Life Insight

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Missouri: The "Clone Me" State?

The campaign to clone human embryos for destructive research has taken some hits this year. Two major journals, *Science* and the *New England Journal of Medicine*, had to admit they printed false claims about progress toward so-called "therapeutic" cloning. Many scientists now admit they simply don't know whether human embryo cloning can work, let alone produce a cell therapy for any disease.

Yet politics can run on its own track, fueled by money and power rather than common sense. Take Amendment 2, a ballot initiative to be placed before the voters of Missouri on November 7. It would give researchers a new *constitutional right* to clone and destroy human embryos – although such activity is punishable as a crime in Canada, Germany, Australia and other nations with much weaker pro-life movements than ours. Many expect the amendment to pass.

The key to success here has been twofold. The first element is money and lots of it. In July the Associated Press reported that supporters had raised $16 million – $15.4 million of it from multi-billionaire James Stowers and his wife. Years ago the Stowers used $2 billion to found the Stowers Institute for Medical Research in Kansas City, which stands to reap enormous benefits if Missouri becomes the cloning capital of the world. One is tempted to say that the man who has everything now wants to buy a state constitution to protect his interests.

The other key is sheer misinformation, spread through the media using those millions of dollars. Voters in the "show-me state" are being shown a fabric of illusions. Let me name three of those illusions.

First, Amendment 2 claims to ban any attempt to "clone a human being." But it defines "clone a human being" as using an already-cloned human embryo to initiate a pregnancy that could result in "the creation of a human fetus" or "the birth of a human being." Since the medical definition of "fetus" begins after the eighth week of development, this creates a large window to clone and grow human embryos and then kill them. The fake ban on "cloning" is really a mandate for abortion by the eighth week.

Second, the amendment says it allows only "stem cell research permitted under federal law." The unwary will think this means real limits, since the federal government does not fund research destroying human embryos. But – surprise! – the amendment defines "permitted under federal law" to mean actions that federal law does not *prohibit* even in the private sector. This means you can do almost anything, since virtually all federal laws against lethal human experimentation deal only with funding. There is no federal law against murdering adults for their stem cells, only state laws. This constitutional amendment would nullify Missouri's state laws, to the extent that they get in the way of "stem cell research."

Third, the web site promoting this "stem cell research and cures initiative" declares that "over 70 diseases and injuries could benefit from stem cell research." In fact patients with over 70 conditions *have* been shown in peer-reviewed journals to benefit from stem cells – but these treatments all use *adult* stem cells, which supporters of Amendment 2 wrongly dismiss as having very limited use. Meanwhile, they themselves talk of "cures" for 70 diseases from cloning and embryonic stem cells, without a scrap of direct evidence for their grandiose claim.

We can all hope that voters in The Show-Me State will realize they are being sold a bill of goods. No one wants to be known as hailing from The Clone-Me State.

--Reprinted from Richard Doerflinger's October 6, 2006 *Life Issues Forum* column.

How NOT to Reduce Abortions and Support Parents

Columnist William Saletan recently endorsed a new Congressional bill which calls for even greater contraceptive access and other measures to reduce the "need" for abortion ("Reducing the Need for Abortion and Supporting Parents Act," H.R. 6067). Mr. Saletan is usually careful with his facts, so it came as a surprise that he asserts: "To lower the abortion rate, we need more contraception" ("Where the Rubber Meets Roe," *Washington Post*, Oct. 1, 2006, at B2).

He illustrates his point with an analogy: "[D]enying that contraceptives reduce your risk of pregnancy is as crazy as denying that an umbrella reduces your risk of getting wet." Well, if at the end of a school day you tell teenagers that the thunderstorm outside will soon pass, but they can try their luck with this stack of umbrellas, many will do so. But some of them will get

soaked when their umbrellas break or slip from their hands in the wind, and all will get a little wet. The problem is that no one gets only "a little" pregnant.

But my point is not to quibble with Mr. Saletan's rhetorical devices. It's his failure to research the subject that troubles me, because he, too, has fallen prey to the Great Contraceptive Fallacy: more contraception reduces unintended pregnancies and abortions. So call me "crazy," Mr. Saletan, but the facts speak for themselves.

Fact 1. Contraceptive use is *already* "virtually universal among women of reproductive age," according to the Centers for Disease Control and Prevention (CDC). The Alan Guttmacher Institute (AGI) reports that 89% of reproductive-age women already are using contraception and 98% have used it in their lifetime. There are still unintended pregnancies and abortions because with typical use, the risk of pregnancy over 12 months is 9% with oral contraceptives and 15% with condoms. (http://womenshealth.about.com/cs/birthcontrol/a/effectivenessbc.htm.)

Fact 2. Contraceptive researchers and social scientists measuring effectiveness in large-scale studies have reached (often reluctantly) the same conclusion: increased availability of contraception, and even emergency contraception, fails to reduce rates of unintended pregnancy and abortion. Studies examining the impact of emergency contraception (EC) are reviewed at http://www.usccb.org/prolife/issues/abortion/FactSheetEC9606.htm. Research in the U.S., Western Europe and China produced remarkable unanimity:

- "No effect on abortion rates was demonstrated with advance provision of EC. … [W]idespread distribution of … EC through health services may not be an effective way to reduce the incidence of unintended pregnancy" (Glasier *et al*., *Contraception* 2004);
- "We did not observe a difference in pregnancy rates. … Previous studies also failed to show significant differences in pregnancy or abortion rates among women with advance provision of EC" (Tina Raine *et al*., *JAMA* 2005);
- "This study adds to the growing literature casting doubt on the increased use of EC as a quick fix for rising abortion rates" (Hu *et al*., *Contraception* 2005);
- "Another commonly held view for which there is no documented evidence is that improving knowledge about and access to Emergency Contraception will reduce the number of teenage pregnancies. … Experience of use so far does not give any evidence of effectiveness" (Williams, *Scottish Council on Human Bioethics*, 2005).

Even AGI's own 2006 "Contraception Counts" report, which ranks states on policies improving contraceptive access as well as on abortion rates, shows *no correlation* between better access to contraception and lower abortion rates.

U.S. researcher Douglas Kirby concludes: "Most studies that have been conducted during the past 20 years have indicated that improving access to contraception did not significantly increase contraceptive use or decrease teen pregnancy" ("Reflections on Two Decades of Research on Teen Sexual Behavior and Pregnancy," 1999 *Journal of School Health* 3:69).

Fact 3. The effectiveness of EC – in the words of prominent EC researcher Anna Glasier – is "unsubstantiated by randomized trials" and "based on rather unreliable data and a great many assumptions" (2004 *Contraception* 69:361-366). Among several recent studies questioning the 89% efficacy claimed by Plan B's manufacturer, one concludes that the "best available estimate" of Plan B effectiveness is only 72% (Stanford and Mikolajczyk, "Methodological review of the effectiveness of emergency contraception," 2005 *Current Women's Health Reviews* 1:119-129). These authors note that the higher estimate of effectiveness is based on two small samples of women: British women using natural family planning (NFP) in the 1960s and women in North Carolina in the 1980s who were trying to conceive. The fertility rate, especially among the British NFP users, was very likely higher than among women in a key World Health Organization study comparing effectiveness of two methods of emergency contraception. This error led to overestimating EC effectiveness. In addition, Stanford notes, "the methods used for comparison do not take into account the normal variation in the timing of ovulation within the menstrual cycle."

Fact 4. Saletan dismisses the "argument" made by opponents" of contraception that "birth control pills, like morning-after pills, can block implantation of an embryo. But there's no evidence that this has ever happened." There's plenty of evidence if one knows where to look.

A leading medical textbook on embryology, Moore and Persaud's *The Developing Human: Clinically Oriented Embryology* (6th ed.), explains that "morning after pills" taken "within 72 hours after sexual intercourse usually prevent implantation of the blastocyst. … *These hormones prevent implantation, not fertilization.* Consequently, they should not be called contraceptive pills. … 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).

The leading textbook on obstetrics, *Williams Obstetrics*, provides a detailed overview of the many female hormones involved in the fertility cycle and their effect on the five major stages of the endometrial cycle. (See 19th ed., pp. 82-93, edited by F. Gary Cunningham, M.D. *et al*., 1996.) The editors describe the miraculous process by which the uterus is prepared each month to be a safe haven for a developing embryo as "unique and astonishing" (at 82). They note that "two thirds of the entire endometrium is shed and regenerated more than 450 times, on average, in the life of most women. … [If] the doubling time of endometrial growth experienced from the 5th to the 20th day of the ovarian cycle were maintained for 1 year, the weight of the endometrium would approach 1 ton!" (at 83) This astonishing process can be disrupted, thwarting the implantation of a newly conceived embryo, by counteracting certain hormones. The most commonly used contraceptive pill is a combination of estrogen and progestin. Here's what *Williams Obstetrics* has to say about its mode of action:

"***Estrogen*** *alone in sufficient dose will inhibit ovulation. … Implantation also is likely inhibited by altering normal endometrial maturation. Although estrogen accelerates ovum transport, progestins cause slowing; thus their possible role in altered tubal and uterine motility is unclear.*

"*… Similar to estrogens,* ***progestins*** *produce an endometrium that is unfavorable to blastocyst implantation. Finally, progestins also can inhibit ovulation. …*

"*The net or* ***combined effect of estrogen and progestin*** *with respect to contraception is extremely effective ovulation suppression* [assuming perfect use! --ed.], *sperm penetration blockage by cervical mucus, and unfavorable endometrium for implantation if the first two mechanisms fail"* (at 1322-23).

Of **progestin-only "mini-pills**" using the ingredient in the Plan B emergency "contraceptive"), *Williams Obstetrics* explains:

"They have not achieved wide-spread popularity because of a much-higher incidence of irregular bleeding and a higher pregnancy rate. As with all forms of progestin-only contraception, when failure results in pregnancy, there is also an increased risk that it is ectopic. … These agents, when used alone, impair fertility without always inhibiting ovulation. This likely results from inducing cervical mucus that impedes sperm penetration and from altering endometrial maturation sufficiently to thwart successful blastocyst implantation. … [If] menses are not disturbed, or only minimally disturbed, ovulation is likely not suppressed, and the pregnancy risk is greater. Actual pregnancy rates with progestin-only pills range from 1.1 to 9.6 pregnancies per 100 women in the first year of use" (at 1333-34).

Robert A. Hatcher, MD, principal editor of the definitive guide *Contraceptive Technology* (18th ed., 2004), has also produced a 171-page *Pocket Guide to Managing Contraception*, available in print and on-line at www.managingcontraception.com. The mechanisms of action of various forms of contraception are described briefly:

- **Combined (Estrogen & Progestin) Oral Contraceptives** (COCs): "Suppresses ovulation. … Also causes thickening of cervical mucus, which blocks sperm penetration and entry into the upper reproductive tract. Thin, asynchronous endometrium inhibits implantation. Tubal motility slowed" (at 101).
- The mechanisms of **progestin-only contraceptive pills** (POPs) are described this way: "Thickens cervical mucus to prevent sperm entry into upper reproductive tract (major mechanism)." *N.B. That action requires 12-24 hours, while sperm in fertile mucus need only 6 hours to be capacitated, that is, capable of fertilizing an ovum.* The description continues: "Effect short-lived–requires punctual dosing. Other mechanisms include ovulation suppression (in about 50% of cycles), thin, atrophic endometrium which inhibits implantation; and slowed diminished mobility. Some POPs in Europe suppress ovulation more than the levonorgestrel, norgestrel and norethindrone pills used in the USA" (at 124).

By "punctual dosing," the authors do mean PUNCTUAL. If a woman is only 3 hours late in taking her daily dose, she is advised to use "backup contraception for 48 hours" and "consider using emergency contraception if [she had] sex in past 3-5 days" (at 127).

- **Depo-Provera injection**. The mechanisms are as follows: "Suppresses ovulation by inhibiting LH and FSH surge, thickens cervical mucus blocking sperm entry into female upper reproductive tract, slows tubal and endometrial mobility, and causes thinning of the endometrium" (at 128).
- **Implanon** (implanted rod with progestin now replacing **Norplant**, which was withdrawn from the market after a successful class action suit based on risks and unpleasant side effects). Its mechanisms: "… thick cervical mucus prevents normal sperm transport"; "inhibition of ovulation"; and "atrophic endometrium" which will inhibit implantation.

A major study by Marta Durand *et al.* (2005 *Contraception* 71:451-457) confirms the frequent failure of levonorgestrel (LNG) to prevent ovulation, when administered as an emergency contraceptive before the LH surge. Instead LNG interferes with the

function of the corpus luteum in preparing the endometrium for implantation.

Durand *et al*. studied levels of glycodelin in the blood and endometrium of women who had been given two doses of LNG 12 hours apart. One group received LNG 3-4 days before the luteinizing hormone (LH) surge. LH triggers both the release of an ovum from the dominant follicle, and the conversion of the residual follicle into a corpus luteum producing progesterone to prepare the endometrium for a possible implantation. LH is necessary to maintain luteal function for the first two weeks. The second group received LNG at the time of the rise in LH, and the third group 48 hours afterward.

In the first group *only*, LNG caused a rise in glycodelin 4 days earlier than normal which produces "antifertility activity" during the fertile window, and also causes "deleterious effects on [progesterone] production by the corpus luteum" (at 455). Reduced levels of glycodelin in the endometrium at the time implantation should occur can inhibit implantation and result in an embryo's death.

Chang-hai He *et al*. compared the effects of 0.75 mg LNG tablets (Postinor and a Chinese version). The pills were taken by women within 8 hours of every act of unprotected intercourse, and again 24 hours later, during the week before and week after their estimated day of ovulation. (1991 *Int J Gynecol Obstet* 36:43-48). Ovulation occurred in all but 14.4% of cycles. He *et al*. state: "Earlier we have reported that ovulation was not affected in four out of six women given 0.75 mg levonorgestrel daily for 7 days starting on day 11 of their cycle. Thus, it would appear that the mechanism of action of postcoitally administered levonorgestrel is not inhibition of ovulation in most women but presumably involves an effect on the endometrium, rendering it unsuitable for implantation" (at 47).

If Mr. Saletan does not believe the evidence published in science journals and medical textbooks, perhaps he would believe in the post-fertilization effect of contraceptives if he reads about it in *The New York Times*.

Coincidentally, that newspaper allowed a statement by James Trussell (described therein as "one of the world's leading experts on contraception") to appear on its pages earlier this year, maintaining that *all* hormonal methods of contraception involve post-fertilization (i.e., *abortifacient*) effects. The *Times* censor must have been asleep at the switch when he allowed this statement by Trussell to slip past him: **"The evidence is about the same for all hormonal methods of contraception. We can't rule out a post-fertility effect for Plan B, and the same is true for the birth control pill"** (quoted in R. Shorto, "Contra-Contraception," *The New York Times*, May 7, 2006).

There are other reasons not to support H.R. 6067 which we'll save for a later day. But it should be clear that more contraception does not decrease unintended pregnancies and abortions. And it should be clear what effectiveness contraception does have may depend on post-fertilization, *abortifacient* effects.

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