ENDEMIC DISEASE, NUTRITION AND FERTILITY IN DEVELOPING COUNTRIES

C. G. N. MASCIE-TAYLOR

Department of Biological Anthropology, University of Cambridge

Introduction

The two main ways in which disease and nutrition can influence fertility are by reducing fecundity or by extending the birth interval. Fecundity refers to reproductive ability, that is the potential to breed, as compared to fertility which denotes actual childbearing (McFalls & McFalls, 1984). Reduced fecundity, which is usually referred to as subfecundity, results from impairment of any of the biological aspects of reproduction, including coital inability, conceptive failure as well as pregnancy loss. Subfecundity is only one factor operating to reduce fertility; other factors include those governing mate exposure (both formation and dissolution of unions as well as exposure to intercourse within unions) and birth control.

There is considerable variation in individual female fecundity, and the number of children a woman gives birth to ranges between zero and over thirty. The estimated maximum population fecundity, which is the average fecundity of individual female members, is about fifteen children per woman for women who use no birth control and have regular sexual intercourse between menarche and menopause. The actual fertility of almost all populations ranges between one and eight children per woman, with about four children per woman being the worldwide average. Thus all populations are subfecund and no population has the conditions for maximum reproduction.

Factors known to cause subfecundity include disease, nutrition, genetic, environmental and psychological components. This paper focuses on the importance of disease and nutrition in modifying fertility in developing countries.

Relationship between disease and fertility

There is evidence that disease has a considerable impact on human reproductive patterns and a number of diseases are thought to cause subfecundity. These diseases can be divided into two groups, sexually transmitted and non-sexually transmitted diseases.

Sexually transmitted diseases (STDs)

There are 21 pathogens for which sexual transmission is a major means of spread. Of these, the most important are the venereal diseases, syphilis, gonorrhoea and genital chlamydia.
Syphilis is caused by the bacterium *Treponema pallidum* and is characterised by a primary lesion (chancre), a secondary skin eruption and a long latent period which may be terminated by a tertiary phase (late syphilis) resulting in lesions of skin, bone or viscera, or by lesions of the cardiovascular and nervous systems. Primary and secondary syphilis are highly infectious stages since spirochaetes are circulating in the blood and other body fluids, including saliva, semen and vaginal discharges.

Accurate rates of syphilis in developing countries are not available but rates of over 10% have been reported in Thailand and over 20% in one study in Papua New Guinea. Syphilis is common throughout Africa and is particularly prevalent in Ethiopia, where in some areas 40% of the population are seropositive.

Syphilis has little or no effect on coital or conception ability but it can lead to increased pregnancy loss. The risk of pregnancy loss depends on two factors, the stage of syphilis the mother is experiencing and the stage of gestation at which the fetus is infected. If the mother is experiencing primary or secondary syphilis then the fetus is unlikely to escape infection because spirochaetaemia is intense. If the maternal infection occurs just prior to or early in pregnancy the fetus is likely to be infected and fetal death is the most probable outcome. On the other hand, if the woman is infected later in pregnancy the child will be born alive but will soon develop severe congenital syphilis. In mothers whose infection is old the spirochaetaemia will be less intense. Consequently children infected early in pregnancy may be born alive and suffer only mild congenital syphilis.

The probability of fetal infection is difficult to calculate because of the interrelationship between the stage of the mother’s infection and the gestational age of the fetus. Stillbirth rates of between 66% and 80% have been reported for syphilitic mothers. Barrett-Connor (1969) reported a 30% fetal death rate and a 70% congenital syphilis rate in live-born infants for women with untreated early syphilis.

McFalls & McFalls (1984) stress that the fetus is at a significant risk of infection only when the mother’s infection is of less than 2 years’ duration when the spirochaetaemia is intense. They say: ‘Syphilis, then, is not a cause of low fertility. Perhaps one child, even two, is lost early in reproductive life due to syphilis, but that is all…. Thus, fertility rates may be lowered somewhat if syphilis is very prevalent, but syphilis is not an important cause of low fertility and/or childlessness’.

Gonorrhoea is caused by the bacterium *Neisseria gonorrhoeae* and it is a disease of young adults. The reported incidence rates of gonorrhoea in developing countries are completely unreliable but the broad picture is that gonorrhoea is very common in many countries, particularly in Africa. For instance, in Nairobi, Kenya, it was found that 17.5% of women attending a family planning clinic and in Uganda 18% of women tested were gonorrhoea positive.

Gonorrhoea is an important cause of subfecundity in both sexes. In men gonorrhoeal infection may result in urethral strictures, obstructive azoospermia and sterility. In addition, seminal fluid abnormalities may occur which reduce fertilising capability. In women the most important complication is salpingitis, also called acute pelvic inflammatory disease (PID). The disease commonly arises during the first or second menstrual cycle after infection and it is estimated that 20% or more of women with cervical gonorrhoea in African countries develop salpingitis. The infection leads to tubal occlusion, ectopic pregnancy, persistent lower abdominal pain and infertility.
Environmental, host and organism related factors affect the risk of developing PID. One very important environmental factor is the type of contraceptive used. The risk of developing PID is several times higher in IUD users than in non-users, and this risk is higher still if the woman has never been pregnant. The mechanism for the increased risk is unknown and several theories have been put forward. One is that bacteria ascend the tail of the IUD. Another theory is that the more prolonged and heavier menstrual flow associated with IUD use facilitates movement of the bacteria to the upper genital tract. In contrast, women using oral contraceptives have a decreased menstrual flow and a concomitantly lower risk of developing PID.

The risk of developing PID is also greater in women with gonorrhoea who have just experienced childbirth or abortion, and Muir & Belsey (1980) have argued that gonorrhoea and other sexually transmitted diseases are important in the aetiology of post-abortal and post-partum infections. McFalls & McFalls (1984) estimate that childbirth or abortion increases the risk of PID in women with gonorrhoea by 50–100%.

In recent years increasing numbers of cases of PID have been shown to be caused not by gonococcal infection but by other sexually transmitted pathogens, the bacterium *Chlamydia trachomatis* and the genital mycoplasms *Mycoplasma hominis* and *Ureaplasma urealyticum*.

*Chlamydia trachomatis* infection rates are thought to be high in developing countries although exact figures are not available. The disease can affect both men and women. In men it can lead to urethral strictures, occlusion of the genital ducts and functional damage to spermatozoa. A recent study showed that chlamydial infection led to an inflammatory response in the male genital tract and 15-4% of men with chlamydial infection were infertile (Wolff et al., 1991). In women chlamydial infections can cause salpingitis and, like gonorrhoeal infections, can lead to tubal occlusion (partial or complete). Luteal progesterone production has been shown to have an inverse relationship to the rate of *Chlamydia trachomatis* inclusions in vitro, and to the degree of resulting inflammation and productive infection in vivo (Tau-Cody et al., 1988). Chlamydial DNA was detected in 49 out of 186 infertile women patients (26-3%) but was present in only eight out of 64 (12-5%) in a fertile control group (Soong et al., 1990). There is also evidence of chlamydial infections resulting in spontaneous abortion.

Genital herpes also has important effects on fecundity. It is caused by herpes simplex virus 2 (HSV-2) and the disease is incurable once a person is infected. The prevalence of genital herpes in developing countries is thought to be similar to or higher than rates in the developed countries. It is estimated that 15–30% of all sexually active adults were infected with HSV-2, while a study of blood donors in Bristol, England, found 18% seropositive.

The virus causes sores in urogenital tissues (vulva, vagina, cervix, penis, testis, urethra and bladder). Sores on the vulva or penis can make intercourse painful or impossible although coital ability returns when the sores heal. There is evidence of a three- to five-fold increase in the risk of spontaneous abortion if a woman had a genital herpes infection during the first trimester of pregnancy. In addition, premature delivery is 20% more common in women infected with genital herpes compared to the general population. Neonatal infection can occur and if maternal lesions are present...
at the time of parturition there is at least a 40% chance that the child will be infected unless it is delivered within 4 hours of rupture of the membranes. Such children have only a 30–40% chance of surviving and most of those that do survive suffer permanent neurological or ocular damage.

Finally, there is an association between genital herpes and cervical carcinoma, and women with HSV-2 virus antibodies have a five-fold increase in risk of developing cervical cancer. For women with cytologically detected infection the risk increases to eight-fold.

Non-sexually transmitted diseases

Diseases producing subfecundity are especially prevalent in developing countries. For instance, diseases common in Nigeria (excluding STDs) include malaria, dysentery, measles, pneumonia, chicken-pox, whooping cough, schistosomiasis, filariasis and tuberculosis, almost all of which can cause subfecundity.

Tuberculosis is a chronic infectious disease caused by the bacterium *Mycobacterium tuberculosis* or, less frequently, by *Mycobacterium bovis*. The body responds to the bacilli by initiating a tissue reaction which results in the formation of nodular lesions called tubercules. The site of the initial infection is usually the lung but the bacilli enter the lymphatic and vascular systems, and thus other organs can be infected. Most bacilli are killed by the immune system and the vast majority of people do not develop tuberculosis. However, if the immune system is suppressed because of, for example, malnutrition, malaria, leprosy, early syphilis or African sleeping sickness, a few of the bacilli not destroyed may be reactivated and disease ensues.

Subfecundity is due to genital tuberculosis, which is one of the extrapulmonary forms of the disease; it causes subfecundity by increasing rates of coital inability, concepitive failure and pregnancy loss. Painful coitus (dyspareunia) may occur in both men and women with genital tuberculosis. Even if the genital organs are not affected, pulmonary tuberculosis may be associated with such shortness of breath on exertion that coitus becomes impossible. Decreased libido and sexual potency have been reported in men and women with pulmonary tuberculosis.

Genital tubercules may interfere with the passage of gametes through the male and female tracts, and genital tuberculosis has been shown to depress sperm motility as well as sperm count. In women tubal involvement is usually bilateral and the fibrosis may be so severe as to result in tubal closure. Even where there is no tubal closure the lining of the fallopian tubes may be so convoluted that spermatozoa are delayed or trapped and degenerate before reaching the ovum.

Menstrual disorders, particularly amenorrhoea, are seen in a substantial percentage (between 5% and 50%) of cases of genital tuberculosis. This amenorrhoea arises from damage or actual destruction of the endometrium rather than by ovulatory failure. The incidence of sterility among women with proven genital tuberculosis varies between 55% and 85%.

Malaria is a disease caused by protozoa of the genus *Plasmodium*. Only four species parasitise humans (*P. malariae*, *P. vivax*, *P. falciparum* and *P. ovale*). Infected people suffer from disabling fevers, chills and headaches, and may also have anaemia which occurs because of the massive red cell destruction. Although early research suggested that malaria had a depressing effect on potency, there is no pathophysio-
logical evidence that the disease affects coital ability. However, the frequency of coitus may be reduced because of the ‘draining of the victim’s strength’.

The relationship between malaria and concepitive ability is unclear. Research in the 1940s showed that if the scrotal temperature exceeds 37°C (35°C is normal) for more than 45 minutes azoospermia will result followed by a recovery period of reduced sperm count (oligospermia) lasting for 2 months. A number of febrile illnesses, including pneumonia, hepatitis and chicken-pox, are associated with reduced sperm count and malaria can be included in this list. A study in an endemic malarial area in Nigeria revealed that 55% of the men were oligospermic.

Eaton & Mucha (1971) suggested that men who are heterozygous for sickle-cell haemoglobin are less likely to experience a fever-induced reduction in sperm count and hence are more fertile in malarious areas than are men with normal haemoglobin. Two factors are important in determining the number of men whose fertility will be affected—the level of malarial endemicity and the frequency of the S allele. In areas of intense transmission (holoendemic) with 40% S allele frequency McFalls & McFalls (1984) estimated that 60% of the male population will experience fevers sufficiently high to alter spermatogenesis. If the S allele frequency were only 15%, then up to 85% would have altered spermatogenesis. In areas with lower endemicity (mesoendemic) and 15% S allele frequency less than 60% of males suffer malaria fevers each year and therefore the effect on male fertility is less than in either of the more endemic areas.

In pregnant women high fevers such as those associated with influenza, typhoid fever, pneumonia and malaria can cause spontaneous abortion, stillbirth and premature birth. Falciparum malaria, which is associated with the highest fevers of the four species of human malaria, is widely cited as a cause of pregnancy loss. Such loss depends on the immunity level of the population. Thus in hyperendemic areas malaria is unlikely to be a cause of pregnancy loss whereas in mesoendemic areas where clinical illness is seen among adults fevers sufficiently high to cause pregnancy loss would be more frequent. In the Black Caribs of Belize, which is a mesoendemic P. falciparum area, women heterozygous for the sickle-cell allele have a fertility rate 1.45 times higher than normal homozygotes.

Anaemia is a common feature of P. falciparum malaria and this haemolytic anaemia may be complicated by a second type of anaemia, megaloblastic anaemia, which is caused by a deficiency of folic acid (an essential nutrient for the manufacture of red blood cells). Pregnant women in malarious areas are particularly prone to this type of anaemia because they must provide folic acid for the replenishment of destroyed red blood cells and for the development of fetal red cells. In areas of West Africa the situation is particularly grave because the normal dietary sources of folic acid (fresh vegetables and meat) are subjected to prolonged cooking which destroys the folic acid. Severe anaemia (defined as less than 6.5 g of haemoglobin per 100 ml of blood) may cause fetal and maternal mortality. Immunity levels are important and primigravidae are more likely to suffer from more anaemia because of their lower immunity.

The malarial parasites can also invade the placenta, and early researchers thought that such placental parasitisation was a cause of abortion, stillbirth and neonatal death. The present consensus is that only heavy placental infection will cause fetal loss.
African sleeping sickness, which is caused by protozoa of the genus *Trypanosoma*, has also been reported to have effects on reproductive potential. Many of the effects on fecundity are similar to those reported for malaria, i.e. infected individuals with sleeping sickness suffer from fevers and temperatures often reach 39.4–40°C and occasionally peak at 41.4°C. Azoospermia is likely although sperm counts in men with sleeping sickness have not been measured. Anaemia is also common in those suffering from sleeping sickness; haemoglobin levels below 9.2 g/100 ml are believed to increase the prematurity rate by 50% and levels less than 8.7 g/100 ml seriously impair both maternal and fetal prognosis.

Sleeping sickness is also thought to produce reproductive problems because the disease leads to defective secretions by the pituitary gland and ovary. Altered pituitary function can adversely affect fecundity in a variety of ways. Pituitary secretions direct thyroid function, oestrogen secretion by the ovary, growth and maturation of the ovarian follicle, ovulation, secretion of testosterone by the testis, spermatogenesis, etc. Menstrual disorders, including amenorrhoea, are frequently observed in women with sleeping sickness and in one study a quarter of the women with the disease had menstrual disorders. Many affected women are probably unable to conceive and women already pregnant before the onset of endocrine dysfunction are likely to experience a premature termination of the pregnancy. In men feminisation (breast development and fat distribution) can occur and impotence has been widely reported.

There has been considerable discussion on the effects of schistosomiasis (bilharzia) and filariasis on male and female fecundity. Most recent results suggest that schistosomiasis has little impact on male or female fecundity (although coital ability might be impaired in those with *S. haematobium*) and rarely makes a woman anovulatory even though schistosomae eggs can lodge in the uterus or in the gonads themselves. Furthermore, although schistosomae eggs have been found close to the maternal/fetal interface, and in the amniotic fluid, there has been no documented instance of finding schistosomae eggs in stillborn infants.

Filariasis might well have a negative effect on male reproductive ability, resulting in loss of coital ability as well as oligospermia, but further confirmatory evidence is needed. The effect of the disease on female fecundity is hard to assess because so little has been written about the disease consequences in women.

The likelihood of female pelvic infection is also increased by abortion, childbirth, female circumcision and the use of IUDs. Abortions in developing countries are commonly carried out by an untrained traditional midwife working in unsanitary conditions and using crude equipment. It is not surprising that many women suffer from haemorrhage and septic infection which may lead to chronic pelvic infections and reproductive problems including sterility. Dyspareunia is a common sequel to septic abortion.

Female circumcision involves removal of part or all (excision) of the clitoris. The labia minora and labia majora might also be removed. Infibulation involves the removal of the clitoris and labia, and the scraped tissue sewn together. There is considerable variation in age at circumcision and the procedures used also differ between countries, ethnic and religious groups.

Female circumcision has short- and long-term complications. Haemorrhage and
infection are major short-term sequelae while in the longer term there is a danger of urinary tract infections and bladder and bowel incontinence resulting from damage to the urethra or anus. Coital ability might be impaired because of the result of excessive scar tissue formation or because the vaginal opening is too small to allow penetration. Even if coitus is possible the opening might be too small for normal delivery and prolonged labour due to obstruction will occur with a high risk of pregnancy loss.

Some authors state that female circumcision causes sterility (Belsey, 1979); others suggest that the greatly narrowed vaginal opening predisposes to infection of the upper genital tract (Epelboin & Epelboin, 1979) while Schrifin, Erez & Moore (1973) proposed that obstruction of the menses can cause endometriosis. Endometriosis causes infertility in 40–50% of cases (Spangler, Jones & Jones, 1971).

Summary

The three ways in which disease reduces fecundity, and thereby influences fertility, are by reduced coital ability, increased concepitive failure and greater pregnancy loss. As Table 1 illustrates, disease is a major cause of subfecundity and can lead to an elongation of birth intervals.

Disease may lower fertility rates other than by subfecundity. The time spent in unions is frequently affected by disease. Women in societies where children are highly valued who suffer from diseases which leave them relatively subfecund (e.g. 2 years in

<table>
<thead>
<tr>
<th>Type of subfecundity and cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coital inability</td>
</tr>
<tr>
<td>Impotence (e.g. African sleeping sickness)</td>
</tr>
<tr>
<td>Complete or partial obstruction of vaginal opening (e.g. severe schistosomiasis)</td>
</tr>
<tr>
<td>Genital deformities (e.g. filariasis)</td>
</tr>
<tr>
<td>Painful coitus (e.g. genital tuberculosis, infibulation)</td>
</tr>
<tr>
<td>Conceptive failure</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Occlusion of ducts (e.g. genital tuberculosis, gonorrhoea)</td>
</tr>
<tr>
<td>Fevers (e.g. malaria, filariasis)</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td>Endocrine problems (e.g. African sleeping sickness)</td>
</tr>
<tr>
<td>Immunological problems (e.g. genital schistosomiasis)</td>
</tr>
<tr>
<td>Damage to ovaries or tubes (e.g. PID)</td>
</tr>
<tr>
<td>Pregnancy loss</td>
</tr>
<tr>
<td>Problems in tubes (e.g. PID)</td>
</tr>
<tr>
<td>Uterine problems (e.g. genital tuberculosis)</td>
</tr>
<tr>
<td>Endocrine dysfunction (e.g. African sleeping sickness)</td>
</tr>
<tr>
<td>Anaemia (e.g. malaria)</td>
</tr>
<tr>
<td>Fevers (e.g. filariasis, malaria)</td>
</tr>
<tr>
<td>Placental transmission site (e.g. syphilis, malaria)</td>
</tr>
</tbody>
</table>
C. G. N. Mascie-Taylor

the case of syphilis) may be deserted or divorced by a mate anxious to have children. Also women who develop a vesico-vaginal fistula following a difficult birth may, because of the unpleasant odour associated with the condition, be rejected by the husband. People with syphilis or tuberculosis are less often chosen as mates and women with debilitating diseases such as tuberculosis might wish to avoid the rigours of pregnancy and so use contraception.

**Nutrition and fertility**

This section only briefly reviews the relationship between nutrition and fertility since the role of nutrition will be dealt with in the papers by Rosetta and Norgan in this volume.

It has long been recognised that maternal nutritional status associates with fertility as well as fetal and infant survival. The relationships between severe food deprivation and fertility have been studied in the natural experiments during acute famine periods in Holland and Bangladesh. In the Dutch famine of 1944–45 births fell by about 50% some 9 months after the famine and similar findings were reported from Bangladesh. However, it is unclear whether the fall in fertility was the result of cessation of menstrual cycling because of marked weight loss, the result of increased psychological stress, reduced levels of intercourse or due to some other factor(s).

There is considerable evidence that a marked loss of weight is usually accompanied by an interruption of reproductive cycles. Frisch (1990) has suggested that moderate weight loss of 10–15% weight-for-height, either through dieting or due to intensive exercise, which is unassociated with anorexia nervosa (where weight loss is to about 30% below ideal weight), results in amenorrhea. However, women do resume menstruating when they are re-fed and approach minimum weight and fat content. Even so women who are regaining weight into the normal range may have a menstrual cycle which has a shortened luteal phase or is anovulatory.

Thus, whilst there is general agreement that severe nutritional deprivation can affect maternal fecundity, the effect of moderate nutritional deprivation, which is the norm in many developing countries, is less clear. In two Bangladeshi studies where maternal weight and weight-for-height were used as indicators of nutritional status there was no significant difference between the lightest and heaviest groups in length of post-partum amenorrhoea although the results were in the predicted direction with the lightest women having a longer period of amenorrhoea. Such studies are complicated by the interaction between nutritional status, lactation and breast-feeding patterns, which are all thought to play a part in the return of ovulation.

Nutritional deficiencies are also responsible for a large proportion of pelvic abnormalities which frequently lead to difficulties in childbirth (cephalo-pelvic disproportion) and high rates of morbidity and mortality in both mother and child. Vitamin D deficiency is responsible for osteomalacia in adults and rickets in children, and general undernutrition is responsible for the contracted pelvis seen so frequently in developing countries.

Another complicating factor is that in many developing countries there is high prevalence of infectious, especially parasitic, diseases, many of which interact with nutritional status. Parasitic infections may affect the nutritional status of their hosts in three principal ways: (1) by bringing about a disturbance in the normal nutritive
processes of the host; (2) by imposing demands on the host which create an extra nutritional cost; and (3) by the loss of nutrients arising from either the feeding activities of the parasite or from damage to the tissues of the host.

For instance, one billion people worldwide are thought to be infected with hookworms which live attached to the intestinal mucosa and feed on mucosal tissue, blood and other tissue fluids. Blood loss occurs during feeding and this loss, facilitated by the feeding activity of hookworms, is considered to be responsible for iron deficiency anaemia, the principal nutritional consequence of hookworm infection. In addition to the loss of iron due to the feeding of hookworms there will also be a concurrent loss of serum proteins which can lead to low concentrations of albumin in the blood.

Disease and nutritional status also interact. Numerous disease attacks and dietary deficiencies lead to exhaustion of the immune defence system and the ability to resist infections such as gonorrhoea is greatly diminished. People with anaemia are more susceptible to infection while those with iron deficiency anaemia are more susceptible to malaria but not to bacterial infection.

Assessment of disease status can be problematic. It is a common belief that infection and disease are one and the same thing. There is an important distinction between infection and disease which has been voiced before. Always to equate infection with disease ignores the great variation in the effects of the agent on the host. For example, the effects of parasites on their hosts is often related to the magnitude of the parasite burden. Thus the prevalence of *Ascaris* in children in Bangladesh is generally thought to be about 80% but not all children will show ascariasis—the signs and symptoms of the disease.

**Conclusions and recommendations**

In the context of research into fertility in developing countries it appears that the impact of disease has been underestimated.

Diseases may and do affect fertility rates in many ways. They may cause subfecundity through coital inability, conceptive failure and/or pregnancy loss.

Disease may affect fertility in yet other ways, including the time spent in unions, coital frequency and contraceptive use.

Poor nutritional status, in general, increases the risk of maternal mortality and low birth weight.

Research in developed countries shows that moderate weight loss of 10–15% of normal weight-for-height can lead to amenorrhoea. The implications of this finding to women in developing countries require further study.

The effects of disease on lactational subfecundity are unclear and further research is required to determine the interrelationships between nutritional and disease status of the mother and child, suckling activity patterns and energy expenditure on lactational subfecundity.

**Discussion**

*Harrison*: Do you know what the effect of sleeping sickness is on the female hormonal cycle? The supposition is that it just produces amenorrhoea. However, an acute phase
of fever is likely to interrupt not only reproductive hormone cycles but all hormone cycles, including the metabolic cycles.

**Mascie-Taylor:** The simple answer to your question is no. Most of the literature is based upon infection in animals; they show lesions in the pituitary area.

**Ulijaszek:** The importance of the paper is that there may be parts of the world in which the effects of lactation, high energy expenditure, work output and poor nutritional status on fertility might simply be swamped by infection.

**Lunn:** It will be extremely difficult to separate the effects of disease and nutritional intake. Usually when you’re ill you don’t eat very well and consequently your energy intake will go down and that will show up on the nutritional status measurement. Also most diseases are going to provoke some sort of acute phase reaction in the body which is going to raise cortisol levels. High cortisol levels are not good for maintaining a normal menstrual cycle.

**Mascie-Taylor:** With syphilis it is the acute phase that is important; chronic syphilis is not debilitating. So it would appear that the acute phase is much more important than the chronic.

**Clarke:** Animal experiments have shown that in populations with a very high nematode prevalence there was no effect on fertility. Is this true of humans too?

**Mascie-Taylor:** There is no evidence that parasites like *Ascaris* or hookworm species have a direct effect upon fertility. Their effects are thought to be indirect in modifying nutritional status, i.e. in preventing food absorption from the gut and increasing nutrient or blood loss. The prevalence of infection, though, is not the most important feature (since an individual can be infected with a parasite and not show any disease). It is the intensity of infection which will have important nutritional implications.

**Harrison:** Distinction between infection and disease is very important. I would not like to make any prediction as to what infection might do to fertility, but I could make a fairly reasonable prediction as to what the disease might do to fertility, however. In general, one would imagine there are various ways that disease would have a marked effect upon fertility, not necessarily by any particular influence with the reproductive cycles but just through general ill-health, the loss of libido or perhaps through pregnancy loss and so on.

**Mascie-Taylor:** We should concentrate on those diseases which have been shown or ought to show a direct effect on the ovarian cycles so that we are dealing with a minimal range of diseases.

**Harrison:** However, if you want to be clear of the complicating results of disease you may not find a place in the Third World which is free of disease, but you might get a place in the Third World free of diseases which affect the menstrual cycle.

**Rosetta:** If we are dealing with very small groups of women in longitudinal studies, is it better to choose women strictly without parasitic or infectious diseases? If we do this they will not be representative of the whole population in many countries.

**Mascie-Taylor:** As far as parasitic infections are concerned, then, it would be possible to have an experimental (treated) and control (untreated) group. However, it is still important to get some idea of what the worm burden is and so the untreated group has to be de-wormed at the end of the study to find out their worm load. Even so, worm burden need not have been maintained at a constant level for the duration of the study.
Thalabard: I suggest a simpler approach. Take a population of normally menstruating women and measure their fecundity and how long it takes them to become pregnant. If we do this we will know exactly how external factors affect the population’s fertility. I assume, of course, that the degree of infection in pregnant women is the same as in non-pregnant women and there is no reason for it to be different.

Mascie-Taylor: It is important to take into account differences in the nutritional status and consider the questions of rural or urban environment, seasonality and all the other factors that may play a part. I think Professor Thalabard's suggestion is too simplistic.

References


