**Is Plan B an Abortifacient?**

*A Critical Look at the Scientific Evidence*

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On September 27, 2007, the Catholic bishops of Connecticut announced that they would allow the four Catholic hospitals in their state to comply with the state’s emergency contraception law that took effect on October 1, 2007. In their statement, the bishops declared, “The administration of Plan B pills in this instance cannot be judged to be the commission of an abortion because of such doubt about how Plan B pills and similar drugs work.” It is a statement that has generated much controversy and criticism from those who are convinced that Plan B is an abortifacient. For example, Fr. Thomas J. Euteneuer, president of Human Life International, has urged the Connecticut bishops to reconsider their position because there is no doubt that Plan B is an abortifacient: “The truth is that there is absolutely no doubt about how the Plan

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B pills work. Just ask the manufacturer, Barr Pharmaceuticals, whose product insert states: ‘This product works mainly by preventing ovulation (egg release). It may also prevent fertilization of a released egg (joining of sperm and egg) or attachment of a fertilized egg to the uterus (implantation).’ It’s that third item that makes Plan B an abortion-causing drug. The same can be said for every chemical contraceptive.”

In order to bring more clarity to this discussion, this essay will summarize and critically review the scientific studies that have attempted to uncover the mechanism of action of levonorgestrel (LNG), the active drug in the contraceptive commonly known as Plan B. What does this drug do? There is mounting and recent evidence—several important papers were published only in the past six months—that suggests that this emergency contraceptive has little or no effect on post-fertilization events.

**Plan B Is an Abortifacient: The Case for the Claim**

Several clinical studies have demonstrated that levonorgestrel (LNG), taken in two doses of 0.75 mg twelve hours apart, can prevent or delay ovulation by abolishing the mid-cycle luteinizing hormone (LH) surge necessary for ovulation. When administered within seventy-two hours of intercourse, Plan B prevented about 85 percent of pregnancies compared with the expected number without treatment. Clearly, LNG—and thus Plan B—is a contraceptive.

But is Plan B an abortifacient? Or, more precisely, can LNG act after fertilization to prevent implantation of the embryo? Two papers purport to present scientific evidence that support an abortifacient effect for Plan B.

In an often-cited study, Kahlenborn et al. reviewed data taken from previously published papers and suggested that Plan B has a post-fertilization effect. The study provides two arguments in support of an abortifacient effect for Plan B. First, its authors argue that there is *theoretical* evidence that suggests that Plan B, as an emer-

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Emergency contraceptive (EC), has an abortifacient effect because ECs adversely affect the implantation process. In support of this claim, they cite studies that showed that oral contraceptives decrease the thickness of the endometrium, the lining of the womb. Since a thinner endometrium has been shown to impede embryo implantation, the authors conclude that Plan B, as an EC, would make implantation difficult, leading to an abortifacient effect. Second, the authors argue that there is statistical evidence that suggests that emergency contraception has a post-fertilization effect. This is found in studies—and there are a handful—that reveal that Plan B taken after ovulation still reduces the expected number of pregnancies. The paper concludes, “These data are highly consistent with the hypothesis that hormonal EC has a post-fertilization effect on the endometrium.”

In a more recent but similar study, Mikolajczyk and Stanford use a computer model to provide statistical evidence that suggests that LNG may have a post-fertilization effect. Again, the authors make their claim because their calculations show that the effectiveness of LNG cannot be explained by attributing the efficacy of the drug to the disruption of ovulation alone. They propose two possible explanations for their findings. First, there could have been an overestimation of the actual effectiveness of the drug or, second, there could be supplementary mechanisms of action for Plan B, including post-fertilization effects. They were unable to distinguish between these two possibilities.

It is important to acknowledge that neither of the studies summarized above demonstrate an abortifacient effect for levonorgestrel. They simply point out that statistically, Plan B taken after ovulation reduces the expected number of pregnancies in a manner that cannot be attributed to its anti-ovulatory effects alone, and that theoretically, Plan B could prevent implantation by adversely affecting the thickness of the endometrium. Together, these point to a possible abortifacient effect for Plan B. However, statistical and theoretical studies do not amount to demonstrative proof.

**Plan B Is Not an Abortifacient:**
**The Case for the Challenge**

Two studies published in the past six months directly challenge the foundations for the claim that Plan B is an abortifacient. First, Novikova et al. question the accuracy of many of the drug studies used for the statistical analyses described above by addressing one problem with efficacy studies of emergency contraception in general: they often are unable to precisely determine the exact relationship between the time of intercourse, the time of Plan B ingestion, and the time of ovulation. In the past, clinical studies that examined the efficacy of emergency contraception relied on the

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Ibid., 467.


women’s self-reporting of their menstrual cycle making calculations of EC efficacy unreliable. In contrast, to complement the woman’s self-report of her menstrual cycle, Novikova et al. included tests to estimate blood hormone levels to calculate the day of ovulation. Significantly, the authors discovered major discrepancies between the women’s self-report of their ovulation date and dating calculations based on hormonal levels in their blood. This led to effectiveness rates for Plan B that were lower than those found in studies where the timing of ovulation was based on the woman’s self-report alone, suggesting that the older studies were quite unreliable. In fact, among the seventeen women who had intercourse in the fertile period of their cycle and took the Plan B after ovulation occurred, the authors could have expected three or four pregnancies. They observed three, suggesting that Plan B was not effective at preventing pregnancy after ovulation. The authors conclude, “These data are supportive of the concept that [LNG] has little or no effect on post-ovulation events but is highly effective when taken before ovulation.” Although the small number of participants in the study prevented the authors from making a definitive statement on the hypothetical post-fertilization effect of Plan B, this paper does suggest that the earlier studies mentioned above may have overestimated the post-ovulatory effectiveness of Plan B.

Next, Lalitkumar et al. question the theoretical basis for Plan B’s abortifacient effect by directly measuring the effect of the drug on the ability of human embryos to implant in the endometrium. They discovered that Plan B does not impair the ability of living human embryos to attach to endometrial tissue grown in a laboratory: the proportion of human embryos that could attach to endometrial tissue exposed to LNG (six of fourteen embryos) was similar to the proportion that could adhere to endometrial tissue that had not been treated with drugs (ten of seventeen embryos). In contrast, of fifteen embryos tested, none could attach to endometrial tissue that had been exposed to mifepristone, the abortifacient commonly known as RU-486. Despite the grave immorality of this experiment, the study shows that Plan B does not affect the ability of human embryos to implant into endometrial tissue, and that the endometrial tissue did not undergo gross changes when exposed to LNG administered after ovulation. The authors conclude that their data “support the view that endometrium exposed to levonorgestrel is still receptive for the human embryo implantation.”

To obtain the most accurate data on both the efficacy and the mode of action of Plan B, larger studies using transvaginal ultrasound in conjunction with the endocrine protocol described by Novikova et al. are needed. However, studies

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9 An earlier study had also found that older studies of contraceptive efficacy were unreliable. See A. Stirling and A. Glasier, “Estimating the Efficacy of Emergency Contraception: How Reliable Are the Data?” Contraception 66.1 (July 2002): 19–22.

10 Novikova et al., “Effects of Levonorgestrel,” 112.


12 Ibid.
of this type are relatively expensive. Nevertheless, the available data cast serious doubt on the statistical claim that Plan B is an abortifacient. Finally, the study by Lalitkumar et al. confirms the earlier findings of Durand et al., who had shown that the endometrial histology of surgically sterilized women taking LNG was indistinguishable from that of controls. Durand et al. had noted, “Of particular importance was the finding that the predecidual changes [in the endometrium] as evaluated by the presence of prominent spiral arteries, which are considered crucial for implantation, were not altered by LNG.” Thus, they had concluded, “peri- and post-ovulatory administration of LNG did not impair corpus luteum function or endometrial morphology.” In the end, despite the theoretical speculation made by some, the direct experimental evidence suggests that Plan B does not adversely affect the thickness of the endometrium and thus would not prevent the implantation of the embryo.

Mounting Evidence

Studies published in the past few months provide mounting evidence that levonorgestrel has little or no effect on post-fertilization events. In other words, given the limitations of scientific certitude, they suggest that Plan B, when administered once, is not an abortifacient. These human studies correlate well with earlier findings in rodents and monkeys that convincingly showed that the postcoital administration of levonorgestrel in amounts several times higher than typical doses given to women does not interfere with the post-fertilization processes required for mammalian embryo implantation. The evidence also addresses what until now has been a nagging, unanswerable question for pharmacologists: Why would levonorgestrel, a progesterone agonist that mimics the effect of progesterone, prevent implantation, when progesterone produced from the corpus luteum immediately after ovulation actually promotes implantation by converting the endometrium to decidua? Answer: It does not.

But what about the manufacturer’s label? Much has been made about the claim made by Barr Pharmaceuticals that Plan B “may also prevent fertilization of a released egg (joining of sperm and egg) or attachment of a fertilized egg to the uterus (implantation).” Labels mean nothing without the scientific data to back up their claims.

\[\text{14Ibid., 232.}\]
\[\text{15Ibid., 227.}\]