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Birth-control methods which can cause abortion

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1. Introduction

Abortion, contraception and sterilisation

Substances and devices are abortifacient if they endanger the fertilised embryo by negatively influencing the endometrium¹ or the hormone balance involved in implantation. Contraception stops fertilisation from occurring. Sterilisation is any procedure by which an individual is made incapable of reproduction².

Although abortion, contraception and sterilisation are separately identifiable, this does not mean that a particular substance or device will perform only one of these functions. While barriers such as condoms and diaphragms would appear to be solely contraceptive, many birth control methods can do more than one thing. Most methods which prevent or delay ovulation also thicken cervical mucus and render the endometrium unreceptive to an embryo seeking to implant. They are thus contraceptive and abortifacient.

Until recently, the British pro-life movement has concentrated on opposing abortions performed under the 1967 Abortion Act. These procedures tend to take place at least six weeks after conception. However, many pro-life organisations, including SPUC, are now concentrating more attention on birth-control devices which cause earlier abortions.

The popularity of contraceptive pills

Pills became the most popular type of contraceptive soon after they were launched. The table below summarises English family-planning clinics' reports³ on contraceptive choice for 2000-2001.

method	usage
pills	42%
male condoms	35%
chemicals	0.4%
implants	0.4%
female condoms	0.2%
rhythm method	0.1%

Data from an omnibus survey shows that, in 1999, over half of women aged 16-49 reported using a non-surgical method of contraception. The survey data also shows that about 12% of women aged 16-49, an estimated 1.4 million women in that age group, had been sterilised. NHS hospital data show that about 55,000 women were sterilised in England in 1993 but this figure has fallen nearly every year since to about 41,000 in 1999.

¹ the lining of the womb

² Dorland's Illustrated Medical Dictionary 27th Edition, WB Saunders Company, Philadelphia 1988

³ NHS Contraceptive Services, England: 2000-01, www.doh.gov.uk/public/sb0127.htm

The Office for National Statistics (ONS) survey data shows that in the year 2008/09 - Approximately 25% of women aged 16-49, an estimated 3.5 million women (3.0 million in England), were using contraceptive pills. Of women attending National Health Service (NHS) community contraceptive clinics in 2009/10, oral contraceptives were the most common form of contraceptive used, accounting for 44% of women (392,000)⁴.

Although contraceptive pills are popular, there are still calls from various quarters for new types of birth-control.

Demands for new types of birth-control

Women who use birth-control want to know that they will not be at increased risk of thromboembolism, breast cancer, or infertility, and that they can regain their fertility at any time.

Feminist activists have their own aims for contraceptive technology.

A woman-centred contraceptive research agenda was the focus of a 1996 Institute of Medicine Committee report. Priority was given to research on methods that act as a chemical or physical barrier to conception and to STDs including HIV; to menses inducers and once-per-month methods; and to male contraceptive methods⁵.

The population control movement supports contraceptives which give the birth-control provider greater influence over the users' fertility. Such techniques include long-acting injections, IUDs and implants.

The future direction of birth-control

Researchers seek methods which are longer-acting, involve a lower dose, are more specific and have fewer side-effects.

Manufacturers are developing formulations with less oestrogen and more progesterone. While a reduction in quantity of oestrogen could reduce side-effects and menstrual disruption, progesterone changes the lining of the womb to prevent implantation and thus can cause an early abortion. There is research into menstrual regulation formulas, which would only be taken if a period was late.

Baird and Glasier predict⁶ that future developments will involve “selective modulators of hormone receptors” which will replace the currently available oestrogens and progestins “in order to avoid their risks, particularly venous thrombosis, while also reducing the incidence

⁴ NHS Contraceptive Services: England, 2009/10; Pg 8.

http://www.ic.nhs.uk/webfiles/publications/003_Health_Lifestyles/nhscontra0910/NHS_Contraceptive_Services_England_2009_10.pdf

⁵ Joseph L. (1999) “Pushing the frontier of science: reflections on an Institute of Medicine Study”, *International Journal of Gynaecology and Obstetrics* 1999 Dec;67 Suppl2:S93-9

⁶ Baird, D. and Glasier, A. (1999) Science, medicine and the future: contraception. *BMJ*; Vol 319; Pp:969-973.

of common diseases such as breast cancer". Baird and Glasier also predict the development of organ-specific drugs to avoid whole-body effects.

Birth-control developers are looking for new approaches which do not involve a regular regime on the part of the woman. These techniques, which are particularly intended for families in the developing world and for adolescents, often work by inducing abortions.

2. The development of contraceptive substances⁷

History

In the early 1900s scientists noticed that the continuing presence of the corpus luteum⁸ stopped further egg-release. In 1921 the Austrian Dr Haberlandt suggested that extracts from the ovaries of pregnant animals might be used as oral contraceptives. Pharmaceutical companies, fearing controversy, were reluctant to use these hormones for contraception. In the early 1950s Margaret Sanger and a wealthy friend provided encouragement and financial resources to researchers, so that the pill was eventually marketed. Margaret Sanger's motivation for this activism was clearly eugenic⁹.

Trials began in Puerto Rico in 1956 and were highly successful until chemists removed oestrogen from the pills, thinking it an impurity. Irregular bleeding and accidental pregnancies then began to occur. Researchers realised that oestrogen was necessary for effectiveness and control of the cycle, and the combined pill was created. The US Food and Drug Administration released Enavid-10, the first combination oestrogen and progestogen birth control pill, in 1960. Enavid-10 delivered as much oestrogen in a day as is now taken in a week and delivered as much progestogen in a day as is now taken in a month in one brand of pill.

Since the mid-1980s, developments have come mainly from research into hormonal methods, including:

- new delivery systems such as implants and hormone releasing intrauterine devices
- better progestogens
- lower doses of oestrogen¹⁰.

⁷ Mostly taken from Guillebaud, J. (1997), *The Pill and other forms of hormonal contraception*, Fifth Edition, Oxford University Press. Dr Guillebaud was the first practising gynaecologist to be given a personal chair in family planning and reproductive health, which he holds at University College, London.

⁸ During ovulation the ovarian follicle ruptures and releases its oocyte. The remains of the follicle form the corpus luteum (yellow body) which contains yellow-pigmented luteal cells which release progesterone and oestrogens. If fertilisation does not occur, the corpus luteum degenerates and a new cycle ensues. If fertilisation occurs, the corpus luteum is prevented from degeneration by human chorionic gonadotrophin released by the embryo and continues to grow and secrete progesterone till the end of the fourth month, after which it slowly regresses. The corpus luteum can reach between one third and one half the size of the ovary by the end of the third month.

⁹ Visit <http://www.all.org/abac/eugen03.htm> to read more about Margaret Sanger.

¹⁰ Baird, D. and Glasier, A. (1999) Science, medicine and the future: *Contraception*. *BMJ*; Vol 319; Pp:969-973.

Progestogen methods are more likely to be abortifacient.

3. How abortifacient birth-control works

Birth-control methods which comprise combinations of oestrogens and progestins can stop the menstrual cycles' hormone changes and thus prevent the maturing of follicles and ovulation. However, these drugs also have other mechanisms which reduce the chances of conception occurring if an egg is released. If conception does occur, there may be further effects such as the prevention of implantation into the uterus which causes an early abortion.

Ovulation, fertilisation and implantation depend on the secretion of progesterone by the ovary at the right time. Progesterone is responsible for the transcription of endometrial gene products which are crucial for implantation. Too high a proportion of progesterone in relation to oestrogen increases the likelihood of an abortifacient effect.

Embryo implantation involves a series of interdependent, hormonally-controlled factors, with the embryo a dynamic participant¹¹. Oestrogen and progesterone regulate these factors both directly and indirectly. It is reasonable to expect that hormones found in birth control methods would adversely affect various implantation factors.

The hormone-receptor interaction is one of the foundations on which the birth control pill was developed. Synthetic hormones act on the same receptors as natural ones and artificial female hormones can mimic or disrupt normal cyclical patterns.

4. Birth control products which are available or under development and which have abortifacient mechanisms

Combined oral contraceptives

These pills contain oestrogen and progestin and are taken daily. They:

- suppress ovulation
- thicken cervical mucus
- change the endometrium making implantation of the newly-conceived embryo less likely and thus can induce an early abortion
- reduce sperm transportation in fallopian tubes.

¹¹ Wilks, J. (2000) A Consumer's Guide to the Pill and Other Drugs (3rd edition). Mandaluyong, Philippines: National Book Store, Inc.

Progestin-only pills

These are taken daily and contain no oestrogen. They do not rely solely on stopping egg release and women who take such pills have natural periods. The pills:

- suppress ovulation
- thicken cervical mucus
- change the endometrium making implantation less likely and thus can induce an early abortion
- reduce sperm transportation in fallopian tubes.

Progestin-only pills have been fancifully described as a “barrier method of family planning which is taken by mouth”¹². The sperm-barrier effect on the cervical mucus reaches its maximum between four and five hours after taking the pill.

Combined injectable contraceptives

These monthly injections of oestrogen and progestin include products such as Cyclofem, Novafem, Mesigyna, Lunelle and Cyclo-Provera. They:

- suppress ovulation
- thicken cervical mucus
- change the endometrium making implantation of the embryo less likely and thus can induce an early abortion
- reduce sperm transportation in fallopian tubes.

Intra-uterine devices (IUDs)

These flexible metal and/or plastic devices are inserted in the uterine cavity. IUDs can be:

- copper-releasing
- inert
- progestin-releasing
- levonorgestrel-releasing

Copper-releasing IUDs can:

- interfere with the ability of sperm to pass through the uterine cavity
- interfere with fertilisation in the fallopian tube
- cause local inflammation in the uterine lining, inhibiting implantation if conception has occurred and thus can induce an early abortion.

Progestin-releasing IUDs additionally:

¹² Guillebaud, J. *op.cit.*

- thicken cervical mucus thus interfering with sperm movement
- produce endometrial changes which may interfere with implantation of the newly-conceived embryo if fertilisation has occurred and thus can induce an early abortion.

Levonorgestrel-releasing IUDs such as Mirena rely more on preventing implantation than devices which were available before them¹³. Levonorgestrel IUDs are associated with irregular menstrual bleeding. Studies have shown that amenorrhoea (the absence of a menstrual period) occurs in 35% of women after 2 years of use. The Mirena IUD was approved by the USA Food and Drug Administration (FDA) in 2000 as a contraceptive device. In recent years it has been approved by the FDA as a treatment for heavy menstrual bleeding in women who use intrauterine contraception¹⁴. Levonorgestrel IUDs are also known to have adverse hormonal effects such as headache, acne, breast tension and functional ovarian cysts¹⁵.

Despite major health problems with IUDs such as the Dalkon Shield, it is claimed that some devices have health benefits since they allow the ovaries to continue releasing oestrogen. Some are also said to combat anaemia¹⁶.

Implants

The original Norplant was first developed in 1983 in Finland. It was an implant that consisted of six small, flexible capsules filled with levonorgestrel, a synthetic progestin, which are put under the skin of the upper arm through minor surgery. Norplant:

- suppresses ovulation
- thickens cervical mucus
- changes the endometrium making implantation of the newly-conceived embryo less likely and thus can induce an early abortion
- reduces sperm transportation in fallopian tubes.

Norplant II (Norplant-2, Jadelle) was subsequently developed and FDA approved in 1996¹⁷. It consists of just two small capsules each containing 75 mg of levonorgestrel in a polymer matrix, instead of six capsules. Also developed and widely used is Implanon which is a single implant injected through a wide bore needle. It is inserted sub-dermally just beneath the skin on the inner side of a woman's upper arm. It works by continually releasing low doses of progestin (etonogestrel) for up to three years. Implanon was FDA approved in 2006¹⁸. The FDA warns that there are complications associated with the insertion of Implanon¹⁹.

¹³ Guillebaud, op. cit.

¹⁴ ¹⁴ <http://www.fda.gov/newsevents/newsroom/pressannouncements/2009/ucm184747.htm>

¹⁵ [No authors listed] (2009) Intrauterine devices: an effective alternative to oral hormonal contraception. *Prescrire Int.* Vol. 18(101):125-30.

¹⁶ *ibid*, page 234

¹⁷ <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm?fuseaction=SearchDrugDetails>

¹⁸ <http://www.medicalnewstoday.com/releases/47600.php>

¹⁹ <http://www.fda.gov/Safety/MedWatch/SafetyInformation/Safety-RelatedDrugLabelingChanges/ucm133061.htm>

It is worth noting that contraceptive implants can be fitted and then forgotten about for long periods. Where birth-control is socially funded, they offer cost-savings “especially when providers are also responsible for the cost of unplanned pregnancies”²⁰.

Progestin-only injectable contraceptives

These progestin injections are given every two or three months and they:

- suppress ovulation
- thicken cervical mucus
- change the endometrium making implantation of the newly-conceived embryo less likely and thus causing an early abortion
- reduce sperm transportation in fallopian tubes.

Depo-Provera uses depot medroxyprogesterone acetate (DMPA) in a 150mg dose into a muscle (usually the buttocks) every 12 weeks. The FDA warns that Depo-Provera can cause loss in bone mineral density and thus “should be used as a long-term birth control method (e.g. longer than 2 years) only if other birth control methods are inadequate”²¹

Noristerat is an injection of a norethisterone ester given every eight weeks and available in the UK^{22 23}.

Emergency Contraceptives

Emergency contraceptives are often referred to as the “morning after pill”. There are two regimes of administration that involve both progestin and estrogen combined or progestin alone – for example, the major regimes are the *Yuzpe* regime, which consists of ethynyl estradiol (EE) in combination with Levonorgestrel (LNG), and LNG alone (Also known as “Plan B”). There is a spectrum of possible modes of action for emergency contraceptives. They could:

- Suppress follicular development
- Suppress ovulation
- Reduce sperm migration
- Prevent fertilisation and zygote development
- Suppress embryo transport
- Change the endometrium making implantation less likely and thus can induce an early abortion

²⁰ Bromham, D. (1996) “Contraceptive implants”, *BMJ* 1996; 312: 1555-1556 (22 June)

²¹<http://www.fda.gov/Safety/MedWatch/SafetyInformation/SafetyAlertsforHumanMedicalProducts/ucm154784.htm>

²² also known as Norigest or Nur-Isterate

²³<http://www.nhs.uk/medicine-guides/pages/MedicineOverview.aspx?condition=Contraception&medicine=Norethisterone%20Enantate&preparationNorethisterone%20200mg/1ml%20solution%20for%20injection%20ampoules>

The literature over the last few years indicates that there is still debate about whether emergency contraceptives are abortifacient. Some claim that a post fertilisation effect (particularly alterations to endometrial receptivity) are the likely mechanism of action^{24 25 26}. However, in most of the recent literature, claims are made that emergency contraceptives act prior to fertilisation by blocking or delaying ovulation, rather than acting after fertilisation^{27 28 29}. Research by Durand et al (2010) has also challenged the view that emergency contraceptives affect endometrial function³⁰.

Mikolajczyk and Stanford have argued in their analysis of the mechanism of action of emergency contraceptives, that there will always be a possibility of post fertilisation effects³¹. In a recent review of the literature by Trussell and Jordan³², they conclude that “it is unlikely that this question can ever be unequivocally answered, and we therefore cannot conclude that ECPs [Emergency Contraceptive Pills] never prevent pregnancy after fertilisation”. Bastianelli *et al*'s review also states that “for those who situate the onset of pregnancy at fertilization—there is no proof that HECs [Human Emergency Contraceptives] always act before this crucial event”³³.

Recently, a new emergency contraceptive has been released onto the market – Ulipristal Acetate. It was approved by the US Food and Drug Administration in August 2010, and has been launched in the USA as “Ella” (Watson Pharmaceuticals Inc.)³⁴ and “ellaOne” in the European Union. It is claimed to be effective for up to 120 hours (five days) after unprotected sexual intercourse³⁵. Ulipristal Acetate works by binding to progesterone receptors to inhibit the effect of progesterone, thus suppressing or delaying ovulation, as well as decreasing endometrial thickness and receptivity. These effects vary according to the timing of drug administration during the menstrual cycle³⁶.

Ulipristal Acetate primarily works by inhibition and delaying ovulation and appears to be significantly more effective in this than other forms of emergency contraception³⁷. However,

²⁴ Grow DR, Iromloo K. (2006) Oral contraceptives maintain a very thin endometrium before operative hysteroscopy. *Fertil Steril*. 2006 Jan;85(1):204-7.

²⁵ Maia H Jr et al (2008) Effect of oral contraceptives on vascular endothelial growth factor, Cox-2 and aromatase expression in the endometrium of uteri affected by myomas and associated pathologies. *Contraception*. 2008 Dec;78(6):479-85. Epub 2008 Aug 9.

²⁶ Meng et al (2010) Effects of oral and vaginal administration of levonorgestrel emergency contraception on markers of endometrial receptivity. *Hum Reprod*. 2010 Apr;25(4):874-83. Epub 2010 Feb 6.

²⁷ Suárez, V.J. et al (2010) Effect of levonorgestrel in the ovulation, endometrium, and spermatozoa for emergency oral contraception. *Rev Peru Med Exp Salud Publica*. 27(2):222-30.

²⁸ Medard LM, Ostrowska L. (2010) Hormonal (levonorgestrel) emergency contraception--effectiveness and mechanism of action. *Ginekol Pol*. 81(7):532-6.

²⁹ Gemzell-Danielsson K. (2010) Mechanism of action of emergency contraception. *Contraception*. 82(5):404-9.

³⁰ Durand et al., (2010) “Hormonal Evaluation and Midcycle Detection of Intrauterine Glycodelin in Women Treated with Levonorgestrel as in Emergency Contraception,” *Contraception* 82: 526-533.

³¹ Mikolajczyk & Stanford (2006) Levonorgestrel emergency contraception: a joint analysis of effectiveness and mechanism of action. *Fertility and Sterility*, vol 88 no. 3 pp 565-71.

³² Trussell J & Jordan B (2006) Mechanism of Action of Emergency Contraceptive Pills. *Contraception*, 74:87.

³³ Bastianelli *et al* (2008) Emergency contraception: A review. *The European Journal of Contraception and Reproductive Health Care* March 2008;13(1):11.

³⁴ Fine P.M. (2011) Update on emergency contraception. *Adv Ther*. 28(2):87-90.

³⁵ Gemzell-Danielsson K, Meng CX. (2010) Emergency contraception: potential role of ulipristal acetate. *Int J Womens Health*. 9;2:53-61.

³⁶ Kim, A. (2011) Ulipristal Acetate (ella): A Selective Progesterone Receptor Modulator For Emergency Contraception. *P T*. Vol. 36(6):325-31.

³⁷ McKeage, K., Croxtall J.D. (2011) Ulipristal acetate: a review of its use in emergency contraception. *Drugs*. Vol. 7;71(7):935-45.

it is structurally similar to Mifepristone (RU-486) and several lines of evidence suggest that it can also work via a post-fertilization mechanism of action which would thus be an abortifacient mechanism of action³⁸. Ulipristal Acetate is marketed as an emergency contraceptive, however, the possibility of its use as an abortifacient means that it can only be thought of as an “emergency contraceptive” if it is taken within the five day period after intercourse.

Despite all of this contention, it seems that there is a growing body of consensus that emergency contraception (with the exception of Ulipristal Acetate) can be regarded as not being abortifacient due to the overwhelming data affirming pre-ovulatory mechanisms of action, and the lack of data demonstrating a post-ovulatory mechanism of action. The suggestion that there is a post-ovulatory and thus abortifacient mechanism of action of emergency contraceptives has no positively supporting data. This is why the International Federation of Gynecology & Obstetrics (FIGO) and the International Consortium for Emergency Contraception (ICEC) have released a document stating that: “inhibition or delay of ovulation is LNG ECPs principal and possibly only mechanism of action” and “review of the evidence suggests that LNG ECPs cannot prevent implantation of a fertilized egg”³⁹.

In order to put all of this contention into perspective, it is worth noting what Rev. Austriaco has consistently said (in a number of publications^{40 41}) with regard to this debate.

“I claim that the cumulative scientific data from numerous labs using a diverse range of scientific approaches and protocols – especially the relatively recent finding that Plan B does not alter the postovulatory hormonal profile of a woman who takes the drug prior to her LH surge – together makes a strong case for the proposal that it is very unlikely that Plan B has a post-fertilization mechanism of action... But is this scientific certitude enough for moral certitude?... in my view, moral certitude includes that certitude that allows the acting person to act even when he may think that it is possible but unlikely that he is mistaken”⁴².

This point is relevant and worth considering, particularly with regard to whether one ought to administer emergency contraceptives to rape victims.

RU486 (also marketed as Mifepristone)

Research was undertaken on mifepristone to see if it could be developed as an oral contraceptive that could be taken either daily or weekly. Daily doses of between 0.5mg and 1mg and a weekly dose of 5mg mifepristone produced changes in the lining of the womb that

³⁸ Keenan, J. A. (2011) Ulipristal acetate: Contraceptive or Contragestive? *Ann Pharmacother.* Vol 45(6):813-5.

³⁹ International Federation of Gynecology & Obstetrics (FIGO) and the International Consortium for Emergency Contraception, “March 2011: Mechanism of Action – How Do Levonorgestrel only Emergency Contraceptive Pills (LNG ECPs) Prevent Pregnancy?” Available at www.cecinfo.org/UserFiles/File/MOA_FINAL_2011_ENG.pdf. Last accessed on September 2nd, 2011.

⁴⁰ Austriaco, N. P. G. (2007) “Is Plan B Abortifacient? A Critical Look at the Scientific Evidence,” *The National Catholic Bioethics Quarterly* 7, no. 4 (Winter 2007): 707.

⁴¹ Austriaco, N. P. G. (2008) “Colloquy: More on Plan B — Fr. Austriaco Replies,” *The National Catholic Bioethics Quarterly* 8, no. 3 (Winter 2008): 421-25.

⁴² Austriaco, N. P. G. (Forthcoming) *Scientific Certitude, Moral Certitude, and Plan B.*

could potentially prevent implantation of a fertilised egg. Encouraged by these findings, the World Health Organisation (WHO) conducted studies in the late 1990s to determine whether these doses would be effective in preventing pregnancy. Interim results indicated that dosage levels that do not disrupt the menstrual cycle apparently do not produce a reliable contraceptive effect. Since the aim of these studies was to find out if mifepristone could be used for contraception without disturbance of the menstrual cycle, these results were considered disappointing and hence research in this area was not continued⁴³.

The recent literature indicates that moderate doses that range from 10-50mg have been reported to be most effective and superior to other hormonal emergency contraceptives^{44 45}. Some researchers have claimed that when taken in moderate doses, mifepristone functions primarily by acting on ovarian function^{46 47}. However, this does not necessarily exclude the possibility of a post fertilization abortive effect associated with preventing implantation.

Antigestogens might also be used for 'once a month' pills if they are given in the early luteal phase of the menstrual cycle so that the formation of a secretory endometrium is retarded without affecting the regular pattern of menstruation. A once-a-month pill that prevented ovulation or implantation would be welcomed by many women from various countries and cultures⁴⁸. In contrast, only a minority of women would be prepared to use a pill taken around the time of expected menses, when implantation of the embryo would already have occurred⁴⁹.

Inducer of missed period

It has also been proposed that mifepristone could be taken only if the menses was overdue ("contragestion"). An inducer of a missed menses acts by disrupting an implanted embryo and induces a very early abortion. A pilot study supported by the World Health Organisation (WHO) reported very few ongoing pregnancies in women given a combination of 600 mg mifepristone and 1mg gemeprost within 10 days of expected menses⁵⁰.

Transdermal contraceptive patches

⁴³ World Health Organisation (1997), "Reproductive Health Research: the new directions" Biennial Report 1996-1997 < www.who.int/hrp/br/1996-97/2.html >

⁴⁴ Cheng L, et al. (2008) Interventions for emergency contraception. *Cochrane Database Syst Rev.* 16;(2):CD001324.

⁴⁵ Jin , J. Et al (2005) Comparison of three single doses of mifepristone as emergency contraception: a randomised controlled trial. *Aust N Z J Obstet Gynaecol.* 45(6):489-94.

⁴⁶ Gemzell-Danielsson K, Mandl I, Marions L. (2003) Mechanisms of action of mifepristone when used for emergency contraception. *Contraception.* 2003 Dec;68(6):471-6.

⁴⁷ Sarkar (2005) The potential of mifepristone (RU-486) as an emergency contraceptive drug. *Acta Obstet Gynecol Scand.* 2005 Apr;84(4):309-16.

⁴⁸ Rimmer et al (1999) Do women want a once a month pill? *Hum Reprod.* 7: 608-611.

⁴⁹ Baird & Glasier *op.cit.*

⁵⁰ WHO (1995) Menstrual regulation by mifepristone plus prostaglandin – results from a multicentre trial. *Hum Reprod;* 10: 308-315.

Contraceptive patches are being promoted because of the potential for contraceptive failure of the pill in everyday use, particularly among young women. Ortho-Evra (or Evra in the UK) are contraceptive patches that are applied weekly. They have a low level combination of oestrogen and progesterone and, thus, similar mechanisms to the oral contraceptive pill⁵¹. The Food and Drug Administration approved them for use in the USA in 2001. However, since then there have been reports of the possible increased risk of venous thromboembolism (VTE) in women who use Ortho-Evra⁵².

Injectable levonorgestrel

Research is taking place on three-monthly 10mg injections of levonorgestrel butanoate. The low dose would:

- expose women to fewer synthetic hormones than depot–medroxyprogesterone acetate (DMPA)⁵³
- result in less suppression of the ovaries so that fewer women would get amenorrhoea⁵⁴.

It is likely that injectable levonorgestrel will have a greater effect on the endometrium thus preventing implantation of the newly-conceived embryo.

Contraceptive vaccines

Scientists have been seeking since the 1970s to use the body's immune system to block conception and terminate pregnancies⁵⁵. The work is considered useful in population control strategies. In China, research on contraceptive vaccines is considered to “contribute to poverty alleviation and improvement of every aspect of human life and sustainable development”⁵⁶. These vaccines are not yet in widespread use.

One contraceptive vaccine has been designed to cause the egg to reject the sperm and is therefore not abortifacient. Abortifacient vaccines include:

- anti-hCG
- trophoblastic antigen.

⁵¹ <http://www.nhs.uk/conditions/Contraceptive-patch/Pages/Introduction.aspx>

⁵² <http://www.fda.gov/Drugs/DrugSafety/PostmarketDrugSafetyInformationforPatientsandProviders/ucm110403.htm>

⁵³ see 4.6 above

⁵⁴ WHO, *op.cit.*

⁵⁵ Lawrence Roberge (1994) “Abortifacient vaccines: technological update, hazards, and pro-life appraisal”, <http://pages.morning-after-pill.com/lroberg/vaccine.htm>26 “Research and development of contraceptive vaccine”, <http://sedac.ciesin.org/china/policy/acca21/218-1.html>

⁵⁶ “Research and development of contraceptive vaccine”
<<http://sedac.ciesin.org/china/policy/acca21/218-1.html>>

Anti-hCG

The anti-hCG vaccine counteracts the natural effects of the human chorionic gonadotropin (hCG) hormone. Embryos produce hCG to signal maintenance of the corpus luteum which provides progesterone and oestrogen which, in turn, maintain the endometrium rather than allowing a period to occur. An anti-hCG vaccine was shown to produce antibodies which inactivated hCG, prevented retention of the corpus luteum, and brought about normal menses⁵⁷ and an early abortion.

In recent years the anti-hCG vaccine was the first vaccine to undergo Phase I and II clinical trials in humans. It is currently in the clinical trial stages but it is foreseeable that it will become available in the future⁵⁸.

Trophoblastic antigen

The cells around the early embryo which form the outer layer of the trophoblast help the embryo implant and later form the placenta. The vaccine trains the woman's body to treat a protein on the trophoblast as foreign and to mount an immune response against it, destroying the embryo before implantation through an early abortion. However, this is only a theoretical concept for contraception that is still in the very early stages of basic research^{59 60}.

5. The birth-control debate

Reasons for supporting and providing birth-control

Those who advocate and supply birth control can do so because of the profit to be made from pharmaceutical sales. Some have concerns about human population levels and others have an ideological belief in the importance of reproductive choice for women. There is also a wish to prevent teenage pregnancy.

⁵⁷ Pal R. "Absence of corpus luteum rescue by chorionic gonadotropin in women immunized with a contraceptive vaccine", *Fertility and Sterility* 76 (2): 332-336, 2001.

⁵⁸ Naz, R. K. Et al (2005) Recent advances in contraceptive vaccine development: a mini-review. *Hum Reprod.* 20(12):3271-83.

⁵⁹ Zhang, Y. C. et al (2007) Contraceptive effect of fusogenic trophoblast surface epitopes. *Acta Academiae Medicinae Militaris Tertiae*.12.

⁶⁰ Frank, H. G. et al (2005) Evaluation of fusogenic trophoblast surface epitopes as targets for immune contraception. *Contraception*.71(4):282-93.

Commercial interests which were involved in the development of contraceptives needed to bury bad news about contraceptive pills. A letter to the editor of the British Medical Journal discussed third generation oral contraceptives⁶¹.

At the end of 1998 three major studies without sponsoring from the industry found a higher risk of venous thrombosis for third generation contraceptives, unlike three sponsored studies. To date, of nine studies without sponsoring, one study found no difference and the other eight found relative risks from 1.5 to 4.0 (summary relative risk 2.4); four sponsored studies found relative risks between 0.8 and 1.5 (summary relative risk 1.1) ... In 1995 four studies found the same risk ... The companies proclaimed that with almost total certainty everything was the result of bias and confounding. Even for a skeptic at the time, that was an unreasonable position: all four studies were reasonably executed and had withstood criticism from the Committee on Safety of Medicines and reviewers of leading journals. Thus, the companies' position ran the high risk of damaging both their product and their credibility ... Since 1995 three multinational companies have used enormous marketing resources to sow confusion ... Many general practitioners, gynaecologists, and family planners were swayed into accepting methodological arguments that sounded logical because of their legitimate concern with good contraception⁶².

A libertarian argument can also be used to support birth-control. It has been suggested by zealous advocates of birth control that, after freedom of speech and worship, and freedom from want and fear, the fifth freedom is from a perceived excess of fertility⁶³.

Birth control and abortion

In a review of Randy Alcorn's "Does the birth control pill cause abortions?"⁶⁴, Dr Joel Goodnough set out to disprove, both scientifically and philosophically, the arguments that the oral contraceptive pill (OCP) was abortifacient. Dr Goodnough's arguments represent some common justifications for use of OCPs and other abortifacient birth control methods. He raises the issues of whether:

- embryo death is frequent and/or can be proved
- an OCP which causes the loss of an embryo is abortifacient
- the benefits of OCPs justify their use if embryo death is unlikely
- OCPs meet the requirement of double effect.

⁶¹ "Third-Generation Oral Contraceptives: Future Implications of Current Use" <http://www.medscape.com/viewarticle/571231>

⁶² Vandembroucke, J P (2000), "BMJ readers should know whose words they read" (letter), *BMJ* 2000; 320: 381 (5 February)

⁶³ D Baird, The fifth freedom, *BMJ* 1965; ii: 1141-1148, cited in D Skegg Safety and efficacy of fertility-regulating methods: a decade of research, *Bulletin of the World Health Organisation*, 1999, 77 (9) pp 713-721.30 Goodnough, J. (2001) "Redux: is the oral contraceptive pill an abortifacient?", *Ethics & Medicine* 17 (2001): 37-51. See abstract of this article in *Bioethics Research Notes* Vol 1303.

⁶⁴ Goodnough, J. (2001) "Redux: is the oral contraceptive pill an abortifacient?", *Ethics & Medicine* 17 (2001): 37-51. See abstract of this article in *Bioethics Research Notes* Vol 1303.

John Wilks responded⁶⁵ to Dr Goodnough and one of his general criticisms was that Dr Goodnough's references were outdated and failed to reflect current knowledge of the process of implantation.

Frequency and proof of embryo death

Dr Goodnough blames all pregnancies which occur while a woman is on the pill on user-error and says there is no evidence of breakthrough ovulation. He suggests it is speculative to say that implantation is inhibited in the case of breakthrough ovulation, and that there is no literature to show this. He also suggests that "the effect of a hostile endometrium may be absent in cases of ovulation when it matters and present in cases of anovulation, when it does not matter".

Wilks cites studies showing significant breakthrough ovulation which is not necessarily attributable to user error and states that modern formulations contain more progesterone and are hence more likely to allow breakthrough ovulation.

Some researchers had argued that a pre-fertilisation mode of action couldn't account for the rate of effectiveness of emergency contraceptives, which was thought to be disproportionately high if the mode of action was exclusively pre-fertilisation. Therefore it was thought that there had to be a post-fertilisation mode of action, namely an implantation effect, in order to account for the observed rate of effectiveness⁶⁶.

Given the difficulties associated with experimentation and setting up direct tests to confirm instances of post-fertilisation modes of action of emergency contraceptives, there is yet to be any concrete evidence that emergency contraceptives act in this manner. Nevertheless, given that there is much biological evidence for the role that the relevant hormones play in endometrial physiology, in addition to statistical evidence concerning the rates of effectiveness, it is plausible to hypothesise that in some instances, emergency contraceptives act after fertilisation by inhibiting implantation.

Inhibition of implantation is not speculative. There is much evidence of reduced endometrial thickness in pill-takers. From a moral point of view, it does not matter how often embryo death occurs and that in each circumstance it actually does. What matters is that a significant risk exists for embryo death to occur.

The abortifacient (or otherwise) nature of OCPs which cause the loss of embryos

⁶⁵ Wilks J (2001), "Response to Joel Goodnough MD, 'Redux: is the oral contraceptive pill an abortifacient?'" , *Ethics and Medicine* 17:2 (2001): 103-115. See abstract of this article in *Bioethics Research Notes* Vol 1303.

⁶⁶ Mikolajczyk, R.T. & Stanford, J.B. (2003) False risk attribution results in misleading assessment of the relationship between suppression of ovulation and the effectiveness of the Yuzpe regimen for emergency contraception. *Contraception: Letters to the Editor*, 67:333-337.

Dr Goodnough concedes that “although there is no direct evidence that this results in loss of the embryo, one cannot prove that it never happens”. He argues, however, that, while an abortifacient is what he describes as “anything used to cause or induce an abortion”, an OCP is merely a contraceptive which, if it fails, can result in the death of an embryo.

He substitutes the intention of the user for an accurate understanding of how the OCP works. Intending or hoping that one’s pill will not cause an early abortion is not a rational or defensible approach if one knows that the evidence says that it can, and often will, do so. As described above, many contraceptives of various kinds can actually cause abortions and should therefore be called abortifacients.

Similarly, a carcinogen causes cancer regardless of its primary intention. Pharmaceutical companies refer to their products’ action on the endometrium in their literature⁶⁷. Many doctors and health professionals openly accept the function of birth control methods to prevent implantation and, by prescribing them instead of alternatives, signal their intention for this to occur.

Weighing OCPs’ benefits against risks to embryos

Goodnough states that the degree of risk to the embryo is unknown, and “with every medication, with every treatment, with every surgery there is an inherent risk of causing harm... If we were to let fear of hurting an individual patient paralyse us into inaction, no one would be helped... If the risk of death is low, the benefits of the OCP justify use. Since the risk of death on the OCP is less than the risk of death in pregnancy, the risk is tolerable”.

This calculation is based on the false assumption that endangering the embryo for one’s own benefit is acceptable, and further flawed by confusing the risk to the mother with the risk of destroying an embryo. Furthermore it suggests that the pill or pregnancy are the only alternatives when, in fact, there are other alternatives which do not endanger lives.

Double effect

Goodnough believes the pill meets the requirements for the principle of double effect because the desired effect is the prevention of conception by prevention of ovulation, and suggests there are no safer alternatives. This is not a valid use of the double effect principle because:

⁶⁷ For example: “The pill ... makes the lining of the womb thinner so that it is unsuitable for pregnancy...”, A small book of questions and answers about the ‘pill’, Wyeth Pharmaceuticals and the Family Planning Association of Australia (publication date unknown); “... altering the lining of the womb so that it becomes difficult for a fertilised egg to implant itself and develop”, Oral contraception today: Your questions answered, Schering Pty Ltd. (publication date unknown).

- An unintended harmful outcome is only allowable if it is proportionate to the benefit being sought. The death of an embryo is a very grave harm to that embryo, of course, and is wholly disproportionate to the supposed good being sought, namely child-free intercourse for the woman and her partner.
- The double effect principle also requires that other means of achieving the objective should be sought to try to avoid the harm. There is no suggestion of any such effort by Dr Goodnough.
- It would seem to be the case that some – perhaps many – users of abortifacient birth control actually want an abortifacient effect if the contraceptive mechanism(s) fail. Double effect is not applicable when the harmful side effect of one’s action is, in reality, an intentional and deliberate consequence.

6. Conclusion

Many up-to-date birth-control methods, as well as those under development, are of the kind which threaten unborn life. Couples relying on these methods will know this. There are alternatives to abortifacient birth-control which do not threaten life.

7. Terminology

Abortifacient (adjective)	Tending to cause abortion
Abortifacient (noun)	A drug or device which intentionally or accidentally causes an abortion
Abortion	The destruction of the developing embryo/foetus in the mother's body, or the fatal expulsion of the foetus or embryo, spontaneously, or by surgical or medical techniques
Contraceptive (noun)	A drug or device to prevent conception
Induced abortion	A deliberately caused abortion
Miscarriage	The unintended death and expulsion of the unborn from the womb (same as spontaneous abortion)
Progestational (adjective)	Helping embryos develop by priming the endometrium (lining of the uterus) to receive an embryo just before menstruation
Progesterone	A naturally occurring progestational hormone which is the principal agent in promoting and

	maintaining gestation
Progestin	The unrefined product extracted from the corpus luteum; any natural or synthetic progestational agent
Progestogen	Any progestational agent
Spontaneous abortion	The unintended death and expulsion of the unborn from the womb (same as miscarriage)
Sterilisation	Rendering a person infertile indefinitely, usually by means of a surgical operation to remove the ovaries or to cut or tie the fallopian tubes or vas deferens
Therapeutic abortion	A deliberately caused abortion undertaken on the grounds of a risk to health