effect. It is likely a part of human nature for us to desire to have strengthened or confirmed what we already believe—or want to believe—and most people instinctively resist information that challenges
their dominant paradigms of thinking. This is particularly relevant in arenas (such as the reproductive arena) where people’s personal desires and personal interests are intermingled with the beliefs they hold.

**More Than a Catholic Issue**

Some believe that moral reflection on contraception is primarily (if not exclusively) a concern for Roman Catholics. It is certainly true that Roman Catholics have consistently been among the most articulate and outspoken critics of contraception, developing many of their critical arguments from a moral theology that they hope will resonate with both Catholics and non-Catholics alike. Nevertheless, it would be a mistake to reduce all religious reflection on contraception to the arguments outlined by Roman Catholics. Many of the assumptions often relied upon when using contraception, as well as contraceptive acts themselves, were vigorously criticized by Protestant thinkers from the time of Luther to the Anglican Church’s 1930 Lambeth Conference, which was the first institutional crack in the Protestant consensus against contraception. Although it sounds rather innocuous to modern ears, one-fourth of the Anglican bishops voted against the Conference’s resolution that “in those cases where there is such a clearly felt moral obligation to limit or avoid parenthood, and where there is a morally sound reason for avoiding complete abstinence, the Conference agrees that other methods may be used, provided that this is done in the light of the same Christian principles.” In a similar development on this side of the Atlantic, in 1931 several Protestant denominations criticized the Federal Council of Churches’ “Committee on Home and Marriage” for its statement defending family limitation and encouraging that laws prohibiting contraceptive sales and education be repealed: the Northern Baptists, Southern Methodists, and Southern Presbyterians claimed that the Committee had no authority to represent them on the subject, and the Presbyterians even withdrew their membership in the FCC for the next ten years. Even since that time, non-Catholic forms of religious opposition to contraception in general and/or the birth control pill in particular have been articulated. A recent example of this is the 2002 book *Open Embrace* by the Protestant couple Sam and Bethany Torode. Clearly, reflective religious opposition to contraception is not something limited to the Roman Catholic community.

Why might reflective Christians be reluctant to embrace contraception in general, or the birth control pill in particular, apart from the pill’s alleged abortifacient effects? With this question in mind, let us briefly consider a few of the different Christian voices that speak to the above-mentioned issues. While much has been written on each of these (see the bibliography at the end of this booklet), we will here just touch upon some of the different positions that have been offered.

**God’s Design for Marriage and the Family**

Various biblical texts seem to be relevant to a theology of marriage and family life: Genesis 1:28, Psalm 127:3-5, Song of Songs, Malachi 2:15, and 1 Timothy 5:4 are but a few. One thing that unites nearly all such texts is that Christians disagree on how best to use them in constructing such a theology. Take, as just one example, the statement to “be fruitful and
increase in number; fill the earth and subdue it” repeated in the early chapters of Genesis (Genesis 1:28, 9:1). Some Christians think this should be understood as an unchanging principle reflecting God’s unchanging purposes for the human race in general and for human marriages in particular. Some argue that this statement is a command, others argue that it is better read as a blessing or benediction, but both see it as having enduring relevance to all human beings—especially married human beings—and not merely to the original (or post-diluvial) inhabitants of the earth. From this, some draw the implication that attempts to limit procreation are not in line with the good and great purposes God has benevolently given to humans. Yet others suggest that using the tools of technology—including contraceptive technology—allows humanity to better fulfill its God-given mandate to “subdue” the earth. Still, other Christians view the statement as in some sense already fulfilled, with the implication that this text does not provide current Christians with any strong moral reason to reproduce. In any event, some argue that whether one interprets the passage as a command or a blessing, the details of how we are to best carry it out are not specified in Scripture. Similar things could be said about other pertinent biblical texts. The point is not that all of these various interpretations are equally good, but simply that as Christians, we need to approach and apply the biblical materials in a careful, humble, and informed manner.

The Purpose(s) of Sexuality

Almost all Christians recognize that God has given sexuality to human beings for a number of related reasons, among which are procreation, unification of husband and wife, and pleasure. This consensus, however, does not continue once questions are raised about the relative priority of each of these purposes, or about the precise nature of the interrelationship between these purposes. Most specifically, there is debate surrounding whether the priority among, and interrelationship between, God's varied purposes for human sexuality imply anything about the inseparability of these purposes. For those who believe that these purposes are inseparable, there are various ways to explain just what this means. For example, most do not think that sexual intercourse is immoral in situations where natural limitations separate these purposes—for example, when infertile or already-pregnant couples engage in sexual intercourse, well aware that this act will not result in the conception of a new child. However, some Christians do believe there is something improper about deliberately separating these purposes via human design and intervention. For example, some think it is not good for married couples to deliberately separate the unitive aspect of sex from the procreative aspect of sex, whether the couple is using either the tools of modern technology to achieve this separation (such as contraceptive devices like the pill or reproductive interventions like in vitro fertilization) or when the couple is using merely the power of planning (such as coitus interruptus or natural family planning). We are aware that even phrasing the issue this way may invite questions (e.g., is it better to accent the similarities between NFP and other forms of family planning, or is it better to accent their differences?). This, too, illustrates the enduring complexity of discussing these issues and the need for careful reflection on them.
Attitudes about Child-bearing and Child-rearing

A cluster of topics related to attitudes about child-bearing and child-rearing impinge upon the question of contraception in general and the pill in particular. For example, a woman may desire and/or be encouraged to use the pill as a way of postponing (or preventing) the birth of children, and sometimes she may desire or be encouraged to postpone (or prevent) the birth of children so that she can flourish alongside men in the modern world. Thus, part of the reason some women take the pill is so that they can have the same sorts of economic and employment opportunities as men.

But should a Christian woman adopt this approach? Some maintain that a Christian woman should be encouraged to work out her salvation primarily in the context of being a godly wife to her husband and a godly mother to her children. On some versions of this view, a woman’s use of the pill can be tantamount to ignoring her distinctively and divinely given feminine capacities. Some Christians question why certain cultural assumptions of achievement—income, education, working “outside the home”—should have any bearing upon the dignity of a human being or should have any claim to being universal standards. They ask why women should feel like they need to act like men (in even the positive, uplifting senses of this phrase) in order to be equal to men in the eyes of God. Those who raise such questions should not be taken as endorsing the idea that women are merely reproductive vessels whose value comes from bearing and raising children. Rather, the animating idea is that women are not merely their own in the same way that men are not merely their own: both have procreative capacities that should not be ignored, and both should be willing to sacrifice what the world calls “success” in order to remain open to the blessings of parenthood. The way this animating idea gets fleshed out in practice may be different in many cases for men than women, but the principle is still the same.

Other Christians, while rejecting the economic justification for using the pill, emphasize that their individual callings from God lead them to be employed in the varied spheres outside of domestic life. These Christians sometimes point out that a woman’s choice between flourishing inside the home or flourishing outside the home is often not an either-or decision. Many women use the pill for a few years and then decide to have children, often choosing to become full-time mothers. Still others may choose to forego having children altogether. Although most of these women are not motivated by the desire to be like men, they seek to recognize the diversity among women’s callings. Using the pill within the context of married life is thus interpreted not as a betrayal of femininity, but as an appropriate way of allowing particular women to live out the lives to which they believe God has called them.

Another example of how the pill relates to attitudes about child-bearing and child-rearing centers around female responsibilities concerning contraception. Some have questioned why it has to be the woman who bears the brunt of the responsibility; why it has to be her body that gets chemically altered on a regular basis in order to achieve the goals of limiting or spacing children. Many have pointed out that the pill can have various negative effects upon a woman’s bodily and emotional health, quite apart from whether it makes her endometrium more hostile to an embryo seeking to implant. While these negative effects may vary from woman to woman in type, duration, and intensity, their possibility should be carefully weighed by any couple considering use of the pill. The proper care of one’s body is an arena of life that we are called to bring under Christ’s direction, if for no other reason
than the fact that we are stewards rather than owners of our bodies (1 Corinthians 6:19-20). It is instructive to note in this context that husbands are admonished to care for their wife’s body as they would their own (Ephesians 5:28). Finally, the woman’s emotional and psychological well-being plays an important role in the overall health of the marriage and/or family of which she is a part. Consequently, she should be very cautious about taking any drug that might disturb or impair that well-being and, by implication, the overall health of the marital and/or familial relationships.

Economic and Social Problems

Some Christians think that contraception is part of the solution to demographic difficulties like the overcrowding of cities, the inequalities between first- and third-world countries, and so on. Others, however, either flatly deny this assertion or suggest that such problems can be solved more effectively in other ways. Some also claim that the introduction of contraception into a society can bring with it a range of new social and economic problems that either add to or exacerbate the problems contraception was introduced to alleviate. To support this claim, these critics point to the rapid increase in certain social problems in Western cultures that began at roughly the same time as the pill: sexually transmitted diseases, divorce, single motherhood, breakdowns of the traditional family unit, and so forth.

Both critics and supporters of the pill point out the conceptual and historical connections between the pill, contraception in general, and other social issues related to human sexuality and human reproduction. Conceptually, for example, contraception has often been closely linked to the issue of abortion, since both involve a dramatic redefinition of reproductive liberty and both have often been justified by conceptualizing children as more of a burden to be avoided than a blessing to be embraced. Even apart from the abortion question, Christians have pointed out the historical fact that many proponents of the pill have not viewed it merely as a tool for alleviating population pressures in third world countries. In addition, the pill’s proponents have often hailed it as an icon for the new sexual moralities, which explicitly reject the biblical world view. The pill’s ability to separate sexual expression from human reproduction enabled extra-marital sexual expression to flourish, “liberating” men and women to have extra-marital sexual relationships without the responsibilities of child-bearing and child-rearing. These facts are sometimes enlisted to encourage resisting the pill, or at least using it only after careful and prayerful deliberation. While it is invalid to fault a technology merely because of the historical circumstances that led to its development, being aware of the original motivations behind a technology like the pill can nevertheless give us a fresh perspective from which to carefully evaluate that technology.

Nature and God’s Design

Regarding the relationship between God’s original design of nature and the current operations of the “natural” world, some Christians emphasize the ways the regular cycles of the female body, or the intricate design of our reproductive systems, bear witness to the
beauty, majesty, and wisdom of God. From this they may conclude that any disruption of this regular cycle is an affront to God’s creative prerogatives in designing human beings in the ways only He knows is best. Or, they may conclude that the cycles and design are there precisely so that we can space and limit the number of children we think God wants us to have, and that taking advantage of this divinely given pattern is an activity in which our Creator expects us to engage. Others, however, emphasize the discontinuity between “nature” as God created it to be and “nature” as it exists now that the fall has occurred. They suggest that the regularities of a woman’s body, and the design and functioning of individual reproductive organs, either cannot be used to support a moral prohibition on altering them or else support a moral mandate to alter them. Since all of nature (and our nature as human beings) was touched by the fall and requires the creative-redemptive activity of God and humans to reverse the effects of the fall, so, too, is this true for this part of nature (and our nature as human beings).

God’s Sovereignty, Human Responsibility, and the Blessing of Children

The convictions of Christians differ and align in unpredictable ways when it comes to integrating divine/human responsibilities in procreation. Some say God is in control of what goes on in the womb and that some (if not all) attempts to control or regulate what goes on there represent an attempt to usurp His rightful dominion in this arena. Others utilize the notion of God’s sovereignty in precisely the opposite direction, arguing that since God is ultimately in control, he has the ability to override or veto any contraceptive action that human beings take. What is important, according to this second representative view, is that human beings act as thoughtfully and conscientiously as they can in reflecting the character and purposes of God—both in the daily decisions they make and in the life-long projects they pursue—and that they trust God to correct them or adjust them if they should become out of line.

Others suggest that the biblical teaching on human responsibility, divine stewardship, and the believer’s “freedom in Christ” (Galatians 2:4; 5:1,13) should govern any approach to the issues of contraception in general and the birth control pill in particular. This is probably the more dominant view among today’s Protestants who reflect on questions like these. As mentioned above, some view contraception as an appropriate method for exercising one’s divinely appointed stewardship over the natural order. If limiting or spacing children is necessary for societies to flourish in the way God intended, and if using contraceptive measures (including but not limited to the pill) is necessary for such limiting or spacing, then contraception is at least morally permissible, and perhaps morally required. Others view contraception as a way of encouraging various other things to flourish—for example, a marriage, an already existing family, an education, a ministry, a career, a lifestyle. As the previous list indicates, not all of these goals are materialistic.

Finally, the way in which children are a “blessing” (Psalm 127:3-5; 128:1-4) is disputed among Christians. Some maintain that this is an unconditional truth regardless of the number, timing, or spacing of children. These Christians point out that it is rather odd to resist deliberately something God regards as a blessing, and they invite us to consider whether we would actively resist other blessings from God (say, a monetary windfall) with the sort of deliberation and diligence with which we resist children. This resistance, they
conclude, is a result of lacking faith in God or of lacking agreement with God’s revealed presentation of the value of children. But others think that this description of children as a blessing does not support such an unconditional stance, especially when understood in light of the culture in which it was articulated. They suggest that large families were especially valued in biblical times in part because of the agricultural nature of the economy and because of the high infant mortality rate, and that it is therefore misleading to argue that children are likewise an unconditional blessing in our modern industrialized world. Even setting this cultural point aside, some suggest that honoring children as a blessing doesn’t speak to the question of regulating their number or spacing.

Hard Cases

The foregoing comments illustrate how even if the pill does not sometimes cause the death of early embryos, this does not automatically mean that using the pill is permissible, and even if it is permissible, this does not automatically mean it is wise or beneficial, all things considered (1 Corinthians 6:12, 10:23). However, we have not considered the assumption that if the pill really is sometimes abortifacient, one should never take or prescribe it. Although this assumption might appear obvious, there seem to be situations where it may not apply. For one thing, it is possible to take a drug with a potential abortifacient effect in a manner that precludes this effect from occurring. For example, a woman who is convinced that the pill sometimes has an abortifacient effect, yet wants to use the pill to regulate her endometriosis (a painful medical condition in which functional endometrial tissue is found outside of the uterus), might choose to use the pill while taking other careful or absolute measures to avoid conception. In cases like these, the idea is to take the necessary steps to ensure that she does not conceive an embryo who might subsequently fail to implant because of an abortifacient mechanism of the pill. Of course, the husband in such a situation would likewise bear full responsibility for implementing these safeguards.

A more difficult case is that in which a woman takes a drug with a possible abortifacient effect, fully knowing that this effect may occur. Though this may appear to be an open-and-shut case of immorality, even here cases can be imagined that are analogous to those in which aborting a fetus in order to save the mother’s life is morally permissible (e.g., when an ectopic, or “tubal,” pregnancy occurs). In such cases, it seems that the mother’s life would have to be at risk were she to become pregnant in order for it to be morally permissible to take the pill, and the pill would have to be the only technology with a realistic chance of saving her life by preventing pregnancy. We recognize that some women may reject this line of reasoning, since they are willing to forfeit their own lives for the sake of their unborn child. While not intending to undermine or condemn that choice, it seems that such sacrifice is not morally required, even if it is morally permitted. However, it would be a rare situation in which the pill was the only option for a woman whose life would be endangered by pregnancy. Even if pregnancy were to pose such a threat, it seems that the woman could almost always use other contraceptive activities to ensure that she does not conceive an embryo who might subsequently fail to implant because of an abortifacient effect of the pill.
Conclusion

This introduction has merely touched upon some of the various considerations to be taken into account when making a decision regarding use of the birth control pill. We have therefore included a bibliography of additional resources at the end of this booklet. We encourage you to read the material in this booklet carefully, approach the decision regarding use of the birth control pill prayerfully, and remember that the question of whether the birth control pill is abortifacient, while important, is but one among many relevant questions.
PERSONAL ACCOUNTS
PERSONAL ACCOUNTS

When my wife and I were married in 1975, we carefully considered the question of whether the birth control pill sometimes causes abortions. At that time, our best reading of the literature led us to the conclusion that the original normal-dose estrogen/progesterone pill (in contrast to the low-dose estrogen/progesterone pill and the progestin-only “mini-pill”) indeed worked by blocking ovulation and not by preventing implantation of the embryo. According to the literature at that time, the possible exception to this might be during a woman’s first cycle on the pill. My wife therefore took the pill for one cycle before we were married and thus was in her second pill cycle at the time of our marriage. Over the years, we were careful to ensure that she be prescribed only the normal-dose pill.

We were aware of the occasional literature claiming that the normal-dose pill might block implantation of the embryo. We also knew women who became pregnant while taking the normal-dose pill and gave birth to a healthy child. These women not only ovulated, but conceived and had a successful implantation! Likewise, people who are not on birth control pills usually do not become pregnant during a cycle in which they are sexually active. Why not? Do they fail to conceive, or does conception occur but the embryo fail to implant? One will never know.

Did we do the right thing? We think we did. We did not have children while we completed our education and training. Later we did. We now have 3 wonderful children, the oldest of whom is applying to colleges.

—Anonymous

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Our experience with the contraceptive pill was short-lived. We were going to get married in a couple of months and wanted to avoid a honeymoon pregnancy. The pill seemed to be the most sensible option at the time. We were careful to choose the least “potent” pill, not because of any moral ideal but because my wife wanted to avoid as many side effects as possible. But once she started taking the pill, she began describing her emotions as “flat.” Her body felt “heavy.” She was agitated and temperamental. She even felt that her sexual drive had been adversely affected.

Around this time, we first heard claims that the pill was sometimes abortifacient. We felt that if there was any question or doubt concerning whether the pill caused abortions, we would err conservatively. So, the abortifacient question, coupled with the fact that we had become concerned about the pill’s side effects, caused us to stop using the pill immediately. Even when we realized that the evidence regarding the abortifacient issue was far from conclusive, we still decided against use of the pill for different and, we felt, no less important reasons.

My wife and I felt that the pill made her more “man-like” by generally flattening out her emotional responses and experiences. Both of us felt that the way the pill altered her body chemistry and
interrupted her natural cycles was in itself ethically questionable since her body was not intended to work that way. After struggling through infertility and learning that some women had become completely infertile because of prolonged pill use, we feared that our inability to become pregnant may have been caused in part by our use of the pill (though later we found this not to be the case). Even so, we felt that there were too many reasons to not use—or even support use of—the pill. We both think it’s important to embrace and understand the beauty of what it means to be a woman, with all her reproductive complexity. For us, the pill does not symbolize the empowerment of women but, rather, exemplifies the antithesis of femininity.

—Anonymous

Though I have long been aware of the controversy concerning whether the oral contraceptive pill (OCP) sometimes causes abortions, I have in the past year become increasingly familiar with the arguments alleging that the pill is sometimes abortifacient. Though I am concerned that some of the articles presenting such arguments refer loosely to “facts” that are somewhat inferential or cannot be readily verified, I am nevertheless hesitant to wholly disregard the possibility that the pill may at times cause the death of an early embryo.

My consideration of this issue thus far has not prompted me to stop prescribing the OCP. Some of the articles claiming that the pill may act as an abortifacient emphasize the fact that ovulation sometimes occurs in pill users, with the conclusion that conception therefore also occurs. However, while ovulation does indeed occur infrequently in women taking the OCP, the pill also functions to inhibit sperm transport, thereby decreasing the likelihood of conception even further. Since it takes a healthy non-contracepting couple an average of 7-8 months to conceive, it would seem that infrequent ovulation combined with impaired sperm transport in couples using the pill would render the chances of conception quite unlikely, at best. At this point in time, there is no way to prove that an abortion sometimes results in the event that conception does occur in a woman who is taking the pill. I am not ready to refuse to prescribe the OCP based on circumstantial, inferential, or theoretical evidence.

I believe that the “principle of double effect” may serve to justify the prescription and use of the OCP (though some would challenge my application to the matter at hand). I believe that the notion of “intent” is a very important factor in the decision to prescribe the pill. While I do prescribe the OCP for the purpose of contraception, I do not prescribe “emergency contraception” (e.g., the “morning-after pill”), which is administered with the intent that an already begun human life will be terminated. I prescribe oral contraceptives to prevent conception, albeit perhaps imperfectly. My intent is not to prevent implantation of an embryo, but to prevent ovulation and conception (though I am coming to believe that inhibition of implantation of an embryo may infrequently occur). I believe that “emergency contraception,” because it is often used to prevent or interrupt the implantation of an embryo, is wrong and irresponsible—both on the part of the patient and her physician.

While I continue to prescribe the OCP, I know my patients quite well and am careful to make those who are Christians or who have pro-life convictions aware that some people believe that the pill sometimes causes abortions. By doing so, I allow them to search their own consciences and reach a
decision on this matter for themselves, since I am not yet fully convinced of the pill’s abortifacient effect.

Early in my Christian walk (though I personally believed that abortion was “wrong”), I continued to take a pro-choice stance on the matter because “who was I to force my beliefs on someone else?”. At that time, a very wise, mature, non-judgmental Christian woman said to me, “Just keep walking with God; He will make the correct position clear to you.” She was right: the inconsistencies in my thinking became clear to me a short time later, and I abandoned my “pro-choice” views. I trust that God, who is Faithful and True, will be so in this instance as well, convicting me if necessary and guiding me to proper action.

—Susan M. Haack, MD, FACOG

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After my husband and I got engaged, we began seriously considering what we should do about birth control. He was planning to be in school full-time for several years, and I was planning on working to provide for us. I felt like we needed to use some form of birth control in order to survive, but I was struggling with the idea of whether or not we should use contraception at all. It seemed like we were trying to control our future too much. I finally decided that if God really wanted us to have a baby, our use of birth control would not stop Him.

Once we had decided that we would use some form of birth control, the huge question became, “Which one?” I had heard that some contraceptives are abortifacient and was very concerned about this possibility. I also wanted a method that was reliable. We began talking to some of our Christian friends and our pre-marital counselor from my church about this matter. I was shocked to hear that most of them had never even considered using any form of contraception other than the pill. They had also not heard that it could be an abortifacient. The few that had actually questioned their doctors about this were told that the pill is not an abortifacient. I knew that it was possible for conception to occur while on the pill and that the embryo might then not implant. That sounded like abortion to me.

Several of my friends had chosen the natural family planning (NFP) method. This method allows couples to know when the woman is ovulating by monitoring her natural secretions. It appealed to me in that the husband is encouraged to actively participate in the training and carrying out of the method. The interesting thing was that even though all of my friends were Protestant, they found that a Catholic pregnancy clinic had the best (and, it seemed, only) counsel, information, and programs pertaining to NFP.

I talked to a woman from this clinic, and she was very helpful. She validated the fact that I was questioning what we should do, rather than just doing whatever was easiest. We were planning to use the natural family planning method, and then I learned that it is often not successful if the woman has irregular periods. I was very irregular and anticipated that the stress of being newly married might exacerbate this condition. So, we again started to consider other methods.
We thought about the diaphragm, but were told that it was not the best route for someone who had never had sex before. We began to think about other barrier methods, such as condoms, but then learned about a Catholic doctor who was very well liked by his patients. Several friends assured me that he would be willing to discuss my concerns about the pill. I immediately made an appointment with him. He listened to my concerns and then gave his counsel. He said that he was completely against abortion and would never perform one. He also said that he believed that the pill is not likely abortifacient if taken consistently. He encouraged me to be diligent in taking it each day at the same time. I was still a bit unsure because of the conflicting information about the nature of the pill’s action; however, in weighing the options and feeling strongly that I did not want to get pregnant in our first year of marriage, I decided to use this form of contraception.

I take the pill consistently just as he suggested and have never missed a day, yet sometimes I still feel unsure of our decision. How can I be against abortion, but then take a pill that could be abortifacient just because having a baby would not be convenient for me right now? Am I being hypocritical? Am I inconsistent in my ethics? I am still not sure. I do know that it is very important to consider this matter carefully. I find it very disheartening that so many who are outspokenly opposed to abortion do not even know that there is some question about whether or not the pill is abortifacient.

We are planning to stop using the birth control pill soon, and we do not think that we will use it again for contraception. This is due to both the moral dilemma and health issues.

—Anonymous

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There are many reasons for not using the pill: there is something special about being in harmony with nature and not fighting against it; it is both wrong and selfish for a man to expect a woman to pump her body full of chemicals, even well-tested chemicals, so that she is available for his pleasure; it’s expensive; mistakes happen and abortions occur; etc.

But the aspect that most strongly persuades me to reject the pill is that not using it results in a better balanced relationship between husband and wife. Any relationship is composed of a wide range of activities—talking; physical intimacy; sex; eating together; going to a movie; enjoying a quiet time alone together, with one spouse reading and another writing or listening to music. How can a couple achieve the best balance of activities in their marriage? So many relationships degenerate because they overemphasize one aspect—e.g., going skiing—at the expense of another—e.g., sharing a quiet evening at home together. Many relationships seem to be more about sex than about sharing all of life’s joys and burdens together. Choosing to use natural means of fertility control helps couples to achieve a natural balance of the many and varied dimensions of marriage. A relationship then doesn’t degenerate into a caricature of itself (two people constantly having sex and never leaving the bed), but, rather, fosters a well-balanced, fully human experience. The natural way is the way to be most fully alive, most fully human, most fully in union with your other.

—Anonymous
My personal experience with the OCP/abortifacient controversy goes back to the early 90’s. I had been prescribing OCPs for a dozen years or so before our family practice group decided to stop prescribing them as a method of birth control (though we continued to prescribe them for medically therapeutic reasons.) The physicians in our group had arrived at this decision after a year-long process of prayer and discernment, informed by medical literature culled from many sources. A half-page story detailing this decision soon hit our local press in western Pennsylvania, resulting in more than a little bit of regional fallout.

Our concerns were primarily focused upon:

1) the abortifacient effect of OCPs, especially of low-dose forms (an effect also linked to most chemical contraceptives, as well as the IUD)
2) the removal of one more barrier (the threat of pregnancy) to promiscuous sexuality and its undesirable consequences
3) the separation of sex from procreation
4) the unwanted (and sometimes unknown) side effects of the chemicals
5) the unwitting/undiscerning/irresponsible view of most Christians on this topic (few believers seem eager to let Scripture truly serve as a guide for their lives, and those that do may sometimes draw the line at the bedroom door)
6) the “medicalization” of what is fundamentally a personal and family issue (family planning), which typically can be managed quite well without direct medical intervention in most cases

We found a large proportion of our patients to be agreeable to choosing other contraceptive options when we explained our reasons for no longer prescribing the OCP. About a third of our patients readily agreed with our stance and expressed relief at being offered other options. Another third of our patients felt inconvenienced, but were nevertheless willing to forego use of the OCP. The remainder of our patients chose to obtain their contraceptive “care” (and sometimes the rest of their care) elsewhere.

We realize that those who believe that the OCP is abortifacient are sometimes perceived as equating promotion and use of the OCP to a subversive plot for eugenics or other devious ends. However, this accusation goes too far, as a plot isn’t required for a large proportion of the population to be led astray—simply the lack of solid training in biblical ethics and a bit of medical misinformation will do it nicely. Yet, I do believe that those wishing to take advantage of this medicalization effect (such as Planned Parenthood) have put forth a calculated effort to advance their own agenda via the OCP and other chemical contraceptives.

Coincidentally, some elements in the medical community have labored in the background to rework the operational definition of “pregnancy” from the traditional “fertilization” moment; thus claiming that pregnancy begins at “implantation.” This, of course, obviates the OCP/abortifacient argument somewhat. There is no scientific basis for this re-definition from embryologic, genetic or anatomic
Within about five years after our decision to no longer prescribe the OCP, I left private practice. I now primarily practice geriatrics, working with patients for whom the OCP issue is, of course, rarely relevant. My former partners continue to speak out against use of the OCP and to promote the benefits of other forms of fertility regulation.

—Richard Schamp, MD
FREQUENTLY ASKED QUESTIONS
(FAQ’s)
Frequently Asked Questions

1. How does the birth control pill work?

This question is at the very heart of the debate over whether the birth control pill sometimes causes abortions. According to FDA-approved information contained in the Physicians' Desk Reference (PDR), the pill has 3 possible mechanisms of action (although the possibility of the third mechanism actually occurring is the subject of great controversy). First, the pill acts to inhibit ovulation (its primary effect), thereby preventing the release of an egg (and the possibility of conception). Second, the pill causes a woman’s cervical mucus to thicken, hindering sperm travel to the egg (thereby also forestalling conception). These first two mechanisms are truly contraceptive. Third, the pill results in a thinning of the endometrium (uterine lining) such that implantation of an embryo (should the first two mechanisms fail and conception occur) is unlikely or impossible. Should implantation of a newly conceived embryo indeed fail to occur, the result would be an abortion.

Those who believe that the pill does sometimes cause the death of an early embryo maintain that this third mechanism comes into play at least on occasion, resulting in an early abortion. Those who do not believe that the pill is abortifacient deny the presence of this third effect and/or point to the very high or complete efficacy of the first mechanism (especially when the pill is taken correctly and in the absence of factors such as illness or drug interaction that could weaken its contraceptive effect). Existing scientific evidence appears insufficient to resolve this matter and is in fact invoked by those on both sides of this debate as offering support for their respective positions.

2. Why is there disagreement regarding the third (abortifacient) mechanism of the pill’s action?

Those holding to the “hostile endometrium” theory assert that the endometrium of a woman on the pill is less suitable for implantation of an embryo than is the endometrium of a non-pill user—and that this decreased suitability remains even if ovulation occurs. According to this theory, such decreased suitability is due to the endometrium being thinned and devoid of essential nutrients—conditions which, though they certainly do not prevent the possibility of implantation, greatly reduce its likelihood. As noted in question one, FDA-approved information on standard birth control pills (combination estrogen and progestin oral contraceptives) indeed states that “changes in the endometrium reduce the likelihood of implantation.” Despite this, some have challenged the very existence of a “hostile endometrium” once ovulation occurs (an event necessary for conception to take place). They point out that even in non-pill users the endometrium is “hostile” until the hormonal release accompanying ovulation renders it suitable for implantation. Although they are not able to offer conclusive scientific evidence that the endometria in pill users who ovulate would be similarly prepared for implantation, they maintain that current understanding of reproductive physiology suggests that this would be the case.
3. What do we know about the nature of the endometria in pill users who have ovulated?

A 1980 Bombay, India study by Chowdhury, et al. reportedly demonstrated that the endometria of pill users who had ovulated remained significantly atrophied (and were thus unlikely to permit implantation of an embryo). Those who dismiss the findings of this study point out that the progesterone level used to establish the occurrence of ovulation in this study (greater than 4 ng/ml) was significantly lower than that typically agreed upon by medical experts (greater than 9 ng/ml). Thus, they reason that ovulation had likely not occurred in these women; hence, the accompanying hormonal release (which they maintain would prepare a woman's endometrium for implantation) also had not occurred. Other studies have similarly been invoked by those on both sides of this debate as offering support for their respective positions.

4. If the pill doesn’t sometimes act to cause an abortion, why does the *Physicians’ Desk Reference* (PDR) say that it does?

Those who believe that the pill is indeed sometimes abortifacient don’t question the information provided in the PDR, but assert that it is in harmony with existing evidence indicating that the pill sometimes causes an abortion. Those who dispute the claim that the pill is abortifacient maintain that, rather than providing factual information, the PDR listing simply serves to increase sales (by indicating a high rate of efficacy) and provide legal protection (in the event of detected miscarriage).

5. Why are women sometimes advised to stay off the pill for a set number of months before trying to conceive?

Those who believe that the pill sometimes causes an abortion advise women not to conceive within the first three months of discontinuing pill use because they believe the pill produces an endometrium that is “hostile” to implantation—and that these hostile effects are not easily reversed, but linger for up to three months. Some physicians who do not believe that the pill is abortifacient encourage delay of pregnancy due to the simple fact that ovulation may not immediately resume upon discontinuing pill use, making it difficult to determine an accurate due date should conception occur. Other physicians who are unconvinced of the pill’s abortifacient effect do not advise their patients to postpone pregnancy after discontinuing pill use, since they determine their patients’ due dates by ultrasound.
ANNOTATED BIBLIOGRAPHY
ANOTATED BIBLIOGRAPHY


This chapter consists of an introduction (by Linda Bevington) and a “debate” between two proponents (Walter Larimore, M.D. and Randy Alcorn, M.A.) of the position that the birth control pill sometimes acts as an abortifacient and four proponents (Susan Crockett, M.D., Joseph DeCook, M.D., Donna Harrison, M.D., and Camilla Hersh, M.D.) of the view that existing evidence does not indicate that the pill sometimes causes early abortions. Larimore and Alcorn here espouse the “hostile endometrium” theory, which asserts that the endometrium in pill users is unlikely to permit implantation of an embryo, thereby resulting in an abortion. They also consider the ratio of normal vs. ectopic pregnancies in women on the pill, concluding that there is a higher incidence of ectopic pregnancies among pill users. They believe that this is indicative of the loss of embryos who try to implant normally in the endometrium. Larimore and Alcorn conclude their portion of the chapter by responding to some of the common reactions of those who disagree with their position.

Crockett et al. begin their segment of the chapter by considering the scientific data relevant to this issue and by questioning the validity of the “hostile endometrium” theory. They also state that while progestin-only pills and Norplant are associated with an increased ectopic pregnancy rate, the standard birth control pill is not linked to such an increase. Before addressing other aspects of this controversy, the authors elaborate on the common ground shared by those on both sides of this debate by presenting a sanctity of life ethic that extends to the moment of conception. Though they challenge the legitimacy of Larimore and Alcorn’s ethical analysis, they ultimately conclude that the abortifacient debate may be rightly regarded as a “disputable matter” (such as that delineated in Romans 14) that should preclude the passing of judgment.


This condensed version of Alcorn’s full-length book presents his (and Dr. Walt Larimore’s) personal struggle regarding the question of whether the birth control pill sometimes causes abortions and summarizes several of the main lines of medical argument germane to this debate. Alcorn presents the “hostile endometrium” theory, which he prefers to characterize by dropping the word “theory” (because it is too weak) and replacing the term “hostile” with the phrase “less
hospitable” (since all one needs to make the central argument is the less strong claim that the endometrium is less hospitable in a woman on the pill). Alcorn argues that those who maintain that the FDA’s conclusions are not supported by scientific studies ought to prevail upon that agency to change their statements, instead of merely asking people to disregard them. He also maintains that differences in intrauterine/extrauterine pregnancy ratios for pill users suggest an abortifacient effect and argues briefly against several medical objections to the alleged abortifacient effect of a compromised endometrium. Alcorn also includes an assessment of the ethical issues raised by this controversy.


This is one of the first publications attempting to counter the alleged evidence that the pill sometimes causes abortions. The article consists mainly of a thorough (and sometimes technical) review of the medical literature. The authors’ conclusion is that “there is no direct evidence in the literature to support the third proposed [abortifacient] mechanism of action [of combined oral contraceptives i.e., the pill].” One of their main arguments is that the thinner endometrium becomes thicker and more receptive to implantation once ovulation has occurred, thereby overriding any negative effects of the pill. The authors advise readers to carefully study each hormonal contraceptive on its own without drawing conclusions from studies where several types of contraception were studied together, and they follow their own advice by discussing progestin-only pills (POPs), progestin implants (e.g., Norplant), injectibles (e.g., DepoProvera), and combined oral contraceptives (COCs). In their discussion of COCs, they argue that an ideal endometrium is not necessary for successful implantation and that there is no need to postulate an abortifacient effect of the pill in order to explain the discrepancy between ovulations and pregnancies among pill users. In their three appendices, the authors 1) present a theological mandate for using contraception in developing nations, not as “population control imperialism” but as an expression of Christian compassion in alleviating suffering, 2) apply Paul’s discussion of “disputable matters” in Romans 14 to the current debate, and 3) offer a careful critique of the Chowdhury study and its use in these debates.


Asserting that it is not sufficient to equate endometrial receptivity merely with endometrial thickness, Wilks considers the role of various “implantation factors” and the manner in which the function of such factors is affected by the pill. Wilks’ premise is that the hormonal content of the pill has an adverse effect on implantation factors such as interleukin-1β (IL-1β) and platelet-activating factor (PAF). Wilks also addresses the likelihood of “break-through ovulation” occurring in women who take the pill, concluding that imperfect patient compliance, gastro-intestinal illness,
and drug interactions increase the incidence of ovulation in pill users.


In considering the medical evidence regarding the question of whether the birth control pill sometimes acts as an abortifacient, Larimore discusses the “hostile (or unreceptive) endometrium” theory and the ratio of ectopic (or tubal) pregnancies versus normal pregnancies in women who take the pill. While acknowledging that the data does not definitively prove that the pill sometimes has an abortifacient effect, he concludes that the evidence is “extremely strong.” Larimore takes issue with those physicians who assert that they need not inform their patients about this controversy until conclusive evidence of an abortifacient effect becomes available. In considering the importance of *intention*, Larimore appeals to the “principle of double effect,” concluding that most arguments seeking to justify a Christian’s use of the pill are invalid.


In this article, Goodnough challenges several premises put forth by those who believe the oral contraceptive pill (OCP) is sometimes an abortifacient. First, he responds to Randy Alcorn’s belief that the OCP may allow ovulation in 10-30% of pill users’ cycles. Goodnough asserts that studies cited by Alcorn (and others who share his position) are incorrectly construed as indicating a significant rate of ovulation in women who take the OCP. Goodnough also posits several challenges to the “hostile endometrium” theory and questions the claim that an unreceptive endometrium boosts the ectopic/normal pregnancy ratio observed in pill users. He also considers the proper definition of the term “abortifacient” and concludes his chapter with an examination of the ethical implications of this dilemma.


In this response to Joel Goodnough’s article in the 17:1 issue of *Ethics & Medicine*, Wilks offers a lengthy defense of his treatment of the question of “breakthrough ovulation” (appearing in the 16:1 issue of *Ethics & Medicine*). Wilks also counters Goodnough’s conclusion regarding the expression of integrins (and the significance of such) in women who take the pill, his views as to the proper definition of an “abortifacient,” his interpretation of the “principle of double effect,” and several other matters that are germane to the abortifacient controversy. Colliton also takes issue with Goodnough’s article, disagreeing with his definitions of “pregnancy” and “abortifacient” (among
other matters) and highlighting pertinent information concerning the pharmaceutical development of the birth control pill. Following Wilks’ and Colliton’s remarks, Goodnough weighs in on their comments, responding to their objections in defense of his original premises.


In this response to Joel Goodnough’s *Ethics & Medicine* (17:1) article, Stanford and Larimore seek to correct what they see as certain inaccuracies about medical facts and mistakes of moral reasoning. They argue that when elective abortions are accounted for, the rate of pregnancies on the pill rises from the rate Goodnough cited. They argue that the “turned on endometrium” theory is not (as Goodnough puts it) “somewhat speculative,” but, rather, “completely speculative.” They claim that Goodnough’s occasional reliance on dated and/or non peer-reviewed sources led to a misrepresentation of their views. For example, they refer back to their original article in *Archives of Family Medicine* to dispute Goodnough’s claim that they had lumped together the progesterone-only mini-pill and the combined oral contraceptives. They also argue that Goodnough did not discuss the feature of the “principle of double effect”—which states that “there must be no other way to produce the good effect”—and they argue that since certain forms of natural family planning are just as effective as oral contraceptives, most arguments in favor of pill use may be reduced to arguments of convenience. The authors assert that when this convenience is compared with the possible abortifacient nature of the pill, the moral thing to do is to avoid risking prenatal life by not taking or prescribing the pill. They emphasize that mere good intentions are not enough, since abortions resulting from pill use are just as real regardless of what the intentions might be.


In responding to Joel Goodnough’s *Ethics & Medicine* (17:1) article, Alcorn argues against what he sees as certain logical and ethical weaknesses of Goodnough’s assertions and seeks to correct a misquotation of Alcorn’s book *Does the Birth Control Pill Cause Abortions?* (Gresham, OR: Eternal Perspective Ministries, 1998). He argues that the central issue in labeling the pill an abortifacient, as well as the central issue in the minds of most concerned people, is the effect of the pill, not the intentions behind its use. Alcorn’s main concern, standing at the heart of his disagreement with Goodnough, is that good intentions and sincerity do not mitigate the seriousness of the fatal results which may occur as a result of pill use. He insists that at the very least, physicians should not withhold evidence that many doctors believe supports the *Physician Desk Reference (PDR)*’s and pill manufacturers’ claim that the pill sometimes prevents implantation. In response to one of Goodnough’s criticisms, Alcorn claims that there is nothing wrong with citing evidence from a study that does not agree with the point being made by the citation, since such is done by every researcher (including Goodnough). He argues that if someone thinks the claims of the *PDR* and pill manufacturers are (as Goodnough says) “speculative” and/or motivated by carelessness or public relations, he has the responsibility to take this accusation to the FDA and pill manufacturers themselves. He claims that to assume that the embryo always implants when breakthrough
ovulation and fertilization occur would be “wishful thinking” and that the burden of proof (henceforth unmet) falls on those who suggest that the endometrium of pill users is not truly hostile. Finally, Alcorn corrects a misquotation of his own book: when Goodnough quoted his statement that the morning-after pill “increases the chances of doing what it [the OCP] already sometimes does—cause an abortion,” he left out the important word “sometimes,” thereby inadvertently misleading the reader to think that Alcorn directly equates the morning-after pill and the OCP.


In her response to Joel Goodnough’s article (*Ethics & Medicine* 17:1), Sister Mirkes objects to Goodnough’s claim that evidence allegedly demonstrating that the oral contraceptive pill (OCP) sometimes causes abortions is indirect, inconclusive, and/or unsubstantive. In doing so, she responds to Goodnough’s treatment of matters such as ovulation rates on the pill, prevention of implantation, the incidence of ectopic pregnancy, and the definition of the OCP. Mirkes also calls into question Goodnough’s application of the “principle of double effect,” reinterpreting this principle in a manner that runs counter to Goodnough’s interpretation. Mirkes concludes her article with the statement that “the prescription of the OCP, even when judged primarily from a medicinal rather than from a moral perspective, is not a good human act.”
INTRODUCTION

LINDA K. BEVINGTON, MA
GUEST EDITOR FOR THE DEBATE

Does the birth control pill (“the Pill”) sometimes cause abortions by acting to prevent implantation of an early embryo rather than to prevent conception? This question has sparked a growing controversy regarding whether use of the birth control pill is morally permissible. Debate over this matter—which likely will escalate as awareness of the issue increases—is primarily fueled by a lack of consensus regarding the mechanism by which the Pill acts. Many physicians and patients would almost certainly reject the use of any contraceptive if they were convinced that it causes abortions.

Physicians and researchers who have devoted themselves to examining whether such a link exists have drawn markedly different conclusions as to whether current scientific evidence suggests that the Pill is indeed abortifacient. Although the authors of the debate presented here disagree over interpretation of the evidence, they are all committed Christians who recognize the magnitude of this issue and its potential implications for Pill-prescribers, Pill-takers, and, of course, the unborn. As a result, they have separately engaged in serious study and evaluation of the available evidence and have prayerfully reflected upon their findings.

This chapter was born out of the recognition that persons committed to upholding the sanctity of life should be provided with the opportunity to examine the evidence and arguments behind both positions in order to facilitate their own prayerful reflection on this critical matter. The authors were therefore invited to produce a manuscript defending their position on this issue. Each author team was provided with the other team’s manuscript so that they could tailor their points to the arguments being presented by those holding the opposing view. Shortly before publication the authors were again given the opportunity to make changes to their manuscript before the final version was published.

In addition to laying out the opposing sides of the birth control pill issue, this debate is designed to address a larger question—namely, how should we develop an ethical position on a life-or-death issue when the scientific data required to draw a definitive conclusion is controversial or not yet available? The ethics of using the Pill, when some believe that it may act as an abortifacient, is merely a case in point. Even if scientific data were to resolve this particular matter tomorrow, the debate would still be illuminating, as we will always lack the data to definitively resolve certain ethical controversies. The authors warrant our deep gratitude and appreciation for their willingness to make this vitally important debate accessible.
Both of the present authors have independently reviewed the medical literature concerning the potential abortifacient effects of the birth control pill (“the Pill”). We (like most in the Christian tradition) would define an “abortifacient effect” as an effect that ends the life of a pre-born child after fertilization or conception. Because we both advocated using the Pill for years, we hoped to discover that it does not cause abortions. Unfortunately, the scientific data led us to the opposite conclusion.¹

Hostile Endometrium

Because space is limited, we will look at just two lines of evidence that indicate the Pill sometimes causes early abortions. First, a large number of medical studies document that the uterine lining (endometrium), the “home” new human beings implant in, is dramatically changed by the Pill. Although this is not direct proof of early abortions, it is indirect proof of high order.

This evidence is so well accepted in the medical world that the Food and Drug Administration’s approved product information for the Pill in the Physicians’ Desk Reference says, “Although the primary mechanism of action is inhibition of ovulation, other alterations include changes in the cervical mucus which increase the difficulty of sperm entry into the uterus, and changes in the endometrium which reduce the likelihood of implantation.”² This latter mechanism means that pre-born children may fail to implant in the uterus, at least on occasion, because of the Pill’s effects. An independent clinical pharmaceutical reference also contains this assertion.³ (Those who say these FDA-approved assertions are false should, in our opinion, prevail upon the FDA to change their statements and not simply ask people to disregard them.)

Reproductive endocrinologists have demonstrated that Pill-induced changes cause the endometrium to appear to be “hostile” or “poorly receptive” to implantation, at least in patients who are infertile.⁴ One group of researchers has written that these “changes have functional significance and provide evidence that reduced endometrial receptivity does indeed contribute to the contraceptive efficacy of [the Pill].”⁵ In other words, secular researchers consistently point out the abortifacient effect of the Pill. To date, no published studies have refuted these findings.

Magnetic Resonance Imaging (MRI) reveals that the endometrial lining of Pill users is consistently thinner than that of nonusers⁶—up to 58 percent thinner.⁷ Recent and fairly sophisticated ultrasound studies have all concluded that endometrial thickness is related to the “functional receptivity” of the endometrium in women—at least in
women undergoing fertility treatments. Other studies have shown that when the lining of the uterus becomes too thin, implantation of the pre-born child (called the blastocyst or pre-embryo at this stage) does not occur. The loss of a pre-born child is obviously abortifacent.

The minimal endometrial thickness required to maintain a pregnancy ranges from 5 to 13 mm, whereas the average endometrial thickness in women on the Pill is only 1.1 mm. These data lend credence to the FDA-approved statement that “changes in the endometrium . . . reduce the likelihood of implantation.” If these indications are wrong, they should be refuted with evidence, not dismissed with wishful thinking.

Some physicians have theorized that when ovulation occurs in Pill-takers, the subsequent hormone production “turns on” the endometrium, causing it to become receptive to implantation. However, there is no direct evidence to support this theory, and there is at least some evidence against it. First of all, after a woman stops taking the Pill, it usually takes several cycles for her menstrual flow to increase to the volume of women who are not on the Pill. This suggests to most objective researchers that, rather than recovering quickly, the endometrium is slow to recover from its Pill-induced thinning. Second, the one study that has looked at women who have ovulated on the Pill showed that after ovulation the endometrium is not receptive to implantation. In determining whether ovulation had occurred, this study looked for serum progesterone levels that are lower than those viewed by many (but not all) current researchers as indicators of ovulation. Therefore, critics of this study say it is possible that the 15 subjects studied with progesterone levels above 4 ng/ml simply had not ovulated. However, it is equally—or perhaps more—plausible to say: It is possible that all (or most, or even some) of the 15 subjects had ovulated.” The bottom line is that this study, the only one of which we are aware, seems to refute the theory (unproved and never published in a peer-reviewed medical publication) that when a woman ovulates on the Pill her endometrium will always and completely normalize prior to implantation.

Intrauterine/Extrauterine Pregnancy Ratio

A second line of evidence of the Pill’s abortifacent effect is this: If the Pill has no post-fertilization effect, then reductions in the rate of intrauterine pregnancies in Pill-takers should be identical to the reduction in the rate of extrauterine (ectopic/tubal) pregnancies in Pill-takers. Therefore, an increased extrauterine/intrauterine pregnancy ratio would constitute evidence for an abortifacent effect.

Two medical studies allow review of this association. Conducted at seven maternity hospitals in Paris, France, and three in Sweden, the studies evaluated 484 women with ectopic pregnancies and control groups of 389 women with normal pregnancies who were admitted to the hospital for delivery during the same time period. These studies were designed, in typical fashion for “case control” studies, to determine the risk factors for a particular condition (here ectopic pregnancy) by comparing one group of individuals known to have the condition with another group of individuals not having the condition. Both of these studies showed an increase in the extrauterine/intrauterine pregnancy ratio for women taking the Pill. Researchers who have reviewed
these studies have therefore suggested that “some protection against intrauterine pregnancy is provided via the Pill’s post-fertilization abortifacient effect,” i.e., the loss of the pre-born child.\textsuperscript{19}

That some other studies using a control group of non-pregnant women have not found a risk of increased ectopic pregnancy for Pill-users is not surprising. This apparent contradiction can be explained by the fact that contraceptive methods do indeed reduce the likelihood of pregnancy—whether normal or ectopic. Therefore, in determining whether there is an association between use of the Pill and an increased risk of ectopic pregnancy in women who get pregnant while taking the Pill, it is important that the subjects studied already be pregnant.\textsuperscript{20} In our opinion (and in the opinion of the majority of the published studies done by secular researchers), the criticism of those who contend that the studies using pregnant controls are invalid is scientifically unfounded and ill-informed.

In other publications and in a much more detailed fashion, we have discussed these and other lines of evidence, citing dozens of scientific studies, as well as researchers and experts in numerous fields. We encourage interested readers to look more deeply into these studies and arguments.\textsuperscript{21} Despite this evidence, some pro-lifers state that the likelihood of the Pill having an abortifacient effect is “infinitesimally low, or nonexistent.”\textsuperscript{22} Though we would both very much like to believe this, the scientific evidence does not permit us to do so. In our opinion, it would prevent most objective observers from doing so also.

**Ethical Implications**

When Christian physicians fail to reach a consensus, how should the Christian public respond? How should Christians make a decision—and how should pastors counsel church members—about taking the Pill, if the risk to unborn children is arguably real, but is believed by some not even to exist?\textsuperscript{22}

This is a significant question, since the Pill is used by about fourteen million American women each year and sixty million women internationally. Thus, even an “infinitesimally low” portion (say one-hundredth of one percent) of 780 million pill cycles per year globally could represent tens of thousands of unborn children lost to this form of chemical abortion annually. How many lives have to be jeopardized for pro-life believers to question the ethics of using the Pill? This is a question with profound moral implications for innumerable Christians.

The following are some of the common reactions we have encountered to the evidence that the Pill causes abortions.\textsuperscript{23}

“I don’t trust the evidence.”

Most of those unsatisfied with the evidence do not appear to have closely examined it. Our natural tendency is to believe whatever supports a position in
which we have vested interests and to disbelieve whatever contradicts it. We should resist this
tendency and allow the evidence to determine our position—no matter how uncomfortable it makes
us. We must each ask, “If this evidence doesn’t convince me, is there any evidence that would? Is it
possible that my vested interests in this issue are blinding me to the evidence? If there wasn’t
something to lose by believing and acting upon the evidence, would I still reject it?”

Note that scientists without pro-life convictions virtually always acknowledge that the Pill
prevents implantation on occasion. Since this conclusion does not threaten them, they consistently
come to it from the existing data. The evidence concerning the Pill is disturbing to pro-life
Christians, but accepting it will help us make more informed and ethical choices.

“If we don’t know how often abortions happen, why shouldn’t I take the Pill?”

Imagine a hunter uncertain whether a movement in the brush is caused by a deer or a child. Should
the hunter shoot? Imagine a woman driving at night who sees a dark figure ahead on the road,
which may be a child or just a shadow. Should she continue to drive or put on the brakes? What if
there is a 50 percent chance that it is a child? 30 percent? 10 percent? 1 percent? How certain does
she have to be before acting? Should not the benefit of the doubt go to life?

We believe the Pill should not be used or recommended to others unless and until it is
proven safe for all unborn children. To date, that proof does not exist. Furthermore, based upon the
ethical principle of double effect, one should not consider as ethical an action that may cause harm
to human life (a bad effect) if there is another way of producing the good effect. In the case of
elective birth control, effective but non-abortifacient contraceptive methods are available, and these
include more than just barrier methods. Multiple studies over the past twenty years have proven the
Creighton University approach to natural family planning to be even more effective than the Pill. In
a more recent controlled study, the researchers demonstrated that (per 100 couples over

one year) the pregnancy probability was 0.14 for perfect use with the Creighton method and 2.72 in
actual use—an efficacy at least as great as the Pill. In addition, the Billings Ovulation Method of
natural family planning is so simple that it is taught around the world to people who cannot read or
write.

Even if there were no effective alternatives to the Pill, it should not be used because the
evidence suggests that it sometimes causes abortions. But the fact is, there are effective non-
abortifacient alternatives to the Pill. Based upon the principle of double effect discussed above, it
seems reasonable to conclude that the Pill should not be used or recommended to those who believe
life begins at conception—unless and until the Pill is proven not to be an abortifacient. It appears
that such studies could be done; however, to date, the needed proof clearly does not exist. Until such
proof is available, the Pill should be considered a possible cause of death for pre-born children and
therefore cannot be ethically used, recommended, or prescribed by Christians for contraceptive
purposes.
“Spontaneous miscarriages are common—early abortions aren’t that significant.”

This argument boils down to: “Since God permits or causes minions of spontaneous abortions each year, it is acceptable for us to cause abortions too.” There is a big difference, a cosmic difference, between God and us! What God is free to do and what we are free to do are not the same. God is the Creator; we are the created. As the maker of human life, God has the right to take it. We do not. Spontaneous abortions in women not taking contraceptives are not our responsibility. Abortions caused by chemicals we choose to take or prescribe are.

“The Pill's alteration of the endometrium hasn’t been proven to cause abortions. It's only a theory.”

No one who is aware of the facts can honestly argue that the Pill does not significantly change the endometrium. Such a change is undeniable. Accordingly, imagine a farmer who has two places where he might plant seed. One is rich, brown soil that has been tilled, fertilized, and watered. The other is hard, thin, dry, and rocky soil. If the farmer wants as much seed as possible to take hold and grow, where will he plant the seed? The answer is obvious.

Some physicians correctly point out that some newly conceived children do manage to implant and temporarily survive in hostile places, such as the fallopian tube. Elsewhere we explain this apparent contradiction. But this in no way changes the obvious fact that many more children will implant and survive in a richer, thicker, more hospitable endometrium than in a thinner, less hospitable one.

“If this is true, why haven’t I heard it?”

There are many answers to this question, including: (1) Concerns about early abortions are not widespread, since preventing implantation is not of any ethical concern except to those who believe human life begins at conception. (2) Published reports of Pill-caused abortions are available, but are spread out in dozens of obscure and technical scientific journals. (3) Medical semantics have played a role in obscuring the Pill’s abortifacient mechanism. In 1976, the word “contraceptive” was redefined by the American College of Obstetricians and Gynecologists (ACOG) to include agents which prevent implantation. (4) The Pill is a multibillion dollar worldwide industry. Its manufacturers have tremendous vested interests in maintaining its status quo, as do most physicians, including pro-life Christians, who prescribe it.

“Using the pill will mean fewer children die in spontaneous abortions.”

Over one-half (some estimates range as high as 78 percent) of fertilized embryos are eventually spontaneously aborted. Some pro-lifers point out that the Pill, by lowering the rate of conception, will lower the total number of deaths due to spontaneous abortions. The logic seems to be that when we use a chemical that kills some children, we can take consolation in knowing that this same
chemical may prevent many other children from ever being conceived and therefore from dying “naturally.”

This is convoluted logic, and again it puts us in the place of God. If there are fewer miscarriages because of the Pill, it is not because the Pill brings any benefit to a pre-born child, but only because it results in fewer children being conceived. It is not that lives are being preserved, but simply that there are fewer lives to preserve. There is less death only because there is less life. Again, we must take responsibility for our choices, not for God’s.

“Without the Pill there would be more elective abortions.”

Suppose for a moment this were true. What is the logic? “If we go ahead and take action that will kill some children now, at least there may be other children, more of them, who won’t get killed.” The same approach could be used to deny drowning children access to a crowded life raft. This sort of pragmatism rings hollow when we put certain human lives at risk, without their consent, for the supposed good of others. However, the premise here is not true, as the small minority who would reject the Pill because it causes early abortions are the same people who would be the last ones to get a later abortion.

“Pill-takers don’t intend to have abortions.”

This is undoubtedly the case. Likewise, most Pill prescribers do not intend to cause abortions. Nevertheless, while the intentions of those taking or prescribing Pill may be harmless, the results can be just as fatal. In this sense, taking the Pill is analogous to playing Russian roulette, but with more chambers and therefore less risk per episode. In Russian roulette, participants usually do not intend to shoot themselves. Their intention is irrelevant, however, because if they play game long enough they cannot beat the odds—eventually someone dies. However, with Pill roulette, it is another person who may die. The fact that a woman will not know when a child has been aborted in no way changes whether or not it happens. The longer she takes the Pill, the greater her chance of having a silent abortion. The more a physician prescribes the Pill, the more likely he or she is to cause and be morally responsible for unrecognized abortions.

“I’m still comfortable prescribing the Pill because it’s primarily contraceptive and only secondarily abortive.”

Even if the Pill does not usually cause an abortion, whenever it does it is just as real an abortion as if that were its primary effect.
“You can't avoid every risk.”

Indeed, some risks are necessary. But Pill-takers unnecessarily put their pre-born children at risk. In fact, the very survival of these children is at stake. Regardless of the actual risk percentage, which is uncertain, a sexually active woman runs a new risk of aborting a child every time she takes the Pill. Furthermore, as discussed above, she has non-abortifacient options for birth control, such as modern natural family planning, that may be more effective than the Pill.

“There may be a risk of the Pill causing an abortion, but if so, it is very small.”

No one knows how small it is, but suppose it is very small. How much risk is acceptable risk? The answer, as discussed above, depends on the alternatives. There is no such thing as a car or a vaccine that poses no risk to one’s children. But there is such a thing as a contraceptive method that does not put a pre-born child’s life at risk. There are effective alternatives, such as natural family planning, that are as effective as—or more effective than—the Pill that do not and cannot cause abortions. It makes ethical, moral, and practical sense for Christians to learn more about these non-abortifacient birth control options. Unfortunately, physicians who ignore or deny the evidence indicating the abortifacient potential of the Pill are not likely to themselves seek such alternatives or to encourage their patients to do so.

“We shouldn’t lay guilt on people by talking about this.”

Scripture makes it clear that we are capable of doing wrong even when we are not consciously aware of it (Lev. 5:14-18; Job 13:23; Ps. 19:12; 139:23-24). We will each give an account to God of all we have done (Rom. 14:10; 2 Cor. 5:10). By coming to terms now with our sin and responsibility, we can to a certain extent preserve ourselves from having to face later judgment (1 Cor. 11:31). Because of the work of Jesus, God freely offers us pardon for everything—including actions taken in ignorance and sincerity that may have terrible and unintended results (Ps. 103:10-14). But to experience that forgiveness, we need to confess, repent of, and turn from our sin (1 John 1:9).

Some assume that one cannot set forth the evidence about the Pill without being guilty of legalism or spiritual abuse. Those who offer this argument do not give either truth or people enough credit. Is truth devoid of grace? Christ was full of grace and truth—so should we be. Are Christians incapable of handling difficult information or accepting God’s provision for guilt? Is it compassionate to hold back disturbing truth from people rather than share it with them so that they can make their own decisions about what to believe and seek the Lord’s guidance as to how to respond? The Christian life is not based on avoiding the truth but on finding and submitting to it.
“We shouldn’t tell people the Pill may cause abortions until we know for sure.”

Informed consent is a widely accepted ethical mandate of modern medicine. The physician’s failure to provide adequate information seriously jeopardizes the patient’s ability to make an informed decision. If this information is consciously withheld, it is a breach of ethics. Not telling women about the Pill’s potential abortifacient effect betrays a disrespect for their intelligence, their moral convictions, and their ability to weigh the evidence and make choices. Not teaching them about non-abortifacient options such as natural family planning is ethically indefensible.

If physicians and pastors make their patients and parishioners aware of evidence indicating the Pill may cause early abortions and later research indicates that this evidence was not valid, what will have been lost? Informed decisions will have been made based on all the available data, and women will have had an excellent option in natural family planning. But if physicians or pastors fail to disclose the evidence to those in their care and it turns out it was valid all along, then they will have withheld vital information that might have kept pre-born children from dying. If we really love our most vulnerable neighbors—the unborn children—we will want to protect and preserve them instead of imperil them through our silence.

Further study of this matter is indeed necessary. We would be delighted if such study contradicted the existing evidence and demonstrated that the Pill is incapable of causing even a single early abortion. However, unless and until such study surfaces, the evidence that the Pill causes unrecognized abortions—at least some of the time—is cumulatively so substantial that we dare not ignore it.

It is hard to imagine a more horrid irony than that followers of Christ would speak out against surgical abortions, yet repeatedly make choices that result in the chemical abortions of their own children. If the Pill causes abortions then the biggest threat to Satan’s success is that people become aware of this truth and act on it. The evil one’s vested interests in our blindness on this issue cannot be overstated.

It is not easy for pastors to speak out on this issue in their churches or for physicians to discuss it with their patients. However, pastors and physicians often have to address unpopular subjects. Why not this one? Some people will be angry and defensive, but most will be thankful and appreciative.

We owe people the truth, spoken in love (Eph. 4:15). “Speak up for those who cannot speak for themselves” (Prov. 31:8). The issue is not whether people will applaud our decision to address this subject; the issue is whether the audience of One desires us to do so. If God does, all other opinions are irrelevant.

To us, the final word on this debate has already been given: “This day I call heaven and earth as witnesses against you that I have set before you life and death, blessings and curses. Now choose life, so that you and your children may live” (Deut. 30:19).

References


11. McCarthy et al., “Female Pelvic Anatomy.”


13. J. L. DeCook et al., “Hormone Contraceptives: Controversies and Clarifications,” April 1999, available from ProLife Obstetricians, P.O. Box 81, Fennville, MI 49408, or by e-mail from prolifeob@aol.com.


19. W.L. Larimore and J.B. Stanford, “Postfertilization Effects”; Thorburn et al., “Background Factors” (the original data was reevaluated by Mol et al., “Contraception and the Risk”).


23. R. Alcorn, “Does the Birth Control Pill Cause Abortions?”


USING HORMONE CONTRACEPTIVES IS A DECISION INVOLVING SCIENCE, SCRIPTURE, AND CONSCIENCE

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Hormone contraceptives include combined oral contraceptives (COCs), injectables (DepoProvera), progestin-only pills (mini-pill, POPs), and the implant (Norplant).

The State of the Science

The idea that hormone contraceptives may occasionally cause a very early miscarriage or “mini-abortion” comes directly from the Food and Drug Administration-approved labeling requirements, arrived at by that government agency in cooperation with the manufacturer’s research literature. The primary mechanism of action of hormone contraceptives is to prevent ovulation; if this mechanism fails, sperm transport to the egg (and thus fertilization) will likely be impeded due to the contraceptive’s thickening of the cervical mucus. Hormone contraceptive literature also reflects the fact that such contraceptives produce a lining of the uterus (the endometrium) that is less vascular and less glandular than that normally seen six days after ovulation, when the fertilized egg would implant. The implication is that if a woman ovulates, and if the sperm gets through the thickened cervical mucus and fertilizes the egg, the altered uterine lining will make implantation difficult—and sometimes impossible—thereby causing the loss of the fertilized egg. This would indeed be a preimplantation abortion. It sounds logical, and the hormone contraceptive literature implies it may happen. (Hormone contraceptive literature is written for marketing purposes [“this contraception will prevent pregnancy”] and for legal protection [“you can’t sue us if you miscarry—we warned you”], as well as for patient education.)

However, this supposed abortifacient action is purely theoretical. The hormone contraceptive literature mentioning the altered uterine lining (the so-called “hostile endometrium”) is only accurate if a woman has not ovulated. If she has not ovulated, there is no chance for pregnancy anyway. If a woman “breaks through” the contraceptive action and does ovulate, a whole new hormone environment comes into play, which has seven days to prepare the lining for implantation. \(^2\) (Even if a woman is not using hormone contraceptives, on the day before she ovulates the lining of her uterus is also unfavorable to implantation, requiring this seven-day transformation.) \(^3\) A burst of hormones, called the follicle-stimulating hormone and the luteinizing hormone, stimulates ovulation. From the day before through the days following ovulation there is an outpouring of natural estrogen...
ten to fifteen times greater than hormone contraceptive levels—increased from 25 pg/ml to 250-
400 pg/ml) and of natural progesterone (twenty times greater than hormone contraceptive levels—
increased from 0.5 ng/ml to 10 ng/ml). Over this period of days, these hormones from the corpus
luteum of the ovaries transform the lining so that it becomes receptive to implantation. This is the
physiologic effect of these hormone levels on endometrial tissue. In the event of ovulation, these
hormones will be present transforming the uterine lining whether a woman is using hormone
contraception or not (since hormone contraceptives are not known to suppress corpus luteum
hormone output). This is likely the reason unexpected pregnancies in women using hormone
contraceptives do as well as any other pregnancies.

The abortifacient theory is not a fact. It fails to account for the essential information about
ovulation and its effect on the uterine lining. The concept of “hostile endometrium” is contrary to
the known physiologic effect of ovulatory

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estrogen and progesterone on the uterine lining. The FDA-approved labeling literature does not tell
people this (the government's or manufacturer's literature does not always tell us everything we
ought to know). Therefore, the misunderstanding is not the fault of concerned pro-life individuals
who are misinformed by the inadequate, and even misleading, FDA-approved labeling literature.

We are aware of the study conducted in the mid-1970s at the Institute for Research and
Reproduction of Bombay. This study claims to have demonstrated that fifteen women who ovulated
while using hormone contraceptives each showed an endometrium that was atrophic—that is, less
favorable to implantation by an embryo. The researchers' criteria for proving that ovulation had in
fact occurred was a progesterone level greater than 4 ng/ml. However, most medical experts agree
that progesterone levels must be greater than 9 ng/ml to indicate the occurrence of ovulation. This
is a glaring weakness in the Bombay study and renders the endometrial biopsy findings meaningless,
since it is very possible that the fifteen subjects studied with progesterone levels above 4 ng/ml
simply had not ovulated.

The abortifacient theory proponents point out another line of evidence that they think
suggests that hormone contraceptives are associated with an early abortifacient effect. They refer to
an increased risk, per pregnancy, of ectopic (tubal) pregnancy. The implication is that if there is an
increased tubal pregnancy rate, then there must also be an increased number of embryos that enter
the uterine cavity and are flushed out due to an inhospitable lining. The scientific literature does
indicate an increased tubal pregnancy rate with progestin-only pills and Norplant. However, this
increase is probably due to the progestin effect of slowing tubal motility—thereby increasing the
likelihood of an embryo implanting in the fallopian tube rather than the uterus. On the other hand,
the literature on combined oral contraceptives and DepoProvera does not show an increased tubal
pregnancy rate over normal.

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Most abortifacient theory proponents have lumped together all of the hormone
contraceptive agents—COCs, POPs, Norplant, and DepoProvera—as one type of agent. Problems
related to rates of ovulation, ongoing pregnancy, ectopic pregnancy, and abortifacient action have
been attributed to all four types alike. The result is a set of erroneous conclusions that impact ethical
decision-making regarding these medications. It is more instructive and accurate to review the
literature concerning these agents separately since they vary in action, complications, and effectiveness.

The present authors have conducted a careful, exhaustive review of the medical literature regarding hormonal contraceptives' mechanism of action, considering the four types of hormonal contraceptives separately. The discussion here summarizes their key findings.

We do not find substantive evidence of abortifacient action with use of hormone contraceptives. POPs are much less effective birth control than the other three types, although they have potential advantages for select patients. POPs and Norplant are associated with higher ectopic pregnancy rates, exposing the user to increased potential for morbidity and even mortality. This may constitute an unacceptable risk for the use of these products. On the other hand, the reviewed literature indicates that COCs and DepoProvera, with consistent and compliant use, are extremely reliable contraceptives. Their effectiveness depends on a high degree of ovulation suppression. Secondarily, they alter cervical mucus to largely impede sperm penetration. If these two mechanisms of contraception fail and conception occurs, the post-ovulatory hormone release from the corpus luteum would be expected to make the endometrium suitable for implantation. We find no evidence of abortifacient action and no demonstrable increase in ectopic pregnancy rates with COCs and DepoProvera.

The question of the mechanism of action of hormone contraceptives is a scientific one, which we as pro-life obstetricians-gynecologists have attempted to address by a thorough review of the existing scientific literature. As discussed above, there is ample evidence for us to believe that certain forms of hormone contraception are not abortifacient.

In light of the above scientific discussion on hormone contraceptives and their mechanism of action, we will now take a moment and address some of the underlying issues that have prompted this research.

The controversy regarding the mechanism of action of the commonly used contraceptives has threatened to split the pro-life medical community. Review of information currently being disseminated reveals some powerful and well-written rhetoric. However, the question of hormone contraceptives' mechanism of action is not one that will be illuminated by rhetoric. The mechanism of action of any medicine will not change based on how we feel about the medicine, who developed it, or on how eloquently its use is defended or opposed. How a medication works is a scientific question, and at least for some of the hormone contraceptives, we have ample information to say clearly how and at what point in the reproductive cycle they exert their action. However, it is not so much the science as the theological and ethical questions that heat this debate within the pro-life community.

The Common Ground

The pro-life community begins this debate by agreeing that human life must be protected from its earliest beginning. As pro-life physicians in a culture that is aggressively pro-abortion, we have all felt the pain and grief associated with advocating respect for and protection of human life from its very beginning. We have all suffered the ad hominem attacks from pro-abortion colleagues, and we also have grieved as we watch our patients devalue themselves and destroy their children. The worldview
of our pro-abortion colleagues is that human life is a product of random chance, as is animal life. So for them, our value as human beings comes from what we can do (especially what we can do for whoever is assigning us value). For the pro-abortion culture, our value is assigned to us by our human society and codified in law.

In sharp disagreement with this prevailing cultural worldview, Christians within the pro-life community typically look at human life differently. The value of human life is not dependent on the value of that human being to any other human being or human society. The value of any human life is measured by the value placed on that life by the One who created it. Therefore, from the beginning, the source of value for human life in the pro-

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 abortion worldview and the pro-life Christian worldview are entirely different. Pro-life Christians typically value human life because God values human life in a way that is distinct from any other life that he created. And God’s bestowed valuation of our life supersedes any assignment of value based on human choice, society, law, or any other human institution.

Christians agree that Scripture teaches that human beings are made in the image of God, by God, and for his purposes, and continue at his pleasure. We as human beings do not have the right before God to terminate the life of any other fellow human being, except as explicitly delegated by God in his Word. It is the love of Jesus Christ that constrains us to care for and love the human beings that he has made. God has designed that new human beings come into existence through, and in the context of, married sexual relationship. Marriage is a publicly witnessed, God-ordained act and institution of promise-giving and promise-keeping. At its core lies an accountability to God, humankind, and spouse for life-giving, truth-telling, faithfulness, and self-sacrifice. Within this refuge of safety lies the only biblical context for sexual intercourse and its designed outcomes, which include childbearing and childrearing. As are all of God’s directives, this directive is for the highest good of the parents as well as of the children.

Human reason has been able to delineate the biological mechanisms by which God creates a new human being: the joining together of a male sperm and a female egg. The point in time at which a new human being is created is at the moment of conception. Therefore, any intentional interruption of the process of development after fertilization constitutes the moral equivalent of an abortion. Any intentionally caused abortion carries the same moral significance as the intentional taking of a human life at any time in the life span of that human being.

We are not free to use every means to achieve our goals. The things that we do must also not be contrary to God’s revelation of his desires for our behavior. For example, it is good to have a child. However, if we commit adultery in order to achieve this good end, then God can and will judge us on the transgression of his moral law, regardless of our good intent. This truth has concrete implications for contraception and abortion. There are times when a conjugal union may occur without its primary purpose being childbirth. However, aborting any child simply because the child is unwanted is outside the limits of moral behavior that God has set, regardless of the “good” that is intended. Because Jesus’ incarnation demonstrated that conception is the time at which God creates a human being in his image, we are constrained by the love of Christ to protect people from their conception to the end of their life as determined by God.
The Controversy

As demonstrated above, most pro-life Christians have much ground in common. However, within the pro-life community there are some who would contend that it is unethical to use any contraceptive mechanism whatsoever, while there are others who would allow for some righteous reasons to contracept. This essay is not meant to address this question directly. Rather, it necessarily makes the assumption that there may be some righteous reasons to contracept.

If there are righteous reasons to contracept, then are there righteous means to contracept? That is, are there contraceptives that work exclusively by preventing conception from occurring? And, given that biological systems are not perfect, do the contraceptives cause no harm to the child if a conception does occur? These are questions that the Christian pro-life community is compelled to face, both out of love for our Lord and out of love for his creatures. This is where the present controversy begins regarding the use of hormone contraception. Some sincerely motivated pro-life physicians have rejected the use of all hormone contraception as abortifacient, based on the following ethical principle: If even one abortion ever occurs in the whole history of the use of hormone contraception, then these contraceptives should never be used and the prescribers and the users are guilty of abortion.

The weakness of this principle is threefold. First, the structure of the statement makes it impossible to scientifically refute. Science is not designed to answer the question of whether or not, under any circumstances, a thing ever happens. Science is a method of modeling or describing reality based on serial observation. However, we cannot sample all of time or history. We cannot prove that an event never happened or never will happen. Yet that is the litmus test that our colleagues have proposed in order to accept the use of hormone contraceptives as a righteous means of contraception.

Second, let us examine this principle in the light of real medical decision-making. Every medication that physicians prescribe, and every surgical procedure that they perform, carries with it real risks of injury or death. Yet, to withhold the medication or not to perform the surgery also carries real risks. If we try to make a decision based on our colleagues’ proposed ethical principle, we will be paralyzed. For example, if a physician knows that one child in a million will die from administering a vaccine, then he or she should never vaccinate, because both the vaccinator and the parents who allow the vaccination would be guilty of murder. However, without the vaccine, the physician knows that 1 out of 100 children will die. So, then, the death of 1 out of 100 children is also the result of the physician’s decision—the decision not to vaccinate. Although this is the greater evil, it may be easier for physicians to blame it on God’s sovereignty. Somehow, to the best of their ability, the physician and the patient must make a judgment as to the balance between the known real risks versus the known real benefits. This vaccination example is useful to help clarify the medical decision-making process that accompanies any therapeutic action.

As illustrated above, any action carries with it real risks and benefits. Likewise, any inaction also carries with it real risks and benefits. How do a physician and patient decide on the best course of action for a patient? The decision-making is an individualized process, based on all the information available to both the physician and the patient, to determine whether or not the benefits outweigh the risks. In this process, we ask the following questions:
• What are the known benefits of using a certain medication or procedure, and how frequently do those benefits occur?
• What are the known risks of using a certain medication or procedure, and how frequently do those occur?
• What are the known risks of not using a certain medication or procedure, and how frequently do those occur?
• What are the known benefits of not using a certain medication or procedure, and how frequently do those benefits occur?

Let us return now to the issue of hormone contraception and ask the four basic questions.
(1) What is the benefit of hormone contraception? It is the ability to have sexual intercourse without the likely possibility of conception. (2) What are the risks of hormone contraception? They include health risks to the woman, the possibility of conception anyway, and health risks to the embryo/fetus. (3) What are the risks of not using hormone contraception? The most significant risk is an increased likelihood of conception if less effective or no alternative methods of contraception are used. This may result in an increased number of abortions, depending on the patient population. (Although our pro-life colleagues who maintain that hormone contraceptives are abortifacient have stated that abortions prevented by the use of contraception are of no consequence in this discussion, we disagree. Perhaps if a physician’s patients are mainly Christians of impeccable moral life, then they would accept an unintended pregnancy as the merciful and gracious hand of a loving Father—which it is. However, in large populations of nominal believers or non-believers, many patients do not hesitate to say that if they found they were pregnant, they would abort.) (4) What are the benefits of not using a hormone contraceptive? The most important is the lack of health risks involved in using such a contraceptive. The present contro-

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versy revolves around the question of what are those health risks to the embryo/fetus.

Those opposing hormone contraception on the basis of the theoretical possibility of it being an abortifacient take the position that a theoretical, yet unproven, risk should carry the same weight and be disclosed in the same fashion as a known, proven risk. It is difficult to see where the line for such thinking will stop, because it depends on who thinks that a risk is theoretically possible. Proven risks are those that have recurrently happened in reality; for them, we can give a frequency of occurrence based on solid evidence from multiple studies over multiple periods of time. We can give the patient facts. Theoretical risks serve the very important function of defining future research directions, but we would be hard-pressed to compel colleagues to disclose risks that they do not think are real, and for which no real evidence exists to substantiate their occurrence.

Third, consider the implications if we generalize this principle to life. What if we were morally forbidden to take any action unless it could be proven under any and every circumstance that no harm will come to anyone at any time from that action? What if we have good reason to believe that doing a certain thing will be of benefit to someone else and have good reason to believe that it will not result in any harm, yet a bad consequence does occur? Is it better for us to abstain from doing good in fear that some evil may result—just so that the blame for such evil does not fall on us? Such fearfulness forgets the heart and core of our belief in the atoning sacrifice of Christ for our sins. This is not to suggest that we have a license to do bad things. Rather, it is to affirm the freedom which comes from the knowledge that our imperfect sinful attempts to do good with all
our heart rest in final judgment at the feet of him who offers to atone for and redeem all things. And it is he who judges heart and mind, not according to appearances, but according to what is right. He is that Creator to whom we are accountable for our medical treatment of his creatures.

We recognize that equally spiritual and honest men and women may consider the same scientific data and hold the same ethical standards but come to differing conclusions on this matter. Thus we face, in the terminology of Romans 14, a “disputable matter” among believers. The principles of how to approach such a vital issue are laid out for us in Romans 14. We call attention to these principles without implication that one side or the other in this “disputable matter” is the “weaker brother,” since we are all weak in many ways. Rather, we seek behavior and attitudes amidst this controversy that will “lead to peace and mutual edification” (v. 19).

First, we are commanded to accept one another “without passing judg-

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ment on disputable matters” (v. 1). We are instructed to “not look down upon” or “condemn” one another (v. 3), nor to “judge your brother” (v. 10). “For who are you to judge someone else’s servant? To his own master he stands or falls. And he will stand, for the Lord is able to make him stand” (v. 4). Rather than judge others, we are instructed to be “fully convinced in our own minds” (v. 5) and to make decisions and perform actions “to the Lord” (vv. 6, 8). For “each of us will give an account of himself to God” (v. 12). It is noteworthy that either of the disputable behaviors discussed in these verses—eating or abstaining—may please or displease God, since God has other priorities: “For the Kingdom of God is not a matter of eating and drinking, but of righteousness, peace, and joy in the Holy Spirit” (v. 17). And finally, in our decision-making on this issue, we must act in faith, for “everything that does not come from faith is sin” (v. 23).

The currently available technology is not sufficient to allow final and definitive scientific resolution of this controversy. However, abundant data are available for evaluation. For each individual, the decision becomes a matter of prayer, of evaluating sufficient pertinent scientific information, and of sensitivity to the Holy Spirit in decision-making. As prescribing physicians, we have a special responsibility in this regard, since many of our patients, especially those with pro-life beliefs, will depend upon our evaluation of this vital subject in making decisions regarding their contraceptive choices. We are obligated to discuss with our patients sufficient factual information to enable them to give their “informed consent.” If a couple decides to use hormone contraceptives as their method of family planning, it is our counsel that they are not using an abortifacient agent.

So, how are we as a pro-life community to respond to potentially divisive questions regarding the mechanisms of hormone contraception in the face of limited scientific evidence? We would draw a parallel to Paul’s treatment of the issue of meat sacrificed to idols in 1 Corinthians 8–10. “So, whether you eat or drink, or whatever you do, do it all for the glory of God.”

References
3. Speroff et al., Clinical Gynecologic Endocrinology.
4. Speroff et al., Clinical Gynecologic Endocrinology.
10. The review paper is entitled “Hormone Contraceptives: Controversies and Clarifications.” Copies of the paper, including a complete set of references, may be obtained on request from ProLife Obstetricians, P.O. Box 81, Fennville, MI 49408, or by e-mail from prolifob@aol.com.
11. McCann and Potter, “Progestin-only Oral Contraception.”
12. McCann and Potter, “Progestin-only Oral Contraception.”

**Editors’ Note:** The Center for Bioethics and Human Dignity recognizes that decisions about prescribing and/or using the Pill should not be based solely on whether such an agent is abortifacient but, rather, should also take into account such factors as its effects on a woman’s health and well-being, and one’s understanding of God’s intended relationship between sex and reproduction.
“The Pill” is the popular term for more than forty different commercially available oral contraceptives. In medicine, they are commonly referred to as BCPs (birth control pills) or OCs (oral contraceptives). They are also called “Combination Pills,” because they contain a combination of estrogen and progestin.

The Pill is used by about fourteen million American women each year. Across the globe it is used by about sixty million. The question of whether it causes abortions has direct bearing on untold millions of Christians, many of them prolife, who use and recommend it. For those who believe God is the Creator of each person and the giver and taker of human life, this is a question with profound moral implications.

In 1991, while researching the original edition of *ProLife Answers to ProChoice Arguments*, I heard someone suggest that birth control pills can cause abortions. This was brand new to me; in all my years as a pastor and a prolifer, I had never heard it before. I was immediately skeptical.

My vested interests were strong in that Nanci and I used the Pill in the early years of our marriage, as did many of our prolife friends. Why not? We believed it simply prevented conception. We never suspected it had any potential for abortion. No one told us this was even a possibility. I confess I never read the fine print of the Pill’s package insert, nor am I sure I would have understood it even if I had.

In fourteen years as a pastor I did considerable premarital counseling. I always warned couples against the IUD because I’d read it could cause early abortions. I typically recommended young couples use the Pill because of its relative ease and effectiveness.

At the time I was researching *ProLife Answers*, I found only one person who could point me toward any documentation that connected the Pill and abortion. She told me of just one primary source that supported this belief and I found only one other. Still, these two sources were sufficient to compel me to include this warning in the book:

Some forms of contraception, specifically the intrauterine device (IUD), Norplant, and certain low-dose oral contraceptives, often do not prevent conception but prevent implantation of an already fertilized ovum. The result is an early abortion, the killing of an already conceived individual. Tragically, many women are not told this by their physicians, and therefore do not make an informed choice about which contraceptive to use.[1]
As it turns out, I made a critical error. At the time, I incorrectly believed that “low-dose” birth control pills were the exception, not the rule. I thought most people who took the Pill were in no danger of having abortions. What I’ve found in more recent research is that since 1988 virtually all oral contraceptives used in America are low-dose, that is, they contain much lower levels of estrogen than the earlier birth control pills.

The standard amount of estrogen in the birth control pills of the 1960s and early ’70s was 150 micrograms. The use of estrogen-containing formulations with less than 50 micrograms of estrogen steadily increased to 75 percent of all prescriptions in the United States in 1987. In the same year, only 3 percent of the prescriptions were for formulations that contained more than 50 micrograms of estrogen. Because these higher-dose estrogen formulations have a greater incidence of adverse effects without greater efficacy, they are no longer marketed in the United States.[2]

After the Pill had been on the market fifteen years, many serious negative side effects of estrogen had been clearly proven. These included blurred vision, nausea, cramping, irregular menstrual bleeding, headaches, increased incidence of breast cancer, strokes, and heart attacks, some of which led to fatalities.[3]

In response to these concerns, beginning in the mid-seventies, manufacturers of the Pill steadily decreased the content of estrogen and progestin in their products. The average dosage of estrogen in the Pill declined from 150 micrograms in 1960 to 35 micrograms in 1988. These facts are directly stated in an advertisement by the Association of Reproductive Health Professionals and Ortho Pharmaceutical Corporation in Hippocrates magazine.[4]

Pharmacists for Life confirms: “As of October 1988, the newer lower dosage birth control pills are the only type available in the U.S., by mutual agreement of the Food and Drug Administration and the three major Pill manufacturers.”[5]

What is now considered a “high dose” of estrogen is 50 micrograms, which is in fact a very low dose in comparison to the 150 micrograms once standard for the Pill. The “low-dose” pills of today are mostly 20–35 micrograms. As far as I can tell, there are no birth control pills available today that have more than 50 micrograms of estrogen. An M.D. wrote to inform me that she had researched many pills by name and could confirm my findings. If such pills exist at all, they are certainly rare.

Not only was I wrong in thinking low-dose contraceptives were the exception rather than the rule, I didn’t realize there was considerable documented medical information linking birth control pills and abortion. The evidence was there, I just didn’t probe deeply enough to find it. Still more evidence has surfaced in subsequent years. I have presented this evidence in detail in my 88-page book Does the Birth Control Pill Cause Abortions? I will now summarize that research.

The Physician’s Desk Reference (PDR)

*The Physician’s Desk Reference* is the most frequently used reference book by physicians in America. The *PDR*, as it’s often called, lists and explains the effects, benefits, and risks of every medical
product that can be legally prescribed. The Food and Drug Administration requires that each manufacturer provide accurate information on its products, based on scientific research and laboratory tests. This information is included in the *PDR*.

As you read the following, keep in mind that the term “implantation,” by definition, always involves an already conceived human being. Therefore, any agent which serves to prevent implantation functions as an abortifacient.

This is the *PDR’s* product information for Ortho-Cept, as listed by Ortho, one of the largest manufacturers of the Pill:

Combination oral contraceptives act by suppression of gonadotropins. Although the primary mechanism of this action is inhibition of ovulation, other alterations include changes in the cervical mucus, which increase the difficulty of sperm entry into the uterus, and changes in the endometrium, which reduce the likelihood of implantation.[6]

The FDA-required research information on the birth control pills Ortho-Cyclen and Ortho Tri-Cyclen also state that they cause “changes in…the endometrium (which reduce the likelihood of implantation).”[7]

Notice that these changes in the endometrium, and their reduction in the likelihood of implantation, are not stated by the manufacturer as speculative or theoretical effects, but as actual ones. They consider this such a well-established fact that it requires no statement of qualification.

Similarly, as I document in my book, Syntex and Wyeth, the other two major pill-manufacturers, say essentially the same thing about their oral contraceptives. (I also relate in the book the results of my phone calls to each of these manufacturers to discuss this issue.)

The inserts packaged with birth control pills are condensed versions of longer research papers detailing the Pill’s effects, mechanisms, and risks. Near the end, the insert typically says something like the following, which is taken directly from the Desogen pill insert:

If you want more information about birth control pills, ask your doctor, clinic or pharmacist. They have a more technical leaflet called the Professional Labeling, which you may wish to read. The Professional Labeling is also published in a book entitled the *Physician’s Desk Reference*, available in many bookstores and public libraries.

Of the half dozen birth control pill package inserts I’ve read, only one included the information about the Pill’s abortive mechanism. This was a package insert dated July 12, 1994, found in the oral contraceptive Demulen, manufactured by Searle. Yet this abortive mechanism was referred to in all cases in the FDA-required manufacturer’s Professional Labeling, as documented in the *Physician’s Desk Reference*.

In summary, according to multiple references throughout the *Physician’s Desk Reference*, which articulate the research findings of all the birth control pill manufacturers, there are not one but three mechanisms of birth control pills:
1. inhibiting ovulation (the primary mechanism),
2. thickening the cervical mucus, thereby making it more difficult for sperm to travel to the egg, and
3. thinning and shriveling the lining of the uterus to the point that it is unable or less able to facilitate the implantation of the newly fertilized egg.

The first two mechanisms are contraceptive. The third is abortive.

When a woman taking the Pill discovers she is pregnant (according to the Physician’s Desk Reference’s efficacy rate tables, this is 3 percent of pill-takers each year), it means that all three of these mechanisms have failed. The third mechanism sometimes fails in its role as backup, just as the first and second mechanisms sometimes fail. Each and every time the third mechanism succeeds, however, it causes an abortion.

Medical Journals and Textbooks

The Pill alters epithelial and stromal integrins, which appear to be related to endometrial receptivity. These integrins are considered markers of normal fertility. Significantly, they are conspicuously absent in patients with various conditions associated with infertility and in women taking the Pill. Since normal implantation involves a precise synchronization of the zygote’s development with the endometrium’s window of maximum receptivity, the absence of these integrins logically indicates a higher failure rate of implantation for Pill-takers. According to Dr. Stephen G. Somkuti and his research colleagues, “These data suggest that the morphological changes observed in the endometrium of OC users have functional significance and provide evidence that reduced endometrial receptivity does indeed contribute to the contraceptive efficacy of OCs.”[8]

In another research journal article, Drs. Chowdhury, Joshi and associates state, “The data suggests that though missing of the low-dose combination pills may result in ‘escape’ ovulation in some women, however, the pharmacological effects of pills on the endometrium and cervical mucus may continue to provide them contraceptive protection.”[9]

Note in some of these citations “contraceptive” is used of an agent which in fact prevents the implantation of an already conceived child. Those who believe each human life begins at conception would see this function not as a contraceptive, but an abortifacient.

In a study of oral contraceptives published in a major medical journal, Dr. G. Virginia Upton, Regional Director of Clinical Research for Wyeth, one of the major birth control pill manufacturers, says, “The graded increments in LNg in the triphasic OC serve to maximize contraceptive protection by increasing the viscosity of the cervical mucus (cervical barrier), by suppressing ovarian progesterone output, and by causing endometrial changes that will not support implantation.”[10]

Drug Facts and Comparisons says this about birth control pills in its 1997 edition:

Combination OCs inhibit ovulation by suppressing the gonadotropins, follicle-stimulating
hormone (FSH) and luteinizing hormone (LH). Additionally, alterations in the genital tract, including cervical mucus (which inhibits sperm penetration) and the endometrium (which reduces the likelihood of implantation), may contribute to contraceptive effectiveness. An independent clinical pharmaceutical reference also contains this assertion. [11]

Reproductive endocrinologists have demonstrated that Pill-induced changes cause the endometrium to appear “hostile” or “poorly receptive” to implantation. [12] Magnetic Resonance Imaging (MRI) reveals that the endometrial lining of Pill users is consistently thinner than that of nonusers [13]—up to 58 percent thinner. [14] Recent and fairly sophisticated ultrasound studies [15] have all concluded that endometrial thickness is related to the “functional receptivity” of the endometrium. Others have shown that when the lining of the uterus becomes too thin, implantation of the pre-born child (called the blastocyst or pre-embryo at this stage) does not occur. [16]

The minimal endometrial thickness required to maintain a pregnancy ranges from 5 to 13mm, [17] whereas the average endometrial thickness in women on the Pill is only 1.1 mm. [18] These data lend credence to the FDA-approved statement that “changes in the endometrium reduce the likelihood of implantation”. [19]

Dr. Kristine Severyn says:

The third effect of combined oral contraceptives is to alter the endometrium in such a way that implantation of the fertilized egg (new life) is made more difficult, if not impossible. In effect, the endometrium becomes atrophic and unable to support implantation of the fertilized egg… The alteration of the endometrium, making it hostile to implantation by the fertilized egg, provides a backup abortifacient method to prevent pregnancy. [20]

Researchers have repeatedly and consistently pointed out this abortifacient effect of the Pill. To date, no published studies have refuted these findings.

Dr. Walter Larimore is a Clinical Professor of Family Medicine who has written over 150 medical articles in a wide variety of journals. In two major medical journal articles, he has addressed the issue of the Pill’s capacity to cause early abortions. [21] In 2000 Dr. Larimore and I coauthored a chapter on this subject in The Reproduction Revolution: A Christian Appraisal of Sexuality, Reproductive Technologies, and the Family. [22] In the same chapter, four Christian physicians present their belief that the Pill does not result in early abortions. We respectfully suggest that their case is not based solidly on the medical evidence.

What Does This Mean?

As a woman’s menstrual cycle progresses, her endometrium gradually gets richer and thicker in preparation for the arrival and implantation of any newly conceived child. In a natural cycle, unimpeded by the Pill, the endometrium experiences an increase of blood vessels, which allow a greater blood supply to bring oxygen and nutrients to the child. There is also an increase in the endometrium’s stores of glycogen, a sugar that serves as a food source for the blastocyst (child) as soon as he or she implants.
The Pill keeps the woman’s body from creating the most hospitable environment for a child, resulting instead in an endometrium that is deficient in both food (glycogen) and oxygen. The child may die because he lacks this nutrition and oxygen.

Typically, the new person attempts to implant at six days after conception. If implantation is unsuccessful, the child is flushed out of the womb in a miscarriage. When the miscarriage is the result of an environment created by a foreign device or chemical, it is in fact an abortion. This is true even if the mother does not intend it and is not aware of it happening.

Despite all the research, including much more presented in my full booklet, there are those who insist that these contentions are incorrect and should not be taken at face value by those concerned about early abortions. In the case of the Pill manufacturers, those who say their FDA-approved assertions are false should, in my opinion, prevail upon the FDA to change their statements, and not simply ask people to disregard them.

**Confirming Evidence**

When the Pill thins the endometrium, it seems self-evident that a zygote attempting to implant has a smaller likelihood of survival. A woman taking the Pill puts any conceived child at greater risk of being aborted than if the Pill were not being taken.

Some argue that this evidence is indirect and theoretical. But we must ask, if this is a theory, how strong and credible is the theory? If the evidence is only indirect, how compelling is that indirect evidence? Once it was only a theory that plant life grows better in rich, fertile soil than in thin, eroded soil. But it was certainly a theory good farmers believed and acted on.

Some physicians have theorized that when ovulation occurs in Pill-takers, the subsequent hormone production “turns on” the endometrium, causing it to become receptive to implantation.[23] However, there is no direct evidence to support this theory, and there is at least some evidence against it. First, after a woman stops taking the Pill, it usually takes several cycles for her menstrual flow to increase to the volume of women who are not on the Pill. This suggests to most objective researchers that the endometrium is slow to recover from its Pill-induced thinning.[24] Second, the one study that has looked at women who have ovulated on the Pill showed that after ovulation the endometrium is not receptive to implantation.[25]

**Intrauterine/Extrauterine Pregnancy Ratio**

Another line of evidence of the Pill’s abortifacient effect is this: if the Pill has no post-fertilization effect, then reductions in the rate of intrauterine pregnancies in Pill-takers should be identical to the reduction in the rate of extrauterine (ectopic/tubal) pregnancies in Pill-takers. Therefore, an increased extrauterine/intrauterine pregnancy ratio would constitute evidence for an abortifacient effect.
Two medical studies allow review of this association.[26] Conducted at seven maternity hospitals in Paris, France[27] and three in Sweden[28], the studies evaluated 484 women with ectopic pregnancies and control groups of 389 women with normal pregnancies who were admitted to the hospital for delivery during the same time period. These studies were designed, in typical fashion for “case control” studies, to determine the risk factors for a particular condition (here, ectopic pregnancy) by comparing one group of individuals known to have the condition with another group of individuals not having the condition. Both of these studies showed an increase in the extrauterine/intrauterine pregnancy ratio for women taking the Pill. Researchers who have reviewed these studies have therefore suggested that “some protection against intrauterine pregnancy is provided via the Pill’s post-fertilization abortifacient effect.”[29]

What accounts for the Pill inhibiting intrauterine pregnancies at a disproportionately greater ratio than it inhibits extrauterine pregnancies? The most likely explanation is that while the Pill does nothing to prevent a newly conceived child from implanting in the wrong place (i.e., anywhere besides the endometrium), it may sometimes do something to prevent him from implanting in the right place (i.e., the endometrium).

Arguments Against the Pill Causing Abortion

I have received a number of letters from readers, one of them a physician, who say something like this: “My sister got pregnant while taking the Pill. This is proof that you are wrong in saying that the Pill causes abortions—obviously it couldn’t have, since she had her baby!”

Without a doubt, the Pill’s effects on the endometrium do not always make implantation impossible. I have never heard anyone claim that they do. To be an abortifacient does not require that something always cause an abortion, only that it sometimes does.

Whether it’s RU-486, Norplant, Depo-Provera, the morning-after pill, the mini-pill, or the Pill, there is no chemical that always causes an abortion. There are only those that do so never, sometimes, often, and usually.

Children who play on the freeway, climb on the roof, or are left alone by swimming pools don’t always die, but this does not prove these practices are safe and never result in fatalities. We would immediately see this inconsistency of anyone who argued in favor of leaving children alone by swimming pools because they know of cases where this has been done without harm to the children. The point that the Pill doesn’t always prevent implantation is certainly true, but has no bearing on the question of whether it sometimes prevents implantation, which the data clearly suggests.

People also often argue, “The blastocyst is perfectly capable of implanting in various ‘hostile’ sites, e.g., the fallopian tube, the ovary, the peritoneum.”

Their point is that the child sometimes implants in the wrong place. This is undeniably true. But again, the only relevant question is whether the Pill sometimes hinders the child’s ability to implant in the right place.
Imagine a farmer who has two places where he might plant seed. One is rich, brown soil that has been tilled, fertilized, and watered. The other is on hard, thin, dry, and rocky soil. If the farmer wants as much seed as possible to take hold and grow, where will he plant the seed? The answer is obvious—on the fertile ground.

Now, you could say to the farmer that his preference for the rich, tilled, moist soil is based on theoretical assumptions because he has probably never seen a scientific study that proves this soil is more hospitable to seed than the thin, hard, dry soil. Likely, such a study has never been done. In other words, there is no absolute proof.

But the farmer would likely reply, based on years of observation, “I know good soil when I see it. Sure, I’ve seen some plants grow in the hard, thin soil too, but the chances of survival are much less there than in the good soil. Call it theoretical if you want to, but we all know it’s true!”

Some newly conceived children manage to survive temporarily in hostile places. But this in no way changes the obvious fact that many more children will survive in a richer, thicker, more hospitable endometrium than in a thinner, more inhospitable one.

(In other publications and in a much more detailed fashion, we have discussed these and other lines of evidence, with hundreds of citations of many scientific studies, as well as researchers and experts in numerous fields. We encourage interested readers to look more deeply into these studies and arguments.[30])

Despite this evidence, some prolife physicians state that the likelihood of the Pill having an abortifacient effect is “infinitesimally low, or nonexistent.”[31] Though I would very much like to believe this, the scientific evidence does not permit me to do so.

Dr. Walt Larimore has told me that whenever he has presented this evidence to audiences of secular physicians, there has been little or no resistance to it. But when he has presented it to Christian physicians there has been substantial resistance. Since secular physicians do not care whether the Pill prevents implantation, they tend to be objective in interpreting the evidence. After all, they have little or nothing at stake either way. Christian physicians, however, very much do not want to believe the Pill causes early abortions. Therefore, I believe, they tend to resist the evidence. This is certainly understandable. Nonetheless, we should not permit what we want to believe to distract us from what the evidence indicates we should believe.

I have mentioned my own vested interests in the Pill that at first made me resist the evidence suggesting it could cause abortions. Dr. Larimore came to this issue with even greater vested interests in believing the best about the birth control pill, having prescribed it for years. When he researched it intensively over an eighteen-month period, in what he described to me as a “gut wrenching” process that involved sleepless nights, he came to the conclusion that in good conscience he could no longer prescribe hormonal contraceptives, including the Pill, the mini-pill, Depo-Provera, and Norplant.
Statement by Twenty Prolife Physicians

Five months after the original printing of my booklet, in January 1998 a statement was issued opposing the idea that the Pill can cause abortions. According to a January 30, 1998, email sent me by one of its circulators, the statement “is a collaborative effort by several very active prolif OB-GYN specialists, and screened through about twenty additional OB-GYN specialists.”

The statement is titled “Birth Control Pills: Contraceptive or Abortifacient?” Those wishing to read it in its entirety, which I recommend, can find it at our web page, at www.epm.org/doctors.html. I have posted it there because while I disagree with its major premise and various statements in it, I believe it deserves a hearing.

The title is misleading, in that it implies there are only two possible ways to look at the Pill: always a contraceptive or always an abortifacient. In fact, I know of no one who believes it is always an abortifacient. There are only those who believe it is always a contraceptive and never an abortifacient, and those who believe it is usually a contraceptive and sometimes an abortifacient.

The paper opens with this statement:

Currently the claim that hormonal contraceptives [birth control pills, implants (Norplant), injectables (Depo-Provera)] include an abortifacient mechanism of action is being widely disseminated in the prolife community. This theory is emerging with the assumed status of “scientific fact,” and is causing significant confusion among both lay and medical prolife people. With this confusion in the ranks comes a significant weakening of both our credibility with the general public and our effectiveness against the tide of elective abortion.

The assertion that the presentation of research and medical opinions causes “confusion” is interesting. Does it cause confusion, or does it bring to light pertinent information in an already existing state of confusion? Would we be better off to uncritically embrace what we have always believed than to face evidence that may challenge it?

Is our credibility and effectiveness weakened through presenting evidence that indicates the Pill can cause abortions? Or is it simply our duty to discover and share the truth regardless of whether it is well-received by the general public or the Christian community?

The physicians’ statement’s major thesis is this: The idea that the Pill causes a hostile endometrium is a myth.

Over time, the descriptive term “hostile endometrium” progressed to be an unchallenged assumption, then to be quasi-scientific fact, and now, for some in the prolife community, to be a proof text. And all with no demonstrated scientific validation.

When I showed this to one professor of family medicine he replied, “This is an amazing claim.” What’s so amazing is it requires that every physician who has directly observed the dramatic Pill-induced changes in the endometrium, and every textbook that refers to these changes, has been wrong all along in believing what appears to be obvious: that when the zygote attaches itself to the
endometrium its chances of survival are greater if what it attaches to is thick and rich in nutrients and oxygen than if it is not.

This is akin to announcing to a group of farmers that all these years they have been wrong to believe the myth that rich fertilized soil is more likely to foster and maintain plant life than thin eroded soil.

It could be argued that if anything may cause prolifers to lose credibility, at least with those familiar with what the Pill does to the endometrium, it is to claim the Pill does nothing to make implantation less likely.

The authors defend their position this way:

[The blastocyst] has an invasive nature, with the demonstrated ability to invade, find a blood supply, and successfully implant on various kinds of tissue, whether “hostile,” or even entirely “foreign” to its usual environment—decidualized (thinned) endometrium, tubal epithelium (lining), ovarian epithelium (covering), cervical epithelium (lining), even peritoneum (abdominal lining cells)…. The presumption that implantation of a blastocyst is thwarted by “hostile endometrium” is contradicted by the “pill pregnancies” we as physicians see.

This argument misses the point, since the question is not whether the zygote sometimes implants in the wrong place. Of course it does. The question, rather, is whether the newly conceived child’s chances of survival are greater when it implants in the right place (endometrium) that is thick and rich and full of nutrients than in one which lacks these qualities because of the Pill. To point out a blastocyst is capable of implanting in a fallopian tube or a thinned endometrium is akin to pointing to a seed that begins to grow on asphalt or springs up on the hard dry path. Yes, the seed is thereby shown to have an invasive nature. But surely no one believes its chances of survival are as great on asphalt as in cultivated fertilized soil.

According to the statement signed by the twenty physicians, “The entire ‘abortifacient’ presumption, therefore, depends on ‘hostile endometrium.’”

In fact, one need not embrace the term “hostile endometrium” to believe the Pill can cause abortions. It does not take a hostile or even an inhospitable endometrium to account for an increase in abortions. It only takes a less hospitable endometrium. Even if they feel “hostile” is an overstatement, can anyone seriously argue that the Pill-transformed endometrium is not less hospitable to implantation than the endometrium at its rich thick nutrient-laden peak in a normal cycle uninfluenced by the Pill?

One medical school professor told me that until reading this statement he had never heard, in his decades in the field, anyone deny the radical changes in the endometrium caused by the Pill and the obvious implications this has for reducing the likelihood of implantation. According to this physician, the fact that secular sources embrace this reality and only prolife Christians are now rejecting it (in light of the recent attention on the Pill’s connection to abortions) suggests they may be swayed by vested interests in the legitimacy of the Pill.
The paper states “there are no scientific studies that we are aware of which substantiate this presumption [that the diminished endometrium is less conducive to implantation].” But it doesn’t cite any studies, or other evidence, that suggest otherwise.

In fact, surprisingly, though this statement is five-pages long it contains not a single reference to any source that backs up any of its claims. If observation and common sense have led people in medicine to a particular conclusion over decades, should their conclusion be rejected out of hand without citing specific research indicating it to be incorrect?

On which side does the burden of proof fall—the one that claims the radically diminished endometrium inhibits implantation or the one that claims it doesn’t?

The most potentially significant point made in the paper is this:

The ectopic rate in the USA is about 1% of all pregnancies. Since an ectopic pregnancy involves a pre-implantation blastocyst, both the “on pill conception” and normal “non pill conception” ectopic rate should be the same—about 1% (unaffected by whether the endometrium is “hostile” or “friendly.”) Ectopic pregnancies in women on hormonal contraception (except for the minipill) are practically unreported. This would suggest conception on these agents is quite rare. If there are millions of “on-pill conceptions” yearly, producing millions of abortions, (as some “BC pill is abortifacient” groups allege), we would expect to see a huge increase in ectopics in women on hormonal birth control. We don’t. Rather, as noted above, this is a rare occurrence.

The premise of this statement is right on target. It is exactly the premise proposed by Dr. Larimore, which I’ve already presented. While the statement’s premise is correct, its account of the data, unfortunately, is not. The studies pointed to by Dr. Larimore, cited earlier, clearly demonstrate the statement is incorrect when it claims ectopic pregnancies in women on hormonal contraception are “practically unreported” and “rare.”

In fact, “a huge increase in ectopics” is exactly what we do see—an increase that five major studies put between 70% and 1390%. Ironically, when we remove the statement’s incorrect data about the ectopic pregnancy rate and plug in the correct data, the statement supports the very thing it attempts to refute. It suggests the Pill may indeed cause early abortions, possibly a very large number of them.

Questions about This Problem

People raise many objections to the issues presented in this appendix, very few of which involve issues of evidential data or scientific fact. However, these objections deserve answers. These are some of the concerns I address in my booklet Does the Birth Control Pill Cause Abortions?[32]

- “If this is true, why haven’t we been told before?”
- “I don’t trust this evidence.”
“If we don’t know how often abortions happen, why shouldn’t we take the Pill?”

“Spontaneous miscarriages are common; early abortions aren’t that big a deal.”

“Taking the Pill means fewer children die in spontaneous abortions.”

“Without the Pill there would be more elective abortions.”

“Pill-takers don’t intend to have abortions.”

“Why not just use high estrogen pills?”

“You can’t avoid every risk.”

“How can we practice birth control without the Pill?”

“I never knew this—should I feel guilty?”

“We shouldn’t lay guilt on people by talking about this.”

“We shouldn’t tell people the Pill may cause abortions because they’ll be held accountable.”

“We’ve prayed about it and we feel right about using the Pill.”

“This issue will sidetrack us from fighting surgical abortions.”

“Prolifers will lose credibility if we oppose the Pill.”

“This puts Christian physicians in a very difficult position.”

“Are there any good alternatives to the Pill?”

Conclusion

The Pill is used by about fourteen million American women each year and sixty million women internationally. Thus, even an infinitesimally low portion (say one-hundredth of one percent) of 780 million Pill cycles per year globally could represent tens of thousands of unborn children lost to this form of chemical abortion annually. How many young lives have to be jeopardized for prolife believers to question the ethics of using the Pill? This is an issue with profound moral implications for those believing we are called to protect the lives of children.
References


[18] McCarthy et al., “Female Pelvic Anatomy”.


[27] Coste et al., “Risk Factors For Ectopic Pregnancy”.


HORMONE CONTRACEPTIVES: CONTROVERSIES AND CLARIFICATIONS

SUSAN A. CROCKETT, JOSEPH L. DECOOK, DONNA HARRISON, AND CAMILLA HERSH

Introduction

Recently, there has been some controversy, and serious questions have been raised by sincere individuals who are concerned that hormone contraceptives may have an abortifacient mechanism of action. This paper will help to clarify the issue based on a thorough review of the available medical literature regarding the mechanism of action of hormone contraceptives. It has been compiled by board-certified practicing ob/gyns, in consultation with perinatologists and reproductive endocrinologists, each being a physician committed to honoring the sanctity of human life from conception. We affirm that as physicians answerable to our Creator and Redeemer, we are responsible to the best of our ability to help, and not intentionally harm, our fellow human creatures. As Christian physicians, we affirm that all life is created by God and that human life is initiated at conception. Fertilization, not implantation, marks the beginning of human life. Disruption of the fertilized egg represents abortion.

The issue of the mechanism of action of commonly used hormone contraceptives has threatened to split the pro-life physician community. Review of information currently being disseminated reveals some powerful and well-written rhetoric. However, the issue of the mechanism of action of hormone contraceptives is not one which will be illuminated by rhetoric. The mechanism of action of any medicine will not change based on how we feel about it, or on who developed it, or on how eloquently it is defended or opposed. How a medication works is a scientific question.

The hormone contraceptives include four basic types: combination oral contraceptives (COCs), injectables (Depo-Provera), progestin-only pills (minipill, or POPs), and implants (Norplant). In this paper, they will, where convenient, be collectively referred to as the “pill.” Most hormone contraceptives are noted to work by 3 methods of action:
1) Primarily, they inhibit ovulation by suppression of the pituitary/ovarian axis, mediated through suppression of gonadotrophin releasing hormone from the hypothalamus.
2) Secondarily, they inhibit transport of sperm through the cervix by thickening the cervical mucus.
3) They cause changes in the uterine lining (endometrium), which have historically been assumed to decrease the possibility of implantation should fertilization occur. This presumption is commonly known as the “hostile endometrium” theory.
A thorough review of the medical literature uncovers ample data to support the first two methods of action, which are contraceptive actions. (Appropriate references will be found in the sections discussing each type of hormone contraceptive.) However, there is no direct evidence in the literature to support the third proposed method of action. This conclusion is shared by the respected gynecologic/endocrinology textbook authors Yen and Jaffe. (1) Nevertheless, for the past nearly 40 years, most authors of “pill”-related scientific literature have routinely repeated the assumption of a contra-implantation effect by this “pill”-primed uterine lining. In light of this large body of literature, some pro-life authors have expressed appropriate concerns that hormone contraceptive methods may include an abortifacient action by hindering implantation. These authors have cited data drawn from this scientific literature to support their claims. Closer scrutiny of the medical literature, however, reveals that the scientists are all simply agreeing that the “pill” produces a thinner, less glandular, less vascular lining. We also agree. However, in an ovulatory pill cycle, the estrogen and progesterone levels are, as discussed below, grossly increased for the seven days prior to implantation. The normal biologic response of endometrium to high levels of these hormones is growth of stroma, blood vessels, glands, and glandular secretions to help prepare the lining for implantation.

An extensive review of pertinent scientific writings indicates that there is no credible evidence to validate a mechanism of pre-implantation abortion as a part of the action of hormone contraceptives. On the contrary, the existing evidence indicates that “on pill” conceptions are handled by the reproductive system with the same results seen with “off pill” conceptions, with the exception of increased ectopic rates seen with POPs and Norplant. Not all the contraceptive agents are equally effective, or even equally appropriate, to be used by doctors and patients concerned with the sanctity of life and maternal welfare. The remainder of this paper is a presentation of the current scientific data, which will allow doctors and patients who are committed to the sanctity of life from the time of conception to make decisions regarding the use of these agents that his or her conscience can be at peace with. We do not assume that everyone, given the same information, will arrive at a uniform decision. However, for the follower of Christ, discernment based on prayer and the evaluation of factual information in the light of Scripture is the basis of ethical decision making.

Normal Physiology

It is helpful at this point to review the basic physiology of the normal ovulatory cycle. Specific endocrinologic details are best found in a text of gynecologic endocrinology. However, in general, after a young woman completes puberty, the levels of estrogen rise and fall twice during each normal menstrual cycle. The pituitary gland releases follicle stimulating hormone (FSH), which causes new, ovum-containing follicles (eggs) to develop in the ovaries during the first half (or follicular phase) of the menstrual cycle. The follicle steadily increases estrogen production, which reaches a peak about one day prior to ovulation. The surge of estrogen stimulates her pituitary gland to secrete another essential hormone, luteinizing hormone (LH), which in turn serves to trigger ovulation (egg release).

Ultrasound can be used to assess the growth and development of the ovarian follicle (cyst around the egg cell) and can indicate the degree of readiness for ovulation. (2) During an ovulatory
cycle the usual cyst size varies from 20 to 28 mm. Non-ovulating follicles rarely exceed 14 mm in diameter. Ovulation is associated with complete emptying of the follicular contents in 1 to 45 minutes. After ovulation, the follicle which has released the egg becomes filled with another type of cell, a luteal cell. The luteal cells proliferate under the influence of pituitary luteinizing hormone (LH) and secrete ever increasing quantities of both estrogen and progesterone.

The follicle (now a corpus luteum) most commonly appears as a smaller, irregular cyst which, if conception has NOT occurred, diminishes in size and ceases to function 2 weeks after ovulation. With subsequent decrease of luteal estrogen and progesterone, the uterine lining (endometrium) is then shed as the menstrual period. However, if conception HAS occurred, the embryo begins, by the time it implants, to secrete another chemical messenger, hCG (human chorionic gonadotropin), which acts like LH to rejuvenate and stimulate the corpus luteum to continue its function until the placenta takes over hormone production 2 months later. The corpus luteum produces, in the six days after ovulation, 10 to 20 times the levels of both estrogen and progesterone seen in a non-ovulatory “pill” cycle. (A preovulatory pill cycle has an estradiol level of 25 pg/ml, a preovulatory normal cycle has an estradiol level of about 40 pg/ml) During an ovulatory cycle, estradiol reaches a peak of 400 pg/ml during the day before ovulation—a ten to 16 fold increase—and peaks again at 275 pg/ml by day 6 after ovulation, which is the day of implantation. Progesterone values rise from a preovulatory 0.5 ng/ml to a peak of 10 ng/ml by implantation day—a twenty fold rise. (41,42) These high levels act on the lining in these seven days to prepare it for implantation and support of the arriving (via the fallopian tube) living embryo. Corpus luteum function continues until 8 to 10 weeks from ovulation, at which time (noted above) the placenta assumes the burden of producing these hormones to support the growing pregnancy.

In the extensive literature we have reviewed, no writer has addressed this very significant question: In a menstrual cycle on the “pill” in which ovulation occurs, what is the histology of the endometrium six days after ovulation (the time of implantation)? Certainly the hormone milieu and endometrial histology will be different from a menstrual cycle on the “pill” in which ovulation does not occur (i.e., the typical atrophic, or “hostile,” endometrium). The FSH-LH-estradiol surge the day before ovulation, and the resulting corpus luteum formation, with its ten to twentyfold estradiol and progesterone output, should produce significant changes in the endometrium. In a normal menstrual cycle, on the day of ovulation the uterine lining (proliferative endometrium) is not receptive to implantation. Seven days of follicle and corpus luteum hormone output transform it to become “receptive.” The same follicle and corpus luteum hormone output, when ovulation occurs in a “pill” cycle, should have a similar salutary effect on the pill-primed endometrium. It is highly probable that the so-called “hostile to implantation” endometrium—heralded (without proof) from the beginning by the “pill” producing companies, echoed (without investigation) by 2 generations of scientific writers, and now adopted (as a scientific fact) by some sincere pro-life advocates—simply does not exist six days after ovulation in a pill cycle. What is currently known about the endometrial response to corpus luteum hormones suggests this conclusion. Research regarding endometrial histology on the sixth day after escape ovulation in on “pill” cycles would add useful information to the current discussion.

Zanatu (51) reports on two women with prolonged infertility (8 to 14 mos.) after DepoProvera injections: “We successfully induced ovulation with the sequential administration of clomaphine
citrate and human chorionic gonadotropin, and pregnancy immediately followed.” This suggests that once ovulation has occurred, the burst of natural estrogen and progesterone from the corpus luteum simply override even the most potent hormone contraceptive, producing a receptive endometrium and resulting in a normal implantation and ongoing pregnancy.

The abortifacient theory proponents propose a second line of evidence that they feel strongly suggests the “pill” is associated with an early abortifacient effect. (3) This refers to an increased risk per pregnancy of tubal pregnancy. The lack of a corresponding increase in intrauterine pregnancy is suggested as evidence of a contrainternal implantation effect of the “pill.” One writer states that “All published data show that the extraterine ratio of pregnancies is increased for women on BCPs . . .” Our own review of the literature has shown this increased ectopic rate to be true of progestin-only pills (POPs) and Norplant. However, we have found absolutely no data in the literature that supports an increased ectopic to intrauterine pregnancy ratio for women using combined oral contraceptives (COCs) or Depoprovera. Comments and references accompany our discussion of these individual agents below.

Two additional lines of reasoning have more recently been offered by abortifacient theory proponents (3, slides 42-48). The first has to do with integrins, an endometrial polypeptide which is felt by some to be associated with endometrial receptivity. These integrins are “conspicuously absent in patients with luteal phase deficiency, endometriosis, and unexplained infertility....In most OC users, the normal patterns of expression of integrins is grossly altered.” This is felt by the proponents to be evidence of potential abortifacient action at the endometrial level. The problem with this theory is that it deals with endometrium in pill cycles that are not ovulatory. As noted previously, an entirely different hormone milieu exists for seven days to prepare the endometrium for implantation in an “on pill” ovulatory cycle, just as it does in a normal (or “non-pill”) ovulatory cycle. We are aware of no studies dealing with integrins in an ovulatory pill cycle.

The second line of reasoning has to do with endometrial thickness in a pill cycle (3, slides 35-41). This position notes from the medical literature that “Recent MRI studies show that pill users have endometrial linings that are 40-60% thinner than women not on the pill,” and “ten recent IVF studies confirm that ‘endometrial thickness is related to the functional receptivity of the endometrium.’” We do not dispute these quotations from the literature. However, as is the case in the previous paragraph, they simply do not apply to endometrial thickness or receptivity in an ovulatory pill cycle, nor do they purport so to apply.

**Definition of Abortifacient**

Abortion, when used as a medical term, refers to the loss of a pregnancy less than 20 weeks gestational age, regardless of whether the termination is intentional or spontaneous. Spontaneous abortions are commonly known as miscarriages. There are literally hundreds of factors that have been implicated in the loss of human pregnancies. For example, aging of the woman, alcohol, infections, RU486, cocaine, genetic disorders, uterine structural anomalies, methotrexate, some prostaglandins and trauma have all been shown to contribute to abortions. There are many more factors that may contribute to fetal loss, but have not been proven to do so. Implicated factors
include almost any environmental substance known to man, including impurities in our air or water, the electronic equipment we sit near, and almost any medication, herb, or chemical. Some of these may actually cause abortion and some may not.

An ethical question is thus raised, “Where do we draw the line in informed consent for responsible disclosure of known risks?” To answer this question as it relates to hormone contraceptives, we must examine what factors would lead us to classify a drug as an abortifacient.

In order to classify COCs as an abortifacient, several things must exist:
1) Conceptions must occur.
2) The abortive effect must be present with proper use as prescribed.
3) Loss of these conceptions must exceed the base-line loss for populations not using this substance, or be shown to occur due solely to the medication itself and not other known factors.
4) The abortive effect should be consistent and reproducible by multiple independent observers.

There are no studies that we are aware of regarding combined oral contraceptives or Depoprovera that demonstrate numbers 2, 3, or 4. (Increased ectopic pregnancies seen with progestin-only pills (POPs) and Norplant will be discussed in the appropriate section below.)

Decision making on the part of a physician prescribing a medicine assumes that the patient will be compliant with the prescribing orders. In our discussion on hormonal contraceptives, patient compliance will be assumed.

Most abortifacient theory proponents have lumped together all of the hormonal contraceptive agents: combined oral contraceptives (COCs), progestin-only pills (POPs), implants (Norplant), and injectables (Depoprovera). The consideration of rates of ovulation, ongoing pregnancy, and ectopic pregnancy, and the assumption of abortifacient action, has been attributed to all alike. This leads to erroneous conclusions which impact ethical decision making. It is more instructive and accurate to review the literature concerning these agents separately since they vary in action, complications, and effectiveness.

A careful, exhaustive review of the medical literature has been done. This reveals the following information regarding the mechanisms of action of the four different types of hormone contraceptives:

**Progestin-Only Pills (POPs)**

Progestin-only pills have been widely studied, particularly by WHO (World Health Organization) sponsored groups for use in underdeveloped countries. The attractive qualities in this context include probable enhancement of breastfeeding (COCs have negative enhancement), and simplicity of use (one daily, no confusing “dummy ” pills or varied colored pills for illiterate populations). In addition, in our cultural context, a high degree of patient tolerance (minimal side effects of nausea,
bloating, etc.), no increase in risk (especially in smokers) of estrogen associated thromboembolism, coronary artery disease, or cerebrovascular disease are seen as indications for POP use. (4) However, estimated ovulation rates range from 14 to 84% (5), varying with age, lactation, and compliance factors. With “typical” use, the average estimated ovulation rate is about 50% (6), and ongoing pregnancy rates are estimated at 5% per year. (7) Pregnancy rates in lactating women and women over 35 years of age are about 1%. (4) Notably, with correct, consistent use, the pregnancy rate for all users is only 0.5% in the first year. (8) To attain this kind of contraception rate, compliance must be unusually exacting. (6) The ectopic pregnancy (EP) rate with POPs may be between 6 to 10% of unintended pregnancies. (6) (In the general population, ectopic pregnancies constitute 2% of all pregnancies.) (9) The increased “per pregnancy risk” for ectopic pregnancy may be a result of progestin action on the tube, reducing the number of cilia on the tubal epithelium, as well as the intensity and frequency of cilia action, thus slowing the rate of ovum transport. (10) Others do not confirm this progestin effect on the tube. (77) Because of the significant ovulation rate and pregnancy rate, and the increased ectopic pregnancy/intrauterine pregnancy (IUP) ratio, the POP may not be a desirable choice for most patients. Elective use of a medication which increases the percent of ectopic pregnancies per conception raises significant ethical questions for many—as does subjecting one’s patient to an increased risk of ectopic pregnancy morbidity or even mortality, when there are equally, or even more effective, alternatives. (In a third world context, additional factors may enter into one’s decision making. See Appendix 1.)

**Progestin Implants (Norplant)**

The implant Norplant shares many similarities with the POP, except that it is considerably more effective birth control than POPs. Compliance is not a factor with the implants. The pregnancy rate with Norplant (0.09% per year) is much lower than the pregnancy rate with POPs (0.5% per year with consistent, correct use; 5% per year with “typical” compliance). (8) Therefore, the ectopic pregnancy rate per 100 women years of use for Norplant users is also much lower than the rate per hundred women years of use for POP users. In the third world context, Norplant would be about 50 times more effective than POPs at preventing ectopies and maternal mortality (often with attendant newborn mortality), and would facilitate improvement in women’s health and newborn nutrition (see appendix 1). In our cultural setting, the parameters for ethical decision making are different: High maternal mortality rates are not present, and infant nutrition does not depend on continuance of a breast milk supply. Like the POPs, Norplant has an increased EP/IUP ratio, estimated at 3 to 5 times the per pregnancy ectopic rate seen in the general non-contracepted population. The general incidence of ectopic pregnancy in the USA is about 2% of reported pregnancies. Between 6 to 10% of unintended pregnancies on Norplant and POPs may be ectopic. This increased ectopic effect has not been well explained. As mentioned above, it may be related to a selective progestin effect on the tube. We see no data to prove or indicate that a number of embryos are produced, with some becoming ectopic pregnancies and with the remainder moving into the uterus to be aborted by an inhospitable lining. But just as in the POP discussion, elective use of a medication with this untoward risk factor, especially when there are hormonal contraceptives with no increased risk factor for either the unborn or the pregnant woman, raises serious ethical questions for many of us.
**Injectable DepoProvera (Depomedroxyprogesterone Acetate)**

The injectable DepoProvera (DMPA) has only been FDA-approved for use in the USA since 1992. It has been used internationally since the 1960’s. Although there is a relative paucity of American medical literature pertinent to the topic, there is considerable world experience with DMPA. The literature regarding this injectable suggests its effectiveness is based on extremely low ovulation rates.

The suppressive action of DMPA is at the hypothalamus or higher to prevent the hypothalamus from giving the signal to the pituitary to release gonadotropins. (22) Secretion of LH and FSH are maintained in the mid-follicular state. Because of this, the ovary does not develop a dominant follicle, so the egg does not mature. There is no LH surge, so there is no ovulation. Ovulation does not occur until serum MPA is at extremely low levels, often 7 to 9 months after injection. (20) By the time ovulation is able to occur, serum MPA is at such low levels (0.1 ng/ml) that it has little effect on the endometrium, and the ovary is producing normal preovulatory levels of estradiol. (17, 23 43, 12, 13, 14, 15, 16, 18, 19, 21, 22, 25, 44, 45, 46, 47, 48, 49, 50) Thus there is no evidence that DMPA produces even a theoretical risk of abortion by “hostile endometrium”.

Several small pharmacokinetic studies utilizing progesterone levels as a major indicator of ovulation have shown a zero ovulation rate 3 months after IM injection of 150 mg of DMPA. (12,13,14,15,16,17,18,19,20) In the practical world, especially the third world, for various reasons the ovulation rate will not be zero (failure to shake the vial, out-dated or deteriorated meds, sub Q rather than IM injection, inadequate dose, and biological patient variation all may enter the picture). However, pregnancy rates indicate DMPA is extremely effective. Five large international studies, including over 8,000 women (21), were used to determine the “Pearl Index” figure of 0.3 pregnancies per hundred women years noted in the chart of “lowest expected pregnancy rates” found in the PDR. The chart is adapted from Hatcher, et al. (8).

There is no evidence that DMPA causes an increased risk of ectopic pregnancy. As noted above, ovulation does not resume until serum progestin levels are extremely low. It is highly unlikely that such negligible progestin serum levels interfere with tubal or tubal cilia motility. In fact, pregnancy is so uncommon with DMPA that statistics on ectopic pregnancy are difficult to find, and, because of very small numbers, difficult to evaluate. The single study identifiable in the literature came from the Downstate Medical Center in New York from 1970 to 1974. The study looked at gross ectopic rates by reported method of contraception. This study calculated the gross ectopic rate for DepoProvera of 1.3%. However, because the total number of ectopics on DepoProvera was so small, and the total number of pregnancies with DepoProvera was also small, the difference between 1.3% and 1% for the baseline ectopic rate of a comparison group was not statistically significant. Tatum (24), commenting on the study, notes, “Since pregnancy on depo-provera was so rare, there were not enough pregnancies to even get a statistically accurate rate.” The significance of these studies is that they show no dramatic increase in ectopic pregnancies. Rather, if pregnancy does occur with DMPA, it is about as likely to be ectopic as a non-contracepted pregnancy.
Combined Oral Contraceptives (COCs)

Background

Over the last 30 years, combined oral contraceptives (COCs) have become one of the most commonly used forms of family planning world-wide. Billions of women have benefited from both the direct contraceptive and indirect health-related benefits of these medications. Ninety-nine percent of American women using hormone contraception use COCs. Although there are risks associated with use of any medication, the risks of COCs are extremely small, especially when patients are appropriately chosen and counseled for use.

Definition of Combined Oral Contraceptive (COC)

Combined oral contraceptives (COCs) are those birth control pills that contain both an estrogenic and a progestational agent. There are several different estrogens and even more progestins available on the market, and there are many ways to combine them into pill formulations. The contraceptive efficacy of these hormones is derived primarily from the actions of progestins in suppressing ovulation and thickening cervical mucus. The estrogenic component serves two purposes. It potentiates the action of the progestogenic component of oral contraceptives, and it stabilizes the endometrium so that irregular shedding and unwanted breakthrough bleeding can be minimized.

In fact, an ideal endometrium does not seem to be necessary for successful implantation, as is evidenced by ectopic pregnancies which may implant in the tubes, endocervical canal, on the ovaries, or on the surface of any intra-abdominal organ, including the bowel or even major blood vessels. Additionally, as noted above, in an “on pill” escape ovulatory cycle, with the required FSH-LH surge, followed by post ovulatory corpus luteum estradiol and progesterone output, one would expect the endometrium to undergo the usual hormonally related changes favorable to implantation, as in any ovulatory cycle. (The endometrium is sufficiently responsive to physiologically balanced hormones such that even the slightly increased estrogen balance in triphasics produces a histologic trend toward secretory pattern.) To have a meaningful discussion regarding the mechanism of action of COCs and to address the “potential abortifacient” question, a review of the pertinent medical literature is necessary. The following discussion is based on our review.

Regarding the Occurrence of Conceptions On COCs With Proper Use

Conceptions do occur, at times, on COCs. We know this, because like all methods of contraception other than abstinence, there is a measurable pregnancy rate. Medical literature often refers to pregnancy rates for contraceptive methods as “failure rates.” So called failure rates for contraceptive methods are classified as perfect use (compliant, consistent) or typical use. Perfect use failure rates imply that the method of contraception has failed, whereas typical use values reflect human noncompliance or other confounding variables (such as pharmaceutical interactions) that contribute to failure. Since this ethical discussion regarding COCs is primarily about the method of action of COCs, we are interested in perfect use failure rates, which most accurately reflect the activity of the
drug itself. The perfect use failure rate most often quoted in the medical literature, including the standardized FDA product labeling of every combined oral contraceptive listed in the PDR, is 0.1 pregnancies for every 100 women years (1300 cycles). Therefore, it can be concluded that at times, ALL of the mechanisms of action of COCs fail.

**Regarding Spontaneous Abortion Rates With “Pill” Pregnancies**

Spontaneous abortions can be divided functionally into “clinical abortions,” i.e., spontaneous loss after pregnancy has been clinically recognized (usually from about 6 menstrual weeks, which is 4 conceptional weeks), and “pre-clinical abortions,” i.e., loss occurring before that time. Pre-clinical abortions can further be divided into “pre-implantation abortions,” i.e., those occurring before the conceptus implants 6 days after fertilization, and those occurring after implantation, but before clinical recognition of the pregnancy. There has been no demonstrated effect of COC use on spontaneous clinical abortion rates. The essential abortifacient argument brought against hormonal contraception is that it causes pre-clinical, more specifically, pre-implantation, abortion due to an inhospitable lining, the “hostile endometrium.” The improbability of this entity, based on known follicle and corpus luteum hormone output during an ovulatory cycle and normal endometrial response to these hormones, has been discussed earlier in this paper.

To look at the controversy from another direction, do “pill” pregnancies have similar outcomes as non-pill pregnancies? Clinically, the answer is “yes.” There is no data to indicate higher clinical spontaneous abortion rates or more problems in ongoing pregnancies. But is there increased loss evident with “pill” pregnancies? From the clinical side, the answer is “no.” (78) From the pre-clinical, especially the pre-implantation perspective, the answer must be sought by evaluating indirect data, since there is no direct data available regarding these loss rates for users of COCs. Most studies evaluating the efficacy of COCs only measure clinically evident pregnancies as an end point. There is scarce literature about ovulation rates on COCs, although more than 40 such studies were reviewed in preparing this paper. Of the COC studies that evaluated ovulation, fertilization and pregnancy rates are almost never evaluated. The reason for this should be obvious: if a patient in a COC study is told that she has ovulated, she will avoid exposure to sperm, thus preventing an unwanted pregnancy.

**Concerning the Outcome of “On Pill” Ovulations**

The first requisite in evaluating this question is to establish a reliable unintended ovulation rate in perfect (compliant, i.e., no missed pills) COC users. In this pursuit, 25 studies were reviewed (several papers contained more than one study). (ref. 59 through 76) These studies used a variety of common COCs, including triphasics, and were about evenly split between the newer very low dose pills (20 mcg estinyl estradiol), and the current standard low dose pills (30 to 35 mcg ethinyl estradiol). Eighteen studies including 3799 cycles showed zero ovulations. Seven studies including 2910 cycles showed 8 ovulations. (Ovulation was indicated by ultrasound and serum chemical markers.) Combining these gives a practical working number of 8 ovulations in 6709 cycles, which equals 15.5 ovulations in 13,000 cycles (a figure used to simplify the arithmetic done below). This is not a scientific metaanalysis. Rather, it is a pragmatic figure, using the referenced peer-reviewed data, that will help provide an informed perspective on the question of pre-implantation loss.
The next necessary information concerns the unintended pregnancy rate with compliant use of COCs. This is well established in the Hatcher table (8) at 0.1 pregnancy per 100 women years, which equals one pregnancy per 1,000 women years, or one pregnancy in 13,000 cycles by compliant “pill” takers. Thus we have, in 13,000 cycles, 15.5 ovulations (from the previous paragraph) and one pregnancy.

Finally, the cervical mucus factor must be considered. The marked change in cervical mucus under the influence of progestins is recognized as a substantial factor in contraceptive effectiveness of the “pill.” As reviewed by McCann (6), studies of cervical mucus changes in women on POPs, which contain only half the dose of progestin found in the COCs, found the mid cycle mucus to be greatly reduced in volume and increased in viscosity and cell content, with an altered molecular structure. (52, 53, 54) The effect is a mucus with low spinbarkeit and poor ferning. This is found to result in little or no sperm penetration in 70-80% of cases. (53) Even in the rare cases when penetration does occur, sperm motility is reduced. (55, 54, 56, 57, 58) One study noted an almost total absence of sperm in the uterine cavity of the progestin treated group, while sperm were present in the uterus of 18 of 19 controls. (55) COCs, with twice the progestin dose of POPs, would produce mucus with similar, if not greater, sperm impedance. (Although COCs also contain an estrogen, ethinyl estradiol, they block follicle activity so well that the actual preovulatory serum estradiol levels on the “pill” are less than normal, i.e.,25 pg/ml vs the normal 40 pg/ml. This level will have a negligible influence on improving cervical mucus.) In the normal cycle, the pre-ovulatory FSH surge will immediately produce an estradiol peak of 400 pg/ml, quickly resulting in production of the optimum fertility enhancing mucus. In an ovulatory “pill” cycle, this estradiol peak almost certainly will override, to some degree, the progestin induced sperm blocking effect on the cervical mucus. It is not known how quickly, or to what degree, this override might take place. Reproductive endocrinologists indicate that ovulation takes place within 12 to 24 hours after the LH-FSH surge with its accompanying estradiol peak and that the newly released egg can only accept fertilization for about 12 hours before it becomes resistant. This leaves a window of about 24 to 36 hours for the transformation of the impenetrable cervical mucus to fertility-favorable mucus that would allow release and transport of sperm to the distal portion of the fallopian tube to fertilize the egg. (And this assumes that sufficient numbers of viable sperm are present in the cervical mucus at the time the mucus becomes favorable.) We know that the cervical mucus factor adds significantly to the contraceptive effectiveness of the “pill”—although to what extent this is true in an ovulatory cycle can only be estimated, since there have been no specific studies done to give us numbers.

The available data referenced in this discussion has, then, yielded this information: 13,000 cycles of compliant COC use results in 15.5 ovulations and one ongoing pregnancy. The basic question in the entire “pill” controversy is, “What happened to the other 14.5 eggs?” Were they the victim of an “inhospitable endometrium” (whose existence in an ovulatory cycle is very questionable), or can they be accounted for in other ways? Let us start with the 15.5 ovulations noted above. Available data indicate that 10 to 15% of our population is infertile. (41, p. 809) Of the original 15.5 eggs, this leaves 13.5 eggs available for possible fertilization and ongoing pregnancy. One could simply take the 13.5 eggs available for fertilization and then apply the normal human fecundity table of 25% per cycle (the fecundity table requires optimum conditions: normal fertility in the man and woman and correct timing of coitus). (28) We would expect 3.3 pregnancies IF the cervical mucus is favorable, the viable sperm are present, and the timing is right. Referencing the previous paragraph, cervical mucus
factors block sperm 80 to 90% of the time in non-ovulatory studies. IF adequate numbers of viable sperm can get free and make it to the waiting egg within the 36 hour receptivity window just 33% of the time, one egg might be fertilized to begin an ongoing pregnancy. We don’t know about the sperm and timing, and the cervical mucus is probably not optimum. If one of these 3.3 remaining eggs is fertilized and thrives, this equals the known ongoing pregnancy rate seen in compliant COC users: One per 1,000 women years (13,000 cycles) of use. The fate of the 15.5 eggs can also be considered from other available data. Again, the 10 to 15% infertility rate leaves 13.5 eggs available for possible fertilization. Recent critically reviewed data indicates that from fertilization to 6 weeks, spontaneous pregnancy wastage is 73%. (26, 27) This leaves 3.5 eggs available for fertilization and ongoing pregnancy. As above, considering mucus, sperm, and timing factors, one egg might be fertilized to become an ongoing pregnancy. This again equals the known pregnancy rate on compliant COC usage: per 1,000 woman years (13,000 cycles) of use. This exercise in arithmetic is not meant to be a statistician’s scientific proof text. It is rather an overview of COC unintended ovulation rates and subsequent ongoing pregnancy rates using available peer-reviewed data to account for the eggs in question.

A recent study by specialists in reproductive technology found, with genetic sampling of morphologically normal embryos, that 24 of 50 had chromosomal abnormalities and would likely not survive. (79) GIFT procedures, introducing multiple viable eggs along with good sperm into the fallopian tube at the optimum time with optimum endometrium, only yield a 30% success rate. 30% of failed fertilizations are due to faulty sperm. There are evidently many naturally occurring reasons for preimplantation loss.

Considering the above information on ovulatory related hormone influences on the endometrium, “on pill” ovulation rates and pregnancy rates, the cervical mucus factor, and known data on human fecundity, fertility, and spontaneous loss rates, it can be appreciated that there is no need to postulate a “pill”-induced preimplantation abortion phenomenon to explain why 15.5 eggs produce one ongoing pregnancy. Known and natural causes can account for the numbers.

Concerning Ectopic Pregnancy and the “Pill”

If COCs are abortifacient in nature by causing the conception not to implant in the uterine cavity, then ectopic pregnancy rates should be at least equal to that in the normal population. One of the well-studied effects of progestins on the fallopian tube is to decrease motility and cilia action, which would inhibit a fertilized ovum from proceeding into the uterine cavity. Therefore, it would be reasonable to expect an even higher EP rate than the normal population (2% of all clinically recognized pregnancies). The literature, at this time, does not show an increased ectopic rate per pregnancy for COCs over non-users.

Any contraceptive method decreases the overall ectopic rate, simply by way of decreasing the number of pregnancies that occur at all. A rough estimate of the numbers involved can be calculated using generally accepted data and simple arithmetic: Using the table of Hatcher (8), and assuming “perfect” (correct, consistent) use for COCs, the sympto-thermal method, and condoms, three matched groups of 100,000 women would experience 100 unintended pregnancies on COCs, 2,000 unintended pregnancies with the ympto-thermal method, and 3,000 unintended pregnancies with condoms. Using the current figure of a 2% ectopic ratio (9), these pregnant women would
experience 2 EP with COCs, 40 EP with the sympto-thermal method, and 60 EP with condoms. Given the potential maternal mortality, morbidity, and damage to fertility with EP, and the absence of any demonstrated increase in abortion due to COC endometrial effect, one might ask if it is ethical to withhold from or discourage the option of COCs for the woman seeking contraceptive information. Certainly this pertinent information should be considered as part of adequate informed consent.

Although there are many references alluding to increased rates of ectopic pregnancies in the context of tubal ligation, IUDs, POPs, and post-coital contraception, a search through over a dozen well-recognized medical texts and multiple journal articles written by experts in obstetrics, gynecology, and contraception has yielded no authoritative opinions that implicate COCs in the etiology of EP. Additionally, the medical society as a whole has had over 40 years of experience with COCs, with virtually millions of women world wide, and a history of billions of cycles of use. In all of this time, although many other risks of COCs have been identified (and are part of the standardized labeling of all COCs in the PDR, as required by the FDA), not one pharmaceutical company, independent clinical researcher, or epidemiologist has published data clearly showing an association between COCs and increased EP rates. In the medico-legal world, product liability implications have an immense influence on standardized labeling. If, in the 40 years of COC experience, there were the slightest hint that the product might cause an increase in EP rates, such warning would have been mandated in the product literature to protect the manufacturer from lawsuits, as well as to warn the user of the possible complication. There are no such warnings in COC labeling. An EP noted in a woman who has compliantly used COCs, and who has no other major risk factors for EP, is so rare that it would be a reportable case.

A careful search of the literature reveals 11 references to ectopic pregnancies in COC users (29-39). Of those 11, one paper (32) calculates the theoretical risk of EP in COC users; the other 10 are clinical papers. Of those 10 references, one (34) is a letter to the editor, which references data from a clinical study (29). References 29 and 31 are written by the identical group of researchers, with identical data, just published in two different journals, in two different languages.

In particular, one reference (35) is often quoted by abortifacient theory proponents as suggesting a “slightly” increased ectopic pregnancy rate on COCs. A critical review of this article reveals this to be a misleading statement. This meta-analysis attempts to draw conclusions from the small amount of data from references 33, 36, 37, and 38. The actual data review compiles data from these 4 studies into 3 categories: 1) one shows a strong, statistically significant decrease in EP in COC users, even supporting a protective effect against EP, 2) the two other groups analyzed showed no statistically significant increase in ectopic rates among COC users, even when these patients were compared to inappropriate control groups of pregnant patients. Therefore, the author's conclusion that suggested a possible increased rate of ectopics with COC use is not supported either in their own meta-analysis data, or by any of the individual papers used in the analysis.

One reference (39) is an epidemiologic study from Beijing, China. This is the only study that gives an ectopic pregnancy rate in women on COCs. The data is expressed in the form of EP per 1000 woman cycles. Unfortunately, there is not enough information in the paper to make a comparative statement about whether this rate is higher or lower than the normal population in
Beijing, since ectopic rates are generally reported as a percentage of clinically recognized pregnancies, not cycles. Also, these authors provided no data concerning other risk factors that these women may have had for EP. In fact, the risk may have been considerable, since the group studied were “sex workers.”

Of the remaining 6 references with clinical data, none were designed to evaluate ectopic rates in COC users. In fact, most of these studies use COC users as their control group in evaluating other contraceptive methods. None of these studies calculated ectopic rates per clinical pregnancy for COC users, and all of them describe their data in the form of the number of women who happened to be taking COCs when they conceived an EP. Several of these studies showed ectopic rates for barrier methods of contraception (which have no impact at all on the endometrium or implantation) to be higher than for COCs. None of these authors conclude that they have data proving ectopic rates to be higher in COC users when compared to non-users. One author (29) makes a suggestion based on 11 patients with ectopies who were on COCs that COCs may prevent intrauterine implantation better than tubal implantation. However, this study has small numbers, too small to reach statistical significance, and uses pregnant women as the comparison group. This is acknowledged by the authors to be a poor choice of a control group, for the reason that they have selected out any women who had spontaneous abortions, early intentional terminations, or EP. Furthermore, even though this author makes this suggestion, he makes it clear that it is not conclusive evidence, as the formulations of the pills and patient compliance were not assessed, and suggests that future research is needed to verify his results.

An analytical error which must be avoided in reviewing the above literature is attempting to calculate an ectopic pregnancy rate based on the numbers in the studies above. This is faulty logic: calculating the number of EP that occur in women taking COCs is not the same as the EP rate (i.e., the fraction of women with ectopic pregnancies among those who became pregnant as a result of COC failure.) The sampling frames are different.

None of the studies listed above even estimate the sample size of “pill” users that are represented by the few ectopic pregnancies reported. None of the studies specifically evaluated patient compliance or the multiple other risk factors that women in the ectopic groups may have had in addition to oral contraceptive use, and most studies do not separate out progestin-only preparations from combined oral contraceptives.

Therefore, at this time, there is no direct evidence that shows that combined oral contraceptives are an independent risk factor for ectopic pregnancy when conception occurs. Given the medical community's long history of experience with COCs, there is no evidence that COCs are an etiologic risk factor for EP. Therefore, the existing medical literature does not support the theory (with its “abortifacient” implications) that COCs are more efficacious in preventing IUP than EP.
Conclusion

Given the above, there is no evidence that shows that the endometrial changes produced by COCs contribute to failure of implantation of conceptions, nor is there evidence that COCs cause an increased per pregnancy ratio of ectopics.

Some Summary Thoughts

1. Ethical decision making by a medical doctor often involves life and death questions. The discussion regarding the ethics of hormonal birth control is an extremely important matter, since 14 million women in the United States alone regularly use these medications (and that is but a fraction of the world wide usage).
2. Ethical decision making on this issue must involve the evaluation of as much pertinent scientific information as possible.
3. Christian ethical decision making requires that the physician be committed to the Authority of Scripture and to the Lordship of Jesus Christ in his/her life and medical practice.
4. Christian ethical decision making on this issue requires that the physician be committed to the sanctity of life of the unborn from the time of conception and to the sanctity of life of the mother.
5. The patient must be a part of this ethical decision making and must be informed of the pertinent issues involved.

The following questions should be addressed by pro-life physicians:

1) Is it appropriate to implicate a medication as an abortive agent without the data to support such a claim? To do so creates needless hostility and division among physicians and patients who genuinely respect life from the moment of conception.
2) Where do we draw the line in informed consent for responsible disclosure of known medical risks vs. a theoretical risk which is not substantiated by current scientific knowledge?
3) Is it accurate to implicate all hormonal contraceptive methods as one regarding their method of action, rather than evaluating each one individually?

We have done our best to evaluate the current literature on the topic, and to present it fairly for consideration by the reader. We do not find substantive evidence that hormone contraceptives include an abortifacient mechanism of action. We recognize that equally spiritual and honest men and women may consider the same facts, but come to differing conclusions on this matter. For the follower of Jesus, the decision on whether or not to use or prescribe hormone contraceptives is a spiritual as well as a medical matter. The principles of how to handle a situation among Christian believers on a very vital subject in dispute is directly addressed in Romans chapter 14 as an issue of individual conscience and responsibility before the Master (see appendix 2).

Many factors play a part in how a family plans and spaces their children. It is not the purpose of this paper to promote nor to oppose hormone contraception. However, if a family, weighing all the
factors affecting their own circumstances, decides to use this modality, we are confident that they are not using an abortifacient.

This paper is not meant to be the “final word” on this issue. If scientific study should validate that a hormone contraceptive agent is partly abortifacient in its action, we would oppose that agent just as we oppose elective medical and surgical abortions. We must constantly examine valid data as it becomes available in our effort to discern what are appropriate methods of family planning to be used or prescribed by those who know that human life begins at the time of conception.
Bibliography


43. Antal EG, Pharm D. BCOP Drug Information Clinical Pharmacist, Pharmacia and Upjohn, personal correspondence.
45. Bodlzieher W et al. A cross sectional study of plasma, kFSH, and LH levels in women using sequential, combined or injectable steroid contraceptives over long periods of time.
60. Csemanticzky G, Dieben T et al. The pharmacodynamic effects of an oral contraceptive containing 3 mg micronized 17 beta-estradiol and 0.150 mg desogestrel for 21 days, followed by 0.030 mg desogestrel only for 7 days. *Contracl* 1996; 54(6):333-8.
Appendix 1

We wish to state emphatically that the following discussion does NOT refer to any program intent on world population control, or what is sometimes called “population control imperialism.” Rather, it refers to compassion and treatment for patients under our care. As Christian physicians, it is our mandate to alleviate, to the extent humanly possible, medical conditions resulting in suffering and death. In third world countries, maternal mortality rates are much higher than in our setting, and the use of hormone contraceptives will mean significantly decreased maternal mortality. For example, 18 countries in Africa have a maternal mortality from 100 to more than 200 times that in the United States. In the US, the maternal mortality rate is 7 maternal deaths per 100,000 pregnancies. In Zambia, it is 764 to 1549 maternal deaths per 100,000 pregnancies. Their hospital based maternal mortality is 543/100,000, or 80 times the US rate. In this context, POPs, the least effective hormonal contraceptive, would result in an immense decrease in pregnancy related deaths. Using the 85 per 100 women years pregnancy rate in non contracepting women from Hatcher (8), using the “typical” 5% unintended “pill” pregnancy rate from Trussel (7), using the highest (10% of pregnancies) ectopic rate (6), using the 543 per 100,000 hospital based maternal mortality from northern Zambia, and using a “normal” ectopic rate of 2%, we would see, among 120,000 women using POPs: 5400 pregnancies rather than 100,000 pregnancies, 540 ectopics rather than 2000 ectopics, 29 late pregnancy-delivery related deaths rather than 543 deaths. These are obviously approximate figures, but they come from simple arithmetic with the most accurate data base available. Additional benefits would include improved health for mothers not subject to the drain of a rapid succession of pregnancies and improved infant nutrition with enhanced and prolonged breast feeding possible before the next newborn displaces its older sibling from the breast (the “kwashiorkor” phenomenon).
Appendix 2

In Romans 14, the issue was eating meat offered to idols, a violation of the First Commandment. In the present controversy, the issue is the killing of unborn children, a violation of the Sixth Commandment. We recognize that equally spiritual and honest men and women may consider the same data, but come to differing conclusions on this matter. Thus we face, in the terminology of Romans 14, a “disputable matter” among believers. The principles of how to approach such a vital issue are laid out for us in Romans 14. We bring attention to these principles without the implication that one side or the other in this “disputable matter” is the “weaker brother,” since we are all weak in many ways. Christ is our only strength. Rather, we seek behavior and attitudes amidst this controversy that will “lead to peace and mutual edification” (v 19). First, we are commanded to accept one another “without passing judgment on disputable matters” (v 1). We are instructed to “not look down upon” nor “condemn” one another (v 3), nor to “judge your brother” (v 10). “For who are you to judge someone else’s servant? To his own master he stands or falls. And he will stand, for the Lord is able to make him stand” (v 4). Rather, we are instructed to be “fully convinced in our own minds” (v 5), and to make decisions and perform actions “to the Lord” (vv 6,8). For “each of us will give an account of himself to God” (v 12). It is noteworthy that neither of the disputable behaviors, eating or abstaining, may please, or displease, God. God, rather, has other priorities. “For the Kingdom of God is not a matter of eating and drinking, but of righteousness, peace, and joy in the Holy Spirit” (v 17). And finally, in our decision making on this issue, “everything that does not come from faith is sin” (v 23).
Appendix 3  
(Refers to page 73, paragraph 2)

The scientific debate surrounding the question as to whether oral contraceptives (OCPs) are abortifacients has focused intently on scientific publications which might lend some insight into the phenomenon of breakthrough ovulation and the subsequent response of the endometrial lining. Of these, V. Chowdhury’s article has been called to the docket frequently to give its testimony.

We have reviewed the 1980 article, “Escape ovulation in women due to the missing of low dose combination oral contraceptive pills” by V. Chowdhury et.al. (2) and have also been in personal correspondence with the authors. We have also reviewed a number of newer research articles on the subject of escape ovulation and ovarian activity on the combined oral contraceptive pills (see list of appendix references). We would like to briefly discuss these below.

In brief, the 1980 Chowdhury article studied “ovulation” in 35 women who were previously sterilized and then asked to take a 30 ug ethinyl estradiol plus norethindrone acetate combination OCP. They were asked to “miss” 2 consecutive pills in a cycle, and then progesterone levels were measured at day 22 of the cycle and endometrial biopsies were also obtained. Chowdhury found that 10 out of 35 women had progesterone levels greater than 4 ng/ml. He concluded that these 10 women had ovulated, based solely on its level of progesterone.

But, is a single serum progesterone level of greater than 4 ng/ml sufficient evidence to prove ovulation? Many authors have addressed this question. The answer is: “Clearly, No”. Let us look at one of these studies more closely; the 1982 article by Hull et.al.: “The value of a single serum progesterone measurement in the midluteal phase as a criterion of a potentially fertile cycle (“ovulation”) derived from treated and untreated conception cycles.” (6) Hull looked specifically at attempts to determine ovulatory cycles by measuring serum progesterone in the midluteal phase (i.e. day 25+/−2 of cycle) of cycles that conceived. He studied conception cycles because those cycles in which conception occurs are the only cycles where we can currently prove conclusively that an ovulation actually occurred; they are the only documentably proven ovulatory cycles. Let’s look at his findings:

“In an extended study a total of 21 untreated singleton conception cycles have been observed with a mean progesterone value of 40.7 nmol/ml (12.7 ng/ml), 95% confidence limits of 28-53 nmol/ml (8.8 to 16.7 ng/ml), and a range of 27 to 53 nmol/ml (8.5 to 16.7 ng/ml). This range was much narrower than for nonconception cycles (3 to 80 nmol/ml, 0.9 to 25.2 ng/ml), which extended significantly above as well as below the conception range, indicating that there is an optimal range for fertility with both an upper and a lower limit. The lower limit is of greater practical importance; and partly to allow for assay variation, we suggest it should be taken as 30 nmol/ml (9.4 ng/ml). It provided a clinically reliable criterion of potential fertility (“ovulation”) in related studies. Our findings in treated conception cycles suggest that a higher value may be needed after treatment with clomiphene or gonadotropins because of the contribution from other stimulated follicles.” (6).

Hull defined the lower limit of progesterone produced in a cycle where ovulation was possible. Below that level of progesterone, ovulation does not happen. Other authors have suggested the
same lower limit of 8-9 ng/ml of progesterone as the lowest limit of a potentially ovulatory cycle (1,3,10,15). Therefore, we corresponded with Dr. Chowdhury in order to obtain more precise information about the actual progesterone levels of his study participants. However, he replied that all the available information about that study was fully published in the paper, and he has no more detailed information than that which is already published. Therefore, we must conclude that we have no idea how many of his 10 patients actually were potentially ovulating (i.e., had a progesterone level high enough to support ovulation.) It is possible that if none of those 10 women had progesterone levels greater than 8 ng/ml, that none of them were actually ovulating. This renders the rest of his results essentially meaningless, because you cannot determine whether or not the endometrium was hostile to implantation in an ovulatory cycle on the OCP unless you determine that you actually HAVE an ovulatory cycle.

The second weakness of the Chowdhury article is the endometrial biopsy histology reporting. Chowdhury states: “The endometrial biopsy showed ‘hormone effect’ as reflected histologically by atrophic glands with excessively stimulated stroma.”

However, Mazur showed that excessive stromal hypertrophy was present in inadvertent endometrial biopsies performed in early gestation (8) and postulated that this was a necessary step in preparing the endometrium for implantation. Also, errors in histology can occur from sampling of the lower uterine segment instead of the fundus (12). Without more description of the actual histology obtained in Chowdhury’s biopsies, it is difficult to tell whether or not his specimens actually show “hostile endometrium.” (Of further interest is an article by Navot (11) who actually used supraphysiologic doses of estrogen and progesterone to support the implantation and early pregnancy of women who were without any ovarian function of their own, but who had been recipients of IVF with donor embryos.)

Chowdhury further states: “In 5 out of 35 women in the first cycle treatment group and in 7 out of the 19 in the fourth cycle treatment group, the endometrium was so scanty that a suitable endometrial tissue sample could not be obtained.” However, there are other reasons as well why a tissue sample cannot be obtained, and it does not always mean “scanty endometrium.” In fact, uterine fibroids, a retroflexed uterus, pain on the part of the patient, and operator inexperience are all frequently reasons for insufficient tissue sampling. In fact, we are forced to conclude that in 14-35% of his data, the endometrial biopsy material is insufficient for meaningful interpretations.

Thus, the question of whether OCPs produce a “hostile endometrium” with breakthrough ovulations and in such instances are functionally chemical abortifacients remains an unanswered question for the following reasons:

1) Chowdhury’s study does not clearly identify a subgroup of patients on the OCP who are clearly ovulating on the OCP. A 4 ng/ml progesterone cutoff is inadequate to indicate ovulation, and his raw data is not available for further review at this time.

2) Even if available, a progesterone level > 9 ng/ml is only “permissive” of ovulation: i.e., a level < 9 ng/ml precludes ovulation, but a level > 9 mg/ml cannot distinguish reliably between ovulatory and nonovulatory cycles. This is because of significant contributions of progesterone production by
luteinized unruptured follicles, which are follicles in the ovary which have not released the egg yet still produce progesterone (6 and others. See literature on polycystic ovarian syndrome). 

3) Chowdhury’s endometrial biopsies are uninterpretable because of the lack of clear documentation of ovulation and the large number of biopsies with no tissue obtained (i.e. 14-35% of his endometrial biopsies had no tissue).

4) Improvements in ovulation detections were not utilized in the Chowdhury study (e.g. LH surge testing and ultrasound demonstration of ovulatory follicles or luteal phase endometrial thickening), limiting the study’s interpretation and utility.

However, the concept behind Dr. Chowdhury’s article is well worth repeating in the current era of availability of ultrasound assessment of ovarian function and evaluation of the endometrial lining, LH and FSH surge testing, and estradiol and progesterone assays. We would propose a new study to reexamine this issue and are currently seeking support to implement this.
References


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THE IMPACT OF THE PILL ON IMPLANTATION FACTORS—NEW RESEARCH FINDINGS

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For health consumers and health care professionals of an orthodox Judeo-Christian or Islamic tradition, as well as those authentically concerned with the universal respect of unqualified human rights, the asserted capacity of the pill to act as an abortifacient, both in its once-a-day and “morning-after” permutations, is one of significant moral weight.

The research on “break-through” ovulation leads moralists, philosophers and human rights’ advocates to question the use of the title “contraceptive” to describe the pill. There is tension regarding this nomenclature. The term “contraceptive” refers to a drug, device or chemical that prevents the joining of the sperm with the female secondary oocyte (commonly referred to as the ovum).

The problem arises because the female sex cell, the secondary oocyte, may be present in the reproductive tract at or near the time of coitus; hence there exists the possibility that fertilization may occur. Yet, as we will see, the pill alters the receptive structure of the endometrium, making implantation problematical.

But are concerned groups justified in moving from a position, which states that the pill sometimes fails to prevent “ovum” fertilization, with the result that new human life may begin, to the position of claiming that the pill has an abortifacient capacity? The first position notes that ovulation occurs in women on the pill and fertilization may occur, but claims there is no evidence that implantation is impeded. The alternate view considers that because ovulation has been detected and the lining of the womb is in an undeveloped state, human life is imperiled.

This is a seismic shift in outlook. What merit is there in this latter claim other than supposition or suspicion? Is the pill tarnished with the title of “abortifacient” on conjecture rather than on fact?

This paper will seek to clarify these issues. I will concentrate in some depth on a variety of the implantation factors associated with the microenvironment of the endometrial epithelium. Discussion will also be focused on the mechanism(s) of hormonal dialogue between the 5–7 day old human embryo (the blastocyst) and the cells which line the endometrium. I will also cover the impact of above normal (supraphysiological) levels of estrogen and progesterone on these implantation factors and the influence of the pill hormones on the integrity of the endometrium. Particular attention will be given to the impact of the pill on cyclical development of endometrial thickness, and the relationship of this uterine feature to the success of implantation of the human embryo. Central to these issues will be a review of the research on “break-through” ovulation (also known as “escape-ovulation”—an event which must occur, otherwise all concerns concerning the pill as an abuser of human rights would be shown to be empty.

This paper is of necessity detailed. I hope that the employment of suitable analogies, as well as bracketed discussion of medical terms or concepts, will make it accessible to the scholar and lay reader alike.
1.1 Executive Summary

The process of implantation of the human embryo into the lining of the womb is a very complex and delicate one. Proper attachment and successful implantation is under the guidance and control of a vast array of “implantation factors” such as interleukin-1 β, platelet-activating factor (PAT), insulin-like growth factor (IGF), leukemia inhibition factor (LIF), and tumor necrosis factor α (TNF α).

Many of these chemical factors participate in a process referred to in the medical journals as “cell-signalling,” a process which involves the new human embryo and the cells of the lining of the womb chemically communicating with each other. The purpose of this chemical communication is to create an optimally advantageous endometrial environment at the time the human embryo attempts to implant.

Aside from this biochemical embryo/uterine-cell communication, successful implantation of the human embryo is dependent also upon a class of molecules known as integrins. Integrins are cell-adhesion molecules found in a “mirror” fashion on both the human embryo and the lining of the womb. These integrins bind onto each other, via gluco-proteins (e.g., fibronectin). The success or otherwise of this binding process is intimately linked to the ongoing success or otherwise of the pregnancy.

The reader will note that I am using the orthodox understanding of the term “pregnancy.” This definition dates the beginning of the pregnancy from the moment of fertilization. I do not use, nor do I accept, the minority view, influenced as it is by the politics of abortion that date a pregnancy from the time of implantation.

1.2 The Re-defining of Pregnancy Terminology

Notwithstanding the embryonic, linguistic and time-honoured orthodoxy of “pregnancy,” increasingly frequent attempts have been made to redefine all aspects of pregnancy, but most particularly, when pregnancy begins. The reason for this move is clear; by redefining pregnancy—when it begins, the nature of the embryo, etc.—the way will be made smooth for the more rapid introduction of RU-486, the morning-after pill, anti-HCG vaccines, anti-implantation factor drugs and other embryocidal drugs. Unwittingly or otherwise, the end result is a semantically based desensitization of the moral conscience of the community.

The following is an indicative selection of quotes to illustrate my point:

The prevention of pregnancy before implantation is contraception and not abortion. (Glasier, NEJM, 1997)

Predictably, some opponents of abortion allege that emergency contraception is tantamount to abortion . . . even if emergency contraception worked solely by preventing the implantation of a zygote, it would still not be abortifacient . . . Pregnancy begins with implantation, not fertilization . . . fertilization is a necessary but insufficient step toward pregnancy. (Grimes, NEJM, 1997)
Emergency contraception works by inhibiting or delaying ovulation or by preventing implantation. Despite some assertions to the contrary, it is not itself a form of abortion.\textsuperscript{15} (Guillebaud, \textit{Lancet}, 1998)

These opinions are starkly at odds with embryology\textsuperscript{20} and etymology.\textsuperscript{21}

Before examining these features and the relational involvement of the pill in more detail, it may be of some benefit to propose an analogy to assist in the understanding of the various implantation factors and the role of integrins.

Consider the example of a space shuttle, low on fuel and oxygen, urgently needing to dock with the space station. The mother ship and the shuttle communicate with each other so that the shuttle knows which docking bay to go to. Importantly, the mother ship knows which bay to make ready. Successful communication is imperative. If this electronic communication fails (disrupted embryonic-uterine “cell-talk”), the shuttle may go to the wrong docking bay, fail to attach to the mother ship, and drift away, with the result that the crew dies from a lack of food and oxygen. Alternatively, the shuttle might go to the right bay, but find that all the docking apparatus is not in place. Again, the attachment between the two fails due to faulty communication, and the crew dies. This role of embryo/endometrium communication is fulfilled by implantation factors such as interleukin, TNF, NDF and PAF. To continue the analogy, integrins could be thought of as grappling hooks that “hold” the human embryo onto the womb whilst the process of implantation is completed.

This, then, is a brief overview of this review paper. I would like now to analyse these issues in more depth, looking at the specific role and activity of the main implantation factors covered in the research literature. As well, I will expand on the interaction between these factors and the steroidal hormones: estrogen and its artificial copies (principally ethinylestradiol, ingested via the pill) and progesterone, plus its artificial copies (norethisterone, levonorgestrel, gestodene and desogestrel).

\subsection*{1.3 The Interleukin System}

The interleukin (IL) system, composed of IL-1\textsubscript{x}, IL-1\textsubscript{B} and IL-1ra, is both hormonally regulated and of endometrial origin (Simon, 1996).\textsuperscript{22} Under normal physiological conditions, progesterone increases the production of IL-1\textsubscript{x} and IL-1\textsubscript{B} from the endometrium\textsuperscript{23} and levels of the IL system reach their maximum during the luteal (post-ovulatory) phase of the menstrual cycle.\textsuperscript{24}

Of the various components of the interleukin system, research suggests that IL-1\textsubscript{B} plays a key role in the proper orientation of the embryo to the uterine lining, a process known as apposition. Recalling our earlier analogy, apposition could be likened to pre-docking maneuvers responsible for correctly aligning the docking ports of mother ship and shuttle.

Within this framework, the role of IL-1\textsubscript{B} is thought to be that of a “signal system” between endometrium and embryo.\textsuperscript{25} “. . . [S]uccess of embryonic implantation relies on a perfect dialogue between good quality embryos and a receptive endometrium.”\textsuperscript{26}

Huang and co-workers (1997) have also reported that the IL system is “an important factor in embryo-maternal molecular communication during the implantation process.”\textsuperscript{27}

Whilst normal levels of the ovarian hormones estrogen and progesterone have a beneficial effect on the levels of IL-1\textsubscript{B}, excessive hormonal levels, known as supraphysiological steroid levels, have been shown to cause a reduction in the levels of IL-1\textsubscript{B}. As a result, the rate of implantation drops significantly. Simon and co-workers (\textit{J Reprod Immun}, 1996) have shown that there is an inverse
relationship between estrogen and progesterone levels and the levels of IL-1β (as estradiol levels increase, implantation success decreases). \(^{28}\)

The direct consequence of these findings, as they relate to the maintenance of pregnancy, is set out by Carlos Simon:

\[
\ldots \text{we have shown prospectively that supraphysiological serum E2 (estradiol) levels during the pre-implantation period are responsible for the impairment of embryonic implantation in patients undergoing IVF. It is possible that above normal (supraphysiological) serum E2 levels impair implantation through disrupting regulation of uterine paracrine factors; specifically, the IL-1 system is one possible candidate when considering what is reported in the present study.}^{29}\]

The term “paracrine” refers to the effect(s) that are caused by hormones but are localized to cells only in the immediate vicinity, \(^{30}\) i.e., the endometrium, rather than the more normal, wider area of bodily influence that characterizes hormones. \(^{31}\)

Simon’s research indicated that excessive estradiol (an estrogen) levels interfere with implantation as a consequence of disruption of the IL-1 system. IVF research has shown that high levels of estradiol (E2) result in a poor implantation rate of 8.5%, whereas reduced E2 levels increased the successful rate of implantation to 29.3%. \(^{32}\)

As Simon and co-workers noted, “High E2 levels, which are known to be interceptive, and altered E2/progesterone ratios, which also are associated with the impairment of endometrial receptivity, are the main factors affecting endometrial receptivity in high responders.” \(^{33}\)

The use of the word “interceptive” is significant. Professor Rahwan, professor of pharmacology and toxicology at Ohio State University, defines interception as the “interference with the implantation (nidation) of an already fertilized ovum, and, from a biological standpoint, must therefore be an early abortifacient approach.” \(^{34}\)

This research by Simon finds its importance within the context of the emerging use of the pill in high doses as a post-coital or “morning-after pill” (MAP). The MAP regime comprises the ingestion, within a time-frame of 12 hours, of approximately 10 times more estrogen and 10–20 times more progesterone than a woman would take via the normal once-a-day pill (depending on the brand used). These increased levels are obviously supra (above) physiological levels.

As previously outlined by Simon, the disruptive effect on implantation rates caused by high levels of estradiol, or incorrect estradiol/progesterone ratios, means it is biologically plausible to suggest the “morning-after pill” (MAP) is an abortifacient-empowered medication because of its capacity to interfere with the interleukin system.

Further supporting this assertion is research by Swahn et al. (1996), which showed that administration of the MAP caused a suppression of the LH surge, decreased the pregnandiol levels and increased the estrone levels (Fig. 1, p. 741). \(^{35}\) These alterations to the normal menstrual cycle hormonal patterns had an impact on the development of the endometrium.

An endometrial biopsy was taken one week after treatment. Although it was difficult to date the biopsy in some women because of the absence of a discernible LH peak, the conclusion was that the endometrium showed significant alterations in endometrial development with a dissociation in maturation of glandular and stromal components. \(^{36}\)
The authors then, in a seemingly contradictory manner, suggest that the “relatively minor changes in endometrial development does not seem sufficiently effective to prevent pregnancy.” This statement would appear to undermine any claim that the MAP acts in part via an abortifacient mechanism. Further reading reveals that the researchers did not investigate the “biochemical effects (of the pill) on molecular levels of the endometrium.” That is, the researchers did not investigate the hormonal impact of the MAP on the various implantation factors.

In my view, this omission negates their attempts to minimize the abortifacient significance of the “relatively minor changes in endometrial development” caused by the MAP. As will be seen later, relying only on measures of endometrial thickness cannot accurately assess the precise conditions needed for successful implantation—this exclusive approach fails to take heed of the implantation factors which are the second, vital characteristic associated with successful implantation.

1.4 Platelet-Activating Factor (PAF)

Another implantation factor which is associated with successful uterine receptivity of the human embryo is platelet-activating factor (PAF). PAF interacts with PAF receptors located on the endometrium. To recall, receptors are biochemical binding sites, located on the surface of cells, which are specifically designed to interact exclusively with a specific chemical, in this case PAF. When PAF attaches to the receptor, a message is conveyed to those cells.

The effect of PAF upon the endometrium is to cause a release of nitrous oxide (NO), leading to vascular dilation and increased vascular permeability of the blood vessels of the endometrium. The fact that chemical blockage of the PAF binding site (receptor) on the endometrium inhibits implantation supports the view that the PAF receptor has a critical role in uterine receptivity.

PAF is also involved in the cyclical development of the endometrium. Not surprisingly, the levels of the receptors for PAF vary throughout the menstrual cycle, with the highest endometrial levels detected during the mid-late proliferative phase (i.e., the days preceding ovulation) and the late secretory phase, when the endometrium is approaching or at its state of maximum monthly development. These findings are consistent with PAF having a preparatory role for uterine receptivity of the human embryo.

As was the case with the interleukin system, control of PAF is under the control of ovarian hormones, estradiol and progesterone. As Ahmed has noted: “PAF production has been shown to be regulated by ovarian hormones . . . .”

Given the role of ovarian hormones on the activity of PAF and its receptor within the endometrium, it is biologically plausible to suggest that irregular uterine hormone levels, caused by the pill, may have a negative impact on uterine preparedness for implantation. Supporting this view is the work of Rabe and co-workers, who reported a decrease in endometrial thickness in women taking the pill during the days when implantation would occur.

Specifically, these researchers showed that there was, for some pill users, a 50% reduction in endometrial development when compared to that seen in the control (non-pill using) group. Therefore, it is reasonable to conclude there is an adverse impact upon the expression of PAF receptors. Indeed, given the hormonal influence exerted by estrogen, it would be biologically illogical to conclude no damage to the expression of endometrial PAF receptors.
1.5 The Effect of Missed Pills on Ovulation

For the pill to exhibit the characteristic of an abortifacient, one biological event is essential: ovulation. The crucial question is this—does break-through (or escape) ovulation occur during regular pill ingestion?

Grimes et al. (Obstet Gynecol, 1994) had previously reported that “suppression of follicular development is incomplete with contemporary low-dose pills.” Grimes’ study was characterized by a high rate of patient compliance, meaning that the women involved in the study adhered to the research protocol of daily ingestion of the pill. Yet, escape-ovulation was detected even within the context of a rigorously scrutinized scientific study.

These facts argue strongly in favour of escape-ovulation also occurring within the general populace of women on the pill. This latter group of women is not necessarily as highly motivated as those participating in a scientific study. To adhere to a tedious daily, monthly, and yearly regime of pill ingestion without supervision is, in the words of one feminist writer, a “bore and a chore.” Because daily pill ingestion is so onerous, patient compliance will be less than the necessary ideal. However, does the occasional failure to take the pill mean that escape-ovulation will increase in some proportional fashion?

In an attempt to determine the frequency of escape-ovulation under more realistic conditions, researchers have constructed experiments that required women in the study to deliberately miss one or more days of the pill. A variety of tests including ultrasound of the ovaries, and measurements of estradiol (E2), progesterone (P) levels, and LH (luteinizing hormone) were used to determine if ovulation had occurred.

Hedon and co-workers (1992) tested 47 young, healthy women who missed between 1 and 4 days’ tablets starting from day 1 of a new cycle. “None of the patients experienced normal ovulation” though one, who missed 3 tablets at the beginning of the cycle, “had a follicular rupture,” but no LH surge or progesterone increases, factors usually associated with normal ovulation. Note that this study was for only one cycle. Limiting the study to one cycle was a weakness because any follicles which may have ruptured during the normal 7 pill-free days between cycles would not be detected.

Earlier, Hamilton (1989) had performed a similar study but extended the observations for two consecutive months. Of 30 women in the study, one had a probable ovulation, due to one deliberately omitted tablet on day one of the second cycle.

More recently, Letterie (1998) published the results of a study employing a new, reduced dosage formulation of the pill. Ten women, divided into 2 groups, used two slightly different formulations comprising a delayed start, limited midcycle use of estrogen and progesterone. Each of the two treatment groups was monitored for 2 consecutive cycles. In total, 30% of cycles exhibited ovulation, all of which occurred in the second cycle.

It is revealing to look more closely at the data for the two groups. In group one, ovulation occurred in 10% of cycles (1 in 10 cycles). This group took 50mcg ethinyl estradiol/1mg norethindrone for days 6–10 and 0.7mg norethindrone for days 11–19. Group two took 50mcg ethinyl estradiol/1mg norethindrone for days 8–12, and 0.7mg norethindrone only for days 13–21—“five ovulation(s) occurred in 10 cycles.” This is an ovulation rate of 50%. This study did not
investigate implantation; all participants used barrier contraceptives or abstinence (private correspondence).37

It should be noted that these research findings, conducted under ideal research conditions, represent the best possible outcome in terms of ovulation suppression by the pill. Yet these results do not faithfully replicate real-life because they do not take into account such common events as gastro-intestinal illness or drug interactions. Stomach upset decreases drug absorption, thus loosening the hold over ovulation otherwise exerted by the pill hormones. Likewise, drug interactions decrease the amount of active pill hormone available to act in a suppressant manner upon the ovaries.58,59 Other researchers and I are of the view that these two issues would contribute to an increase in the frequency of escape-ovulation.60

1.6 Pill Control Over Ovarian Follicular Development

Based upon my 20 years experience as a community pharmacist, I believe the commonly held view is that the pill fully stops ovulation (anovulation). Yet this view is wrong. The recent work by Rabe et al. (1997) contradicts this common misunderstanding. Following are some salient points from this research.

• Pre-ovulatory follicular cysts (> 20mm) occurred in 7.3% of 329 pill users enrolled in the study.61 This size of follicle is identified with an increased rate of escape-ovulation.62
• For non-pill users, the rate of follicular cysts was 13.9%.
• Some women, notably those on triphasic formulations, had follicles measuring 60mm.
• Estradiol was present at higher levels (in pill users with enlarged follicles) than in non-pill users (who also had enlarged follicles). The respective levels were 153 pg/ml and 126 pg/ml.63

The estradiol level of 153 pg/ml, seen in pill users with enlarged follicles, is important, as it is close to the “threshold level of 150 to 200pg/ml,” which, if persisting for approximately 36 hours, triggers ovulation.64

As a summary of their research, Rabe noted: “Analysis of the ovarian activity in the current study demonstrated that the total number of developing follicles increased rather than diminished during OC use, without marked differences between OCs.”65

This research underscores the pill’s precarious hold over ovulation suppression. It is an event endeavouring to occur. The intervention of a variety of “lifestyle” factors such as missed doses, drug interactions or gastrointestinal upset can act to loose the hold exerted by the pill over natural ovarian function.

As a footnote to this discussion, the FDA approved, in late 1998, a low-dose estrogen formulation of the pill (norethindrone acetate, 1 mg; ethinyl estradiol, 20 mcg). Similar low-dose estrogen formulations are also now available in Australia.66 The frequency of escape-ovulation can only be expected to increase in situations of reduced hormonal ingestion.

1.7 Endometrial Thickness and Implantation

Thus, the question arises: will a low-dose pill, more inclined than not to permit escape-ovulation, increase the frequency of implantation failure due to an under-developed endometrium? The
medical literature indicates that there is a critical thickness of the endometrium needed to sustain implantation of a human embryo.

Isaacs (Fert Steril, 1996) reported that an endometrial thickness of at least 10mm or more around the time of ovulation “defined 91% of conception cycles.” Spandorfer (Fertil Steril, 1996) noted that 97% of abnormal pregnancies, defined as Fallopian tube lodgment or spontaneous abortion, had endometrial thickness of 8mm or less. Shoham (Fert Steril, 1991) reported that a mid-luteal thickness of 11 mm or more “was found to be a good prognostic factor for detecting early pregnancy,” but no pregnancies were reported in an ovulation induction programme “when the endometrial thickness was less than or equal to 7mm.”

The mid-luteal phase of the menstrual cycle, around day 20, is referred to in the medical literature as the window of expected implantation.

Gonen (Journ In Vitro Fert Embryo Transf, 1990) also reported that “endometrial thickness was significantly greater in the group of patients who achieved pregnancy than in the group who did not.” Implantation failure was associated with endometrial thickness of approximately 7.5mm, success with endometrial thickness of approximately 8.5–9mm.

These study results, which indicate a normative endometrial thickness of around 8.5mm for successful implantation, are central to any claimed interceptive/abortifacient capacity of the pill. Research findings from Rabe and co-workers (1997) underscore this point.

Rabe reported that study subjects who took the triphasic levonorgestrel/ethinylestradiol formulation had the highest percentage of follicular cysts with a diameter greater than 20mm, but they failed to develop a median endometrial thickness in excess of 6mm. To recall, follicles of this size are “thought to be associated with increased risk of escape ovulation.”

The importance of these events is clear: follicles of a suitable size can develop in women taking the pill daily, but endometrial thickness has been shown to be underdeveloped. In the event of follicle rupture and release of an “ovum,” implantation of a human embryo would be greatly hampered. Rabe confirms this very point: “. . . the occurrence of pregnancy would be unlikely because accessory contraceptive mechanisms such as cervical hostility and endometrial suppression are usually in effect.”

It must be pointed out that in this quote Rabe has falsely defined pregnancy as beginning at implantation. Pregnancy begins with the fertilization of the female sex cell (ovum) by sperm, the restoration of the full complement of 23 pairs of chromosomes and thereby the creation of a new human person.

Based upon these findings, a number of issues present themselves:

- An endometrial thickness around 8.5mm has been shown to be associated with successful implantation.
- Low dose triphasic formulations of the pill, the most popular in Australia, fail to completely stop follicular development, the precursor stage to the release of a female sex cell.
- Break-through ovulation is an event straining to occur, even with daily pill ingestion.
- If break-through ovulation were to occur, implantation might fail because of an endometrium that is too thin.

It is important to note that these four observations exist independently of the impact of the pill on the various implantation factors involved in cell-signaling.
1.8 Integrins

As the aforementioned research indicates, the last few years have seen a remarkable unveiling of the process of implantation of the human embryo into the uterine tissue. A large body of evidence now exists which demonstrates that the process of implantation, rather than being an accidental event dependent on chance, is in fact a multi-factorial, cascading bio-molecular, physiological and hormonal event of spectacular intricacy, complexity, refinement and interdependence. Implantation is not, as one might suppose, akin to two pieces of Velcro fortuitously touching and gripping together. Rather, implantation is, in every sense, as complex, and therefore susceptible to interference, as is the clotting mechanisms of the cardiovascular system.

Besides PAF, the interleukin system and other factors mentioned briefly in the introduction, the class of cell adhesion molecules known as integrins also play a critical role in successful implantation of the human embryo into the endometrium.

As the description of the molecule suggests, the role of integrins is to bind cells together. Etzioni has suggested that integrin facilitated cell adhesion is “a process that is essential for anchorage” of cells to each other (Lancet, 1999).

There are a variety of different types of integrins found within the body—one that plays an essential role in implantation is known as ανβ3. The medical literature now contains many research papers demonstrating the vital role of this integrin in the process of binding the 5–7 day old human embryo to the endometrium (lining of the womb).

Somkuti and co-workers (Fert Steril, 1996) for example reported that integrins “might prove useful as markers of normal endometrial receptivity” because they have been shown to be absent in women with unexplained infertility and endometriosis. Similarly, Lessey (Am J Reprod Immunol, 1996) reported that “aberrant expression of this integrin is associated with infertility in women.” Widra (Mol Hum Reprod, 1997) noted “the absence of endometrial ctv133 during the critical period of implantation . . . in women with unexplained infertility and endometriosis.” Others had also commented on the absence or diminution of ανβ3 in women with recurrent pregnancy loss or unexplained infertility.

Assessing the role of the pill, Somkuti (1996) compared endometrial sampling from women on the pill with samples from non-users and reported integrin expression “to be altered grossly in OC users.” Complementing this work were the observations of Yoshimura (1997): “. . . a loss of normal ανβ3 expression is associated with primary infertility and milder forms of the disease. These observations suggest that this integrin plays a significant role in the implantation process.”

Eric Widra and colleagues (1997) at Georgetown University investigated the role of physiological levels of estrogen and progesterone on the endometrial levels of ανβ3. They reported that estrogen caused a down-regulation in the expression of ανβ3, an important finding in the light of the fact that “expression of the ανβ3 integrin may, in fact, be necessary for normal implantation to occur.”

Castelbaum and co-workers, (J Clin Endo Metab, 1997) reported the endometrial expression (presence) of ανβ3 was “reduced by E2 treatment and further suppressed by E2 plus P . . . .” These results indicate a link between the impact of hormones on the expression of integrins and the role of integrins in implantation. Whilst the inter-relationship between hormones, integrins and implantation is not yet fully understood, sufficient evidence exists to conclude that the inter-
relationship is significant from the perspective of implantation. This is because implantation occurs only “on or about day 20 of an idealized 28-day menstrual cycle” and the αβ integrin “is expressed on endometrial epithelial cells only at the opening of the implantation window, on postovulatory day 6.”

1.9 Insulin-Like Growth Factor (IGF)

The IGF system is an important growth factor, playing a key role in the monthly development of the endometrium and in the process of implantation. There are two subsets, IGF-1 and IGF-2. The first is believed to facilitate the mitotic action of estradiol [E2] in the endometrium, whilst IGF-2 “expressed abundantly in mid-late secretory endometrium, may be a mediator of progesterone action.” Aside from this hormonal aspect, the most abundant expression of IGF-11 is in the columns of the invading trophoblast in the anchoring villi.

From this it can be seen that IGF has a promotional effect upon the process of implantation. But IGF is in turn regulated. “The biological actions of IGFs are modulated by a family of binding proteins (IGFBPs). The demonstration of IGF and IGFBP transcripts [copying facilities] in pre-implantation embryos indicates that the influence of IGFs and IGFBPs in fetal development begins even prior to implantation.”

Thus far, it can be seen that these factors have a key role to play in both the preparation for and process of implantation. As Han et al. have noted: “Presumably, IGF-II and IGFBPs are used for cell-to-cell communications between fetal trophoblasts and maternal decidual cells at the feto-maternal interface for placental development and/or function.”

Against this background, the role of the hormones in the pill, particularly their influence on implantation, is important. A number of researchers have shown that the pill causes an increase in IGFBP-1 levels and a decrease in plasma concentrations of IGF-1. More specifically, during the pill-free week “IGFBP-1 was significantly lower on the medication-free day than on day 14 of the cycle . . . . The short absence of exogenous estrogen and progestin during the medication-free week also affected IGF-1 levels, which were significantly increased.”

The superabundance of IGFBP induced by the pill has, from an implantation perspective, significance. Giudice has reported that: “IGFBP’s bind IGF’s with high affinity and, for the most part, inhibit IGF bioavailability to their receptors for action in their target organs.” Thus, the supraphysiological levels of IGFBP, induced by the pill, may be detrimental to the process of implantation via an inhibitor action on the levels of IGF. Giudice highlights this point: “IGFBP-1 has been shown to inhibit trophoblast invasion into decidualised endometrial stromal cultures, suggesting that this IGFBP-1 is a maternal ‘restraint’ on trophoblast invasion.”

Aside from the indirect anti-implantation effect of excessive levels of IGFBP upon IGF, IGFBP also has a direct, anti-attachment effect upon the human embryo. “ . . . IGFBP-1 specifically binds to the first trimester trophoblast and that it binds to the α5β1 integrin in the trophoblast. Furthermore, it inhibits trophoblast attachment to fibronectin, another RGB ligand found in the placental bed.”

In summary, the pill causes an increase in IGFBP levels, leading to a decrease in IGF levels. This may have a negative impact upon implantation. IGFBP also may have a direct effect at the level of trophoblast/endometrial integrin binding. More research is required to understand fully the roles of IGF and IGFBP. This represents a new, emerging field of research into the multitudinous factors involved in the process of implantation. Whilst the above research indicates that the pill facilitates an anti-implantation endometrial environment, confirming evidence is yet to be found. Hence there exists a reasonable suspicion only, a point made by key researchers in the field.
1.10 Conclusion

This discussion has had as its focus the multifactorial nature of embryo implantation. On occasion, this discussion has required detailed analysis of the relevant factors influencing the success of this event. Sometimes it is not possible to speak of these events, centred as they are on the maintenance of human life, without a certain measure of complexity and detail. To those readers who have struggled with this material, I apologize.

This paper does not presume to be the final word on this complex and evolving branch of medical knowledge. New research appears almost monthly to illuminate further and sometimes confuse this emerging medical discipline. Nevertheless, I hope I have briefed the reader on issues related to the first right of all humans—the right to stay alive. Some may seek to discount the interceptive/abortifacient capacity of the pill. For three reasons, this would be a scientifically precarious position to adopt.

[21]

First, I am of the view that the preceding evidence strongly argues the case in favour of the pill possessing an interceptive/abortifacient capacity. At the very least, the evidence is repetitive and circumstantial. Indeed, how more clear and straightforward could the issue be than the following statement from Eric Widra and colleagues? “Demonstration of complimentary integrin expression on preimplantation embryos has further buttressed the argument that these molecules are important for the initiation of pregnancy.”

Second, even researchers view as the new arena of “contraceptive” research the interrelated system of implantation factors. Carlos Simon and colleagues (Fert Steril, 1998), after discussing the interdependent relationship between the interleukin-1 system, the αvβ3 integrin adhesion system and implantation, conclude by stating that the interleukin-1 system would be a promising new area of research apropos the development of new “contraceptives.” Given this sentiment, I am of the view that anti-interleukin chemicals will be the RU-486 of the next decade.

Third, and most tellingly, the abortifacient capacity of the pill is recognized by those who support abortion. Consider the following, taking from the Guttmacher Report. “The best scientific evidence suggests that ECP’s [emergency contraceptive pills] most often work by suppressing ovulation. But depending on the timing of intercourse in relation to a woman’s hormonal cycle, they—as is the case with all hormonal contraceptive methods—also may prevent pregnancy either by preventing fertilization or by preventing implantation of a fertilized egg in the uterus” (my emphasis).

Need any more be said?

References


23. Ibid., 180.

24. Ibid., 166.

25. Ibid., 180.


27. Huang, op. cit.


29. Loc cit.

31. Ibid., p. 773.

32. Simon, C., *Fert Steril* (1998); 237, Table 3.

33. Ibid., p. 238.


36. Ibid., p. 742.

37. Ibid., p. 743.

38. Ibid.


44. Sat, op cit., p. 99.


47. Ahmed, op cit., p. 841.


49. Ibid., table 4, p. 46.


51. Ibid., p. 33.


56. Ibid., p. 39.

57. Private Correspondence (Nov 11, 1998).


60. Rabe, op. cit., p. 48.

61. Ibid, p. 43.

62. Sullivan, H., Furniss, H., et al., “Effect of 21-day and 24-day Oral Contraceptive Regimens Containing Gestodene (60ug) and Ethinyl Estradiol (15ug) on Ovarian Activity,” *Fert Steril.* 72:1 (1999), 115-120: “Ovulation was defined as the presence of a follicle-like structure that was <13mm in diameter and ruptured within 48 hours combined with serum 1713-E2 and progesterone concentrations of >30pg/ml and >1.6ng/ml, respectively, in the same cycle.” Ibid., p. 116.

63. Rabe, op. cit., p. 45.


65. Ibid., p. 48.


72. Gonen, Y., Casper, R.F., *Journ In Vitro Fert Embryo Transf* 7:3 (1990), 146-52 (8.5mm +/- 0.4mm vs. 7.5mm +/- 0.2mm, P, 0.01).

73. Rabe, Table 3, p. 44.

74. Ibid., Figures 4 & 5, p. 46.

75. Ibid., p. 43.

76. Ibid., p. 48.


81. Ibid., p. 484.


88. Widra, op. cit., p. 566, Table 1.

89. Ibid., p. 563.


92. Loc. cit.

93. Castelbaum, op. cit., p. 140.


95. Guidice, p. 135.


100. Westwood, p. 533.


103. Guidice, p. 142.

104. Guidice, p. 141.

105. Widra, op. cit., p. 563.


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INTRODUCTION

Christians are increasingly being exposed to the medical and theological debate concerning the potential abortifacient effect of the birth control pill (the Pill). Some argue that the Pill, in both of its forms (the oral combined oral contraceptive [COC], containing estrogen and progesterone hormones, and the oral progestin-only pill [POP], containing only progesterone hormone) has an abortifacient effect, at least some of the time. By “abortifacient effect,” they mean that the Pill causes the unnatural and unrecognized death of preborn children sometime between conception and “patient recognized pregnancy”—the time when the woman realizes that she is pregnant, either by signs or symptoms. A patient-recognized pregnancy can be clinically confirmed by physical examination, ultrasound or laboratory testing. By “preborn child” is meant the developing human life that secular physicians medically label, depending upon the stage of development, as a morula, a zygote, a blastocyst, a preembryo (sic), a conceptus, or an embryo.

Other physician experts argue that the possibility of the Pill causing an abortifacient effect is either “non-existent” or “infinitesimally small.” For the purposes of this paper, the former group will be called the “abortifacient theory proponents” or “proponents” and the latter group will be called the “abortifacient theory opponents” or “opponents.” It appears to this author, that among practicing physicians and among those obstetrician-gynecologists who have studied the subject and written opinions, the majority are in the “opponent” camp. However, it also appears that more information has been published and distributed by the “proponents.” Further, the only studies that have been accepted for publication in national and peer-reviewed medical journals represent the “proponent” position.

Some opponents use the term “mini-abortion” to refer to the abortion of a preborn child prior to or just following implantation. Proponents have objected to this term, declaring that it appears to devalue the preborn. Opponents say that the term “mini-abortion” is only meant to indicate that the microscopic preborn child is much, much smaller than at later stages of development. For the purposes of this paper the term “abortifacient effect” or “abortion” will be applied to the death of human life from conception to the point of that life being able to live outside of the mother’s womb.

National groups, ministries, commissions or publications have published information that appears to favor one of several positions: (1) The “proponent” view appears to be supported by the American Academy of Natural Family Planning, the American Life League, Eternal Perspective Ministries, Human Life International, One More Soul, Pharmacists for Life, the Study of Abortion Deaths Commission and the journal Life Advocate; (2) A “neutral” view seems to be supported by the Christian Medical and Dental Society and the WELS Lutherans for Life; (3) The “opponent” view is supported by a group consisting of twenty-three well-respected academic and
private-practice, pro-life, obstetrician-gynecologists and has been expanded by a group of four obstetrician-gynecologists. National groups that are currently discussing or debating the issue, but have yet to publish or publicly release an opinion, include (but may not be limited to) the National Right to Life, the American Association of Prolife Obstetrician-Gynecologists (AAPLOG), the American Association of Prolife Family Physicians (AAPFP), the Physicians’ Resource Council of Focus on the Family, the Family Resource Council (FRC), The Center for Bioethics and Human Dignity and the Catholic Medical Association (CMA).

PREMISES

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…The term “contraception” is the process by which conception is prevented (“contra” = “against”; “ception” = a root word for the word “conception”). Some would differentiate “natural” contraception (such as modern, medical natural family planning) and “artificial” contraception, based on the concepts of cooperating with versus suppressing natural fertility processes. "Birth control” is a process by which birth is prevented, whether conception occurs or not. For example, a medical abortion is birth control but not contraception. Using these definitions, birth control methods that are “natural contraceptives” include abstinence, periodic abstinence, and natural family planning (a variety of methods), and birth control methods that are “artificial contraceptives” include the diaphragm, condom (male or female) and spermicidal sponge, creams and gels.

Among Christians there are a variety of theological views concerning the morality of contraception. There are those who would contend that it is unethical to use any contraceptive mechanism or method. Others believe it is unethical to use most “unnatural” or “artificial” forms of contraception. Still others believe virtually any form of contraception is ethical. It appears that the majority of those who have published on this issue (at least since 1950) would allow for ethical reasons to contracept. This paper is not meant to discuss the ethics of contraception, as this has been done elsewhere, but makes the assumption that birth spacing using contraception can be ethical, following the principles outlined by Meilaender and Turner. However, for those who hold that valuable human life begins at conception, then ethical birth control must be exclusively contraceptive: e.g., it must (1) work exclusively (or, some would say nearly exclusively) by preventing conception from occurring and (2) cause no harm to the conceived but preborn child.

Finally, for the purposes of this paper, it is assumed that the principle of “double effect” is more likely to help Christians determine moral or ethical actions in medicine than the principle of utilitarianism.

THE MEDICAL EVIDENCE

Both proponents and opponents seem to agree that the risk of an abortifacient effect with the POP and Norplant® (subcutaneously implanted progesterone rods) are such that, in general, it would be unethical to use or prescribe these products as a contraceptive. In other words, the POP and Norplant® appear to have an abortifacient or post-fertilization effect, at least some of the time. Of POPs, opponents have stated, for example, that “POPs are much less effective birth control . . . although they have potential advantages for select patients.” They go on to say, “POPs . . . are
associated with higher ectopic (tubal) pregnancy rates, exposing the user to increased potential for morbidity and even mortality. This may constitute an unacceptable risk for the use of these products.” Proponents have said, “For POPs . . . postfertilization effects are likely to have an increased role.” However, proponents and opponents derive different conclusions when it comes to the COCs or injectible progesterone (i.e., Depoprovera®). Since COCs are used much more frequently than Depoprovera®, this paper will examine the COC. The following arguments for and against an abortifacient effect of the Pill were distilled from four excellent reviews on the subject.

The ‘Hostile’ or ‘Unreceptive’ Endometrium Theory

Proponents cite a large number of medical studies that document that the uterine lining (endometrium), the “home in which newly conceived human life implants and develops,” is dramatically changed by the Pill. They cite scores of studies that seem to document that the endometrial structure, biochemistry and function are all dramatically changed by the Pill. They feel that most of these studies conclude that the pill-induced endometrial changes render the endometrium “hostile” or “unreceptive” to implantation “at least some of the time.” Proponents also point to secular research opinion that these endometrial “changes have functional significance and provide evidence that reduced endometrial receptivity does indeed contribute to the contraceptive efficacy of (the Pill).” Proponents believe that no published studies have refuted these findings.

Although proponents admit, and opponents point out, that this is not direct proof of an abortifacient effect of the Pill, it is felt by the proponents to be indirect proof of “a very high order.” They state that the presumption that these pill-induced endometrial changes reduce the chance of implantation and increase the chance of an unrecognized, pill-induced abortion of the preborn is so well-accepted in the medical world that the Food and Drug Administration’s (FDA’s) approved product information for the Pill in the Physicians Desk Reference (PDR) says, “Although the primary mechanism of action is inhibition of ovulation, other alterations include changes in the cervical mucus, which increase the difficulty of sperm entry into the uterus and changes in the endometrium that reduce the likelihood of implantation.” To proponents, this is an FDA admission of the abortifacient effect of the Pill.

Further, proponents cite Magnetic Resonance Imaging (MRI) studies that show that the endometrial lining of Pill users is significantly thinner than that of non-users. They also cite nine recent and fairly sophisticated ultrasound studies that have all concluded that endometrial thickness is related to the “functional receptivity” of the endometrium in women who are infertile. Some of these studies, they say, show that when the endometrium becomes too thin, at least in infertile women, implantation of the preborn child does not occur. They point out that the minimal endometrial thickness required to maintain a pregnancy in infertile patients ranges from 5 to 13mm, whereas the average endometrial thickness in women on the Pill is 1.1 mm. They feel that these data lend credence to the FDA-approved statement that there are Pill-induced “changes in the endometrium which reduce the likelihood of implantation.”

Opponents reply that the assertion that any “hostile” endometrium causes unintended abortions of preborn children in women on the Pill has absolutely no direct supporting medical evidence. Opponents claim that the “hostile endometrium theory is unproven assertion.”
Further, they state that the FDA-approved statements about the Pill-induced changes to the endometrium are accurate only when the woman does not ovulate (“ovulation” is the process whereby the ovary releases an egg [ovum] into the abdominal cavity). They believe that if the woman taking the Pill has a “breakthrough ovulation,” that a whole new hormone environment comes into play.\textsuperscript{10,11} They feel that the hormonal changes occurring after ovulation have seven days to act on the lining of the uterus (the endometrium) to prepare it for implantation.\textsuperscript{10,11} They feel that these hormones will normalize the endometrium, whether the woman is on the Pill or not.\textsuperscript{10,11} They feel this is the reason that unexpected pregnancies on the Pill do as well as any other pregnancies (at least after the pregnancy is clinically recognized).\textsuperscript{10,11}

Proponents counter that the opponent’s theory that a breakthrough ovulation on the Pill will normalize the endometrium has no supporting medical studies.\textsuperscript{2} Further, they point out that after a woman stops taking the Pill, it can take several cycles for her menstrual flow to increase to the volume of women who are not on the Pill,\textsuperscript{38} suggesting to them that the endometrium is slow to recover from its Pill-induced thinning.\textsuperscript{2} They also cite an older study that looked at women who ovulated on the Pill.\textsuperscript{39} This study showed that after ovulation the endometrium did not appear to be receptive to implantation. Proponents feel that this study directly refutes the theory that a breakthrough ovulation on the Pill will normalize the lining of the uterus and supports the potential that the Pill causes unrecognized loss (death) of preborn children, at least some of the time.\textsuperscript{2}

**Ectopic Pregnancy Risk on the Pill**

Another argument proposed by the proponents is this: They argue that if the Pill has no abortifacient (postfertilization) effect, then the reduction in the rate of intrauterine pregnancies (IUPs) in Pill-takers should be identical to the reduction in the rate of extrauterine (ectopic or tubal) pregnancies (EUPs) in Pill-takers.\textsuperscript{2} They argue that if there is an increased extrauterine/intrauterine pregnancy (EUP/IUP) ratio, this would constitute conclusive evidence for an abortifacient effect.\textsuperscript{2}

Proponents cite at least two medical studies that have shown an increased EUP/IUP ratio.\textsuperscript{40,41} They point out that these data came from seven maternity hospitals in Paris, France\textsuperscript{41} and three in Sweden\textsuperscript{40} and involved a total of 380 women with ectopic pregnancies and 380 pregnant controls (women who become pregnant while using the Pill).\textsuperscript{2} Proponents point out that secular researchers who have reviewed these studies have suggested that at least some of the Pill’s birth control effect is provided via a postfertilization (or abortifacient) effect.\textsuperscript{41,42}

Opponents point out,\textsuperscript{11,12} and proponents admit,\textsuperscript{2} that EUP studies that compare women with EUP to a non-pregnant control group do not show an increased risk of EUP for Pill-users.\textsuperscript{11} Opponents feel that comparing EUP patients with pregnant controls results in unreliable data and conclusions.\textsuperscript{11} Therefore, opponents totally discount the EUP data that compares EUP patients with pregnant controls. However, there is, as yet, no published, peer-reviewed research that substantiates the opponents’ opinion. Further, proponents assert that only the data comparing EUP patients to pregnant controls is valid. They substantiate their claim by pointing to published secular research opinions that state that, “... when considering the situation where a woman became pregnant during contraceptive use, one should focus (exclusively) on pregnant controls.”\textsuperscript{41,42} Therefore, proponents say, the elevated IUP/EUP ratios in women on the Pill is strong evidence that the Pill is associated with an abortifacient effect, at least some of the time.\textsuperscript{2,3}
Conclusions About the Medical Evidence

Proponents and most opponents seem to agree that the use of POPs and Norplant® as contraceptives are, in general, unethical. Thus, the debate and controversy seem to swirl around COCs, which are the most common form of birth control (exclusive of sterilization) used by women.

Concerning the EUP data, even proponents have to admit that the risk is very small. They estimate that the absolute risk of an EUP in a woman on the Pill would be 1 to 20 per year for every 1000 women using a COC for an entire year. Only one study in the medical literature, from Zimbabwe, has reported an absolute risk of EUP in women on COCs and it reported a rate of 1 per 2000 women who used the COC for one year. Assuming that these data could be generalized to other women, if a woman took the Pill for 20 years, then she would have only a 1% risk of EUP. Proponents point out that there are women who would never choose a medical abortion, even once in 20 years, and that this data “proves” that the risk of an abortifacient effect of the Pill is not “infinitesimally small” and certainly not “nonexistent.” Further, proponents argue that the unreceptive endometrium in Pill-takers is much more likely to cause unrecognized abortion of the preborn child than the recognizable EUP.

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However, for the most part, proponents and opponents agree that their arguments about the data are qualitative and not quantitative. In general, both sides agree that there is no direct proof—no “cause and effect” proof—that the endometrial changes cause unrecognized abortions in women on COCs. The proponents clearly believe the evidence is strong, or extremely strong. The opponents believe the evidence is nonexistent, or extremely weak. Both sides admit that it is not possible from the current data to predict just how often an abortion might occur.

Proponents argue that even if the effect is rare, there are so many millions of women on the Pill, that even a very rare effect could abort countless preborn children. Further, they say that the abortifacient effect can potentially occur in any woman who is taking the Pill: e.g., that when a woman takes the Pill she is playing a “form of Russian roulette with her preborn child.” They feel that the longer a woman takes the pill, the greater the chance she has of the Pill causing an unrecognized abortion. Opponents point out that for any particular woman they would predict that the risk of an unrecognized abortion is “infinitesimally small.”

Should Women be Informed About This Controversy?

Many reproductive scientists have defined pregnancy as occurring at the point of or at some point after implantation. However, this definition does not change the fact that many patients identify the start of human life as beginning with fertilization. For many of these patients, a form of birth control that may allow fertilization and then cause loss of the preborn child is unacceptable. Regardless of the personal belief of the physician or provider about the mechanism of the action of the Pill, it is important that patients have information relevant to their own belief and value systems. Some physicians have suggested that post-fertilization loss attributed to the Pill would not need to be included in an informed consent until it is either definitely proved to exist or proven to be a common event. However, rare but important events are an essential part of other informed consent
discussions in medicine, primarily when the rare possibility would be judged by the patient to be important. For example, anesthesia-related deaths are extremely rare for elective surgery (<1:25,000 cases); nevertheless, it is considered appropriate and legally necessary to discuss this rare possibility with patients before such surgery because the possibility of death is so important to patients. Therefore, for women for whom the induced loss of a preborn child is important, failure to discuss this possibility, even if the possibility is judged to be remote, would constitute a failure of informed consent.

There is a potential for negative psychological impact on women who believe human life begins at fertilization, who have not given informed consent about the Pill, and who later learn of the potential of post-fertilization effects of the Pill.46 The response to this could include disappointment, anger, guilt, sadness, rage, depression or a sense of having been violated by the provider.47

DO INTENTIONS MATTER?

Opponents seem to agree with proponents that if the Pill does have an abortifacient effect, that this effect would be a bad effect, a bad consequence.11 Proponents say this bad consequence of taking or prescribing the COC is probable, at least on occasion. Further, they point out that the longer a woman takes the Pill, the greater her chance of having an unrecognized, pill-caused abortion. Opponents say this bad consequence is very unlikely. Therefore, those not versed in the technical intricacies of these medical arguments and unable to decide “which side is right” are left with the dilemma of deciding whether to take or prescribe the COC until or if the medical controversy is resolved.

Opponents have argued that physicians who prescribe the Pill and women who take the Pill do so almost universally to prevent ovulation and that the Pill prevents ovulation the vast majority of the time it is taken (although they concede that there is breakthrough ovulation on the Pill). Opponents point out that physicians who prescribe the Pill and patients who take it intend that the COC be contraceptive. Opponents argue that this intention, which is good and ethical, supersedes any potential rare and unintended bad consequence—such as a possible abortifacient effect. Proponents have argued that the effect is bad, no matter what the intention.

Indeed, intention is viewed as important in medical ethics since it not only can help to determine whether an action is right or wrong, but has been used to help define the nature of the act itself and the kind of person who is performing the act.34,35 Therefore, Christian ethicists point out that it is not always bad to produce bad consequences.34,35 They point out that morality is not just about consequences. There are times when good consequences can actually be ethically bad, based upon a bad or unethical intention. On occasion, bad consequences can be ethical if based upon good intention. However, to know when it is morally possible to produce bad consequences, Christian ethicists often resort to an ethical principle called the principle of “double effect.”24,48

The Principle of the Double Effect

The principle allows the performance of an act (such as prescribing or taking the Pill) that has good and (potential or actual) bad consequences only if the following conditions are met:34,48

1. The act of prescribing or taking the Pill must be ethical—it must be morally good (or, at the very worst, morally neutral). In other words, the act itself must not intrinsically be a bad act.
2. The person prescribing or taking the Pill must intend for the action of the medication to be moral (or good). In other words, he or she in no way intends a bad effect or consequence.

3. The good effect of the Pill (its birth control effect) does not follow a bad effect (i.e., an abortifacient effect). In other words, a bad effect cannot be a means to a good effect.

4. If there is a bad effect or consequence, then there must be sufficiently serious moral reason(s) for allowing the bad effect to occur. In other words, the good effect that is intended has sufficiently moral and ethical value to justify allowing or tolerating the bad effect.

5. And further, as a corollary to number four, there must be no other way of producing the good effect.

This principle has a long and rich history in western ethics and medicine and is increasingly called upon in modern medical ethics to determine the rightness or morality of actions with good and bad effects. The principle of double effect is often opposed by the principle of “consequentialism” or “utilitarianism.” The latter principle is increasingly popular in medical ethics and teaches that the rightness or wrongness of an act is determined primarily by its consequences or results.

Application of the Principle of Double Effect to the Pill Data

Based upon the principle of double effect, then, is it ethical or not to take or prescribe the Pill given this scientific controversy? To be an ethical action, all of the above conditions will need to be met. With the COC, are they?

As discussed in the assumptions section of this paper, for the purposes of this paper it is assumed that birth spacing with good intention and with contraceptive agents can be ethical. Therefore, by definition, condition one is met. In addition, for the purpose of this discussion, it is conceded and/or assumed that virtually all prescribing physicians and women taking the Pill are doing so with good intention. Therefore, condition two is also met.

Since most proponents and opponents agree that an abortifacient effect of the Pill, should it occur, is likely to occur only a minority of the time (if at all, say the opponents), then condition three is met—in the sense that the vast majority of the time the good effect of the pill does not depend upon a possible (or even probable) bad effect (an abortifacient effect). Therefore, for this discussion, it is declared that condition three is met; however, it is also conceded that this is a debatable point.

Condition four of the principle of double effect is hotly debated by the proponents and the opponents. It is not the purpose of this paper to repeat the fullness of this discussion; however, to summarize two of the arguments:

1. Opponents argue that women who do not have access to the Pill are more likely to become pregnant and then more likely, in industrialized societies, to choose abortion and, in primitive societies, to die from pregnancy. Thus, they imply, condition four is met. Proponents argue that this premise is not true, as only a small minority would choose not to take the Pill because it causes early abortions, and these same people (presumably Christians and other theists) would in all likelihood be the very last ones to try to obtain a medical abortion.
2. Opponents state that studies indicate that up to 80% of conceived embryos naturally fail to implant and they point out that the Pill, by lowering the rate of conception, will lower the total absolute number of deaths of the preborn. They seem to be saying that if the Pill kills some children, consolation can be had under condition four in knowing that the Pill prevents many other preborn children from ever being conceived and therefore from dying “naturally.” Proponents argue that if there are fewer abortions because of the Pill, it is not because the Pill brings any benefit to a preborn child, but only because it results in fewer preborn children being conceived. They imply that it is not that lives are being preserved, but simply that there are fewer lives to preserve and that humans are instructed in Scripture to take responsibility for their choices, not for God’s.

Were our discussion to end at this point, the controversy certainly might be considered “unsettled,” or “debatable.” It certainly could be considered to fall under the category of “disputable matters” discussed in Romans 14:1-21. Objective, knowledgeable Christian observers would in all likelihood line up on one side or the other of the argument, based upon a variety of subjective and objective criteria. However, the principle of double effect has one last condition that must be considered and that condition relates to alternatives. In other words, the principle makes the condition that there must be no other way to produce the good effect. Indeed, there may be.

Natural Family Planning—A Viable Option to the Pill

Only over the last decade has modern, scientific natural family planning (NFP) become established in the medical literature. Nevertheless, many physicians and most women view natural family planning only as the old-fashioned and mostly ineffective rhythm method. The old joke goes something like this: “What do you call a couple who uses the rhythm method for birth control?” The answer: “Parents!”

Many are surprised to learn that one form of NFP, developed at Creighton University (called the NaPro™ method), has been medically studied over the last 20 years and has been reported in one meta-analysis to be even more effective than the Pill at preventing pregnancy. This meta-analysis reported five studies that recorded 1,876 couples who used the NaPro method for a total of 17,130.0 couple of months [sic] of use. The method and use effectiveness rates for avoiding pregnancy were 99.5 and 96.8 at the 12th ordinal month and 99.5 and 96.4 at the 18th ordinal month, respectively. The discontinuation rate was 11.3% at the 12th ordinal month and 12.1% at the 18th ordinal month. Obviously, in the populations studied, the method is highly effective as a means of avoiding pregnancy in both its method and use effectiveness. The method effectiveness has remained stable over the years of the studies, but the use effectiveness for avoiding pregnancy appears to have improved over the study period. Another form of NFP, the Billings Ovulation Method, is so simple to teach and use that it is taught around the world, even to people who cannot read or write.

NFP is said to promote love, romance, communication, prayer, spirituality, and learning about natural, God-created reproductive mechanisms. A very great advantage of NFP is that it is said to foster communication and understanding between the man and the woman, develop cooperation between them and a sharing of the responsibility in this important matter of their children. In all these ways it is said to improve a couple’s relationship and help them to grow in love and fidelity to each other.
These medical and sociological facts about NFP appear to nullify condition five of the principle of double effect. Since there is a viable, safe and effective alternative to the Pill, this fact would appear to dissolve most arguments that the Pill, until proved to be non-abortifacient, should be or can morally be used by Christians. In fact, assuming that NFP is only as effective as the Pill (and not more effective), it would appear that most arguments to use the Pill, in view of the fact that it may have an abortifacient effect, would be reduced to arguments of convenience at the potential expense of preborn human life.

**Future Research**

Without question, more medical research on this controversy is needed and would be instructive to physicians, ethicists and theologians. Others have begun to call publicly for such research to be done.\[1,2,10,13,22\] In particular, studies are needed that evaluate women who get pregnant while taking the Pill. Medically, two separate types of research need to be done with these women: one type would evaluate the development of the preborn child from the point of conception to the point of implantation; the second would evaluate gestation from the point of implantation onward.

**From the Point of Conception to Implantation**

Direct evidence of a postfertilization, preimplantation abortifacient effect would require methods to measure directly the rate of fertilization and the loss of the preborn child before implantation in women on the Pill. Transcervical tubal washings have been used in women on IUDs to quantify the rate of ova fertilization\[55\] and could theoretically be done in women on COCs. However, it is probable that most Christians would view such research as unethical.

Other than the washings, there is no currently accepted and proven method to measure the loss of the preborn child prior to implantation. However, a number of techniques and methods to quantitate preimplantation conception are being investigated. Promising research involves the measurement of maternal hormones that appear to be produced or altered after fertilization.\[56,57,58\] The most promising research involves the identification and measurement of a substance called the “early pregnancy factor.”\[59,60,61\] It is reasonable to predict that this research will assist in the answer of this question in the very near future.

**From the Point of Implantation**

Direct evidence of an abortifacient effect on the preborn child after implantation and prior to signs or symptoms of pregnancy would require measurement with ultrasensitive assays for \(β\)HCG (a hormone that can be measured in the blood or urine of the mother).\[2\] There is also the possibility of being able to measure other pregnancy-related hormones.\[62\] Studies using these ultra-sensitive assays have been done with normally fertile women not using birth control,\[63,64,65,66\] as well as with women using nonhormonal methods of birth control.\[67\]

Using these established methods to detect very early pregnancy, women on the Pill (the COC) could be studied and the loss of their preborn children (from implantation onward) could be demonstrated and compared to already published studies of the “natural” losses of normally fertile
women using no birth control. Studies such as these, in women on the Pill, would be expensive and would necessarily have to involve a large number of women. An additional obstacle is that it is unlikely that pharmaceutical companies would fund such research. Nevertheless, it would appear reasonable for the proponents and opponents to join together to carry out such research.

If this study showed that there is increased loss of the preborn in women on the Pill, as compared to women not using any birth control, then the case of the proponents is established. If this study showed that there is no measurable loss of the preborn in women on the Pill, then the case of the opponents is established. However, a third possibility exists: the proposed study could show that there is a significant loss of the preborn in women on the Pill, but that the loss is less than that seen in noncontracepting women. If so, then another ethical debate would be forthcoming and appropriate. Such a discussion is beyond the scope of this paper.

CONCLUSION

There is currently a significant controversy about whether the Pill causes early, unrecognized abortions of preborn children. It does appear theoretically possible (even probable) that research could be done to begin to settle the controversy and this research is critically needed. However, until such research is available, those who feel ethically comfortable with prescribing the Pill should inform their female patients of this possible effect and allow their patients to decide whether they should or should not use this form of birth control. Further, physicians or pharmacists who feel ethically constrained from prescribing or dispensing the Pill should be supported. Whether they should be encouraged or compelled to inform patients who still desire to use the Pill to a healthcare provider who can prescribe or dispense the Pill is beyond the scope of this discussion.

There appear to be viable, safe and effective forms of NFP. NFP is a natural method of contraception that never has an abortifacient effect. It appears that most physicians and patients are not aware of this option and that the vast majority of those who prescribe the Pill have never been educated about modern medical NFP. Efforts should be undertaken by national groups to educate Christian women and physicians about these options.

Finally, based upon the principle of double effect, it appears reasonable to conclude that the Pill should not be used or recommended to those who believe life begins at conception unless and until the Pill is proven not to be an abortifacient. It appears to be a reasonable conclusion that such studies could be done and that proof could and should be forthcoming; however, to date that proof clearly does not exist.

Until such proof is available, one way or the other, the Pill should be considered a possible cause of death to

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preborn children. It is reasonable to hypothesize that if the Pill was in development today and if the preborn child was considered truly human under the law, then it would be unlikely that the FDA would allow the Pill to be approved for public use until the manufacturers had studied and established whether or not (and, if so, how often) the Pill causes the death of preborn children.

Since, in the final analysis, the choice to prescribe or use the Pill may be legitimately considered a potential life and death decision for the preborn, it seems reasonable to let God’s Word be the final one: “This day I call heaven and earth as witnesses against you that I have set before you life and death, blessings and curses. Now choose life, so that you and your children may live” (Deut 30:19).
References


12. DeCook, J.L., Crockett, S.A., Harrison, D., Hersh, C., “Hormone Contraceptives: Controversies and Clarifications,” *Prolife Obstetricians* (PO Box 81, Fennville, MI, 49408)


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REDUX: IS THE ORAL CONTRACEPTIVE PILL AN ABORTIFACIENT?

JOEL E. GOODNOUGH, MD

Randy Alcorn has written a very thought provoking book which examines the evidence and concludes that the combined estrogen and progesterone oral contraceptive pill (OCP), or birth control pill as he prefers to call it, sometimes causes abortion of the early embryo. He believes that the OCP prevents a clinically recognizable pregnancy not only through prevention of conception, or fertilization, but also through prevention of successful implantation of the embryo into the wall of the uterus when conception does occur.

The central question that Alcorn asks is whether the OCP exclusively acts as a contraceptive or whether it sometimes prevents implantation and therefore causes abortion (pg. 12 of his book). He cites a number of mechanisms of action for the OCP in preventing pregnancy: inhibition of ovulation; thickened cervical mucous and decreased sperm transport; prevention of implantation of the embryo; and long-term effects that have an abortive effect even after discontinuation of OCP use. He then goes on to say that if the OCP sometimes prevents implantation and therefore causes abortions, it is more Christ-like to not use it (pg. 54 of his book). In other words, the OCP should not be used because it is an abortifacient. But are his conclusions appropriate? Does the OCP cause early loss of the embryo at all, infrequently, or frequently? And if it does cause abortions, does that make it an abortifacient? If we cannot decide if the OCP causes abortions, what should we do? To answer these questions, we must assess the pill's ability to prevent fertilization and then try to determine the consequences to the embryo when the pill fails to do so. Finally, we have to decide how to live in an imperfect world with risks.

Ovulation Rates on the OCP

Alcorn believes that the OCP may fail to prevent ovulation in 10-30% of cycles (pg. 22 of his book). The significance of ovulation, of course, is that it may lead to conception and open up the question of the possible abortive effect of the OCP. He bases his conclusion on studies that show a high level of ovarian activity in women taking the OCP. Ovarian activity is not synonymous with ovulation, however. Spona, et al. showed ovarian activity, as evidenced by the presence of follicles and unruptured corpus luteum cysts, in up to 35% of patients correctly taking the OCP. Ovarian activity is not synonymous with ovulation, however. Spona, et al. showed ovarian activity, as evidenced by the presence of follicles and unruptured corpus luteum cysts, in up to 35% of patients correctly taking the OCP. Some of these patients also had a rise in their serum progesterone levels, indicating corpus luteum function. Ultrasound studies, however, failed to show ovulation in any of these patients, a phenomenon called the luteinized unruptured follicle syndrome. In a similar study, Crosignani, et al.
showed ovarian activity in patients who correctly took low dose and very low dose OCP, but again there was no evidence of ovulation.\textsuperscript{6}

Alcorn also cites a statement by Stephen Killick to support the argument for ovulation on the OCP. In his paper, Killick opens his introduction with the statement: “It is well established that newer, lower-dose regimes of combined oral contraceptive (OC) therapy do not completely suppress pituitary and ovarian function.” The question of whether this ovarian function could result in ovulation, however, is the very basis for Killick’s study. Killick then goes on to show that he can \textit{artificially induce} ovulation in cycles where ovarian function occurred as a result of intentionally missed pills. This result, however, is in contrast to his clinical trials, which show no ovulation in over 150 cycles when the OCP was taken correctly.

Rossmanith, et al.,\textsuperscript{8} in a German study, showed ovarian activity based on progesterone levels in 2.9\% and 4.1\% of cycles of women who were on very low dose OCP. Combined analysis with ultrasound and hormone levels, however, showed no ovulations in any of the cycles. He concludes that “although the capability of these [follicles] to grow and ovulate has been demonstrated in previous studies [he cites Killick’s study], the likelihood that this occurs is very limited. This view is substantiated by the findings of the current and previous investigations, which clearly showed that there was no evidence for ovulation occurring.” Finally, there are at least three studies, which show that missing up to four pills in a row does not result in ovulation.\textsuperscript{9}

It is particularly distressing that Alcorn refers to studies in order to make a point even though one would be hard pressed to find actual support for the point within the context of the studies. Wilks\textsuperscript{10} cites a study by Letterie in which women began taking a combined OCP six to eight days late in the cycle. In addition, they took the combined OCP for only five days followed by a progesterone-only pill (POP) for another nine days. The purpose of the study was to determine if other formulations of pills would be effective at preventing ovulation. The study showed a 30\% ovulatory rate, primarily in the second cycle studied. Now, why would Wilks even mention this study in his discussion on the effect of missed pills on ovulation? Is he suggesting that women who start their pills a week late, take them for only five days, and then switch to a progesterone-only (POP) pill for nine days are at a similar risk of ovulating as women who take the combined OCP and that, therefore, the OCP should not be used because conception and embryo loss is possible? That would be like saying that safe, routine driving has the same potential of causing a fatal accident as driving the wrong way on a one-way street; therefore, we should not drive at all. If his point is that studies, to be truly valid, have to look at the second cycle in the study, then Spona addresses that in his previously referenced study when he looked at three cycles. In either case, Letterie’s study would only be representative of a woman totally misusing the OCP. It should be noted that Letterie showed in 1992 that missing four OCPs in a row in various times in the cycle resulted in no ovulations.\textsuperscript{11}

The point is not that ovulation never occurs, for we know that pregnancy occurs on the OCP. In carefully monitored studies with highly motivated women, the rate of pregnancy on the OCP is .1\% per year whereas the observed rate in the general population is 3\% per year.\textsuperscript{12} This
difference is due to breakthrough ovulation when errors are made in taking the OCP or to decreased absorption of the OCP secondary to medications or illness. The point is that it is wrong to assume, based on evidence of ovarian activity, that there is a high frequency of ovulation, especially when the OCP is taken responsibly and correctly.

Prevention of Implantation

Alcorn believes that if ovulation and conception occur on the OCP, the embryo is at risk of being aborted due to changes in the endometrium (the lining of the uterine wall) that are hostile to implantation of the embryo. He does a wonderful job of reviewing the literature that shows that the OCP produces changes in the endometrium (pgs. 14-18 of his book). It is speculative, however, to say that these changes result in inhibition of implantation of the embryo and therefore result in abortion. Alcorn refers to this as the third mechanism of action of the OCP. The literature that he quotes describes the endometrium in women on the OCP as being hostile to the embryo, but no literature actually shows that death of the embryo results. The Physician’s Desk Reference, for instance, states, “Although the primary mechanism of this action (contraception) is inhibition of ovulation, other alterations include changes in the cervical mucus (which increase the difficulty of sperm entry into the uterus) and the endometrium (which reduce the likelihood of implantation).” No references are cited to support the speculation that implantation is actually inhibited, however. Alcorn states that because women sometimes get pregnant on the OCP, this means that the third mechanism sometimes fails. The embryo sometimes implants and survives despite the changes in the endometrium. But in light of the fact that there is no definitive information on whether the embryo implants or not, he could just as easily assume that the embryo always implants and survives despite seemingly hostile changes in the endometrium. Or, more accurately stated, he could say that the embryo implants and survives as frequently in those on the OCP as in those not on the OCP, since embryo loss occurs in an estimated 70% of fertilizations in women not taking the OCP. Fifteen percent of these embryos die immediately after fertilization, 15% fail to implant, and 40% are lost after implantation.

Animal studies in rodents have shown the failure of embryos to implant in an endometrium created to be similar to that which is found on the OCP. The author of the study, however, cautions that “mechanisms of implantation vary markedly between species, making extrapolation [to humans] difficult.” He goes on to point out that the human embryo, unlike other species, has a unique invasiveness and ability to implant in extrauterine locations such as the fallopian tube.

Alcorn’s assertion that the OCP causes abortion is based on the observation that the OCP causes this seemingly hostile endometrium. The thinner endometrium that is seen on ultrasound may be a direct result of the progestin in the OCP, but it could also be a result of suppressed ovarian hormones. Although it is true that the endometrium has a hostile appearance when the OCP succeeds in preventing ovulation, it does not follow that the endometrium is hostile when the OCP fails and ovulation occurs. Ovulation and endometrial condition are not independent factors. Ovulation occurs as a result of rising follicle stimulating hormone (FSH), luteinizing hormone (LH), and estrogen levels. After ovulation, progesterone levels...
rise. These rising hormone levels are responsible for making the endometrium receptive to the embryo. One would therefore expect the endometrium in an ovulatory cycle on the OCP to be more receptive than the endometrium in an anovulatory cycle on the OCP where there is no rise in hormone levels. Stephen Killick, for example, was able to demonstrate normal ovulatory endometrium in cycles where pills were intentionally missed and ovulation artificially produced. Finally, it has been shown that the embryo is capable of sending signals prior to implantation that create a more favorable endometrium. This effect would not be seen in an anovulatory cycle where no embryo exists.

In the absence of large numbers of studies addressing the status of the endometrium in an ovulatory cycle on the OCP, the expectation that the endometrium is more receptive in such cycles is somewhat speculative, although in my opinion reasonable. It is incorrect, however, to assume that the embryo sometimes implants and survives despite the third mechanism of creating a hostile endometrium since that mechanism may not even be a factor in ovulatory cycles. Alcorn states: “When you say the effect of preventing implantation is absent in some cases, you are implying it is present in some cases” (pg. 37 of his book). That is simply not true, for it may be that the third mechanism is absent in all cases of fertilization. The effect of a hostile endometrium may be absent in cases of ovulation when it matters and present in cases of anovulation, when it does not matter.

Alcorn compares the situation of the hostile endometrium and the embryo with that of dry, thin soil and a seed. “Surely no one believes its chances of survival are as great on a thin rocky path as in cultivated fertilized soil” (pg. 44 of his book). My thought, having read that statement, was “surely no one believes that the human embryo is in any way analogous to a seed or that the human endometrial lining is in any way analogous to dirt.” If one insisted on using the example of the seed and the hostile soil, it may be more analogous to say that in times of drought, the soil is dry and barren. Because of the drought, there is also no seed production. When conditions are more favorable and there is seed production, the soil also responds and becomes more favorable. In addition, the seed sends signals to the soil to make it even more fertile. It is interesting to note that, at one time, the pioneers who came through Illinois thought that the treeless prairie must have infertile soil. After all, they speculated, the lack of trees must mean that the soil is hostile to seed germination. Things are not always as they seem.

Integrins are molecules believed to be markers of endometrial receptivity. Alcorn cites a study that shows changes in integrin expression in the endometrium of OCP users that are unfavorable to implantation. No attempt was made, however, to ascertain whether these patients were anovulatory or ovulatory. The critical question, again, is whether these unfavorable changes in integrin expression occur in ovulatory cycles on the OCP. That same study cites another study in which integrin levels were measured in cycles where the postcoital contraceptive (the morning-after pill) approach was used. That is, high dose OCP was used for a short time after coitus. In these cycles where ovulation was documented, the integrin expression was no different than that seen in non-OCP users. The author cautions that this could be due to sampling the endometrium too late in the cycle. It is tempting to argue that the cycle in which postcoital OCP is used after ovulation is an approximate representation of what happens when a woman misses pills and ovulates. Would it then be reasonable to speculate that integrin expression
in the endometrium of ovulatory women on the OCP may be normal? Probably not, since the “morning-after pill” is so different from the OCP in timing and dose.

Alcorn, however, does attempt to equate the so-called morning-after pill with the OCP (pg. 35 of his book). “It is significant that this ‘morning-after pill’ is in fact nothing but a combination of several standard birth control pills taken in high dosages. When the announcement was made, the uninformed public probably assumed that the high dosage makes birth control pills do something they were otherwise incapable of doing. But the truth is, it simply increases the chances of doing what it already does—cause an abortion” (emphasis his). But how can he reach that conclusion? The “morning-after pill” is four pills in one day, nothing at all like the OCP where one pill is taken every day to prevent ovulation. In addition, those four pills are taken at any time in the cycle that they might be needed. The mechanism of action of the “morning after pill” is not at all clear. It may prevent ovulation in some cases and prevent implantation in others, depending on when in the menstrual cycle it is taken. This is far different from the OCP in both mechanism of action and intent. Any medication, if taken in a way other than for which it was designed, can have a different adverse effect. That certainly does not mean that the adverse effect is present when the medication is taken correctly.

Alcorn proposes yet another means of preventing implantation (pg. 33 of his book) when he quotes from My Body, My Health: “Estrogen and progestin may also alter the pattern of muscle contractions in the tubes and uterus. This may interfere with implantation by speeding up the fertilized egg’s travel time so that it reaches the uterus before it is mature enough to implant.” He then goes on to say that “this is the same contraceptive effect Dr. Speroff referred to as ‘peristalsis within the fallopian tube.’” Speroff, however, was referring to animal studies rather than human studies. In fact, Speroff states: “Moreover, when fertilized donor eggs are transferred to women who are on hormone supplementation, there are a number of days during the treatment cycle when the blastocysts will implant. This crucial difference between animal and human physiology is of more than academic importance. There has been speculation concerning the use of drugs that could accelerate tubal transport as a means of providing contraceptive by ensuring that the egg would reach the uterus when it was in an unreceptive state. Although this may work in animals, it is of doubtful value in the human because perfect synchrony is not required.”

The Incidence of Ectopic Pregnancy

Alcorn, quoting Dr. Walter Larimore, suggests that the use of OCP does not lower the incidence of ectopic pregnancy to the same degree that it prevents intrauterine pregnancy (pg. 31 of his book). This would imply that the endometrium on the OCP is more hostile than the tubal lining in an ovulatory cycle on the OCP. Larimore believes, in other words, that the OCP is better at preventing implantation in the right place than it is at preventing implantation in the wrong place. He refers to a study by Mol, et al. to back up his belief. In a meta-analysis, Mol showed that the OCP protects against both ectopic and intrauterine pregnancies. But when pregnancy does occur, there is a slightly increased risk of ectopic pregnancy on the OCP. Mol, however, offers an explanation for this
observed increased risk that is different from the explanation offered by Larimore. He points out that the meta-analysis includes the progesterone-only pill, which is known to have higher rates of ovulation and which is also known to slow tubal transport, thereby increasing the risk of implantation in the tube. This is an effect of progesterone, as opposed to estrogen, which speeds up tubal transport.

In addition, Weiss et al. suggest that the apparent increased risk of ectopic pregnancy may be explained by the definition of the control group. The control group consisted of pregnant women seeking abortion. Those women who had a tubal pregnancy diagnosed prior to seeking abortion would not be included in the control group since they did not present to the abortion clinic to have an abortion. The pregnant control group in the study would therefore have an under-representation of tubal pregnancies.

Larimore refers to another study to support his belief that the OCP prevents the embryo from implanting in the right place, leading to a relative increased risk of ectopic pregnancy. The author of the study, however, does not come to the same conclusion: “Use of combined oral contraceptives at time of conception was not associated with a significant increase in the risk of ectopic pregnancy. However, four cases and two pregnant controls used the minipill (POP), and this excess among cases is consistent with previous reports suggesting a higher proportion of extraterine pregnancies in minipill failures.” Larimore again lumps the progesterone-only minipill in with the combined estrogen and progesterone OCP. This is in contrast to the author’s observation of no increased risk of ectopic pregnancy with the combined estrogen and progesterone OCP. In fact, the author goes on to say: “Our interpretation of the present results is that methods such as the combined pill provide the maximum protective effect against ectopic pregnancy by preventing ovulation. . . .”

Another source for the Alcorn/Larimore proposal is a letter to the editor in The Journal of the American Medical Association (JAMA) in which a study was described showing an increased rate of ectopic pregnancy relative to the rate of [43]

intrauterine pregnancy on the OCP. Although studies had shown increased risk on the progesterone-only minipill (POP), this study showed an increased risk on the estrogen and progesterone combined OCP. The author of the study concluded that the new lower dose OCP may allow ovulation and prevent implantation in the uterus, but not in the tube. The author who replied to the letter agreed, but also said that the observed increase risk of ectopic pregnancy could be due to some other OCP associated risk of ectopic pregnancy, such as pelvic infection.

**Long-Term Effects**

Alcorn quotes from Linacre Quarterly when he suggests that the OCP may have a prolonged effect on the endometrium or cause chromosomal abnormalities in the embryo after a woman has stopped taking the OCP, both of which would cause increased risk of miscarriage. He then suggests that this is the reason that it is recommended that pregnancy be avoided for three months after stopping the OCP. Numerous studies, however, have shown no increase in the risk of miscarriage in women who
conceive immediately after stopping the OCP. In those miscarriages that do occur, studies have shown no increase in the incidence of chromosomal abnormalities in the aborted tissue. Women are advised to avoid pregnancy for at least one spontaneous cycle for one simple reason: The OCP can delay resumption of spontaneous ovulation after stopping the OCP, making it difficult to determine the due date for the pregnancy. It is more accurate to determine the due date based on a spontaneous last menstrual period rather than relying on a pill-induced period.\textsuperscript{24}

The OCP: Contraceptive or Abortifacient?

Based on the ovulation studies it is clear that the OCP is capable of living up to its name of being an effective contraceptive. When used correctly, it effectively prevents ovulation and therefore conception. It is also clear, however, that it sometimes fails to do so. Although there is no direct evidence that this results in loss of the embryo, one cannot prove that it never happens. Does this mean that the OCP is an abortifacient, as Alcorn contends?

An abortifacient, according to \textit{Taber's Cyclopedic Medical Dictionary}, is anything used to cause or induce an abortion. Is the OCP an abortifacient? Or is it a contraceptive that has the potential for failure, a failure that may result in the death of the embryo? A gun becomes a murder weapon when used intentionally to kill someone. A car becomes an instrument of homicide when driven by a drunk. When used correctly and for the purpose for which they were designed, the gun and the car are simply agents of sport and transportation, respectively. It would be absurd to say that because the gun and the car sometimes kill people, they are in essence agents of homicide. In the same way, the OCP is not an abortifacient simply because it may have the potential to abort.

In saying that the OCP is an abortifacient, Alcorn confuses function and essence. By design, by intent, and by primary function, the OCP, when properly

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used, is in essence a contraceptive. The fact that it may fail to act as it was designed does not change its essence. We see this same confusion of function and essence in pro-abortion arguments when it is argued that the unborn is not fully human because she does not function as a human being. The pro-life response is that we are not human because we function as humans. Rather, we function as humans because we are human. The unborn is a human person who is not yet fully functional. Similarly, the OCP is a contraceptive even though it may not always be a fully functional contraceptive.

Alcorn states that “to be an abortifacient does not require that something always causes an abortion, only that it sometimes does” (pg. 23 of his book). The first part of his statement is true, for the success of an agent does not define its essence. That is to say, a hunter who uses his sport to kill people is a murderer if even just one of ten attempts at murder succeeds. On the other hand, if a hunter accidentally kills a fellow hunter, he is not a murderer. In the same way, a medication that is used to prevent conception is not an abortifacient even if it sometimes causes abortion. A medication that is designed and intended to cause abortion, however, is an abortifacient.
What to Do?

What are we to do with the information at hand? Since the OCP may pose an unknown degree of risk to the embryo, should we use it as a form of birth control? Randy Alcorn believes that it is more Christ-like to not use the OCP.

There are other analogous situations to which we can turn for guidance. With every medication, with every treatment, and with every surgery, there is an inherent risk of causing harm. Physicians decide to treat or not to treat based on risks versus benefits. If the treatment causes some deaths but the number of deaths is acceptable compared to the benefits of being free of the disease or condition, then we say that the benefits outweigh the risks. We know that many treatments can result in the death of the patient, but we prescribe them nevertheless because statistically patients benefit from the treatment and the risk is justified. The harmful effect of the treatment is tolerated because the benefits of the treatment are proportionately great. We may not know if a particular patient will be harmed or helped from a treatment, but we know that if we prescribe that treatment to enough patients, more patients will be helped than hurt.

When I was an intern, I found myself one day in an elevator alone with the chairman of my department. I was troubled by my timidity in treating patients and expressed my fear of hurting someone. My chairman, Dr. Fred Zuspan, told me something that I lean on to this day. Looking at me, he said: “Joel, that’s fine, but now you have to start worrying about not helping someone.” That is what medicine is about. If we were to let fear of hurting an individual patient paralyze us into inaction, no one would be helped.

In prescribing the oral contraceptive, some women (viz., one in 100,000) and perhaps some embryos will die. It is a matter of degree of risk. If the risk of death is low, the benefits of the OCP justify use. Since the risk of death on the OCP is less than the risk of death in pregnancy, the risk is tolerable. Alcorn does not object to the use of the OCP based on the risk of death to the woman taking the pill. Yet, her risk of death is known and acceptable whereas the risk of death to the embryo is unknown and to Mr. Alcorn unacceptable. The death of the woman, however, is just as much my responsibility as the death of an embryo. The fact that she consents and the embryo does not in no way lessens my responsibility.

Be aware, however, that responsibility, as I am using the word, does not imply desire or intent. Although I know that deaths do occur as a result of my prescription, I do not want that particular patient to die. When I prescribe the OCP, I do not want an embryo to die. The death of the embryo, should it occur, is the undesired result of intending to prevent fertilization. If the risk of death is acceptably low and the benefit of contraception is felt to be high, then prescribing the pill is an acceptable practice of medicine.

Some would argue that the concept of risk and benefit does not apply since the OCP does not directly benefit the embryo while at the same time posing some risk. This is one of the
interesting aspects of reproductive medicine, however. Everything that a pregnant woman ingests and every activity that she undertakes poses a potential risk to her baby. Yet, most of her daily activity and some of what she ingests does not directly benefit her unborn baby. She drinks a cup of coffee, takes a Tylenol, and drives to work, all for her benefit. She does this believing that the benefits to her outweigh the risks to her baby. Similarly, physicians prescribe medications for the pregnant woman for her benefit, believing that the benefits outweigh the risks to the baby.

The issue of hormonal therapy and breast cancer is a good illustration of the decision-making process that physicians must go through in deciding how to best treat a patient. We know that breast cancer is linked in some way to estrogen. Breast cancer is much more common in women than in men, for instance, and breast cancer is many times estrogen receptor positive, meaning it will be stimulated by estrogen. Yet, the preponderance of evidence suggests that prescribing estrogen to menopausal woman does not substantially increase the risk of breast cancer. The studies on the subject are somewhat mixed in results, but for the most part are reassuring. On the other hand, the benefits of taking estrogen after menopause are well documented and consistent. What should we do? After all, we cannot prove that estrogen therapy never causes breast cancer, and future studies could show that estrogen clearly causes breast cancer. If we wanted to be completely safe, I suppose we should not prescribe estrogens at all. That approach, however, would deny to many women the benefits provided by taking estrogen.

The answer is that we try to practice evidence-based medicine. Evidence-based medicine is best described by the following quote from Danforth’s Obstetrics and Gynecology:

"Excellent medical practice should be inspired by love and guided by science. Both are essential. If a clinician practices scientific medicine without compassion, he or she becomes an automaton. On the other hand, if a clinician is compassionate but unscientific, he or she may be as dangerous as a well-intended parent feeding chicken soup to a child with meningitis. Evidence-based medicine is not only well-intended, it is well directed."

— David A. Grimes

So we look at the studies and act on what we know to be true given the present state of knowledge. We do not act on speculation or unfounded fears. We inform the patient and then give our best recommendation, letting her make the final decision. We prescribe estrogen, not yielding to unproven fears of cancer, while at the same time keeping our eyes on future studies.

Perhaps a more commonly encountered dilemma is one faced by all of us when driving a car. We know that a certain number of children are struck and killed by cars each day. On any particular day, we could be driving a car that kills a child. Yet, we still get in our car each day to go to work or play. We do this knowing that the risk is acceptable and the benefits of being able to drive outweigh the risks to children. Alcorn feels that, unlike using the OCP, driving a car is an acceptable risk because he feels that there are no alternatives. He states that “there is no such thing as a car or a house that poses no risk to your children. But there is such a thing as a contraceptive method, which does not put a child’s life at risk. There are safe alternatives to the Pill that do not and cannot cause
abortions” (pg. 60 in his book). Alcorn asks: “Is it a Christ-like attitude to say ‘Because taking the Pill may or may not kill a child, I will therefore take the Pill?” (pg. 55 in his book).

But just as we choose our mode of transportation based on the risks versus convenience, can we not choose our contraception based on risks versus convenience? Just as we could choose not to use the OCP because of a perceived unacceptable but unknown risk to children, could we choose not to drive a car based on a known risk to children? If our driving sometimes causes the death of innocent human beings, the Christ-like thing to do, perhaps, would be to not drive at all. Yet Mr. Alcorn does not say this. Apparently walking, which is much less convenient and efficient, is not for Mr. Alcorn an acceptable alternative to driving just as condoms, which are much less convenient and efficient, are, to some not an acceptable alternative to the OCP. Yet, are we not responsible for the death of a child we have killed with our car in the same way that we are responsible for the death of the embryo we have killed with the OCP? We did not want the death, but we chose a course of action with the foreknowledge that it could happen and knowing that we had a safer alternative course of action.

Another well-known risk is hunting. We know that a certain number of hunting accidents occur each year resulting in a number of deaths. Alcorn asks the rhetorical question: “If a hunter is uncertain whether a movement in the brush is caused by a deer or a person, should this uncertainty lead him to shoot or not to shoot?” (pg. 55 in his book). The answer, I assume, that Alcorn would give is to not shoot. But to be consistent with his conclusion that we should not take the OCP, he would have to conclude that, to be totally safe, we should not hunt at all. There are safer alternatives to hunting, such as photography, and if hunting sometimes causes the death of innocent human beings, the Christ-like thing to do, perhaps, is not to hunt at all.

The Principle of Double Effect

Walt Larimore feels that the OCP does not satisfy the conditions necessary for successful application of the Principle of the Double Effect. This principle allows the performance of an act that has good and bad effects under certain conditions:

1. The act itself must be morally good or at least neutral.
2. The person performing the act must intend the act to be morally good.
3. The good effect must not follow a bad effect.
4. The good effect that is intended must have sufficient moral value to justify tolerating the bad effect.
5. There must be no other way of producing the good effect.
Larimore concedes that the first and second conditions are met. He feels that the third and fourth conditions are debatable and the fifth is not met. That is to say, sometimes the birth control effect is accomplished through the pill’s abortifacient effect (3), convenience of birth control is not of sufficient value to justify the deaths of embryos (4), and there are effective, safer alternatives to the pill (5).

The problem is in his definition of the good effect. He refers to the pill as the birth control pill (BCP) because he feels that it controls birth, not conception. He naturally concludes, therefore, that the desired effect is birth control. The effect of birth control may in part be from abortion and there are other forms of birth control that do not cause abortion.

If, however, one feels that the pill is a contraceptive through its prevention of ovulation, the pill is referred to as the oral contraceptive pill (OCP). The desired effect is not simply birth control but rather prevention of conception by prevention of ovulation. As a matter of fact, physicians frequently prescribe the OCP to prevent ovulation for medical reasons that have nothing to do with birth control. If the desired effect is prevention of conception by prevention of ovulation, it is not accomplished by a bad effect and there are no alternatives that are safer. The conditions necessary for successful application of the Rule of Double Effect are met.

**Summary**

I have examined Randy Alcorn’s work on the mechanism of action of the OCP. Although I found some of his statements to be in error or misleading, he does raise a very important question about the OCP. Does it cause abortions? The only thing clear to me is that the answer to this question is unclear.

Alcorn’s contention that it does cause abortions is speculative, being based primarily on the observation that the OCP creates an endometrium that appears to be hostile to implantation when it functions as it was designed to do—prevent ovulation. What is not clear is what happens to the endometrium when the OCP fails to do what it was designed to do and ovulation occurs. I have cited some studies that suggest that the endometrium is more normal when ovulation does occur on the OCP, but this does not prove that implantation is as successful as in those not on the OCP. And just how good is the embryo at implanting into a hostile environment if it does exist anyway?

I have also found some real problems with Mr. Alcorn’s interpretation of the studies on ovulation rates and tubal pregnancy rates with the combination estrogen and progesterone OCP. At times, he seems to come to a conclusion that is different from that of the authors of the studies. It is not possible, based on these studies, to conclude that the OCP causes abortions. In fact, based on more recent studies, it appears that the OCP, when taken correctly, approaches 100% effectiveness in preventing ovulation.

I certainly do not want to present myself as being an expert in the area of contraceptive technology. The paucity of good studies that specifically address the question of the OCP’s potential to cause abortions makes it impossible to be an expert. It is clear to me, however, that it is not
possible to say that the OCP causes abortions. I am comforted to some degree by the fact that, as pointed out by Alcorn (pg. 67 in his book), the majority of Focus on the Family’s Physician’s Resource Council also concluded in 1997 that there is no direct evidence that the pill causes abortions.

As a physician, I have seen countless women who have benefited from the OCP. The benefits go beyond adequate birth control to a better quality of life. They have more manageable periods, less cramps, improved acne, less premenstrual symptoms, less chance of tubal infection, less chance of ectopic pregnancy, less benign breast disease, less rheumatoid arthritis, better bone density, and a decreased risk of ovarian and uterine cancer. The studies on cervical and breast cancer are mixed, but overall the results are reassuring. The safety of the OCP, one of the most studied drugs ever, is well established. Just as I am not willing to deny estrogen to menopausal women based on unfounded fears of breast cancer, I am also not willing to withhold the benefits of the OCP based on unfounded fears of causing abortion.

Alcorn would say that this is not good enough. “Show me the evidence, direct or indirect, that the Pill never causes abortions” (pg. 73 in his book). This, of course, would be impossible. If he were to apply this same standard to other risks, he would never drive his car or give his children a headache medication. He would not use a cellular phone either, since there is no evidence that says cellular phones never cause brain tumors.

If we cannot decide if the OCP causes abortions, perhaps we can determine what we are dealing with. What is the OCP? Is the OCP an abortifacient, as Alcorn asserts? Or is it a contraceptive that has the potential for failure, a failure that may result in the death of the embryo? I think it is clear that, when used for the purpose for which it is designed, the OCP is a contraceptive both in design and intent.

One solution to our dilemma would be to simply not use the OCP, as Alcorn suggests. I would not criticize someone for taking that approach. In the face of a lack of credible evidence that the OCP does cause abortions, however, I would wonder about someone’s consistency if they advocated not using the OCP while at the same time driving to work, hunting, taking estrogen after menopause, or engaging in any other activity with potential risks, whether real or perceived.

Another approach would be to use the OCP but use it responsibly. In carefully monitored studies with highly motivated women, the rate of pregnancy on the OCP is .1% per year whereas the observed rate in the general population is 3% per year. This difference is due to breakthrough ovulation when errors are made in taking the OCP or to decreased absorption of the OCP secondary to medications or illness. If the OCP has the capacity to cause abortions when it fails to prevent ovulation, it makes sense to increase its capacity to do what it is designed to do—prevent ovulation. This means acting responsibly when using the OCP. This means remembering to take the pill as it was designed and using back-up contraception when an error is made or when taking a medication that might interfere with the OCP. This means using back-up contraception when one has an illness with nausea and vomiting. This is no different than handling a car or gun responsibly.
Do the benefits of taking the OCP outweigh the risks? The answer is yes if one considers only the woman taking the pill. But what about the embryo? We simply cannot quantify the actual risk to the embryo with our present state of knowledge, but we know that we can lessen that risk by taking the OCP responsibly. In my judgment, responsible use of the OCP results in a risk to the embryo that is tolerable.

Finally, Alcorn made a good point about informed consent. Perhaps we should tell our patients about this controversy. But what exactly do we tell them? Is there a high risk or low risk of causing abortion? I tell them that there is an unknown risk, but that the risk can be reduced to a tolerable level through responsible pill taking. I have advised some of my patients to take the OCP continuously rather than cyclically. By avoiding the pill-free week, one would theoretically lower the risk of accidental ovulation due to missing those pills most commonly forgotten, the first few pills of the pack. There are no health risks associated with taking the OCP in this manner. Spona advocates a similar approach in suggesting a shorter pill-free interval to minimize side effects and increase effectiveness of the OCP.

References


2. Abortion is the termination of pregnancy by any means prior to viability of the fetus.

3. I follow the position of the American College of Ob-Gyn (ACOG) in defining the beginning of pregnancy as the time of implantation. Pregnancy is a maternal condition unrelated to the condition of the embryo. One can have a pregnancy that is abnormal with no embryo (molar pregnancy), for instance. On the other hand, one can have an embryo in the laboratory in which case no one is pregnant. This in no way means that the embryo prior to implantation is less significant in any morally relevant way. Contrary to ACOG, however, I define conception as meaning fertilization rather than implantation. In other words, I consider conception to refer to the beginning of the embryo’s existence (usually by fertilization), not the beginning of a pregnancy. Defining the terms pregnancy, conception, and abortion is not just an academic exercise. Since ACOG defines conception and pregnancy as beginning at implantation of the embryo, the embryo cannot be aborted prior to implantation since the pregnancy has not yet begun. Therefore, destruction of the embryo prior to implantation is not an abortion. Similarly, a contraceptive is anything that prevents fertilization and implantation since conception is at implantation. This confusion of terms can be used to mask moral issues. For this paper, to avoid confusion over moral issues, I will continue to use the term abortion to describe the loss of the embryo both prior to and after implantation.


5. The follicle, from which the egg is released, develops in response to rising follicle stimulating hormone (FSH). After ovulation, the follicle develops into the corpus luteum, which produces progesterone.


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25 I use the concept of want and intent interchangeably. Beauchamp and Childress, in Principles of Bioethics, make the point that intentionality is based on what is willed rather than what is wanted. One can will or intend that something should happen while not wanting it to happen. The result is simply tolerated in order to achieve another desired effect. This is in contrast to the “Rule of Double Effect,” which states that the undesired effect is not intended. Foreknowledge does not imply intent. I find it easier, though sometimes problematic, to equate intent with want.


28 Ibid.


Joel Goodnough, MD is a gynecologist who practices in Arlington Heights, Illinois, USA.
RESPONSE TO JOEL GOODNOUGH MD, “REdux: IS THE ORAL CONTRACEPTIVE PILL AN ABORTIFACIENT?”

Editor’s Note: The controversy surrounding the question of the oral contraceptive pill’s abortifacient properties continues. Ethics & Medicine is delighted to have a role in hosting this on-going dialogue.

The two articles and response below would normally be included in a “letters to the editor” section; however, since E&M does not have a regular letters section (though, if sufficient readers wrote in, we would gladly add one) and since these responses are so lengthy, we include them below in this special section.

JOHN WILKS, B. PHARM MPS

In the spring issue of Ethics & Medicine, Dr. Joel Goodnough provided readers with an in-depth critique of Randy Alcorn’s text Does the Birth Control Pill Cause Abortions? He canvassed many issues, including putative breakthrough ovulation rates whilst on the pill, the pill’s claimed impact on implantation, its influence on the frequency of ectopic pregnancy and an extended discourse centred on Alcorn’s assertion that the pill has an abortifacient capacity. This last point flowed into a discussion of the merits of reclassification of the pill. Dr. Goodnough concluded that Alcorn was wrong in much of his data analysis, and therefore wrong in deducing that the birth control pill can reasonably be said to act against implantation. As a consequence, Dr. Goodnough concluded that Alcorn was in error for suggesting the pill had an abortifacient capacity.

In presenting his counterarguments, Dr. Goodnough took strong exception to the interpretation placed upon a paper by Letterie, referenced by Alcorn in his text. My involvement in this debate has occurred because I had also cited the same work in my Ethics & Medicine paper in January 2000 and am also heavily criticised by Dr. Goodnough for apparently committing the same misdemeanour as Alcorn.

I consider that Dr. Goodnough has misunderstood the purpose and point of the Letterie paper I cited and consequently has misrepresented, albeit unwittingly, the argument I—and therefore Alcorn—was advancing. Further, I take the view that Dr. Goodnough has used dated references to support his position, relying on papers which do not properly reflect our current knowledge of the process of implantation. As well, I am concerned that Dr. Goodnough has chosen only one paper I referenced on the topic of breakthrough ovulation, yet in my original work I referenced three. These errors and omissions require correction.

To begin, Dr. Goodnough finds it “particularly distressing” that Alcorn (and I) should draw conclusions from studies “even though one would be hard pressed to find actual support for the point within the context of the study” (p.38). Dr. Goodnough signatures his concern by criticising
my (and Alcorn’s) use of a paper from Letterie. This study reported a breakthrough ovulation rate of 30% when a new formulation of the pill was tested. Dr. Goodnough asks: “Now, why would Wilks even mention this study in his discussion on the effect of missed pills on ovulation?” (p.38). He suggested that this formulation of the pill is a nonsense formulation, disconnected from normal pill use, and hence tangential to the core of the debate over breakthrough (or escape) ovulation. And his reason? Because this formulation requires a woman to start the tablets a week into her cycle, take a combined oestrogen/progesterone tablet for the first part of the cycle, then switch to a progesterone-only pill during the latter part of her cycle.

This conclusion by Dr. Goodnough contains two substantial misunderstandings. First, Dr. Goodnough does not inform the reader that this hormonal approach to birth control, rather than being an extreme example of atypical pill use, was actually a clinical trial by a researcher who was testing a “novel” approach to birth control, intended to determine if a restricted regimen may offer both an effective method of contraception and a means of further reducing both estrogen and progestin content per cycle and the possible short and long term adverse side effects of these hormones.\(^4\)

I had placed my use of this paper within the overall topic of the effect of missed pill tablets, the consequence this omission might have on breakthrough ovulation and the subsequent possibility of conception. Hence my use, and Alcorn’s, was contextually valid. I was reporting on a style of the pill that is a forthcoming product currently under evaluation, and certainly not, as Dr. Goodnough suggested, only ‘representative of a woman totally misusing the OCP (oral contraceptive pill).’\(^5\)

But Dr. Goodnough might reply that this new formulation is not currently used, and on this point he would be right. So, does my point, and hence Alcorn’s, now deflate? No, because in my Ethics & Medicine paper I also cited works by Van der Vange (1986) and Grimes (1994), the former briefly and the latter extensively. Both researchers demonstrated that in motivated, healthy women taking contemporary formulations and faithfully adhering to the normal procedure of daily ingestion, breakthrough ovulation occurs. My citation of these two works, but Dr. Goodnough’s omission of the same, constitute my second concern with his criticism of my and Alcorn’s publications.

As the work by Van der Vange and Grimes has demonstrated, breakthrough ovulation is not necessarily associated with “errors” made by the woman, or attributable to reduced absorption due to illness or concurrent medication use. His suggestion that particular events such as these are the usual explanation for escape ovulation does not accord with the research (p.39). Yet surely the occurrence of breakthrough ovulation during proper pill use is the key to this bioeth-

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ical debate. If the pill is not an anovulant, as research has shown, then fertilization can occur, resulting in the creation of a new human life. Informed academic debate requires that all the facts are made known.

Since the publication of my paper in this journal, I have become aware of another paper, published in September 1980, which supports my and Alcorn’s position. This Indian study
compared the incidence of breakthrough ovulation in a group of previously sterilised but normally ovulating women—who were instructed to intentionally miss two pill tablets during the first and fourth cycles of the study—with non-sterilised women who did not miss any tablets. Their respective progesterone levels, as a marker of ovulation, as well as endometrial development and cervical mucus structure were observed and compared. In this study, their daily pill formulation comprised 1 mg of norethisterone (the artificial progesterone) and 30mcg of ethynyl estradiol (the artificial estrogen).

Three fundamental points emerged from this study. First, and most damaging to Dr. Goodnough’s hypothesis, was the possible “occurrence of ovulation” in one of the 10 women in the control group, as indicated by the woman’s progesterone levels. To recall, the control group was instructed not to miss any tablets. Endometrial sampling in the control group showed that in 8 of 10 women there was a hormonal effect evident, and for the remaining two women the endometrium was so poorly developed that a biopsy could not be taken (p.244). One would presume that if there were insufficient endometrium to provide a biopsy sample, there would also be insufficient endometrium to accommodate implantation.

Second, since this 1980 Indian study, the dosage of the progesterone-like component of the pill has reduced markedly, and in Australia at least, no formulation now contains 1 mg of norethisterone for the full 21 days, though this amount was given for 21 days in the Indian study.

The implication to be drawn from this dosage reduction is simple: as a result of the reduction of the “progesterone” component in contemporary formulations, breakthrough or “escape” ovulation is more likely than not to increase because the pharmaceutical hold over ovulation has been loosened. This is a biologically plausible conclusion to make.

Third, this study gave a valuable insight into the effects of the real-life event of missing the pill occasionally during a cycle. When women missed only two tablets during their cycle, escape ovulation was indicated in 14% of women during the first cycle and in 36% of the same women during the fourth cycle. The omission of two tablets, either intentionally (as in this study) or accidentally (as in real life), indicates the precarious hold that the pill has on ovulation. In real life, busy schedules, illness, or a complexity of other human factors may negate regular pill use, leading to breakthrough ovulation and the possibility of conception.

So what fate awaits the human embryo conceived as a result of breakthrough ovulation? Is implantation hindered, or does it proceed uneventfully? This is a vital question because if the former occurs, then the pill is no longer a contraceptive (contra = against), but, rather, is an abortifacient.

Alcorn had recommended this change in description, noting that the hormones in the pill lower the level of a crucial molecule known as an integrin, which normally binds the embryo to the endometrium so as to facilitate implantation. Dr. Goodnough does not agree that a diminished integrin level is necessarily proof positive of an abortifacient capability because “no references are cited” by Alcorn (p.39). But there is good reason for this apparent omission by Alcorn—researchers

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consider it unethical to investigate the structure of the endometrium whilst an embryo seeks to implant. Therefore, in my view, Dr. Goodnough’s argument is invalid.

Is Alcorn isolated in his advocacy of a change in description of the pill from “contraceptive” to “abortifacient”? No. Embryologists such as Moore and Persaud, authors of The Developing Human: Clinically Oriented Embryology (6th ed., W.B. Saunders Company, 1998) and Ralf Rahwan, Emeritus Professor of Pharmacology and Toxicology, Ohio State University, support a change in title based upon the recognised capacity of the pill to interfere with implantation. Note that, in the citation from Moore and Persaud, they are speaking about the medication known as the “morning-after” pill. Since the “morning-after” pill, or more correctly, the post-coital abortifacient pill (PCAP) has mechanisms of action in common with the daily pill, it is acceptable, in my view, to apply Moore and Persaud’s definition to the daily birth control pill. Both medications employ the same hormones, though the PCAP is administered at an excessive “body-shock” dose.

Equally unsound is Dr. Goodnough’s dismissal of Alcorn’s statement that the pill will negate implantation because of changes to the endometrium. Dr. Goodnough labels Alcorn’s assertion “speculative” (p.39). The doctor is wrong.

We know that there is an optimum level of endometrial thickness necessary for successful implantation. In 1996, Issacs reported that an endometrial thickness of at least 10mm or more, around the time of ovulation, “defined 91% of conception cycles.” That same year, Spandorfer noted that 97% of abnormal pregnancies, defined as Fallopian tube lodgement or spontaneous abortion, had endometrial thickness of 8mm or less. Shoham reported in 1991 that no pregnancies were reported in an ovulation induction program “when the endometrial thickness was less than or equal to 7mm.”

These results have been obtained from IVF studies, and whilst the message apropos endometrial thickness is relevant to this debate, the drugs used are not. We still need to know what happens to the structure of the endometrium when the pill is taken.

Addressing this specific point was a research paper by Rabe (1997), who studied subjects who took the triphasic levonorgestrel/ethinylestradiol formulation of the pill. Women in this research project failed to develop a median endometrial thickness in excess of 6mm yet they had the highest percentage of follicular cysts with a diameter greater than 20mm. Follicles of this size are “thought to be associated with increased risk of escape-ovulation.”

The importance of these events is clear: follicles of a pre-ovulatory size can develop in women taking the pill daily, but endometrial thickness has been shown to be underdeveloped. In the event of follicle rupture, release of an ‘ovum’, and, fertilization/conception, implantation of a human embryo would be greatly hampered. Rabe confirms this very point: “...the occurrence of pregnancy would be unlikely because accessory contraceptive mechanisms such as cervical hostility and endometrial suppression are usually in effect.”
Dr. Goodnough also relied upon Fallopian tube implantation to argue that embryonic implantation is a robust event, resistant to suspected damage to the endometrium induced by the pill. Therefore the pill cannot act as an abortifacient. But again, he is incorrect in his central premise. Embryonic Fallopian tube implantation occurs because Fallopian tube tissue has the same integrin expression and window of implantation timeframe as the endometrium.\textsuperscript{18} Implantation is not akin to two pieces of Velcro randomly touching and sticking.

Alcorn also suggested that the combined pill might increase Fallopian tube contractions, hastening embryo transport to the extent that the embryo arrives prematurely at the surface of an under-prepared endometrium and, subsequently, fails to implant. Again, this would qualify the pill as an abortifacient. Dr. Goodnough disagreed with Alcorn’s proposition and cited a 1994 paper by Dr. Speroff to refute Alcorn. Dr. Speroff advised that “perfect synchrony (between embryo arrival and endometrial maturation) is not required.” (p.42)

Both Dr. Goodnough and Speroff are mistaken. Space precludes a detailed refutation, so I refer the reader to my previous paper on this matter for a comprehensive presentation and merely mention that contemporary researchers consider it mandatory that bio-chemical communication takes place between the embryo and the maternal endometrium prior to implantation. This precise, structured maternal/embryonic communication has been varying referred to as “a signalling system,”\textsuperscript{19} embryonic “dialogue,”\textsuperscript{20} “molecular communication”\textsuperscript{21} and “cross-talk.”\textsuperscript{22} Piccinni has summarised the complexities of the pre-implantation process as requiring “exquisite dialogue”\textsuperscript{23} between the human embryo (at the blastocyst stage) and the maternal endometrium.

Finally, we approach what I consider to be a most unpleasant aspect in Dr. Goodnough’s paper: his apparent embracing of relativistic morality. He says: In prescribing the oral contraceptive, some women (viz., one in 100,000) and perhaps some embryos will die. It is a matter of degree of risk” (p.44).

How does one respond to this statement? Initially, it should be pointed out that Dr. Goodnough appears to foresee only two “alternatives” for husbands and wives: the pill or pregnancy, and if a few unborn children’s lives are the trade-off factor this is seemingly acceptable. Apparently the solution to the “who lives” question is answered on the basis of risk/benefit analysis.

Dr. Goodnough also sought to defend his views by suggesting that the Principle of Double Effect validated the use of the pill. His advocacy of this position is erroneous because the foreseen “bad” effect of the pill—that some embryos might die—is a fully avoidable consequence: husbands and wives need only to practice one of the many methods of natural birth regulation.

For those wishing to assuage any technological interventions into this area of marriage, the Billings method, proven to be as reliable as the pill, and safer for both mother and unborn child, is an excellent option, as is the sympto-thermal method. Other couples may choose to avail themselves
of new technologies such as ovulation monitors, now within the price range of many. These are reliable options, which create no hazards for an embryo “unexpectedly conceived,” because these methods do not interfere with endometrial development, embryo/endometrial implantation factors, or Fallopian tube movements. Yet Dr. Goodnough does not mention these in his discourse on the justified marital use of the pill.

This is unfortunate because an endearing feature of these methods is that they are fully “Christ-like” on at least three points. These non-drug approaches do not violate the tenant “Thou shalt not kill”; they maintain what God has joined together in marital intercourse (the unitive, pleasurable and procreative dimensions); and they do not lead married couples into a form of Onanism, wherein the unitive, pleasurable and procreative characteristics are separated (Gen. 38:8-10).

In my scientific opinion, Dr. Goodnough has erred substantially and frequently in his critique of Randy Alcorn’s book. He has misinterpreted some key reports, omitted others, and seemingly been unaware of many. The end result is that the ancient dictum “first do no harm” takes a battering.

References
2 Letterie GS. A regimen of oral contraceptives restricted to the periovulatory period may inhibit folliculogenesis but inhibit ovulation. Contraception. 1998; 57:39-44.
4 Letterie, p.43.
5 Goodnough, p.38.
7 Ibid., p.243. It was greater than 4ng/ml.
8 “Postcoital birth control pills (“morning after pills”) may be prescribed in an emergency (e.g., following sexual abuse). Ovarian hormones (estrogen) taken in large doses within 72 hours after sexual intercourse usually prevents implantation of the blastocyst, probably by altering tubal motility, interfering with corpus luteum function, or causing abnormal changes in the endometrium. These hormones prevent implantation, not fertilization. Consequently, they should not be called contraceptive pills. Conception occurs but the blastocyst does not implant. It would be more appropriate to call them ‘contraimplantation pills.’ Because the term abortion refers to a premature stoppage of a pregnancy, the term abortion could be applied to such an early termination of pregnancy.” p.532
9 Rahwan RG. Chemical contraceptives, interceptives and abortifacients. Division of Pharmacology, College of Pharmacy, The Ohio State University, Columbus, Ohio 43210 USA, 1998.
10 I have devised this term to describe the effect that the PCAP has on women. The fact that women frequently experience nausea and vomiting post-ingestion is an obvious gauge of the pharmaceutical assault accessioned by this drug regimen.


15 Rabe, Table 3, p. 44.

16 Ibid., p.43.


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WILLIAM F. COLLITION, JR, MD

I am writing to rebut Joel E. Goodnough’s “Redux: Is the Oral Contraceptive Pill an Abortifacient?” It is particularly distressing that Dr. Goodnough’s thinking is inconsistent and always reflects his enthusiasm for the estrogen/progesterone combination birth control pill (BCP). The start of his reference 3 reads: “I follow the position of the American College of Ob-Gyn (ACOG) in defining the beginning of pregnancy as the time of implantation.” Dr. Goodnough pledges allegiance to the ACOG’s inane definition of pregnancy, which declares that a healthy woman who engages in non-contraceptive coitus at her peak fertile period with a healthy man and as a result has a tiny baby girl or tiny baby boy traversing her fallopian tube on her/his way to the endometrial cavity isn’t pregnant. That is obviously a ludicrous proposition. The same endnote comments: “This confusion of terms can be used to mask moral issues.” This was the purpose of the ACOG in changing the definition (circa 1970) that has traditionally been defined as “being with child”. The ACOG definition makes the IUD, BCP’s, POP’s (progesterone-only pills), and so-called “emergency contraception” (morning-after pills) non-abortifacients. If the woman isn’t pregnant, why does she need this last treatment? Dr. Goodnough concludes the footnote as follows: “I will continue to use the term abortion to describe the loss of the embryo both prior to and after implantation.” But he can’t have it both ways. Either it’s the ACOG or the truth.
On p. 39, without citation, Dr. Goodnough claims an estimated 70% pregnancy loss in healthy women not on BCP’s. While I am not ready to accept this high pregnancy loss in healthy women, it is ludicrous to suggest that implantation in a BCP-induced atrophic endometrium would match that of the lush, glycogen-rich, God-prepared endometrium found in normal cycling women. Dr. Goodnough always stresses correct use of the BCP. This is wishful thinking when Planned Parenthood and its allies are pushing this mode of contraception on teenagers. Besides, every experienced obstetrician has delivered a baby for a patient who will swear that she was taking her newer low-dose pills faithfully and in the prescribed fashion when she conceived. This chain of events is called a “pill pregnancy.”

On p. 44, Dr. Goodnough states, “A medication that is used to prevent conception is not an abortifacient even if it sometimes causes abortion.” This is pure semantic gymnastics. Gould’s Medical Dictionary (5th edition) defines “abortifacient” as follows: “(1) causing abortion, (2) a drug or agent inducing expulsion of the fetus.” From the moral perspective, it matters not how often the loss of totally innocent life occurs, only that it does occur. The doctor also overlooks a fundamental truth: that the BCP and abortion are both anti-life. This truth in no way equates the moral evil contained in these actions. To articulate this proposition more fully, the BCP and all contraceptive actions posited with or remote from coitus are designed to thwart the reproductive good obviously contained in intercourse and placed there by our Creator. Induced abortion ends a totally innocent life already conceived. On p. 45 Dr. Goodnough adds: “Yet, the preponderance of evidence suggests that prescribing estrogen to menopausal women does not sub-

stantially increase the risk of breast cancer.” He then states: “future studies could show that estrogen clearly causes breast cancer.” The future is now. Chris Kahlenborn, an Ohio internist, has written a book entitled Breast Cancer: Its Link to Abortion and the Birth Control Pill, filled with evidence suggesting that it is exogenous estrogen that has caused the doubling of the incidence of that disease in the last 5 decades. More recently, an AP article reported that a government scientific advisory committee has recommended that the chemical estrogen be added to the list of cancer-causing agents. We can be grateful that Dr. Goodnough supports fully informed consent for patients, as does Randy Alcorn.

Later (p. 48) the doctor states: “In fact, based on more recent studies, it appears that the OCP, when taken correctly, approaches 100% effectiveness in preventing ovulation” (no citation provided). Dr. Don Gambrell, a renowned gynecological endocrinologist who agrees with Dr. Goodnough on the issue at hand, has noted a 14% incidence of breakthrough ovulation in patients on the 50 microgram pill. He and a group of pro-life ob/gyns supporting the non-abortifacient nature of the BCP, noted that our literature documents a 3-5% (some say as high as 9%) incidence of “pill pregnancies” on the low dose pills. This is proof positive of, at a minimum, a like incidence of breakthrough ovulation. How many more conceptions occurred and were lost to an iatrogenic BCP-induced abortion because of an unfavorable endometrial pattern? That is unknown. However,
Ever since becoming involved in [the abortifacient] debate in the first months of 1988, I have been curious about why the discoverers of the birth control pill (BCP), from the earliest days of their work, described three mechanisms of action for their products: inhibition of ovulation, thickening of the cervical mucous (with both of these being truly contraceptive actions), and endometrial changes making implantation unlikely (an abortifacient action). Did they have animal or human studies to prove this? Our conferees of the opposing view suggested that they did not. This third mode of action, they suggested, was merely a marketing ploy to ensure women of the complete effectiveness of the BCP. In October 1999, I had the privilege of meeting Elora J. Weringer, Ph.D., a biologist with connections to the Pfizer Company. I inquired of her the location of the early studies of Gregory Pincus, D.Sc. and John Rock, M.D. She referred me to the Worcester Foundation for Experimental Biology, located in the city of the same name in Massachusetts. Several long distance calls to that fair city indicated that the Foundation had broken up into several different disciplines, and that none of the librarians whom I contacted had any information about the location of Dr. Pincus’ early studies. Subsequent to that point, the Holy Spirit entered the scene. Via an e-mail originating from Janet E. Smith, a philosopher at the University of Dallas, I received a copy of an article which appeared in the New York Times (June 25, 2000). It was written by Barbara Seaman and entitled “The Pill and I: 40 Years On, the Relationship Remains Wary.” The Pill was the brainchild of Margaret Sanger, the founder of Planned Parenthood and an unconquerable fighter for women’s rights. In circa 1950 she was introduced to Gregory Pincus, a reproductive scientist. She raised approximately $150,000, mostly from her friend, Katherine McCormick, an heiress to a farm-machinery fortune. She urged Pincus to get started on a universal contraceptive. Twenty years earlier researchers had established that hormones could prevent ovulation in rabbits and other species. Mrs. Seaman notes that Dr. Pincus was interested in a progesterone-only pill because he was wary of estrogen, as it was already understood to increase cancer risks. She adds:

“But there is a problem with progesterone-only contraceptives: they produce irregular and unpredictable spotting, or conversely, a complete absence of menstruation.” She labels this “menstrual chaos.” Pincus eventually put estrogen back into the BCP. How did Mrs. Seaman learn all this? She has made a study of Dr. Pincus’ papers, which are now housed in the Library of Congress. She notes: “They comprise approximately 44,000 items, filling 213 containers on 85.2 feet of shelf space. They reveal an awesome scientific and entrepreneurial brinkmanship, and make one wonder why Pincus didn’t burn the evidence.”

Read Barbara Seaman’s article. It is sordid. I won’t trouble you with the details. Her article informed me that the object of my search was a 20-minute subway ride from my home with no
downtown parking problems. Gregory Pincus was the steroid guru of his day and was internationally acclaimed. I reviewed only 4 boxes (#’s 93,107,142, and 145), but the contents were most revealing.

With regard to the issue of the abortifacient nature of the BCP, the following correspondence is enlightening. It is a letter from Albert Segaloff, M.D., dated September 4, 1964. He must have been an editor for Steroids, an international journal. He writes: “Dear Goody, I am enclosing your manuscript on ‘Further Studies on Implantation Inhibitors.’ I want to thank you for submitting this most fascinating paper on a very interesting series of compounds to Steroids.” The opening paragraph of this paper co-authored by Upendra K. Banik and J. Jacques of the Worcester Foundation for Experimental Biology and the College de France reads: “Twenty-three compounds injected on day 1 or days 1 through 3 of pregnancy in rats have been tested as possible inhibitors of implantation. Among them eight have proven active at total doses of 1.5 mg per rat or less. Administration of some of the active compounds by gavage has also led to implantation inhibition. The group of compounds found to be active were also the most potent in uterotrophic assay in immature mice. Among them a highly active compound, A-nor-androstane-2a, 17a-diethynyl-2b, 17b-diol (V) has been examined in detail. It appears to act primarily by causing expulsion from Fallopian tubes and uterus of the free, pre-implantation ova (sic), and was ineffective in the usual sterilizing dose in terminating implanted embryos.”

In 1965 in the World Health Organization Technical Report Series No. 303 appeared an article titled “Mechanism Of Action Of Sex Hormones And Analogous Substances: Report of a WHO Scientific Group.” On page 17, paragraph 5.1 reads: “Both the steroid hormones and the synthetic analogues, when used during long periods, have effects on the reproductive tract that need evaluation. In the normal female, endogenous hormones are secreted cyclicly (sic), involving the interrelated rise and fall of oestrogen and gesto-

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gen; this seems to be a protective mechanism of considerable significance. If there is continuous exposure to even low doses of oestrogens, either endogenous or exogenous, pathological effects are produced, the endometrium becoming hyperplastic. On the other hand, if progestogens and gestogens are given continuously at even low levels, amenorrhea and sterility result, with regression of the endometrium to a thin layer having scant if any secretory activity.” This finding has been known for a long time.

In Dr. Pincus’ files was a paper by Professor L. T. Samuels, a temporary member of the WHO Scientific Group. On page 5 of his paper we note: “Excess oestrogens can interfere with either fertilization, blastocyst formation, or implantation, depending on the time after ovulation when the high level occurs.” And later on the same page he adds: “The retention and rate of development of the blastocyst in utero has long been known to be progesterone-dependent. Oestrogens inhibit the blastocyst stimulating effect of progesterone. It is, of course, well known that excess oestrogens prevent implantation, just as they prevent gestogen-induced deciduoma formation in experimental animals.” I found several other references verifying these findings, but sufficient to clinch the long-known fact of the abortifacient nature of sex steroids under certain circumstances is another letter from Victor A. Drill, M.D., Director of Biological Research for G.D. Searle & Co. It is dated July 14, 1954 and addressed to Dr. Pincus, On page 2, one finds this paragraph: “We will not
send any compounds for anti-ovulatory or anti-implantation tests this month. If you need any for
the following month, this, of course, will be indicated on your list of requested numbers of
compounds.16

References
5  DeCook, J.L., Mcllhaney, J., et al. “Hormonal Contraceptives: Are They Abortifacient? A Physician’s Report.” (Sparta, MI: Frontlines Publishing, January 1988).  (For more information, call (616)887-6256 or e-mail order@frontlines.org.)

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RESPONSE BY JOEL E. GOODNOUGH, MD

I would like to thank Mr. Wilks for his response to my article. The purpose of my article was to critically evaluate Mr. Alcorn’s book, since his writings are appearing in churches and Christian bookstores, and there are very few other writings available to a mainstream audience on the subject.

I reviewed the studies that he referenced in order to determine the quality of his work. My article was not meant to be an in-depth comprehensive study on the entire issue. I am not a research scientist but rather just a physician who is seeking information and attempting to apply it to my practice. Mr. Wilks is therefore correct when stating that I omitted some studies, but I considered those I did include sufficient to show that Mr. Alcorn’s conclusions on the OCP are suspect.

Mr. Wilks has concerns about my comments directed at his use of the Letterie study. If one reads carefully, it can be seen that I did indeed point out to the reader that the purpose of the study “was to determine if other formulations of pills would be effective at preventing ovulation.” Mr. Wilks, however, says that he used this study as an example of the consequences of missed pills. His is an inappropriate use of a study that was designed to answer a different question and not at all typical of a woman who misses one or two pills.

Mr. Wilks feels that I was too limited in my discussion on the endometrium and that the studies I used were “dated,” being from 1981, 1989, 1994, 1996, and 1997. He then goes on to cite a study from 1980 as being particularly damaging to my arguments. The issue of the receptivity of the endometrium in an ovulatory cycle on the OCP is complex and not yet resolved.
Mr. Wilks continues the argument about ovulation rates. I did not think that it was necessary to repeat the information contained in my article that showed a difference between ovarian activity and rise in serum progesterone on the OCP versus actual ovulation. I cited five studies in my article that showed the OCP to be 100% effective at preventing ovulation, even when some pills are missed. One of these studies referenced three other studies showing the same conclusions. The problem of failure arises primarily when a woman forgets to start her pills on time and goes longer than seven days without taking a pill. It seems to me that this is an area where we can focus our efforts while the research on the endometrium continues.

I agree with Mr. Wilks when he says that the pill has a precarious hold on ovulation. There is, after all, a 3% pregnancy rate on the OCP. But this can be improved through proper use and with new formulations that shorten the pill-free interval, thereby lessening the consequence of error. In addition, there is now a monthly injectible combined estrogen-progesterone formulation that is very successful at preventing ovulation, primarily because a woman does not have to remember to start her pills each month.

I was prepared for criticism, but surprised by Mr. Wilks’ impression that I am embracing moral relativism. The fact that it is wrong to intentionally kill an innocent human being is a moral absolute. The problem lies in the determination of whether embryos are dying at all, infrequently, or frequently as a result of our intentions to prevent fertilization. If they are dying frequently, it is too high of a cost to pay. If they are dying infrequently, a risk versus benefit analysis is appropriate, just as it is for any other medication or activity that involves unintended risk to human life.

I would also like to thank Dr. Colliton for his comments. I agree with him completely as to the moral status of the embryo, regardless of how one defines pregnancy. I will let the theologians decide on the issue of the OCP thwarting the reproductive good contained in intercourse, placed there by our Creator. I disagree with him on the issue of estrogen and breast cancer, but the purpose of my inclusion of this in my article was simply to illustrate the decision-making process of risks versus benefits that a physician has to go through on a daily basis.

Joel Goodnough, MD is a gynecologist who practices in Arlington Heights, Illinois, USA.
TO THE EDITOR

WALTER L. LARIMORE, MD
JOSEPH B. STANFORD, MD, MSPH

Dr. Goodnough’s commentary (Ethics & Medicine 17:1) is an important addition to the current debate about the oral birth control pill’s (the Pill’s) postfertilization effects—which would be tantamount to an abortifacient effect to those who believe that valuable human life begins at fertilization (conception). However, we are concerned about several inaccuracies pertaining to medical facts in this paper and believe your readers will find this information useful.

Dr. Goodnough states that the rate of pregnancy on the Pill “. . . in the general population is 3% per year.” Unfortunately, the data to which he refers did not account for elective abortions. In other words, women who get pregnant on the Pill and then abort are not counted in these data. One national analysis, based upon 1992 data from the United States, that did account for the underreporting of elective abortions reported that the unintended pregnancy rates during the first year of Pill use were at least 4% for “good compliers,” 8% for “poor compliers,” and up to 29% for some users. We find that most Pill users and prescribers are unaware of these facts. Dr. Goodnough discusses what we have called the “turned-on-endometrium theory.” The proponents of this hypothesis feel, like Dr. Goodnough, that “One would therefore expect the endometrium in an ovulatory cycle on the OCP (oral contraceptive pill) to be more receptive than the endometrium in an anovulatory cycle on the OCP.” We have discussed elsewhere data that may refute this hypothesis. Dr. Goodnough does admit that this is only a theory and as such “is somewhat speculative.” We feel it is more accurate to report that the “turned-on-endometrium” theory is completely speculative. There is, to our knowledge, no published, peer-reviewed data that supports this theory.

Dr. Goodnough inaccurately discusses both our views and the data about the increased risk of ectopic pregnancies in women who get pregnant on the Pill. We feel it is unfortunate that he only used an outdated secondary source of our data (he used the 2nd instead of the current 4th edition). We feel your readers may have been better served if Goodnough had used the primary reference—a peer-reviewed, systematic review that we published in the Archives of Family Medicine, an American Medical Association journal. Unfortunately, this oversight led to several inaccurate statements. We will cite only one example: Goodnough says that we “. . . lump the progesterone-only minipill (POP) in with the combined estrogen and progesterone OCP.” This is not true. In our paper, we clearly stated that of the available studies, we specifically excluded any that even might have included women taking POPs mixed into the COC group. We said, “Therefore, of the five available publications, only two allow review of the association of COCs with ectopic pregnancy. These two studies from seven maternity hospitals in Paris, France, and three in Sweden involved 484 women with ectopic pregnancies and 289 pregnant controls and suggest that at least some protection against intrauterine pregnancy is provided via postfertilization preimplantation.
effects.”

“Our evidence-based and systematic review of this topic concluded, “Therefore, COC use seems to be associated with an increased risk of ectopic implantation or unrecognized loss of (embryos). We considered this level II (good to very good) evidence.”

For the reader seeking objective information, the peer-reviewed, systematic review of a subject may be of more value than a commentary, which may be more affected by the bias of the authors. This bias can be amplified in single-author commentaries—such as Dr. Goodnough’s. Likewise, we have some concerns about Dr. Goodnough’s ethical conclusions: Dr. Goodnough indicates his belief that prescribing a medication with a potential postfertilization effect, such as the Pill, is acceptable under the principle that “…if we prescribe (OCPs) to enough patients, more patients will be helped than hurt.” Indeed, in the practice of medicine, some risks are necessary. But Pill takers unnecessarily put pre-born children at risk. In fact, the very survival of these children is at stake. Regardless of the actual risk percentage, which is uncertain, a sexually active woman runs a new risk of aborting a child, in an unrecognized fashion, every time she takes the Pill.

Furthermore, as we discuss below, she has a non-abortifacient option for birth control, such as modern, scientific, natural family planning (NFP), that can be as or more effective than the Pill. Dr. Goodnough discusses a patient’s consent to use the Pill and states, “The fact that she consents and the embryo does not in no way lessens my responsibility.” This does not lessen his responsibility, but increases it. If Goodnough believes that the embryo is fully valuable human life, how can he allow someone else’s consent to put that pre-born child at risk to control his choice to prescribe the Pill? Even if the Pill does not usually cause an abortifacient effect, whenever it does it is just as real an abortion as if that were its primary effect.

Dr. Goodnough reviews our discussion about the Principle of Double Effect. Unfortunately, his incomplete review of the topic did not address what we consider to be the most important point of this principle: The argument about a possible abortifacient effect of the Pill “…certainly could be considered to fall under the category of disputable matters discussed in Romans 14:1-21. Objective, knowledgeable Christian observers would in all likelihood line up on both sides of the argument based upon a variety of subjective and objective criteria. However, the fourth principle of double effect has a corollary that must be considered. That corollary relates to alternatives. In other words, the principle is now being interpreted by some authors to make the contention that there must be no other way to produce the good effect.”

Goodnough does not discuss this information with his readers. Your readers should certainly be aware that several forms of natural family planning (NFP) have been found to have effectiveness rates comparable to oral contraceptives. One method that was developed at Creighton University in the United States has been medically studied over the last 20 years and has been reported in a large meta-analysis to be 96.8% effective at preventing pregnancy, taking into account user and teacher errors. As mentioned above, the Pill is at best 94% effective in actual use. The most recent study of this scientific approach to NFP concluded that pregnancy probabilities using this form of NFP
compared favorably with those of other methods of family planning and that women did not need
to have regular cycles to use NFP successfully.

Another effective form of NFP, the Billings Ovulation Method, is taught around the world
in all sociocultural situations and used successfully even by people who cannot read or write. NFP is
noted by its users and advocates to promote love, romance, communication, prayer, spirituality and
learning about natural, God-created reproductive mechanisms. Other advantages of NFP are that it
fosters communication and understanding between the man and the woman, develops co-operation
between them and a sharing of the responsibility in this important matter of their children.\textsuperscript{11}

In all these ways NFP improves a couple’s relationship and helps them to grow in love and
fidelity to each other. There is no evidence that the Pill provides these same benefits. Since there is a
viable, safe, and effective non-abortifacient alternative to the Pill, this fact would appear to dissolve
most arguments that the Pill, until scientifically proven to be non-abortifacient, should be or can
morally be used by Christians for birth control. In fact, assuming that NFP is only as effective as the
Pill (and not more effective), it would appear that most arguments to use the Pill, in view of the fact
that it may have an abortifacient effect, would be reduced to arguments of convenience (for the Pill-
user or prescriber) at the potential expense of pre-born human life.

Lastly, Goodnough indicates that the intent one has in prescribing or using the Pill is an
important consideration. He contends, “If the desired effect is prevention of conception by
preventing ovulation, it is not accomplished by a bad effect and there are no alternatives that are
safer.”\textsuperscript{1} Indeed, most Pill prescribers don’t intend to cause an unrecognized abortion. Nevertheless,
while the intentions of those taking or prescribing the Pill may be harmless, the results can be just as
fatal. In this sense, taking the Pill is analogous to playing Russian roulette, but with more chambers
and therefore less risk per episode. In Russian roulette, participants usually do not intend to shoot
themselves. Their intention is irrelevant, however, because if they play the game long enough they
cannot beat the odds—eventually someone dies. However, with Pill roulette, it is another person
who may die. The fact that a woman will not know when a child has been aborted in no way
changes whether or not it happens. The more Pills she takes, the greater her chance of having a
silent abortion. The more a physician prescribes the Pill, the more likely he is to cause an
unrecognized abortion.

\textbf{References}
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TO THE EDITOR

Randy Alcorn, MA

I read with interest Dr. Goodnough’s article “Redux: Is the Oral Contraceptive Pill an Abortifacient?” *Ethics & Medicine* 17(2001):37-51 on the abortifacient effect of the oral contraceptive pill (OCP). One thing Dr. Goodnough and I share in common is that we both very much hope his theories are correct—and that my view of the evidence is eventually disproved. As one whose wife took oral contraceptives, and who for many years recommended oral contraceptives in premarital counseling—and who doesn’t want for a moment to believe children may have been killed by my actions taken out of ignorance—I would certainly like to believe Dr. Goodnough’s position. If one day he is proved right, I will rejoice. Unfortunately, the evidence I’ve found, through painstaking research, does not support his conclusions. Furthermore, he made a number of factual errors of which the objective reader and a peer-reviewed journal such as *Ethics & Medicine* would want to be informed.

Some of the weaknesses of Dr. Goodnough’s article have been pointed out by Dr. Walter Larimore, in his letter to the editor of *Ethics & Medicine*. These include, but are not limited to, the following: 1) In citing the 3% pregnancy rate for first-year pill takers, Dr. Goodnough fails to take into account the fact that women who get pregnant while taking the pill and then get abortions are counted statistically as if they’ve never gotten pregnant at all, making the actual first-year pregnancy rate in pill takers much higher, 2) Dr. Goodnough’s “turned on endometrium” theory is completely speculative, and he presents no scientific evidence supporting it, 3) By using a dated version of my book (1998, instead of the 2000 revision, which is three revisions later) and by drawing my quotes from a now long-dated email exchange with Dr. Larimore rather than from Dr. Larimore’s subsequent article in *Archives of Family Medicine*, Dr. Goodnough significantly misrepresented several of my and Larimore’s conclusions.

My major concerns about Dr. Goodnough’s article involve its considerable logical and ethical weaknesses, as well as one particularly serious misquotation of my book.

Dr. Goodnough asks, “Is the OCP an abortifacient? Or is it a contraceptive that has the potential for failure, a failure that may result in the death of the embryo?” (It’s interesting that he narrows it down to these two choices, an apparent admission that the Pill may indeed result in the death of a child—which, ironically, is the central point that I present in my book.) He cites a medical dictionary’s definition of an abortifacient being something deliberately used to cause an abortion. Then he argues that the Pill isn’t an abortifacient. But this isn’t the point. The issue isn’t what the OCP should be called, it’s what the OCP can do. My book’s title is not “Should the Birth Control Pill Be Called an Abortifacient?” but “Can the Birth Control Pill Cause Abortions?” The latter question, not the former, is what this is all about.
Dr. Goodnough gives considerable attention to semantics. He insists, “a medication that is used to prevent conception is not an abortifacient even if it sometimes causes an abortion.” But the young men and women who talk to me about this issue are never concerned about labels and terminology. Their question is simple—can taking the Pill result in the unrecognized death of a pre-born child? Though at times he seems to deny it, at other times Dr. Goodnough appears to admit the answer is yes. Given what he regards as the positives of OCPs, he considers this a risk worth taking. Many couples, however, do not.

One of my main points is that couples have the right to know this information, and the medical community has the legal and ethical obligation to inform them. This is why Dr. Larimore and I and others have simply encouraged physicians and health care systems to provide full information to patients. If the patient is interested, show them the evidence, and let them come to their own conclusions. This is the crux of informed consent. But is it ethical for a physician to withhold evidence that many people—including other well-respected physicians—believe supports the contention of the Food and Drug Administration (FDA) and the OCP companies that the Pill sometimes prevents the implantation of a newly conceived child? Conscientious Christians who put their pro-life convictions above their personal convenience are not unusual, and they are not stupid. They can handle the evidence and reach their own conclusions. They will be held accountable for their choices, just as we will be held accountable for whether or not we present them with the full body of evidence.

As I clearly state in the book, usually the birth control pill does not cause abortions. As far as I am aware, no one argues that it usually acts as an abortifacient. The question is whether it sometimes causes the death of a child. How often it does so, no one knows—some say it is infrequent, some say it may not be as unusual as we’d like to believe. But the moral question is, how much risk to an innocent child are we willing to run for the sake of convenience? We may come to different conclusions, to be sure, but unless the evidence is laid on the table, people can’t bring their own ethical values to bear on these matters involving themselves and their children.

Dr. Goodnough says, “It is particularly distressing that Alcorn refers to studies in order to make a point, even though one would be hard pressed to find actual support for the point within the context of the study.” If by “support for the point” he means that the authors cited don’t state the conclusion “oral contraceptives cause abortions,” obviously that is true. I’ve researched and written fifteen books and many articles. It is standard practice in presenting one’s research to selectively cite Plato, C. S. Lewis, The New York Times, or The New England Journal of Medicine without implying that they necessarily agree with your particular conclusion. If we limited our citations only to those who have reached the same conclusion as we have, it would keep us from presenting evidence for any new or unpopular viewpoint. I present dozens of threads of evidence, documented in 138 endnotes. That some of them would not agree with my conclusions or share my ethical concerns is obviously true.
cause abortions.” I disagree with his conclusions, but I do not find it distressing that he pulls data from sources, which make no claim whatsoever to support his conclusions.

Dr. Goodnough admits that many sources, including *The Physicians Desk Reference (PDR)*, refer to the effects on the endometrium as “reducing the likelihood of implantation.” He then calls such statements “speculation.” I always find this interesting. The disclosure of the medical information contained in the PDR is mandated by no less an authority than the FDA. The information presented is more than a marketing ploy or a legal caveat. Anyone convinced that the manufacturers’ claims that the Pill sometimes prevents implantation are not truthful statements based on science, but false statements motivated by carelessness or public relations, has the responsibility to take this serious accusation to the oral contraceptive companies (all of whom make this claim) and the FDA. Dr. Goodnough and others should not expect either physicians or the general public to simply disregard this medical information from qualified research departments in favor of the more desirable (for pro-lifers) belief that the Pill really can’t do what the pharmaceutical researchers all claim it can.

Dr. Goodnough says, “in light of the fact that there is no definitive information on whether the embryo implants or not, [Randy Alcorn] could just as easily assume that the embryo always implants and survives despite seemingly hostile changes in the endometrium.” I would certainly like to make this assumption, as it would relieve me of any sense of moral obligation. Unfortunately, the assumption seems to be based on wishful thinking, not scientific observation or logic. It is clearly not equally valid to draw either conclusion after looking at what everyone, even Dr. Goodnough, agrees are “seemingly hostile changes in the endometrium” caused by OCPs. To admit that this appears to be true and then to say—without producing any evidence to support it—that one might just as well conclude the embryo “always implants and survives” is nonsensical, isn’t it? If the endometrium appears to be hostile, clearly the burden of proof falls upon those, such as Dr. Goodnough, who argue it is not (or, who argue that conception and a hostile endometrium are not mutually exclusive). Dr. Goodnough needs to produce evidence to show that a *seemingly* hostile endometrium is not a *truly* hostile endometrium. But he fails to do so. In the absence of such evidence, aren’t we forced to assume that the endometrium is indeed what it seems to be—hostile to implantation? To present these conclusions as equally valid, in the absence of evidence supporting what is contradictory to empirical observation, is untenable.

Among those who have no vested interests, I have virtually never found anyone arguing that the Pill cannot or does not hinder implantation. The *only* people I’ve found who make that assumption are those who have vested interests in doing so—pro-lifers who use, prescribe, or recommend oral contraceptives, but do not (understandably) wish to believe they can jeopardize human life.

My most serious concern with Goodnough’s article was a misrepresentation of my argument, followed by a striking misquotation from my book. The misrepresentation is claiming that I “attempt to equate the so-called morning-after pill with the OCP.” In fact, I do not equate the two—I simply point out that the morning-after-pill is not some novel chemical invention, but four standard OCPs taken together (suggesting that the pills already have something in them which raises the frequency of an abortifacient effect as the dosage increases). But to back up his
misrepresentation of my point, Dr. Goodnough quotes me as saying that the morning-after-pill “increases the chances of doing what it [the birth control pill] already does—cause an abortion.”

That does indeed sound like I’m equating the two. Dr. Goodnough follows by expressing dismay that I could say such a thing. When I read the quotation, I too was dismayed. Why? Because I knew what other readers wouldn’t—I did not say this. What I actually said, in all five editions of the book (Goodnough quotes from the second)—was this: the morning-after-pill “increases the chances of doing what it [the OCP] already sometimes does—cause an abortion.”

Dr. Goodnough left out the all-important word “sometimes.” This makes it appear I was claiming the OCP, like the morning-after-pill, acts primarily as an abortifacient. That would be an erroneous claim, of course. Indeed, readers of Dr. Goodnough’s article now believe I was making that very claim. Anyone who could have read what I actually said would know I was not. Unfortunately, this correction will never reach most of those who read the article or who will read it in the future. I am disappointed that such a misquotation was not corrected during the peer-review process of Ethics & Medicine. I can only hope Dr. Goodnough did not also leave out critical words when he cited other sources, but I have no assurance this is the case.

I am not straining out gnats here. It is one thing to misunderstand an author and in the process misrepresent his position to others. It is another thing to actually revise what an author has said, in this case leaving out a critical operative word, resulting in misrepresenting the author and misleading the reader. I trust that was not Dr. Goodnough’s intention, of course. But it certainly was the result.

This critical gap between intentions and results leads naturally to my final and most serious concern about Goodnough’s theories, one that lies at the heart of my disagreement with him. He says, “When I prescribe the OCP, I do not want an embryo to die. The death of the embryo, should it occur, is the undesired result of intending to prevent fertilization” (p.45).

First, we should remember that some patients would consider the risk of carrying an unwanted child as less serious than the risk of killing an unwanted child. They will think in terms not simply of the preferences of adults to not have children, but the welfare of children themselves. We certainly all want physicians to have clear consciences—but let’s not forget their patients also have consciences, and it is of paramount importance that the patient be able to act in good conscience, informed by their physician of the existing evidence, and the interpretations of not one, but both, schools of thought.

But my main concern is with this matter of intentions. As a college ethics professor and author of several books on ethical issues, I’ve interacted with people in hundreds of different vocations. Interestingly, I have found that the logic of “sincerity and good intentions makes something right” seems more prevalent among medical professionals than any other group.

I certainly agree that most women taking the Pill don’t intend to get abortions. In fact, I’m convinced that 99% of them are unaware this is even possible. (This is precisely the problem, and why we need true informed consent by Pill takers.)
The fact remains that while the intentions of those taking the Pill may be harmless, the results can be every bit as fatal. A nurse giving a child an injection may sincerely intend no harm to a child. But if she mistakenly injects him with a fatal poison, her good intentions will in no way lessen the tragedy. Whether the nurse has the heart of a murderer or a saint, the child is equally dead. The best intentions do nothing to reverse the most disastrous results.

Even if the Pill doesn’t usually cause an abortion, whenever it does do so it is just as real an abortion as if that were its intended effect. So, I certainly believe that when he prescribe OCPs, Dr. Goodnough does not want an embryo to die. But I find that irrelevant to the question at hand. The chances of the embryo’s (I prefer the term “pre-born child’s”) death are in no way lessened by the prescribing physician’s or the mother’s or anyone else’s intentions.

By all means, let us be sincere and intend only to do good. But we must never argue for the legitimacy of a course of action based on our sincerity and good intentions. We must act instead in light of the actual evidence that indicates what consequences may come from the action itself. Whether or not an action is moral depends on a number of factors, not least of which is the possible impact on the welfare of a human being. This is particularly true when it involves an innocent human being who is unable to speak up for himself and for whom we are commanded by God to act as advocates (Proverbs 31:8-9).

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THE ORAL CONTRACEPTIVE PILL AND THE PRINCIPLE OF DOUBLE EFFECT

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The question of whether the low-dose combined estrogen and progesterone oral contraceptive pill (OCP) is an abortifacient has evoked considerable discussion within the pro-life medical community. The main lines of the debate bifurcate between one contingent of pro-life physicians who oppose abortion and contraception and another who oppose abortion but not contraception. Some clinicians and ethicists in the first group object to the combined OCP based on good indirect evidence that it could cause the death of an early embryo. Others in this group broaden their moral opposition to include the primary effect of the OCP: direct suppression of fertility for procreative (family-planning) purposes. For the latter, moral objection to the OCP would continue even if it were proven that it was not an abortifacient.

Pro-life physicians in the second group morally oppose abortion but, because they view hormonal contraception as an effective method of family planning that also has impressive health benefits, do not take moral issue with the low-dose combined oral contraceptive (COC) and, therefore, prescribe it to their female patients. Furthermore, these physicians maintain that, given the lack of direct, and compelling indirect, evidence for its abortifacient character, prescription of the OCP is morally justified.

Background

In a previously published article in Ethics & Medicine (17:1), Joel Goodnough, M.D. weighs in on the abortifacient question and its implications for obtaining informed consent from OCP-users. He concludes that the most commonly prescribed oral contraceptive, the COC, is designed and intended to suppress ovulation and, therefore, to prevent conception. Though he admits that the COC has the potential for failure due to user error or decreased absorption, he also maintains that, if such a failure were to occur and result in the death of the embryo, it would be an unintended adverse side effect. Goodnough argues that, while physicians may want to inform their patients of this possibility in obtaining their properly informed consent, the most reasonable way of dealing with the moral ambiguity is not to discourage the use of OCPs altogether but to encourage, instead, responsible pill-taking.

Goodnough bolsters his thesis, first, with scientific documentation from the primary research of pertinent studies in the literature and, second, with moral corroboration from the principle of double effect. Using the latter, he concludes that, in the act of the prescription or use of the COC, the good of conception control is what the physician-prescriber (or the COC-user)
intends (directly wills) while the evil of the possible death of the embryo is what the moral agent accepts as an unintended side effect and, therefore, what lies outside his/her intention.

My contribution to the ongoing debate outlined above is twofold: first, to

advance data from relevant medical/scientific literature which calls into question and encourages re-examination of Goodnough’s conclusion that indirect evidence for the COC’s post-fertilization effects is negligible (part one); second, to challenge his use of the principle of double effect to morally justify the prescription of the COC (part two).

Part One: Critique of the Scientific Evidence

No direct evidence; no substantive indirect evidence: “[I]t is not possible to say that the combined OCP causes abortions.” Goodnough insists that, first, there is no direct evidence for the abortifacient character of the COC and, second, the indirect evidence for such a position is inconclusive and/or negligible and based on “unfounded fears.”

Response: I certainly agree that there is no direct evidence that the COC causes abortions. And if its post-fertilization effects (its anti-implantation mechanisms operative pre-, peri-, or post-implantation) were studied directly, it would either involve techniques and procedures that are immoral by virtue of destroying early embryonic life or involve studies that would be moral but non-definitive since they would include indicators such as the Early Pregnancy Factor (EPF) [a pregnancy-associated immunosuppressive protein detected in maternal sera by rosette inhibition assay that, to date, provides a less than acceptable accuracy index]. However, in the rest of this segment, I hope to substantiate that there is good indirect evidence that post-fertilization effects play a small, yet not negligible, role in loss of embryonic life induced by the COC. The principal deficiency of the indirect evidence is a paucity of published data that prevents the quantification of that risk in absolute terms.

A) Ovulation rates on the pill: Goodnough argues that there is no evidence to support the occurrence of ovulation in excess of that of the pregnancy rate for normal use of the OCP (3% for 100 woman years). The pertinent studies he cites show that, while there is evidence of ovulatory or ovarian activity among COC users, there is no evidence of ovulation. Only studies that include progesterone-only pill (POP) users along with COC users show breakthrough ovulation.

Response: It is relevant to point out that evidence of ovarian activity on the OCP does not necessarily include normal ovulation. Nevertheless, pertinent literature demonstrates that, to determine whether ovarian activity does include ovulation, it is critical to study more than three cycles. The available evidence suggests that breakthrough ovulation may become more common with increasing duration of OCP use. In investigations involving 4 or more cycles, ovulation did occur. Breakthrough ovulation was more likely in women using OCs with lower doses (or no dose) of estrogen and with women whose use of the OCP is imperfect rather than perfect. Ovulation rates (ORs) for COC users range from 1.7% to 28.6% per cycle. The former figure comes out of a 6-cycle study (Grimes et al.) that, because it is based on ultrasound investigation, supplies
incontrovertible evidence for ovulation; the 28.6% figure is based on a 4-cycle study (Chowdhury et al.) that, although it provides less conclusive evidence since it is hormonally based, does demonstrate that, with imperfect or normal use, 10 out of 35 women ovulated by the fourth cycle and, with perfect use, 10% or 1 out of 10 women showed a rise of progesterone suggesting ovulation. ORs for POP users for 6 or more cycles range from 33% to 65% per cycle.

As for failure of the hormonal contraceptive to prevent pregnancy, it is necessary to account for the underreporting of elective abortions. If this is considered, the rates of pregnancy on the OCP are estimated at 4% for “good compliers” and 8% (increasing to a possible 29%) for “poor compliers.” Logically, these adjusted pregnancy rates must be taken into account in attempting to make the best estimate possible of breakthrough ovulation rates on the OCP.

B) Prevention of Implantation: Goodnough enlists four arguments to defend his position that the OCP’s effects on the endometrium do not cause the loss of the embryo.

First, he agrees that it makes sense to postulate that the endometrium during an anovulatory cycle on the OCP is less normal, but that it does not make sense to argue the same during an ovulatory cycle. Due to the active presence of endogenous sex hormones associated with ovulation, the endometrium of an ovulatory cycle on the OCP would be more normal and proportionately less likely to be hostile to implantation.

Response: Where are the peer-reviewed data to support Goodnough’s postulate regarding the state of the endometrium during ovulatory cycles on the OCP? Just from a common sense perspective, does it seem reasonable to hypothesize that, after perhaps prolonged OC use and its corresponding deleterious effects on the endometrium (average endometrial thickness in OCP users is 1.1 mm), the same endometrium, following breakthrough ovulation, will immediately spring back from its atrophied, decidual state to that of a normal, non-pregnant (non-secretory) state or even to a normal pregnant (secretory) state? Some IVF studies demonstrate that implantation following embryo transfer does not occur in an endometrium that is less than 6 mm thick.

Second, Goodnough insists that the claim that the OCP-induced changes in the endometrium actually prevent embryo implantation is speculative. While the literature describes the OCP as effecting an endometrium that is inhospitable to implantation, “[n]o literature actually shows that death of the embryo results.” Whatever embryo loss occurs following breakthrough ovulation and fertilization, “despite seemingly hostile changes in the endometrium,” occurs “at the same rate as the embryo implants and survives in non-OCP users.” (sic)

Response: As I already noted, I agree that there is no direct evidence for OCP-induced embryo loss. However, in the definition of the mechanisms of the OCP’s action (contraception), it is clear that the COC’s efficacy is guaranteed by a combination of the pill’s effects from both its estrogenic and its progestational agents (that is, from the pill’s pre- and post-fertilization effects). In the first place, the COC prevents a clinically recognized pregnancy by the estrogenic/progestational effects of its primary mechanism: inhibition of gonadotropin secretion via an effect on both pituitary and hypothalamic centers. The progestational agent suppresses luteinizing hormone (LH) secretion and the estrogenic agent suppresses follicle-stimulating hormone (FSH) secretion via the prevention
of the selection and emergence of a dominant follicle. In the second place, the COC assures “good contraceptive efficacy” (translated: prevents a clinically recognized pregnancy) by effects of the pill’s (progestational)

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secondary mechanisms: changes in the endometrium (creating “a decidualized bed with exhausted and atrophied glands”) which make it unreceptive to ovum [sic] implantation; changes in cervical mucus so that it becomes thick and impervious to sperm transport to the uterus; and, changes in the secretion and peristalsis within the fallopian tube that alter embryo transport (and that provide “possible . . . additional contraceptive effects”).

It is well known clinically that, during use of OCPs, the regular withdrawal bleeds (“menstrual” bleeds) are lighter than natural menses. A lighter menses indicates a thinner endometrium. Further, once a woman discontinues the use of OCPs, it takes more than one cycle for her menstrual flow to return to the normal level of flow that occurs without OCs. This is clinical evidence that the endometrium does not return immediately to its full thickness when the OC is discontinued altogether. It seems far less likely that it could return to full thickness during a cycle in which the OC is still being taken, albeit, perhaps irregularly.

Due to the COC’s almost perfect rate of contraceptive effectiveness during perfect use, Speroff et al. go on to say that the occurrence of a clinically recognized pregnancy while on the pill is most likely not due to any failure of the pill to act as it is estrogenically and progestationally designed, but to extrinsic factors that have nothing to do with the action of the OCP. When taken correctly, the COC approaches 100% contraceptive efficacy, that is, it is almost 100% effective in preventing a clinically recognized pregnancy. Contraceptive failure is most likely due, then, to failure of its users to strictly adhere to the prescribed regimen (such as missing days), to interference from other medications, or to pill use accompanied by “vomiting and diarrhea.”

Furthermore, does not the claim that the embryo implants and survives at the same rate it does in non-OCP users, despite “seemingly” OCP-induced hostile endometrium, imply that all embryo loss following breakthrough ovulation on the pill is due completely to natural causes and has nothing to do with the effects of synthetic hormones? Does not such a claim run directly contrary to the authoritative conclusion of gynecological textbook authors that the COC’s efficacy in preventing a clinically recognized pregnancy is due to the comprehensive action of its pre- and post-fertilization effects? Is it not disingenuous to argue that, if a pregnant COC user, under the perhaps prolonged influence of synthetic estrogenic and progestational steroids and their post-fertilization effects, experiences any early embryo loss, it will only be the result of spontaneous abortions and at the same rate as that of a pregnant woman not on the OCP? Again, where is the evidence to support this argument?

While data demonstrate that women who experience a clinically recognized pregnancy while on the OCP experience subsequent spontaneous abortions at rates similar to those of women not on the pill, to argue this way in reference to unrecognized pregnancies cannot be substantiated. As Stanford and Larimore point out, “. . . available evidence suggests that the mechanisms of early establishment and maintenance of pregnancy and later maintenance of pregnancy are qualitatively and substantially different.”23
Third, it has been shown that even if the endometrium is in a less receptive state when the human embryo reaches it, the embryo could still implant (and obviously does sometimes implant as evidenced in women who get pregnant on the pill). In humans (in contrast to some animals) there are several days—a window of days—when the embryo could successfully implant, including a time before and after the optimal time for implantation. As Leon Speroff argues, the use of drugs that, speculatively, could provide contraceptive efficacy by accelerating tubal transport of the embryo would be “of doubtful value in the human because perfect synchrony is not required.” In other words, the arrival of the human embryo to the implantation site and an optimally receptive state of the endometrium need not be synchronous and, as a result, accelerating the embryo’s transport through the tube would not contribute to the COC’s efficacy due to the flexible window of implantation in humans.

Response: That some embryos do implant in the endometrium of women taking COCs is obvious from those women who get pregnant on the pill. But this says nothing about whether an embryo is more or less likely to implant in endometrium that has been decidualized and atrophied from the COC compared to implantation in normal endometrium in a woman not taking COCs. The Chowdhury et al. study (cited above) showed that, in women who ovulated secondary to missing two low-dose COCs, the lutenized endometrium was found to be nonsecretory. Such evidence strongly suggests that fewer embryos will be likely to implant in this situation.

Fourth, Goodnough insists that integrin studies showing an appreciable decrease of integrin expression in the endometrium of OCP users are relevant to the question of the anti-implantation possibility of the pill only if the data originate from ovulatory cycles on the pill.

Response: Somkuti et al. report “significant alterations in cycle-dependent integrin expression” in the endometrium of OCP users, but they do not specify whether the women tested are in ovulatory or non-ovulatory cycles on the pill. But if, for argument’s sake, one concedes that decreased integrin expression only occurs during anovulatory cycles on the pill, how reasonable is it to claim that a COC user who conceives during an ovulatory cycle will move from a grossly altered level of integrin expression to one that is normal in such a brief period of time? Certainly, there are no data to support such a complete recovery within one follicular phase. Furthermore, would Somkuti et al. have concluded that this diminishment of integrin expression contributed to the pill’s efficacy, that is, prevention of a clinically recognized pregnancy, if they were not referring to integrin expression during ovulatory cycles on the combined OCP?

C) The Incidence of Ectopic Pregnancy: Goodnough points out that one of the benefits of OCP use is “less chance of ectopic pregnancy.” He points out that certain studies demonstrate an increased risk of ectopic pregnancy on the OCP for several reasons. For one, they include POP users along with COC users. Since the POP slows down the transport of the embryo, it would naturally lead to higher incidence of tubal pregnancies. If a study consisted of COC users only, Goodnough argues, the results would vindicate his claim that the COC protects against ectopic pregnancy at least as well as it prevents uterine pregnancy.
Response: Stanford and Larimore point to two large studies\textsuperscript{29,30} whose participants are COC users only: one conducted in seven maternity hospitals in Paris, France; the other in three Swedish hospitals. Collectively these investigations, involving 484 women with ectopic pregnancies and 289 pregnant controls, suggest that “at least some protection against intrauterine pregnancy is provided via postfertilization effects,”\textsuperscript{31} namely, via ectopic pregnancy.

Since risk of ectopic pregnancy also involves varying degrees of health risk for the women involved, it is important, from the perspective of obtaining adequate informed consent and respecting individual beliefs, to determine, as accurately as possible, the COC’s absolute risk of causing extrauterine pregnancy. Adapting the model of Franks et al.,\textsuperscript{32} and assuming an odds ratio (relative risk) for an extrauterine pregnancy for an OCP user of 1.1 to 13.9, Stanford and Larimore predict that a woman on the COC has an absolute risk of an ectopic pregnancy due to postfertilization effects “ranging from 0.7 . . . to 19.9 . . . per 1000 woman-years.”\textsuperscript{33} For POP users, presuming an odds ratio for an extrauterine pregnancy of 79.1, one could predict an “absolute risk of 4 to 99 ectopic pregnancies per 1000 woman-years.”\textsuperscript{34}

D) The Definition of the OCP: Goodnough states, “[b]y design, by intent, and by primary function, the OCP, when properly used, is in essence a contraceptive. The fact that it may fail to act as it was designed does not change its essence.”\textsuperscript{35} And “. . . a medication that is used to prevent conception is not an abortifacient even if it sometimes causes abortion.”\textsuperscript{36} The way physicians can “render the risk to the embryo tolerable”\textsuperscript{37} and morally justify the prescription of the OCP is to encourage its responsible use and, on the part of the physician, to prescribe it continuously rather than cyclically, eliminating the pill-free interval.

Response: Goodnough defines the combined OCP in the literal, more narrow sense of that which is \textit{contra} or \textit{against} conception. In this view, the practice of contraception is conception control, not birth control. But in defining the OC narrowly, Goodnough sets himself outside the more comprehensive, mainstream definition of the OCP—an agent designed to prevent a clinically recognized pregnancy—assigned to it by users, designers/researchers and physician-prescribers.

If queried, users would probably not define the OCP as a pharmacological drug that prevents them from ovulating, but as one that prevents them from getting pregnant. N. Van der Vange addresses this point in his study of ovarian activity with the use of selected low-dose COCs:

Pearl-Index data, claimed by the manufacturers of these low-dose preparations, indicate that protection against pregnancy is indeed maintained. The present study may introduce some doubts about these figures (his study found a relatively large number of ovulatory cycles with the low-dose COC: triphasic LNG [30 mcgs EE; 50 mcgs levonorgestrel]). However, the \textit{mode of action of these OCs is not only based on ovulation inhibition, but other factors are involved such as cervical mucus, vaginal pH and composition of endometrium} (italics mine).\textsuperscript{38}

The action of accessory contraceptive mechanisms just alluded to gives credence to the definitional accuracy of the vernacular term, the “birth control pill,” when referring to the OC. Women who
take the pill for family planning purposes do so to avoid getting pregnant. Abortion statistics substantiate that fact by revealing that half of the women who have an abortion were on the pill when they got pregnant. In other words, when the OCP fails to do what it is intended to do and what it is designed to do, namely, prevent a clinically recognized pregnancy, many women “rectify” the contraceptive failure with abortion.

Standard texts describing the mechanism of the COC and professional inserts written by pharmaceutical designers that accompany the pills corroborate this populist definition. The COC acts both to suppress ovulation and to prevent uterine implantation. Its dual end is realized not only by the primary estrogenic mechanism of anovulation but by its secondary progestational mechanisms that, besides preventing the surge-like release of LH necessary for ovulation, also prevent sperm transport to the uterus, alter fluid secretion and peristalsis of the fallopian tubes, and alter the uterine endometrium in a way that makes implantation of the early embryo less likely. When the contraceptive nature or essence of the pill is defined in this broader, more comprehensive sense, it is clear that the way the OCP is designed to act—in a pre-fertilization and post-fertilization manner—corresponds exactly to the commonplace definition of the OCP’s essential nature, which is to prevent a clinically recognized pregnancy or to control birth.

Part Two: Is Prescription of the COC Morally Justified by the Principle of Double Effect?

Because of its implications for moral analysis, Goodnough is right to hone in on the correct definition of the COC. First, by defining its design and intent as the suppression of ovulation, he suggests that the moral object of the action of prescribing the COC—precisely what the physician is intending in that action—is a morally good one and one that could be done for a good motive. What the physician is doing, i.e., what he intends, is the suppression of ovulation (its content) chosen under the guise of the good (its form). In short, according to Goodnough’s analysis, to offer the COC user temporary, reversible infertility is a good thing and, therefore, the moral object of the act of prescribing the hormonal contraceptive is a good one. Second, defining the essence of the COC as suppression of ovulation, Goodnough also implies that the principal motive of the physician for prescribing it, to prevent conception, is also morally good. The physician intends the act’s foreseen good effects (prevention of conception) and only permits or accepts its foreseen but unintended evil effects (prevention of implantation).

Third, it is impossible, from a moral perspective, to define what it is that one wills or intends in the action of prescribing the oral contraceptive unless and until one understands the pill’s intended effects versus its unintended side effects. If, as Goodnough argues, the COC is essentially defined as an anovulant, that is, that its principle effect is to prevent conception by suppressing ovulation, then what he or any other physician intends (in se intentum) by prescribing the combined OCP is the good of the suppression of ovulation. However, Goodnough considers the COC’s side effects, like that of the risk of death of the early embryo in the event of breakthrough ovulation and fertilization, to lie outside the intention (praeter intentionem) of the physician. By not intending but only accepting the foreseen evil side effect of a possible abortion, Goodnough appears to be arguing that the physician is fulfilling his duty to avoid those evil effects as far as possible.
Fourth, understanding the “nature” of the COC alerts the physician to the morally ambiguous nature of the act of prescribing it. Goodnough defines the COC as a medication that, in essence, prevents conception, but one that also has the potential for failure that could result in the death of the early embryo. Hence,

[18]

a physician-prescriber is able to foresee that the action of prescribing the COC has both beneficial (morally good) and harmful (morally bad) effects: the foreseen good effects—the prevention of conception and other health benefits, and the foreseen bad effect—the risk of the loss of early embryonic human life. Whether prescription of the pill is a morally good thing to do—in the presence of this morally evil effect—is the question that the principle of double effect can help to answer.

Given Goodnough’s definition of the COC and its implications for the way he would define the moral object of and motive for the action of prescribing it, the following is a suggested specification of his employment of the principle of double effect:

1) The intended object of the act of prescribing the COC—the suppression of ovulation—is a morally good one, i.e., it facilitates the patient’s family planning goals, facilitates genuine gynecological health and, therefore, contributes to human fulfillment of the COC-user.

2) The motive of the prescribing physician—to prevent conception of a new human being—is a morally good one (i.e., it advances human fulfillment since it conforms with the woman’s plans to, say, avoid unplanned pregnancies). The physician’s motive is to will the foreseen good effect while only permitting or accepting the foreseen (but rarely occurring) evil effect (risk of the death of the early embryo).

3) The foreseen good effect of the action—the suppression of conception along with other health benefits—is realized not by means of the foreseen action’s bad effect—the possibility of the death of the early embryo—but by means of the introduction of synthetic sex steroids that alter events of the ovulatory and menstrual cycle. [In other words, the death of the embryo is not the means to the suppression of ovulation; the action of the synthetic sex steroids is.]

4) The foreseen good effects of the action—an effective, convenient, and safe method of conception control and a host of health benefits—are equal to or greater than the foreseen but rare occurrence of the death of an embryo.

5) The physician has no other effective means than the use of COCs to realize the ends of conception control and other pill-specific health benefits.

Response: As I outlined above, Goodnough defines the COC in a literal, narrow manner that fails to encompass its broader, more comprehensive mechanisms of action and essence, viz., the prevention of a clinically recognized pregnancy. With an inaccurate understanding of the essential
nature of the COC in place, his definition of the moral object of the act of prescribing the OC will also necessarily be faulty. Objectively speaking, then, what the physician-prescriber intends in the act of prescribing the COC is the prevention of a clinically recognized pregnancy. And directly willing the prevention of a clinically recognized pregnancy means that the physician wills that the pill achieve that end through its primary and secondary mechanisms of action, i.e., through both its pre- and post-fertilization effects.

Understood correctly, then, the moral object of the act of prescribing the COC is evil, not good. It is critical to this discussion to note that directly intending to prevent a clinically recognized pregnancy by the prescription of the COC is illicit based on two distinct immoral acts, risk of abortion and suppression of fertility. First, one ought never to prescribe a medication that could directly risk causing the death of another human being, and second, one ought never prescribe a medication that works against the good of the patient by suppressing rather than promoting, specifically, the human procreative good and, by extension, overall physical and psycho-somatic health and human fulfillment.

Of course, if the act of prescribing the COC is immoral by virtue of its directly intended object (even if you define it, as Goodnough does, as suppression of ovulation only), one cannot proceed to the subsequent conditions of the principle of double effect without incurring moral inconsistencies. Referencing my previous construction of Goodnough’s appeal to the principle of double effect, and presuming for illustrative purposes that the directly intended object of the act is suppression of ovulation only, these contradictions would include the following:

1) The object of the act of prescribing the COC, suppression of ovulation, is described as moral when it is immoral;

2) The motive for the act, the control of conception, could be morally acceptable given the presence of psychological, financial, and health reasons justifying the spacing of children. (Keep in mind, however, that a morally upright motive will not transform an action that is immoral by virtue of its moral object into a morally good act);

3) The foreseen effect of the act—conception control—is evil not good, and it is sometimes realized by the evil means of post-fertilization effects;

4) The “good” effect of conception control cannot be equal to or greater than the evil effect of birth control since both effects—the anovulant and abortifacient—are evil; and

5) The prescription of the OCP is not the only means of obtaining the end of effective conception control; there is another means to avoiding conception that is moral since it accords with the good of the human beings involved, both providers and users, and it brings with it its own set of other health benefits.

**Summary Response:** Based on these contradictions, my objections to Goodnough’s use of the principle of double effect (PDE) are threefold. First, in his description of the requisites for the
correct application of the PDE, Goodnough opts to make explicit in his fifth criterion what is typically unexpressed but always presupposed by the principle, namely, that “there must be no other way of producing the good effect.” Since this criterion is central to adjudicate legitimate appeal to the principle, it is appropriate to state it upfront. It immediately restricts invocation of the PDE to cases where the good goal of the agent can be achieved only through a morally mixed means, that is, through an action that realizes both good and bad effects. In other words, if, in the case under consideration, there would be an effective way to suppress ovulation or to avoid pregnancy that does not bring with it the evil of an abortifacient effect, one would be obligated to choose that option rather than the OC.

Laboring under the aegis of that requisite, I maintain that Goodnough inappropriately invokes the PDE to justify the prescription of the OC, since there is an alternative, that is, an effective medical and moral means of avoiding pregnancy. Evidence of the medical efficacy of a natural method of family planning, a meta-analysis of the Creighton Model NaProEducation Technology (five studies involving 1,876 couples), reveals that, when this system of natural procreation education is used to avoid a pregnancy, its method effectiveness at the 12th ordinal month is 99.5% and its use effectiveness is 96.8%; at the 18th ordinal month, its method and use effectiveness is 99.5% and 96.4% respectively. These statistics compare favorably with the efficacy of the OC.

Although a moral comparison/contrast between the use of a natural method and the OCP for family planning purposes would entail a discrete article, the following is sufficient here. Only natural methods of family planning afford a method of avoiding pregnancy that does not bring with it the risk of the induced death of the early embryo (a moral ambiguity associated with OCs that Goodnough recognizes and about which he has moral reservations). Further, when a couple avoids the conception of a new human being by respecting the natural rhythms of their fertility, they choose a means to their end that conforms exactly to a comprehensive understanding of human nature and the procreative/personal aspect of human fertility. As Leon Kass warns, the principal norm against which we need to adjudicate any sort of reproductive technology, including “the pill,” is whether it constitutes a fulfillment rather than a “defilement of our given nature as procreative beings...” The reality is that only with natural methods of family planning (as opposed to steroidal hormonal methods) is a couple able to promote the truth of their procreative nature, the truth of marriage as a community of love and life, and the truth of their marital intercourse as acts that are at once life- and love-giving.

Second, even if for argument’s sake we concede, from one side, that one can legitimately invoke the PDE in respect to the prescription of the OC and, from the other, that the abortifacient effect of the OC is an unintended side effect, the nature of the evil effect of the act, i.e., the death of an early embryo, would not be outweighed by the good effects of a convenient method of family planning and of ancillary health benefits. Or, stated another way, the good effects of convenient family planning and possible health benefits are not of a sufficient moral value to justify the bad effect of risking the death of an embryonic human being. There is a clear disproportionality between the good and bad effects of the act of prescribing the OC and, as a result, Goodnough’s argument fails to fulfill the proportionality requisite of the PDE.
Third, in assessing the moral object of the action of prescribing the OC, and, again, conceding for argument’s sake that the “what” of the action is suppression of ovulation as Goodnough defines it, it is necessary to analyze the moral nature of the kind of act that suppresses ovulation. One cannot describe the suppression of ovulation as good unless one views fertility and the normally functioning reproductive system as some sort of pathology. But, in what sense are a woman’s natural menstrual and ovulatory cycles a disease? Should not working cooperatively with a woman’s reproductive system so that it can function optimally be a premiere goal of gynecological medicine? And, if this analysis stands, the prescription of the OC, even when judged primarily from a medicinal rather than from a moral perspective, is not a good human act. That is, prescribing the OC is not in the best health interests (physical and moral) of the patient, nor is it, by logical extension, in the best professional interests of the health professionals who are bound to promote the integral good of every patient.

References

This discussion of the question of the abortifacient character of the oral contraceptive focuses on the perfect use of the “low-dose combined” oral contraceptives, i.e., those containing 30 or 35 micrograms (mcgs) of ethinyl estradiol [EE]). It does so because, first, most women using hormonal contraception are on this form of “the pill.” Second, there appears to be more substantive agreement that the other contraceptive formulations may have greater risks of incurring breakthrough ovulation, ectopic pregnancy, or hostile effects on the endometrium and, therefore, may have greater risks than the “low-dose combined” pill (COC) for post-fertilization effects. The other contraceptive formulations that are referred to in contrast to the COC include: the lower dose combined pill (20 or 30 mcgs of EE); progestosterone-only pill (no estrogenic component); emergency contraceptives (2 doses:120-200 mcgs of EE or no EE but 1.5 mg levonorgestrel); injectable contraceptives such as DepoProvera (no estrogenic component) and imperfect use of any kind of hormonal contraceptive.


4 I adhere to the scientifically sound definition of fertilization as the beginning of pregnancy and abortion as any event (natural or induced) that causes the death of the developing embryo or fetus and that occurs from post-fertilization up to and including the end of the third trimester. Early abortions, those that occur before implantation and the secretion of hCG, are clinically unrecognizable pregnancies, while those occurring after the verifiable presence of hCG are clinically recognizable pregnancies. Consequently, what I intend by the phrase “death of the early human embryo” in the context of post-fertilization effects of the combined OCP is an induced abortion of the early embryo.

5 B Ashley, KD O’Rourke. Health Care Ethics: A Theological Analysis 4th ed (Washington, DC: Georgetown University Press) 1997. Also see my argumentation in Part Two of this article.


7 Ibid., 47.

8 Ibid., 48.

9 Ibid.


17 Goodnough, “Redux,” 40.


22 Ibid.

23 Larimore and Stanford, “Postfertilization Effects,” 130.


26 Goodnough, “Redux,” 46.


31 Larimore and Stanford, “Postfertilization Effects,” 133.


33 Larimore and Stanford, “Postfertilization Effects,” 129.

34 Ibid., 130

35 Goodnough, “Redux,” 43-44

36 Ibid., 44.

37 Ibid., 49.


41 The relevant question is, how precisely does the practice of hormonal contraception abuse rather than properly use the human procreative power? The answer exposes how contraception contradicts the human meaning or purpose of the act of marital intercourse and how it denies the truth of the procreative/sexual natures of male and female human beings and of their acts of conjugal intercourse. First, the practice of contraception treats fertility, or the procreative aspect of the act of marital intercourse, as a one-dimensional reality or as mere biology. But just as the human being is a body-soul unit, human fertility, a bodily power, has, at once, a biological and a personal meaning. The need to propagate the human species which is certainly a biological need, is also, at once, a basic human need that, when properly fulfilled, promotes the good of the whole person. Second, since the suppression of any basic human need contradicts the well-being of the whole person, and since contraception directly suppresses the procreative need of the human being, the practice of contraception acts against human welfare. Stated another way, the fulfillment of the cadre of basic human needs specifies how it is a human being realizes him or herself. These human needs, including the need to procreate, tell the husband and wife what they ought to do or what goods they ought to promote in order to be themselves, that is, to realize themselves—how they ought to act in order to be a fulfilled and happy couple. Third, contraceptive intercourse fails to recognize that the personal, self-giving dimension of marital intercourse is the inseparable correlate to its biological, life-giving dimension. To untether the life-giving dimension of fertile marital intercourse from its self-giving falsifies the comprehensive meaning of the act of marital love. Predictably, to some degree or another, the very love and intimacy that one had hoped to protect and promote through suppression of fertility necessarily collapses in upon themselves. Accordingly, the mutual human growth and flourishing that every couple hopes for, and that should follow on the heels of the “one flesh” relationship of their genital intercourse, will be truncated or will, to varying degrees, fail to materialize.


43 From a practical perspective, the use of natural methods of family planning has the potential to promote a cadre of health benefits beyond that of the moral regulation of birth. The CREIGHTON MODEL FertilityCare® System, for example, not only provides the benefits of an effective holistic method of both achieving and avoiding a pregnancy as the circumstances of a marriage require, but it also provides a charting system that acts as an elemental diagnostic tool for tracking, evaluating, and maintaining gynecological health. Tracking one’s reproductive cycles, as part of the science of NaProTECHNOLOGY® (Natural Procreative Technology), allows the woman and her physician to maintain good gynecologic health by monitoring and evaluating anomalies including infertility, miscarriage, irregular cycles, hormonal imbalance, PMS, ovarian cysts, and unusual bleeding.

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POSITION STATEMENTS
Focus on the Family Position Statement: Birth Control Pills and Other Hormonal Contraception

From the outset, Dr. Dobson would emphasize as foundational his strict concurrence with the biblical teaching that every child is a blessing from God. The entire ministry of Focus on the Family has been grounded upon this truth. While affirming that human life begins at fertilization (the union of sperm and egg), his interpretation of Scripture leads him to believe that the prevention of fertilization is not morally wrong. However, he would oppose any method of birth control that acts after fertilization and terminates a conceived human life by preventing its implantation in the womb. For example, the intrauterine device, or IUD, as it is commonly called, is thought to interfere with implantation of the fertilized egg, and, therefore, may terminate human life in its very early stages.

Birth control pills (also known as oral contraceptives, OCPs, or “the pill”) have become a focus of controversy because of concerns that they may occasionally terminate human life after fertilization. Hormonal contraception is a very complex matter, not only because it involves multiple biological effects, but also because many different types and formulations of pills and medications are currently available. Dr. Dobson has been careful not to arrive at any conclusions regarding this highly technical subject without first consulting with respected colleagues in the medical field. You may be aware that a panel of doctors representing a broad range of specialties serves on Focus on the Family’s Physician Resource Council (PRC), which advises the ministry regarding medical concerns.

Based on a review of the available literature, the PRC has concluded that birth control pills which contain only the hormone progesterone do not reliably prevent ovulation (the release of the egg from the ovary). This is also true of Norplant, a device implanted under the skin which slowly releases progesterone. With these methods, the pregnancies which do occur have a greater chance of being ectopic—that is, outside the uterus. This may be evidence that these contraceptives act in some cases to disrupt the normal implantation of an early pregnancy and not merely to prevent conception. Thus, the use of Norplant and the progesterone-only pill is problematic for those of us who believe that human life begins at conception.

The most commonly prescribed birth control pills, called “combined” oral contraceptives, contain both estrogen and progesterone. These medications, as well as Depo-Provera injections, seem to work primarily through suppression of ovulation. They also cause the mucus at the opening of the uterus to be thickened and, therefore, less likely to be penetrated by sperm. If combined oral contraceptives and Depo-Provera work only through these mechanisms, they are functioning as true contraceptives because they prevent the sperm and egg from uniting. However, there is controversy as to whether they also bring about changes (primarily within the uterus) that could increase the likelihood of losing a fertilized egg if ovulation and conception should occur.

Pro-life physicians who have carefully and conscientiously studied this issue have come to different conclusions regarding the interpretation and implications of the relevant scientific data. After two years of extended deliberation and prayer, the PRC has not been able to reach a consensus as to the
likelihood, or even the possibility, that these medications might contribute to the loss of human life after fertilization. The majority of the experts to which Dr. Dobson has spoken feel that the pill does not have an abortifacient effect. A minority of the experts feel that when conception occurs on the pill, there is enough of a possibility for an abortifacient effect, however remote, to warrant informing women about it.

Focus on the Family encourages further investigation of the mechanisms of action of the pill and other hormonal contraceptives, and specifically calls upon the medical community to undertake research to prove or disprove the hypothesis that the combined oral contraceptive pill occasionally interrupts human life in its very early stages. You may be certain that we will monitor new developments in this area of research.

Dr. Dobson respects the integrity and pro-life convictions of those who hold differing opinions regarding the mechanism and use of oral contraceptives and asks the pro-life community at large to do likewise. He also recognizes that scientific reasoning is not the only factor that may influence one’s viewpoint regarding the use of birth control and advises couples to examine the facts prayerfully as they consider the acceptability of any approach to family planning.
Christian Medical & Dental Associations Ethics Statement:
Possible Post-conceptional Effects of Hormonal Birth Control

CMDA holds firmly that God is the Creator of life, that life begins at conception, and that all human life is of infinite value. We support measures to protect life from its earliest beginnings.

CMDA recognizes that there are differing viewpoints among Christians regarding the broad issue of birth control and the use of contraceptives. The issue at hand, however, is whether or not hormonal birth control methods have post-conceptional effects (i.e., cause abortion). CMDA has consulted many experts in the field of reproduction who have reviewed the scientific literature. While there are data that cause concern, our current scientific knowledge does not establish a definitive causal link between the routine use of hormonal birth control and abortion. However, neither are there data to deny a post-conceptional effect.

Because this issue cannot be resolved with our current understanding, CMDA calls upon researchers to further investigate the mechanisms of action of hormonal birth control. Additionally, because the possibility of abortive effects cannot be ruled out, prescribers of hormonal birth control should consider informing patients of this potential factor.

We recognize the difficulties of providing informed consent while handicapped by lack of definitive information. However, counseling of patients may simply involve asking if they have concerns about potential post-conceptional effects of these methods of birth control. In cases where concern exists, an explanation may follow that includes the known mechanisms of action (e.g., inhibition of ovulation and decreased sperm penetration), as well as the concern about the unanswered question of whether hormones negatively affect the very early stages of life.

CMDA respects and defends the right of our colleagues to refuse to prescribe hormonal birth control when they do so with the concern of a post-conceptional effect.

We recognize that scientific reasoning is not the only factor that influences opinions about the use of hormonal birth control. But, while additional investigation is needed, current knowledge does not confirm or refute conclusions that routine use of hormonal birth control causes abortion. CMDA will continue to monitor new developments.
FAMILY RESEARCH COUNCIL PRESS RELEASE:
WOMEN NOT FULLY INFORMED ABOUT BIRTH CONTROL PILL,
THANKS TO THE AMA

“Each woman has the right to know what’s good for her health and acceptable to her conscience,” FRC’s Dr. John Diggs says.

WASHINGTON, D.C. - The American Medical Association (AMA) voted overwhelmingly this week against a proposal to inform women of the abortion-inducing potential of the birth control pill. Citing the AMA’s dedication to enhancing the patient/physician relationship, the Alabama doctor who submitted the proposal during the AMA’s annual meeting believes full disclosure about the birth control pill should be made to patients to help them make choices.

The prescribing information for Ortho Tri-Cyclen, a popular oral contraceptive, enumerates three pathways by which the pill works: suppressing ovulation, preventing fertilization and precluding the implantation of an already fertilized egg. The third one constitutes an abortion. The third function is conspicuously excluded from information made available to patients.

“If manufacturers are telling doctors that oral contraceptives can keep a new member of the human family from being nourished, why isn’t that information being passed on to patients?” Family Research Council Advisory Board Member John Diggs, M.D. said Friday. “The AMA is doing a great disservice to women by refusing to fully inform them of their birth control options. Since informed consent is a basic medical ethic, it should be standard operating procedure to tell women that the birth control pill can cause an abortion. Each woman has the right to know what’s good for her health and acceptable to her conscience. If the AMA has suppressed its conscience, it shouldn’t draw American women into its own ethical lapses.”

According to a member of the AMA’s executive committee, lobbying by the American Society of Reproductive Medicine largely contributed to the AMA’s decision.

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AMERICAN LIFE LEAGUE:
A DECLARATION OF LIFE BY PRO-LIFE PHYSICIANS

Introduction:

Birth control pills, Depo-provera injections and Norplant implants achieve their anti-fertility effects primarily by causing temporary sterilization, secondarily by causing abortion by preventing the implantation of the approximately week-old human from successfully attaching or “implanting” into the wall of the mother's womb, and thirdly by acting as a contraceptive barrier to sperm by thickening the cervical mucous. That some drugs promoted as contraceptives may really cause abortion has not been clear to many Americans for whom abortion presents serious moral questions.

Background of the Pill:

Gregory Pincus, co-developer of the Pill, credits a visit from Planned Parenthood’s founder Margaret Sanger who promised research money for the development of the Pill. i

Sanger, who supported abortion, was concerned about developing a Pill as a means of curbing the “population explosion.” ii

Like Sanger, Pill supporters who shared Sanger's demographic concerns, such as Dr. Robert Kistner of Harvard, were less concerned about means than ends: “Our efforts to control population growth should not lead to mass guilt about methodology. It would be tragic if an effective postcoital pill or long-term progestational agent were declared illegal because of its abortifacient effect.” iii

Conflict of Values: Guilt Would Be a Problem for Some.

In 1962 Dr. Mary Calderone, then Medical Director of Planned Parenthood said that: “if it turns out that these intrauterine devices operate as abortifacients, not only the Catholic Church will be against them, but Protestant churches as well.” iv

Legal problems existed because the language of pre-Roe anti-abortion laws was such that the “broad language of statutes and cases would suggest that to use pre-implantation means on a pregnant woman would be unlawful ... manufacturers, distributors or sellers of the pre-implantation means might be prosecuted under statutes prohibiting the manufacture, distribution or sale of abortifacients.” v
Technology Meets Biology:

Planned Parenthood’s Dr Abraham Stone, noted in 1952 that any mechanical, chemical or “... biologic method that would prevent ovulation or fertilization merely prevent life from beginning ... Measures designed to prevent implantation fall into a different category. Here there is a question of destroying a life already begun.” vi

The federal Department of Health, Education and Welfare also acknowledged this in a survey of birth control research: “All of the measures which impair the viability of the zygote at any time between the instant of fertilization and the completion of labor constitute, in the strict sense, procedures for inducing abortion. Administration of compounds whose mechanism of action is of this character to man either as an investigative procedure or as a practical birth control technique poses legal questions that have as yet not been resolved.” vii

The problem was that most of the promising research included anti-implantation or abortion causing actions. viii

Facts vs. Semantics:

With biology such a stubborn thing, Pill promoters turned to semantics for a solution. Swedish researcher Bent Boving, at a 1959 Planned Parenthood-Population Council symposium noted that: “Whether eventual control of implantation can be reserved the social advantage of being considered to prevent conception rather than to destroy an established pregnancy could depend upon something so simple as a prudent habit of speech.” ix

The advice was not isolated. At the 1964 Population Council symposium Dr. Samuel Wishik pointed out that acceptance or rejection of birth control would depend on whether it cause an early abortion. Dr. Tietze, of Planned Parenthood and the Population Council suggested, as a public relations ploy, “not to disturb those people for whom this is a question of major importance.” Tietze added that theologians and jurists have always taken the prevailing biological and medical consensus of their times as factual, and that “if a medical consensus develops and is maintained that pregnancy, and therefore life, begins at implantation, eventually our brethren from the other faculties will listen.” x

In 1965 the American College of Obstetrics and Gynecology (ACOG) responded with its own semantic answer: “CONCEPTION is the implantation of the fertilized ovum.” xi

Not everyone accepted these manipulations. Dr. Richard Sosnowski said he was troubled: “… that, with no scientific evidence to validate the change, the definition of conception as the successful spermatic penetration of an ovum was redefined as the implantation of a fertilized ovum. It appears to me that the only reason for this was the dilemma produced by the possibility that the intraterine contraceptive device might function as an abortifacient.” xii
The Pill and Abortion:

The federal Food and Drug Administration approved the Pill for limited use in 1960. First generation Pills allowed ovulation in 6.8% of menstrual cycles. (Because of health problems, the Pills’ high levels of estrogen were reduced, but less estrogen allows greater breakthrough ovulation.)

After much study a 1969 FDA Advisory Committee said the Pill's “high degree of contraceptive effectiveness [was] brought about through interference with several phases of the reproductive process. An influence on the hypothalamus ... is probably responsible for the ... inhibition of ovulation. ... The second major effect is on the endometrium. The progestin acts as an anti-estrogen causing alteration in endometrial glands and as a progestin, causing pseudodecidual reactions. Both of these alter the ability of the endometrium to participate in the process of implantation.”

Longtime Planned Parenthood associate Dr. Lewis Hellman chaired the advisory committee, and Dr. Christopher Tietze of PP and the Population Council was a committee member along with other PP members. xiv

And former PP President Dr. Alan Guttmacher is also on record as recognizing the triple mode of action for the Pill. xv

Pill Labeling:

In December, 1976 the federal FDA proposed mandatory patient package inserts accompany all Pill prescriptions: “The Food and Drug Administration will regard as misbranded and subject to regulatory action any oral contraceptive that is shipped in interstate commerce ... after April 6, 1977 without labeling that is substantially the same as set forth in this notice.” Thus, the FDA required Pill manufacturers to tell physicians that the Pill included a mode of action that every physician would understand from his medical training to be an early abortion: “Combination oral contraceptives ... Although the primary mechanism of action is inhibition of ovulation, alterations ... in the endometrium (which reduce the likelihood of implantation) may also contribute to contraceptive effectiveness ... progestin oral contraceptives are known to ... exert a progesterational effect on the endometrium, interfering with implantation, and, in some patients suppress ovulation.” xvi

Physician package inserts for the Pill are still required in 1998, and they still use language that indicates the Pill, Depo-provera and Norplant inhibit implantation. These chemicals “harden” the lining of the womb (uterus) creating a hostile environment and thus make it harder for the tiny multicelled human being from implanting in the wall of the womb. This constitutes abortion at approximately one week of life. There is no definitive medical agreement as to what percent of times per monthly cycle this occurs.

We, the undersigned physicians, do therefore declare that the pill and similar birth control products act, part of the time, by design, to prevent implantation of an already created human being. These
products clearly cause an early abortion and are—despite the semantic gymnastics of their ardent apologists—abortifacient.

We further declare that the so-called emergency contraceptive products being promulgated on the American people work in the same fashion and are also abortifacient.

References:


ii Margaret Sanger, *Family Limitation*, 1st ed., 1914, 15-16, Margaret Sanger Collection, Library of Congress (MSCLC); Sanger Speech, Washington DC, (MSCLC) speech was first given in 1916 and delivered 119 times; letter from Sanger to Hanna Stone, 3/10/32 copy to Marjorie Provost (Sanger’s handwriting) Sophia Smith Collection, Smith College.


v Sybil Meloy, “Pre-Implantation Fertility Control and the Abortion Law,” *Chicago-Kent Law Review*, vol. 41 (1964):183, 205-06. Planned Parenthood recognized in its amicus brief for Roe v. Wade that criminal abortion laws could be applied to the IUD because of its potential to prevent implantation. PPFA’s physician’s group’s (APPP) amicus brief on p. 44 cited Cybil Meloy and also said that prosecutors had not used state anti-abortion laws to outlaw the use of IUD’s.


week before and including the beginning of implantation, and the next greatest is in the week immediately following.”


xi ACOG Terminology Bulletin, Terms Used in Reference to the Fetus, Chicago, American College of Obstetrics and Gynecology, no. 1, September 1965.


xiii Joseph Rovinsky, MD, “Clinical Effectiveness of a Low Dose Progestin-Estrogen Combination,” Obstetrics and Gynecology, vol. 23, no. 6, June 1964, p.845, citing Goldzieher at al., JAMA, 180:359, 1962 “In 6.8 percent of menstrual cycles they have studied on patients on norethindrone medication, urinary pregnandiol excretion reached levels ordinarily found only in the postovulatory phase of a normal menstrual cycle.”


xvi Fed. Register vol. 41, no. 236, Tuesday, December 7, 1976, 53634.

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(This declaration was created by the department of Public Policy of American Life League, Inc. It was circulated to members of the medical and scientific communities for review and endorsement. A number of physicians have signed statements of agreement with the declaration. Their signed statements are on file at American Life League, P.O. Box 1350, Stafford, VA 22555. For a list of signatories, access: http://www.all.org/news/docsigs.htm.)
The rights of conscience of any person being a duly licensed pharmacist, who shall object on personal, ethical, moral or religious grounds to the performance of any act in the normal course of professional performance or dispensing, shall be respected.

Further, such a refusal to perform any act or the omission of any act based on such a claim of conscience, shall not form the basis for any claim for damages or any recriminatory or discriminatory action against such a person.

Any such person making such a claim of conscience, or who states a willingness or intention to make such a claim of conscience, shall not be denied employment, or discriminated against in any manner related to employment because of such a claim of conscience.

The Pharmacist’s Model Conscience Clause was adopted and approved by the PFLI Board of Directors in 1988. It was the first—and remains the only—one of its kind for the profession of pharmacy. It uniquely addresses the needs of pharmacists for recognition of their sincerely held religious, moral and ethical convictions which preclude the misuse of the gift of medications in manners contrary to the God-given dignity of the profession. Nothing less will do.

Any attempt to dilute or weaken the Conscience Clause does a disservice to the profession as well as an injustice to the many pharmacists who have courageously fought to have the Conscience Clause implemented in their workplaces. For standing by their principles, many of these brave professionals have paid the price of ostracism, calumny, vilification, persecution, reprimands, censure and dismissal.
RECOMMENDED READING
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The Abortifacient Debate:


DeCook, Joseph L.; McIlhaney, Joseph; et al. Hormonal Contraceptives: Are They Abortifacients? Sparta, MI: Frontlines Publishing, 1998. (To order an abbreviated version, e-mail: order@frontlines.org)


Theological Considerations Regarding the Morality of Contraception:
Brown, Harold O.J.; Budziszewski, J.; Chaput, Charles J.; Chevlen, Eric; Hinlicky, Sarah E.;


**History of the Church’s Views on Contraception:**


**History of the Pill:**


**Possible Health Implications of Pill Use:**


**Resources Describing “Natural” Alternatives to the Pill:**

Billings Method (BOMA-USA): www.billingsmethod.com


Couple to Couple League International: www.ccli.org

Creighton Model Fertility Care System: www.creightonmodel.com

Fertility Awareness Method: http://www.fwhc.org/birth-control/fam.htm


One More Soul: http://onemoresoul.com/


