

Birth Control Pills: Contraceptive or Abortifacient?

Currently the claim that hormonal contraceptives [birth control pills, implants (norplant), injectables (depoprovera)] include an abortifacient mechanism of action is being widely disseminated in the pro-life community. This theory is emerging with the assumed status of "scientific fact," and is causing significant confusion among both lay and medical pro-life 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.

This paper is meant to provide some clarifying information on the issue based on current knowledge and experience regarding the mechanism of action of hormonal contraceptives. It has been compiled in consultation with, and by cooperative effort of, several practicing obstetrician-gynecologists, perinatologists, and reproductive endocrinologists (all among the undersigned), each being a physician committed to the sanctity of human life from conception.

We begin with the recognition that within the Christian community there is a point of view which holds that artificial birth control per se is wrong. We would consider this a personal matter of conscience and belief, and this paper is not intended to argue for or against this issue.

In this discussion we accept the time honored definition that conception occurs when a sperm penetrates an egg. Disruption of the fertilized egg after this point represents abortion. We consider fertilization, not implantation, to be the beginning of human life.

Most literature dealing with hormonal contraception ascribes a three-fold action to these agents. 1) inhibition of ovulation, 2) inhibition of sperm transport, and 3) production of a "hostile endometrium," which presumably prevents or disrupts implantation of the developing baby if the first two mechanisms fail. The first two mechanisms are true contraception. The third proposed mechanism, IF it in fact occurs, would be abortifacient. (Note: the developing baby at the time of implantation is called a "blastocyst," and will be referred to as such in this paper. "Endometrium" is the lining of the uterus into which the blastocyst implants.)

The entire "abortifacient" presumption, therefore, depends on "hostile endometrium" actually being hostile to the blastocyst, resulting in the loss of blastocysts that would otherwise prosper and grow. Since there are no scientific studies demonstrating the validity of this presumption, abortifacient proponents appeal to the writings of scientists and clinicians involved in the production or study of these contraceptive products. Nearly all of these sources freely use the term "hostile endometrium" to describe the changes which occur in the uterine lining when these medications are used. And most make the presumption that these changes contribute to birth control effectiveness. On the surface, this would seem to be nearly incontrovertible evidence that the "pill" is, at least occasionally, an abortifacient. However, we again emphasize that there are no scientific studies that we are aware of which substantiate this presumption.

Let us examine this "abortifacient presumption" by asking several questions:

1) What is meant by the term "hostile endometrium?" Where did it come from? Is it actually "hostile?"

The term "hostile endometrium" originated as a descriptive term for the less vascular, less glandular, thinner lining of the uterus produced by these hormones. The early pill literature from the late 1950's established the descriptive term. 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 pro-life community, to be a proof text. And all with no demonstrated scientific validation.

All pill manufacturers list this "hostile endometrium" presumption in their drug literature, implying it is a safeguard against pill failure. (Each company's literature says essentially the same thing as they comply with FDA labeling requirements) Understandably, their literature has a marketing agenda. However, to our knowledge, not one company will offer data to validate the "hostile endometrium" presumption. It should be noted that intertwining histologic fact (changes in endometrium) with presumptive action (makes the endometrium hostile) leads to a conclusion compatible with pill marketing strategies, but not necessarily compatible

with truth.

The fact that scientific authors in general all use the term "hostile endometrium" to refer to pill induced changes to the lining of the uterus adds nothing to establish the validity of the presumption that these changes cause loss of blastocysts. They are simply using long established descriptive terminology standard in the literature

2) Does the blastocyst require a "friendly endometrium" to thrive, or even to survive?

The nature of the blastocyst is important to this discussion. There is much we do not understand about the role of the blastocyst in implantation. But we do know it 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 it's usual environment [e.g., 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. Pill company literature estimates 3 to 5 pregnancies per 100 women per year for pill users. Many of these women take the "pill" an additional month or two before finding out they are pregnant. These pregnancies generally progress with no more difficulty than non-pill pregnancies. To our knowledge, there are no studies showing that the spontaneous abortion rate in these cases is any greater than in pregnancies with a "friendly endometrium."

The blastocyst regularly and successfully implants on tubal ciliated epithelium (commonly referred to as tubal, or ectopic, pregnancies) Approximately 1% of pregnancies in the USA are tubal pregnancies. The tubal epithelium is a tissue with an entirely different function and structure than the endometrium. Unlike endometrium, it has no glands with secretions, no rich vascular stroma. Yet these pregnancies implant and generally thrive until interrupted by treatment or rupture of the fallopian tube due to size constraints.

3) Is there actual clinical evidence of early miscarriage in pill users?

The typical clinical picture of spontaneous abortion (heavy bleeding, severe cramping, passage of tissue) is rarely, if ever, seen by most practicing physicians dealing with pill pregnancies, and is not substantiated in any literature we are acquainted with. The "hostile endometrium is abortifacient" proponents theorize that the losses are pre-implantation, and thus would have no tell-tale clinical or laboratory findings. However, since the actual rate of demonstrable ovulation for women on the pill roughly approximates the pregnancy rate for women on the pill, this type loss would seem extremely unlikely.

4) What is the conception rate for women on hormone contraception?

It is impossible to say. Ovulation suppression rates vary from about 95% with the combined 35 mcg estrogen pill to about 50% with the minipill or norplant in place 3-4 years. Cervical mucus factors enter in. Most pill literature estimates 3 to 5% pregnancies per year for combined OCs, less for depoprovera, more for norplant, and minipills.

One may get an idea of the frequency of conception on hormonal contraceptives by considering the ectopic (tubal) pregnancy rates. The ectopic rate in the USA is about 1% of all pregnancies. Since an ectopic pregnancy involves a preimplantation 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 rarely reported. This would suggest conception on these agents is also 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 very uncommon occurrence.

5) Is it possible that hormonal contraceptives may be responsible for the loss of blastocysts in some instances? In Medicine, anything is possible. Does the known medical information suggest that "on-pill" conceptions have a higher rate of blastocyst loss than normal "non-pill conceptions?" We believe the answer is "No."

There are 1,200,000 medical and surgical abortions of unborn babies that take place every year in the United States. The "hormonal contraception is partly abortifacient" theory is not established scientific fact. It is speculation, and the discussion presented here suggests it is error. How happy the abortionists must be to find us training our guns on a presumption, causing division/confusion among pro-life forces, and taking some of the heat off the abortion industry. Ought we not rather be spending our energies to eliminate the convenience destruction of the innocent unborn?

In Summary:

1. We know of no existing scientific studies that validate the "hormonal contraception is partly abortifacient" theory.
2. There is regular successful implantation of the invasive blastocyst on surfaces a great deal more "hostile" than "hostile endometrium" (e.g., fallopian tube lining). "Hostile endometrium" is not a demonstrated clinical reality.
3. The almost total absence of reporting of ectopic pregnancies associated with hormonal contraception would indicate the rarity of actual conception by patients using these modalities. (Minipill and norplant apparently are less effective in preventing pregnancies and ectopics).
4. 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 hormonal 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.
- 5) This paper is not meant to be the "final word" on this issue. If scientific study should validate that a hormonal 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 is abortifacient vs what is appropriate birth control to be used or prescribed by those who hold to the sanctity of human life from the time of conception.

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Co-signators (alphabetically) All signators are specialists in obstetrics and gynecology, and a number have sub-specialty recognition and/or are on the faculty of teaching hospitals or Universities. This information may be distributed freely to Crisis Pregnancy Centers or other individuals or groups who may have an interest in the subject matter.

Watson A. Bowes, Jr, MD, Professor, Maternal-Fetal Medicine, Chapel Hill, N.C.
Matthew J. Bulfin, MD, general OB-GYN, Ft. Lauderdale, Florida
Byron Calhoun, MD, Maternal-Fetal Medicine, Tacoma, Washington
Steve Calvin, MD, Asst. Prof Maternal-Fetal Medicine, Minneapolis, Minnesota
Denis Cavanagh, MD, Professor, Gynecologic Oncology, Tampa, Florida
Curtis Cook, MD, Maternal-Fetal Medicine, Asst Clin. Prof, Grand Rapid, Mi.
Susan A. Crockett, MD, Assistant Clinical Professor, San Antonio, Texas
Steven Cruikshank, MD, Professor OB-GYN, Dayton, Ohio
Joseph L. DeCook, MD, general OB-GYN, Holland, Michigan
Bill Dodds, MD, Reproductive Endocrinology, Asso. Clin. Prof, Grand Rapids, Mi
R. Don Gambrell, Jr., MD, Clinical Professor, Augusta, Georgia
David Hager, MD, Professor/Consultant OB-GYN, Lexington, Kentucky
Donna Harrison, MD, general OB-GYN, Berrien Springs, Michigan
Camilla Hersh, MD, Clinical Professor OB-GYN, Vienna, Virginia
Antony Paul Levitino, MD, .Asso. Prof., OB-GYN, Rensselaer, New York
Robert W. Lobel, MD, Assistant Clinical Professor, Urogyn, Albany, N.Y.
Joe McIlhaney, MD, Obstetrician-Gynecologist, Austin, Texas
Gwendolyn Patterson-Hobbs, MD, OB-GYN Clinical Associate, Vienna, Va.
William Stalter, MD, Associate Clinical Professor OB-GYN, Dayton, Ohio
Roy Stringfellow, MD, general OB-GYN, Colorado Springs, Colorado
Robert L. Weeldreyer, MD, general OB-GYN, Holland, Michigan



HOLLAND GYNECOLOGY & OBSTETRICS, P.C.

Joseph L. DeCook, M.D.

L. Carl Jurgens, M.D.

November 9, 1999

Dear Pro-Life Colleague or other friend interested in Life Issues:

We have previously sent you our literature research paper entitled "Hormone Contraceptives: Controversies & Clarifications." Since completing the paper we have become aware of one study done in about 1977, published in 1980 which challenges a point that we made on page 3, paragraph 2 regarding the status of the endometrium in a birth control pill cycle in which there has been escape ovulation, (and thus a different hormone milieu than exists in a birth control pill cycle with no ovulation). This topic is important because it bears on the quality of the endometrial lining that may exist in an ovulatory pill cycle. We have reviewed the original article and commented upon it in light of a number of other articles bearing on the same topic. Our discussion is contained in appendix 3 of our paper. Appendix 3 is enclosed for your consideration. Once again, this material has reference to the second paragraph on page 3 of our original paper. We would be glad to attempt to answer any questions you may have. If you need another copy of our original paper, please let us know.

Sincerely,

Joseph L. DeCook, M.D., F.A.C.O.G.
Donna Harrison, M.D., F.A.C.O.G.
Camilla Hirsch, M.D., F.A.C.O.G.
Susan Crockett, M.D., F.A.C.O.G.

JDC/jmu

Hormone Contraceptives: Controversies and Clarifications

Appendix 3 (Refers to Page 3, Paragraph 2, of our original paper)

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 his 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 this 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, (6): "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." 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 which conceive 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 (12.7ng/ml), 95% confidence limits of 28-53nmol/ml (8.8 to 16.7ng/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 80nmol/ml, 0.9 to 25.2ng/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 (9.4ng/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 8-9ng/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 8ng/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, frequently uterine fibroids, a retroflexed uterus, pain on the part of the patient, and operator inexperience are all 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 interpretation.

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 4ng/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 ng/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 an egg, yet still produce progesterone. (6 and others. see literature on polycystic ovarian syndrome.)

3) Chowdhury's endometrial biopsy data are uninterpretable because of the lack of clear documentation of ovulation, and the large number of biopsies with no tissue obtained (ie 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, 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.

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