

Correspondence

The place of birth

Sir,

I was intrigued by John Davis's passing reference in his annotation¹ to 'one set of assumptions leading to eugenics as the other points back towards what is called social Darwinism', although I am not sure about the aptness of the contrast. The term 'social Darwinism' is widely interpreted² and social Darwinists have included advocates both of collectivism and competitive individualism.

However, nowadays it is variation in planned family size rather than in fecundity and infant and childhood mortality that provides the opportunity for genetic change. Eugenic, or dysgenic, trends depend in developed countries on the minority of couples who have more or fewer than the 2 children, which are now the target of a majority.^{3, 4} There will always be some men and women who are infertile; some who, for one reason or another have no liking for children, or for medical or genetic problems or fear of nuclear war feel they should not have any children. This provides the opportunity for some couples to aim at a third or fourth child, without the birth rate exceeding replacement rate.

It is to be hoped that the families of more than 2 children will be those in which the children will, in Professor Davis's phrase, 'achieve maturity and independence as a result of good genes, good fortune, and a good upbringing'. There are indications that such hopes are fulfilled once family size is effectively planned throughout the community. This is to be expected, since on the whole men and women enjoy doing what they do well. This includes rearing children as well as the practice of trade, sport, or hobby. So it is mainly those who enjoy the rearing of their first 2 children who will want to plan a third and fourth. This process might well be called social Darwinism, natural selection operating in the context of the planned family.

References

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Klinefelter's syndrome in adolescence

Sir,

We read with interest the paper by Ratcliffe *et al.*¹ and should like to add some of our own observations, some of which have been published but seem to have escaped the attention of the authors. Klinefelter's syndrome can be diagnosed in early age not only in mentally-retarded children or by screening for abnormal karyotypes, but also by performing a buccal smear on any child who presents with hypogonadism² or cryptorchidism.³ We have followed up 24 boys with Klinefelter's syndrome, 18 of whom were diagnosed prepubertally throughout puberty into adulthood.⁶ Onset of puberty, as judged from testicular enlargement and presence of pubic hair, occurred between 11 and 14 years in the 18 patients. By age 17 years pubic hair, penile length, and height had reached the adult stage in all patients, but arrest of testicular growth was noted at mid-puberty, 13 years, with maximal mean (\pm SD) volume attained being 3.5 ± 1.5 ml. The first conscious ejaculation was reported to have taken place between 13 and 16 years in ten patients and in the remaining four between 17 and 18 years of age. Sperm counts obtained after age 18 showed azoospermia or severe oligospermia in every patient, except one who had a sperm count of 30×10^6 /ml. This man fathered 2 children.⁴ The hypothalamic-pituitary-gonadal axis, assessed by luteinising hormone-releasing hormone and human chorionic gonadotrophin stimulation tests, was found to be normal in prepuberty and during early pubertal stages. From mid-puberty the basal levels of plasma follicle-stimulating hormone and the response to luteinising hormone-releasing hormone showed a gradual increase above the normal. Towards late puberty (>15 years) basal and peak levels of luteinising hormone were above normal with a concomitant decrease in the basal level of testosterone and in its response to human chorionic gonadotrophin.

In many subjects the syndrome is benign and the patient can lead a normal life including army service and marriage.⁵

References

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Dr Ratcliffe and co-workers comment:

The single case with a sperm count of 30×10^6 per ml (and who fathered 2 children) confirms our statement that a note of caution should be introduced in the prognosis of future sterility. It would have been of interest to know how many of the 24 boys with Klinefelter's syndrome that they have seen have developed gynecomastia as this feature, for both patients and parents, would greatly affect a benign image of the condition. Furthermore, if cases are ascertained from an endocrine clinic, selection may exclude those presenting with educational or psychiatric problems, each of which we have encountered in our follow-up of 20 cases identified in the 1967-79 newborn survey in Edinburgh.¹

Reference

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Oral rehydration in acute infantile diarrhoea

Sir,

Sandhu *et al.*¹ reported a significant rise in serum sodium concentration in 4 of 7 infants treated with an oral rehydration solution containing 125 g/l of glucose-polymers (which yields 730 mmol/l free glucose on hydrolysis) and 90 mmol/l of sodium. One patient developed severe hypernatraemia with this solution (serum sodium 162 mmol/l). The authors suggest that such hypernatraemia could be secondary to (1) the high sodium content in the oral rehydration solution; (2) the low sodium diarrhoea caused by infections secondary to agents such as rotavirus; (3) the malabsorption of sugar with resultant water loss secondary to the infectious process. (The glucose-polymer was implicated by the authors in the one patient with severe hypernatraemia).

From these assumptions, Sandhu *et al.* recommended that the sodium concentration in oral rehydration solutions be reduced to as low as 25 mmol/l, particularly in temperate climates.

Several studies have demonstrated the effectiveness of the WHO/UNICEF solution (sodium 90 mmol/l, glucose 20 g/l) in treating diarrhoeas of multiple aetiologies, including rotavirus.² We have successfully used the WHO/UNICEF solution, both for children in

hospital and for ambulatory patients in the the USA without inducing hypernatraemia.^{3,4} The glucose concentration of oral rehydration solutions should be maintained between 56 and 140 mmol/l (10 and 25 g/l) to obtain optimal absorption of water and sodium.⁵ Increasing this concentration beyond this range could potentially aggravate the diarrhoea by osmotic mechanisms, as seen in this study, leading to the increased loss of diarrhoeal stool with a low sodium content. Unfortunately no stool electrolyte studies were carried out.

Sandhu *et al.* recommended the high glucose-polymer concentration, instead of the WHO/UNICEF recommended monomer glucose, to improve the nutritional benefit of oral rehydration. This can be achieved more appropriately by introducing food as soon as rehydration is completed and appetite has been restored, generally within the first few hours.^{3,6} They also suggest that the metabolic responses of patients in temperate climates (implying well-nourished patients) may be different from those of endemically undernourished children, although our studies^{3,4} do not support this nor are we aware of any that do.

The WHO/UNICEF solution, containing 90 mmol/l sodium and 20 g/l glucose has been shown to be safe and efficacious, provided the solution is used appropriately. It is important that free water be allowed after the initial rehydration period.

We disagree with the suggestion that a solution with a lower sodium content should be used to prevent hypernatraemia. The WHO/UNICEF solution can be used safely in both well-nourished and undernourished populations. The study of Sandhu *et al.* only confirms the predicted results when oral electrolyte solutions containing a marked excess of osmotically-active carbohydrates are used.

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