France pasteurisation of raw milk cheeses is not feasible for cultural, social, and economic reasons."

To me, the managing director of a cheesemonger, this strikes right at the heart of the matter. Pasteurisation is not the obvious answer it might seem. Good cheese can be made from pasteurised milk. Exceptional cheeses can be made only from raw milk: the flavours are so much more alive and vibrant. Pasteurised cheeses always have a dull, subdued character. Not only that, but raw milk cheeses retain more calcium and vitamins and so provide a healthier diet. It is thought that the calcium helps to lower the amount of cholesterol absorbed into the body, thus minimising the risk of cardiovascular disease. Raw milk cheeses probably also harbour fewer pathogens than pasteurised milk cheeses, thus making them "safer." Finally, it was Louis Pasteur himself who said, "I would rather that a child ate bread which had been dragged through the dirt than to grow up on an over sterile diet."

The correct answer is to ensure that the quality of the milk is good. Pasteurisation should be seen as a last resort—an admission of failure of good hygiene—leading to poorer quality, less nutritious cheeses with a greater risk of contamination by pathogens after their production.

Overall, cheese is a safer product than most. As Desenclos and colleagues say, problems are rare. Some illnesses have occurred, which led the chief medical officer to announce, "As a precaution it is recommended that soft cheese, whether made from pasteurised or unpasteurised milk, should be avoided by pregnant women, the very young and the elderly. Pasteurised or unpasteurised hard cheese has not given any cause for concern and is quite safe to eat." Note the references to groups at risk, soft cheeses, and pasteurisation. It is unhelpful for the editorial to perpetuate the myth that universal pasteurisation is the answer.

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1 Desenclos J-C, Bouvet P, Benz-Lemoine E, Grimont F, Desqueyroux H, Rebière I, *et al.* Large outbreak of Salmonella enterica serotype paratyphi B infection caused by a goats' milk cheese, France, 1993; a case finding and epidemiological study. *BMJ* 1996;312:91-4. (13 January.)
2 Rampling A. Raw milk cheeses and salmonella. *BMJ* 1996;312:67-8. (13 January.)

Prenatal and postnatal prevalence of Turner's syndrome

No scientific evidence for study's conclusions

EDITOR,—The members of the board of the Danish Cytogenetic Central Register were surprised to read the paper by Claus Højbjerg Gravholt and colleagues on the prenatal and postnatal prevalence of Turner's syndrome.[1] The authors have seriously misinterpreted the register's data.

The purpose of the paper was to study the prevalence of Turner's syndrome in Denmark and to assess the validity of prenatal diagnosis. The authors attempted to do this by using data on Turner's syndrome in the Danish Cytogenetic Central Register, which includes 100 prenatal and 215 postnatal Turner's syndrome karyotypes. The main outcome measures were the "prevalence of Turner's syndrome karyotypes among prenatally tested fetuses and Turner's syndrome among liveborn infants."

The basic scientific problem is that Gravholt and colleagues compared the prevalence of prenatal cases of 45,X and 45,X/46,XX karyotypes with the postnatal prevalence of Turner's syndrome. This is scientifically meaningless, being a comparison of incongruent measures, since (*a*) only few cases of 45,X/46,XX karyotypes detected prenatally will be diagnosed postnatally as being cases of Turner's syndrome and (*b*) Turner's syndrome is a clinical diagnosis and only a fraction of women with a 45,X or 45,X/46,XX karyotype will be diagnosed postnatally as having Turner's syndrome. Consequently, it is obvious that Gravholt and colleagues would find the "prenatal prevalence of Turner's syndrome higher than the postnatal."

The paper contains no information about the number of cells counted in prenatal cases of 45,X/46,XX mosaicism and the percentage distribution of the two cell lines. There is no information about genetic counselling of the couples associated with such findings and the reasons why the couples might choose to continue or end their pregnancy. There is no long term follow up of children with 45,X/46,XX mosaicism diagnosed prenatally and no information about possible abnormal findings in fetuses aborted because of such mosaicisms. Furthermore, it is wrong to say that "the diagnosis of Turner's syndrome was revised" (postnatally). All eight so called "revisions" were in cases of mosaicism, a condition that can never be ruled out, even from an apparently normal blood karyotype.

Thus there is no scientific evidence to conclude that the study "challenges the predictive value and specificity of prenatal examination techniques in the diagnosis of Turner's syndrome." The insinuation "that perfectly healthy fetuses could have been legally aborted over the years because of false positive prenatal diagnoses of Turner's syndrome" also lacks scientific evidence.

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1 Gravholt CH, Juul S, Naeraa RW, Hansen J. Prenatal and postnatal prevalence of Turner's syndrome: a registry study. *BMJ* 1996;312:16-21. (6 January.)

No reason to doubt standard of prenatal diagnosis

EDITOR,—As participants in the European collaborative research on mosaicism in chorion villus sampling, we wish to comment on Claus Højbjerg Gravholt and colleagues' paper on the prenatal and postnatal prevalence of Turner's syndrome.[1] The authors claim to have assessed the validity of prenatal diagnosis, but their conclusions on the magnitude of the predictive value are based on incomplete data.

Calculation of the predictive value requires that every karyotype obtained on prenatal diagnosis can be classified as false positive, true positive, false negative, or true negative. To do this, the prenatal karyotype must be compared with the karyotype obtained postnatally or after abortion, not with the phenotype of a small proportion of the population investigated. Gravholt and colleagues reported 100 "possible Turner's karyotypes" diagnosed prenatally by either amniocentesis or chorion villus sampling. Calculation of the positive predictive value, however, relied exclusively on the 24 liveborn infants. The follow up karyotypes of the fetuses from spontaneous (five) and induced (71) abortion are not considered in the paper.

In their considerations about the predictive value the authors do not distinguish between mosaicism diagnosed on chorion villus sampling and mosaicism diagnosed on amniocentesis; they thus ignore the fact that the importance of a finding of mosaicism on chorion villus sampling is different from that on amniocentesis owing to the occurrence of confined placental mosaicism. A finding of 45,X/46,XX mosaicism on chorion villus sampling often leads to amniocentesis to evaluate its importance further. Were repeat 45,X/46,XX diagnoses in the same pregnancy properly excluded in the calculations?

Gravholt and colleagues found that the proportion of possible Turner's karyotypes occurring in mosaic form was the same among postnatal diagnoses that were ascertained because of an abnormal phenotype as among prenatal diagnoses. This just shows that not only non-mosaic but also mosaic sex chromosomal aberrations can result in an abnormal phenotype. Genetic counselling after a finding of sex chromosomal aberration is never easy because the phenotypes are so variable—even more so when mosaicism is considered. The couple's decision about the pregnancy is based not only on the karyotype(s) but on several aspects evaluated as a whole, such as the proportion of abnormal cells, the result of ultrasonography if performed, the outcome of prior pregnancies, and the family's situation.

Misinterpretation or manipulation of data causes confusion. There is no evidence to raise doubts about the standard of prenatal diagnosis.

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1 Gravholt CH, Juul S, Naeraa RW, Hansen J. Prenatal and postnatal prevalence of Turner's syndrome: a registry study. *BMJ* 1996;312:16-21. (6 January.)

Author's reply

EDITOR,—Quality assessment sometimes yields unpleasant results or, as in the case of our study, unpleasant questions. The Danish Cytogenetic Central Register gives a unique opportunity to assess the validity of prenatal screening, although the information is incomplete.

Both Claes Lundsteen and colleagues and Johanne M Hahnemann and Lars O Vejerslev criticise our study for mixing "pure" Turner's karyotypes with mosaicisms. Table 1 therefore shows the results for 45,X karyotypes separately from those for other karyotypes. When amniocentesis was used (and correction was made for cases ascertained after ultrasonography) the prevalence of the 45,X karyotype diagnosed prenatally was four times that diagnosed postnatally, and this again raises the question of the risk of a