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Debate

VARIATION OF HUMAN SEX RATIOS AT BIRTH BY THE SEX COMBINATIONS OF THE EXISTING SIBS, AND BY REPRODUCTIVE STOPPING RULES: COMMENTS ON GARENNE (2009)

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Summary. Garenne (2009) presented data on the sex ratio of a present birth by the numbers of previous brothers and sisters. In unisexual sibships, the probability of a further girl increases with the number of previous girls; and the probability of a further boy increases with the number of previous boys. Garenne noted that there is an asymmetry in that the effect is stronger with regard to girls than boys. He was uncertain of the cause of this. Here I suggest a potential solution to this problem. Garenne also seems to imply that parental reproductive stopping rules *cause* heterogeneity of sex ratios. I suggest that they may reveal it – but do not cause it. Moreover, I suggest that the effects of such stopping rules may be counter-intuitive.

Introduction

Garenne (2009) presented data based on 2 million births from maternity histories in sub-Saharan Africa. The offspring sex ratio (proportion male) of parents with no previous children was 0.511. Offspring sex ratios following unisexual male sibships rose roughly monotonically with the number of previous boys to 0.529 in those parents with seven previous sons (and no daughters). Sex ratios following unisexual female sibships declined roughly monotonically with the number of previous girls to 0.476 in those parents with seven previous daughters (and no sons). Similar data had been presented by Malinvaud (1955) in respect of 4 million French births. Both these authors concluded that couples vary in their probability of producing a boy. Garenne (2009, p. 400) wrote: 'However, these analyses do not clearly answer the question of whether these effects are more likely to be biological or behavioural.' Here I consider his discussion of these matters.

Theoretical background

The probability p that a birth will be male is potentially subject to three different forms of variation, namely Poisson, Lexis and Markov variation. These will be described later. Moreover analysis is made complicated by the fact that couples operate 'stopping rules' (also to be described later) by which decisions to reproduce further are based on the sex(es) of existing offspring. These rules falsify the assumption of randomness that would otherwise simplify analysis.

Definitions

Lexis variation. Here p is constant within a given couple, but varies across couples.

Markov variation. Here p varies within couples according to the sex (or sexes) of previous births. Where p increases with previous male births, Markov variation is called 'positive'; where p decreases with previous male births, it is called 'negative'.

Poisson variation. Here p varies from one pregnancy to the next within couples regardless of the sexes of the existing sibs and has the same mean for all couples. Poisson variation may be called 'chaotic', where p varies randomly across this mean within each individual couple. Poisson variation may be called 'systematic', where p varies from one pregnancy to the next in parallel across all couples (as in the declines in sex ratio associated with birth order, maternal age, paternal age and duration of marriage). In his account of the forms of variation to which p may, in principle, be subject, Edwards (1960) confined himself to systematic Poisson variation, neglecting the possibility of chaotic Poisson variation. However, it is here suggested that the chaotic Poisson variance of p is of greater magnitude than the systematic Poisson variance of p.

Reproductive stopping rules

There are two important forms of stopping rule. The Type I rule is where couples wish for one or more children of one sex and cease reproducing when they have arrived. The Type II rule is where couples wish for given numbers of representatives of both sexes among their progeny, and cease reproducing when they have arrived.

Co-existence of these forms of variation and stopping rules

All these forms of variation and stopping rule may co-exist and interact, and no statistical test has been devised for the independent presence of each. In comparison with binomial expectation, the variances of the distributions of the combinations of the sexes (and the correlations between the sexes of sibs within sibships) are independently increased by Lexis and positive Markov; and decreased by Poisson and negative Markov variation (see, for example, Weatherburn, 1949, or Feller, 1950).

The empirical evidence

Markov variation

I have previously suggested that there is no decisive evidence for the existence of Markov variation (either negative or positive) of p in mammals, including human beings

(James, 2000a, 2009a). For the purpose of simplicity, the possibility of Markov variation will here be provisionally ignored. In contrast, there is overwhelming empirical evidence for the existence of both Lexis and Poisson variation: this is now summarized.

The evidence for Poisson and Lexis variation

At the outset it should be acknowledged that it is unclear whether some (or most) sources of sex ratio variation may be usefully categorized as predominantly Lexis or Poisson. Influences that operate throughout the reproductive life may reasonably be taken as Lexis; in contrast, those that vary across individual cycles, or from one cycle to another, may certainly be taken as Poisson. So, for instance, the variations in sex ratios by race (James, 1987) and dominance (Grant, 1990) may be classified as Lexis. And variation by time of insemination within the cycle (James, 2008) and by side of ovulation (Fukuda *et al*., 2001), may be regarded as Poisson. However, the variations by, for example, war (James, 2009b) and by stress (e.g. Catalano *et al*., 2006) are not so readily classifiable. This is so because in some sibships, such sources of variation operate at the times of all conceptions; in other sibships they operate at the times of some conceptions only. In other words, these sources of variation act as Lexis in some sibships, and Poisson in others. However, there are three important points here, viz:

(1) both Lexis and Poisson variation exist and;
(2) they have counteracting effects on variances and correlations and;
(3) overall, the Lexis variation slightly outweighs the Poisson variation.

This latter conclusion may be drawn from inspection of Geissler's huge quantity of nineteenth century German data as reproduced by Edwards (1958). In those data, the variances of the distributions of the combinations of the sexes within sibships are super-binomial for all sibship sizes.

Adverse exposures occasioning both Poisson and Lexis variation

Tables 1 and 2 illustrate the wide range of variables with which offspring sex ratio has been reported to vary. Table 1 lists selected human paternal chemical and occupational exposures associated with reported low offspring sex ratios. Table 2 lists pathologies in male and female patients that reportedly are associated with significantly biased offspring sex ratios. (It should be emphasized that in no case is it suspected that the offspring sex is causally responsible for the parental pathology.) Thus it seems that there must be substantial Lexis variation even though it is not readily quantifiable. Attempts to estimate this variation have been made by Edwards (1958), James (1975) and Pickles *et al*. (1982). Their estimates of the Lexis standard deviation were respectively 0.05, 0.045 and 0.051; however, it has been questioned whether this apparent agreement is a spurious effect of various forms of flawed estimate (James, 2000a, 2009a).

Poisson variation

As noted above, this may be categorized into systematic and chaotic.

Table 1. Reports of low offspring sex ratios associated with selected adverse human paternal exposures

Exposure	Source
Chemical	
Dioxin	Mocarelli *et al.* (2000)
Dibromochloropropane (DBCP)	Potashnik & Yanai-Inbar (1987)
Fungicides	Garry *et al.* (2002a,b, 2003)
Methylmercury	Sakamoto *et al.* (2001)
Borates	James (1998, 1999)
Alcohol	Dickinson & Parker (1994)
Cigarette smoking	Koshy *et al.* (2010)
Occupational	
Professional driving	Dickinson & Parker (1994)
Professional diving	Rockert (1977); Lyster (1982)
Non-ionizing radiation	James (1997)
Astronaut/pilot of high performance aircraft	Snyder (1961); Goerres & Gerbert (1976); Little *et al.* (1987); Irgens & Irgens (1999)

It will be noted that under a wide variety of circumstances, adverse exposures to men are reportedly associated with the production of daughters. I know of only two forms of adverse paternal exposure associated with the production of sons, viz celiac disease (Khashan *et al.*, 2010) and hepatitis B carrier (Chahnazarian *et al.*, 1988). To these may be added (some) exposures to war (though it is not clear which parent is affected by war). Some conflicts (notably World Wars I and II) were associated with additional male births. Other more recent hostilities were reportedly associated with additional female births. An attempt has been made to summarize and explain these disparate findings (James, 2009b).

Systematic Poisson variation. The systematic variation (that associated with parity, duration of marriage and ages of spouses) is clearly minuscule (James, 1987). Indeed, though most univariate analyses suggest that sex ratio declines with each of these (highly intercorrelated) variables, multivariate analyses have not finally established whether – or to what extent – such declines are independent. For the present purpose, it is sufficient to note that within sibships, there is a general very slight overall decline in sex ratio with time. As will be seen, it is important to distinguish this effect from that of chaotic Poisson variation.

Chaotic Poisson variation. Two presumably independent sources of chaotic Poisson variation will be mentioned here. The first source is that associated with time of insemination within an individual cycle (James, 2009b). The second source of chaotic Poisson variation is that associated with side of ovulation (Fukuda *et al.*, 2001): here p varies from one cycle to another within the same woman. The magnitude of the variance of p associated with these two sources seems to be appreciable, in contrast with that associated with the systematic Poisson variation.

Table 2. Directions of significantly biased sex ratios associated with selected pathologies

Pathological condition	Male patients	Female patients
Celiac disease	High (Khashan *et al.*, 2010)	Low (Khashan *et al.*, 2010)
Constipation	NK	High (Czeizel *et al.*, 2010)
Cytomegalovirus positivity	NK	Low (Shields *et al.*, 2002; Piazze *et al.* (1999)
Dermatoses of pregnancy	NA	High (James, 2000b)
Extrauterine pregnancy	NA	Low (James, 1995)
Fatty liver of pregnancy	NA	High (James, 1995)
Hepatitis B carrier	High (Chahnazarian *et al.*, 1988)	High (Chahnazarian *et al.*, 1988)
Hepatitis C	NK	High (European PHC Network, 2005)
HLA B 15 positivity	Low (Astolfi *et al.*, 2001)	NK
Hyperemesis gravidarum	NA	Low (James, 2001a)
Lupus	NK	High (James, 2007)
Measles	NK	High (Langaney & Pison, 1979)
Multiple sclerosis	Low (James, 1994)	High (James, 1994)
Non-Hodgkin's lymphoma	Low (Olsson & Brandt, 1982)	Low (Olsson & Brandt, 1982)
Placenta accreta	NA	Low (James, 1995)
Polycystic ovary syndrome	NA	High (Kitzinger & Willmott, 2002)
Pre-eclampsia	NA	High (James, 1995)
Testicular cancer	Low (Moller, 1998; Jacobsen *et al.*, 2000; Gundy *et al.*, 2004)	NA
Varicella	NK	Low (Miller, 2002, personal communication)
Toxoplasmosis	NK	High (Kankova *et al.*, 2007)

NA=not applicable. NK=not known.
It is acknowledged that there may be bias within the table occasioned by selecting only results that met arbitrary significance levels. Moreover, considerations of publication bias would suggest that some of the cited conclusions – especially the uncorroborated ones – may prove to be false. However, some of the data have been extensively replicated, e.g. those relating to adverse obstetric conditions, hepatitis B status, cytomegalovirus status and testicular cancer; and also those relating to men exposed to various forms of chemical exposure and gravitational change.

Garenne's treatment of his data

The asymmetry

As noted above, the probability that a current birth will be male reportedly rises with the number of prior boys and decreases with the number of prior girls (Malinvaud, 1955; Garenne, 2009). In both sets of data the effect was not

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symmetrical, being significantly stronger for females. Garenne (2009) accordingly fitted his data to an asymmetric log-gamma function. He suggested (p. 405) that this asymmetry deserves further research, and that it is probably explained by biological factors. However, I think I may already have identified the cause of this asymmetry: I suggest that it lies in the systematic Poisson variation described above (viz the very slight decline in p within sibships with time) (James, 1975). The argument is as follows.

Malinvaud (1955) published data on nearly 4 million births in France in 1946–50. He gave the sex ratios of these births by the numbers of pre-existing boys and pre-existing girls. He showed that these data are fairly well fitted by the linear relationship:

$$p_i = 0.5145 + 0.003n_i - 0.005m_i,$$

where p_i is the probability that the present pregnancy will yield a boy, n_i is the number of pre-existing boys and m_i is the number of pre-existing girls. This formula apparently exemplifies the asymmetry that elicited the comments of Garenne (2009). I suggested (James, 1975) that this formula might be re-arranged thus:

$$p_i = 0.5145 - 0.001(n_i + m_i) + 0.004n_i - 0.004m_i.$$

In other words, it is suggested that symmetry (as between the sexes) is restored (or the asymmetry explained) if it is assumed that the probability of a boy declines very slightly (by 0.001) within each couple for each preceding live birth. Justification for this assumption exists in the systematic Poisson variation identified above. Moreover, Garenne's data are also susceptible to such adjustment. So I suggest that the asymmetry does not have the causal significance that Garenne is inclined to ascribe to it.

Garenne's suggestion of 'behavioural' determinants of sex ratio heterogeneity

Garenne (2009, p. 400) was uncertain whether the heterogeneity of p (the probability of a male birth) among couples may have biological or behavioural determinants. He inferred (if I understand) that since the heterogeneity exists in sub-Saharan Africa (where birth limitation is not widespread), the cause is biological. However, I now suggest that birth limitation (as exemplified in stopping rules) can have only very limited effects on sex ratio heterogeneity. The argument is as follows:

1. If p were distributed binomially within and across couples, such stopping rules would have no effect on sex ratio heterogeneity. Nor would they have any effect on population sex ratio.

2. If p were subject only to Lexis variation across couples, then the most probable result of such stopping rules would be further children of the same sex as the prior sib(s). Cessation or continuation of reproduction would depend on the stopping rule employed by each individual couple. Such a process would affect sex ratio heterogeneity in the following ways. In societies in which boys and girls were equally valued, natural boy-producing parents wanting girls would presumably be roughly equalled by girl-producing parents wanting boys. Here the effect of the stopping rule is slightly to increase the heterogeneity of p with increase of parity. However, in

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societies valuing one sex (e.g. boys) over the other, girl-producing parents would (on average) have more children than boy-producing parents. This effect may be thought of as counter-productive, and the overall population sex ratio would be lower than if parents did not operate such a rule. Again, the effect is very slightly to increase the heterogeneity of p with parity.

3. If p were subject only to Poisson variation within couples, then the most probable result of such stopping rules would be further children opposite in sex to their existing sib(s). But cessation or continuation of reproduction would have no appreciable effect on heterogeneity of p.

4. Lastly if (as is suggested here), Lexis and Poisson variation co-exist (the Lexis very slightly outweighing the Poisson), then the effect of stopping rules (of either sort) on sex ratio heterogeneity are unclear, but, in any case, must be of very small magnitude. For instance, two recent studies on large samples of Scottish and Danish births failed to find significant correlations between the sexes of sibs within sibships (Maconochie & Roman, 1997; Jacobsen *et al*., 1999). So, for practical purposes, p may be considered as distributed binomially in these populations. Thus, though it may be agreed that the heterogeneity of p across couples has biological rather than (or as well as) behavioural causes, it would seem invalid to infer this from the paucity of family limitation in sub-Saharan Africa.

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