Does the Birth Control Pill Cause Abortions?

A Condensation
By Randy Alcorn

"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 *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 lutenizing 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 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 Minipill, 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 prolife 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.

[1] Randy Alcorn, *Prolife Answers to ProChoice Arguments* (Multnomah Publishers: Sisters, OR: 1992, 1994) 118.

[2] *Danforth's Obstetrics and Gynecology* (Philadelphia, PA: J. B. Lippincott Co., 1994, 7th edition), 626.

[3] Nine Van der Vange, "Ovarian Activity During Low Dose Oral Contraceptives," published in *Contemporary Obstetrics and Gynecology*, edited by G. Chamberlain (London: Butterworths, 1988), 315-16.

[4] *Hippocrates*, May/June 1988, 35.

[5] *Oral Contraceptives and IUDs: Birth Control or Abortifacients?*, Pharmacists for Life, November 1989, 1.

[6] *Physicians' Desk Reference* (Montvale, NJ: Medical Economics, 1998).

[7] The *PDR*, 1995, page 1782.

[8] Stephen G. Somkuti, et al., "The Effect of Oral Contraceptive Pills on Markers of Endometrial Receptivity," *Fertility and Sterility*, Volume 65, #3, March 1996, 488.

[9] "Escape Ovulation In Women Due To The Missing Of Low Dose Combination Oral Contraceptive Pills," *Contraception*, September 1980; 241.

[10] G. Virginia Upton, "The Phasic Approach to Oral Contraception," *The International Journal of Fertility*, volume 28, 1988, 129.

[11]. Kastrup, EK, ed. *Drug Facts and Comparisons*, Annual Edition (St. Louis: Facts and Comparisons, 1997).

[12]. Abdalla HI, Brooks AA, Johnson MR, Kirkland A, Thomas A, Studd JW. "Endometrial Thickness: A Predictor Of Implantation In Ovum Recipients?" *Human Reprod* 1994;9:363-365.

[13] Bartoli JM, Moulin G, Delannoy L, Chagnaud C, Kasbarian M. "The Normal Uterus On Magnetic Resonance Imaging And Variations Associated With The Hormonal State." *Surg Radiol Anat* 1991;13:213-20; Demas BE, Hricak H, Jaffe RB. "Uterine MR Imaging: Effects Of Hormonal Stimulation." *Radiology* 1986;159:123-6; McCarthy S, Tauber C, Gore J. "Female Pelvic Anatomy: MR Assessment Of Variations During The Menstrual Cycle And With Use Of Oral Contraceptives." *Radiology* 1986; 160: 119-23.

[14]. Brown HK, Stoll BS, Nicosia SV, Fiorica JV, Hambley PS, Clarke LP, Silbiger ML. "Uterine Junctional Zone: Correlation Between Histologic Findings And MR Imaging." *Radiology* 1991;179:409-413.

[15]. Abdalla, et al., "Endometrial thickness"; Dickey RP, Olar TT, Taylor SN, Curole DN, Matulich

EM. "Relationship Of Endometrial Thickness And Pattern To Fecundity In Ovulation Induction Cycles: Effect Of Clomiphene Citrate Alone And With Human Menopausal Gonadotropin." *Fertil Steril* 1993;59:756-60; Gonen Y, Casper RF, Jacobson W, Blankier J. "Endometrial Thickness And Growth During Ovarian Stimulation: A Possible Predictor Of Implantation In In-Vitro Fertilization." *Fertil Steril* 1989;52:446-50; Schwartz LB, Chiu AS, Courtney M, Krey L, Schmidt-Sarosi C. "The Embryo Versus Endometrium Controversy Revisited As It Relates To Predicting Pregnancy Outcome In In-Vitro Fertilization-Embryo Transfer Cycles." *Hum Reprod* 1997;12:45-50; Shoham Z, et al. "Is It Possible To Run A Successful Ovulation Induction Program Based Solely On Ultrasound Monitoring: The Importance Of Endometrial Measurements." *Fertil Steril* 1991;56:836-841; Noyes N, Liu HC, Sultan K, Schattman G, Rosenwaks Z. "Endometrial Thickness Appears To Be A Significant Factor In Embryo Implantation In In-Vitro Fertilization." *Hum Reprod* 1995;10:919-22; Vera JA, Arguello B, Crisosto CA. "Predictive Value Of Endometrial Pattern And Thickness In The Result Of In Vitro Fertilization And Embryo Transfer." *Rev Chil Obstet Gynecol* 1995;60:195-8; Check JH, Nowroozi K, Choe J, Lurie D, Dietterich C. "The Effect Of Endometrial Thickness And Echo Pattern On In Vitro Fertilization Outcome In Donor Oocyte-Embryo Transfer Cycle." *Fertil Steril* 1993;59:72-5; Oliveira JB, Baruffi RL, Mauri AL, Petersen CG, Borges MC, Franco JG Jr. "Endometrial Ultrasonography As A Predictor Of Pregnancy In An In-Vitro Fertilization Programme After Ovarian Stimulation And Gonadotrophin-Releasing Hormone And Gonadotrophins." *Hum Reprod* 1997;12:2515-8; Bergh C, Hillensjo T, Nilsson L. "Sonographic Evaluation Of The Endometrium In In-Vitro Fertilization IVF Cycles. A Way To Predict Pregnancy?" *Acta Obstet Gynecol Scand* 1992;71:624-8.

[16]. Abdalla HI, et al., "Endometrial thickness"; Dickey, et al., "Relationship Of Endometrial Thickness"; Gonen, et al., "Endometrial Thickness And Growth"; Oliveira, et al., "Endometrial Ultrasonography As A Predictor"; Bergh, et al., "Sonographic Evaluation Of The Endometrium".

[17].The 5mm figure is from Glissant, A, de Mouzon, J, Frydman R. "Ultrasound Study Of The Endometrium During In Vitro Fertilization Cycles." *Fertil Steril* 1985;44:786-90. The 13mm figure is from Rabinowitz R, Laufer N, Lewin A, Navot D, Bar I, Margalioth EJ, Schenker JJ. "The value of ultrasonographic endometrial measurement in the prediction of pregnancy following in vitro fertilization." *Fertil Steril* 1986;45:824-8

[18].McCarthy, et al., "Female Pelvic Anatomy".

[19].*Physicians' Desk Reference*; Kastrup, *Drug Facts.*

[20] Kristine Severyn, "Abortifacient Drugs and Devices: Medical and Moral Dilemmas" *Linacre Quarterly*, August 1990, 55.

[21].Walter L. Larimore and Joseph Stanford, "Postfertilization Effects of Oral Contraceptives and their Relation to Informed Consent." *Archives of Family Medicine* 9 (February, 2000); Walter L. Larimore, "The Abortifacient Effect of the Birth Control Pill and the Principle of Double Effect," *Ethics and Medicine*, January 2000.

[22] Walter L. Larimore and Randy Alcorn, "Using the Birth Control Pill is Ethically Unacceptable," in John F. Kilner, Paige C. Cunningham and W. David Hager (eds), *The Reproduction Revolution* (Grand Rapids, MI: W.B. Eerdmans, 2000), 179-191.

[23] Susan Crockett, Joseph L. DeCook, Donna Harrison, and Camilla Hersh, "Using Hormone Contraceptives Is a Decision Involving Science, Scripture, and Conscience," in John F. Kilner, Paige C. Cunningham and W. David Hager (eds), *The Reproduction Revolution* (Grand Rapids, MI: W.B.

Eerdmans, 2000), 192-201.

[24].Stanford JB, Daly KD. "Menstrual And Mucus Cycle Characteristics In Women Discontinuing Oral Contraceptives (Abstract)." *Paediatr Perinat Epidemiol* 1995;9(4): A9.

[25].Chowdhury V, Joshi UM, Gopalkrishna K, Betrabet S, Mehta S, Saxena BN. "'Escape' Ovulation In Women Due To The Missing Of Low Dose Combination Oral Contraceptive Pills." *Contraception* 1980;22(3):241-7.

[26].Thorburn J, Berntsson C, Philipson M, Lindbolm B. "Background Factors Of Ectopic Pregnancy. I. Frequency Distribution In A Case-Control Study." *Eur J Obstet Gynecol Reprod Biol* 1986;23:321-331 (the original data was reevaluated by: Mol, et al., "Contraception and the Risk"); Coste J, Job-Spira N, Fernandez H, Papiernik E, Spira A. "Risk Factors For Ectopic Pregnancy: A Case-Control Study In France, With Special Focus On Infectious Factors." *Am J Epidemiol* 1991;133:839-49.

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

[28]. Thorburn, et al., "Background Factors Of Ectopic Pregnancy".

[29]. Larimore and Stanford JB. "Postfertilization Effects"; Thorburn et al., "Background Factors"; (the original data was reevaluated by: Mol BWJ, Ankum WM, Bossuyt PMM, Van der Veen F. "Contraception And The Risk Of Ectopic Pregnancy: A Meta Analysis." *Contraception* 1995;52:337-341); Mol, et al., "Contraception and the Risk".

[30]. Alcorn, "Does The Birth Control Pill Cause Abortions?"; Larimore WL, Stanford JB. "Postfertilization Effects Of Oral Contraceptives And Their Relation To Informed Consent." Larimore WL. "The Growing Debate about the Abortifacient Effect of the Birth Control Pill and the Principle of the Double Effect." *Ethics and Medicine*: in review.

[31]. DeCook JL, McIlhaney J, et al. *Hormonal Contraceptives: Are they Abortifacients?* (Sparta, MI: Frontlines Publishing, 1998).

[32] Randy Alcorn, *Does the Birth Control Pill Cause Abortions?* Fifth edition (Gresham, OR: Eternal Perspective Ministries), 50-73.

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