

Missing women—revisited

Reduction in female mortality has been counterbalanced by sex selective abortions

See also *Papers*
p 1319

The concept of “missing women,” which was presented in an editorial I wrote in this journal 11 years ago, refers to the terrible deficit of women in substantial parts of Asia and north Africa, which arises from sex bias in relative care.¹ The numbers are very large indeed. For example, using as the standard for comparison the female:male ratio of 1.022 observed in sub-Saharan Africa (since women in that region receive less biased treatment), I found the number of missing women in China to be 44m, in India 37m, and so on, with a total that easily exceeded 100m worldwide, a decade or so ago. Others used different methods and got somewhat different numbers—but all very large (for example, Stephan Klasen’s sophisticated demographic model yielded 89m for the countries in question).²

How have things moved more recently? At one level they have not changed much. The ratio of women to men in the total population, while changing slowly (getting a little worse in China and a little better in India, Bangladesh, Pakistan, and west Asia), has not altered radically in any of these countries. Even though the total numbers of missing women have continued to grow (Klasen’s 89m is now 93m for the same countries and 101m for the world as a whole³), this has resulted mainly from the absolute growth in population.⁴

But another more important and radical change has occurred over the past decade.^{5 6} There have been two opposite movements: female disadvantage in mortality has typically been reduced substantially, but this has been counterbalanced by a new female disadvantage—that in natality—through sex specific abortions aimed against the female fetus. The availability of modern techniques to determine the sex of the fetus has made such sex selective abortion possible and easy, and it is being widely used in many societies. Compared with the normal ratio of about 95 girls being born per 100 boys (which is what we observe in Europe and North America), Singapore and Taiwan have 92, South Korea 88, and China a mere 86 girls born per 100 boys. Given the incompleteness of birth registration in India that ratio is difficult to calculate, but going by the closely related ratio of girls to boys among young children (below 6) we find that the female:male ratio has fallen from 94.5 girls per 100 boys in the census of 1991 (almost in line with the ratio in Europe and North America) to 92.7 girls per 100 boys in the census of 2001.

The drop may not look particularly high (especially in comparison with China or Korea), but further

grounds for concern exist. Firstly, these could be “early days,” and it is possible that, as sex determination of the fetus becomes more standard, the Indian ratio will continue to fall. This is quite possible despite the fact that the Indian parliament has outlawed sex determination of the fetus (except when it is medically required) precisely to prevent its abuse for sex selective abortion. Secondly, variations within India are gigantic, and the all India average hides the fact that in several states—in the north and west of India—the female:male ratio for children is very much lower than the Indian average and lower even than the Chinese and Korean numbers.

Most interestingly, a remarkable division seems to run right across India, splitting the country into two nearly contiguous halves.^{6 7} Using the European female:male ratios of children (the German figure of 94.8 girls per 100 boys was used as the dividing line), all the states in the north and the west have ratios that are very substantially below the benchmark figure, led by Punjab, Haryana, Delhi, and Gujarat (between 79.3 and 87.8 girls per 100 boys). On the other side of the divide, the states in the east and the south of India tend to have female:male ratios that equal or exceed the benchmark line of 94.8, with Kerala, Andhra Pradesh, West Bengal, and Assam leading the pack with 96.3 to 96.6 girls per 100 boys. The solitary exception in this half is Tamil Nadu, with a figure just below 94, but that too is close to the European dividing line of 94.8 and well above the numbers for every northern and western state.

The higher incidence of sex-specific abortions in the north and the west cannot be explained by the availability of medical resources (Kerala or West Bengal do not have fewer of these than Bihar or Madhya Pradesh). The difference does not lie in religious background either, since Hindus and Muslims are divided across the country, and the behaviour of both groups conforms to the local pattern of the region. Nor can it be explained by the income level (since the list of deficit states includes the richest, such as Punjab and Haryana, as well as the poorest, like Madhya Pradesh and Uttar Pradesh). Nor can it be explained by variations in economic growth (it includes fast growing Gujarat as well as stagnating Bihar). Even female education, which is so effective in cutting down sex bias in mortality does not seem to have a similar effect in reducing sex bias in natality (as is readily seen from the deficit in high education Himachal Pradesh or Maharashtra or Gujarat, not to mention China, South Korea, Singapore, or Taiwan).

The remarkable division of India (splitting the country into two disparate halves) is particularly puzzling. Are there differences in traditional cultural values that are hidden away? Is there any cultural or deep political significance in the fact that religion based parties have been able to make much bigger inroads precisely in the north and the west and not in the east and the south? A simple but imperfect indication of this can be seen in the fact that in the last general elections (held in 1999), 169 of the 197 parliamentary members of the Hindu right wing parties were elected precisely from northern and western states? Or is all this purely coincidental, especially since the rise of religion centred politics and the emergence of female feticide are both quite new in the parts of India where they have suddenly become common. We do not know the answer to any of these questions, nor to a great many others that can be sensibly asked.

Sex bias in natality calls for intensive research today in the same way that sex bias in mortality—the earlier source of “missing women”—did more than a decade ago when I was privileged to write in these pages.

Amartya Sen *master of Trinity College*

Master's Lodge, Trinity College, Cambridge CB2 1TQ

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Giving aspirin and ibuprofen after myocardial infarction

Clinical consequences are still unknown

Aspirin is a highly effective antiplatelet agent that is used by millions of people to reduce cardiovascular morbidity and mortality.^{1 2} However, a recent pharmacodynamic study showed that ibuprofen, a non-aspirin, non-steroidal, anti-inflammatory drug, can inhibit the antiplatelet effects of aspirin.³ This effect occurred in people who took daily ibuprofen before taking aspirin or in those taking ibuprofen regularly. Participants who took a single daily ibuprofen after aspirin did not exhibit an inhibitory effect. It is therefore possible that ibuprofen, if taken regularly or before daily aspirin, could reduce or even negate the beneficial effects of aspirin. If this interaction is clinically relevant it could have enormous public health implications because non-aspirin non-steroidals are among the most commonly used drugs in the world.⁴

Designing studies to address the clinical relevance of this interaction presents some unique challenges. The ideal study would accurately measure the use of aspirin and ibuprofen, both prescription and non-prescription, and their frequency of use in the period immediately preceding the outcome. Reliance on prescription records or one time assessments of medication use at baseline are likely to be inaccurate for several reasons. Firstly, many patients obtain ibuprofen and aspirin without a prescription.⁵ Secondly, even among “users” of ibuprofen, many take the drug only for a short time, sporadically, and less than three times a day.⁶ Thirdly, adherence with prescribed aspirin is suboptimal, and many patients who are prescribed aspirin will not be taking it regularly.^{7 8} A final caveat is that ibuprofen itself has antiplatelet properties³ and could itself reduce the risk of myocardial infarction.⁹ Therefore, even if ibuprofen interferes with the antiplatelet effects of aspirin it is not clear that this would result in a net increase in risk of myocardial infarction.

With these methodological challenges in mind, three papers have recently been published that examine the clinical effects of combining ibuprofen and aspirin. Macdonald and Wei, using an electronic prescription database, found that the rates of both all cause mortality and cardiovascular mortality were higher among patients with cardiovascular disease who were prescribed aspirin and ibuprofen versus aspirin alone; diclofenac and other non-steroidals did not show this effect.¹⁰ Although the study did not measure the use of non-prescription non-steroidals, this limitation would probably have biased the results towards the null hypothesis and therefore is an unlikely explanation for their findings. By considering patients exposed to non-steroidals only if they had an active prescription for the drug, the authors tried to minimise the misclassification inherent in using a single prescription for a non-steroidal as an indicator of long term use. They did not, however, use a similar approach for aspirin prescriptions. Further acknowledged limitations of the study included the relatively small sample size and inability to adjust for potentially important confounders that are likely to be more common among users of non-steroidals, such as higher body mass index and lower levels of physical activity.¹⁰

Kurth et al examined data from the physicians' health study.¹¹ They found that doctors randomised to aspirin who also reported on an annual survey that they had used non-steroidals (not specifically ibuprofen) for at least five days in a one month period, but not those reporting less frequent use, had an increased risk of subsequent myocardial infarction relative to those randomised to aspirin and not reporting any non-steroidal use. This shows that frequency of non-steroidal use is an important factor. However, only six myocardial infarctions occurred among users of frequent non-steroidals in the aspirin group. Also, because actual use of aspirin

Papers p 1322

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