microscope using the rather high electron flux rates required for high resolution. Undoubtedly, there must exist fiendishly high electric field gradients on a highly local level caused by electron–atom collision–induced ionization, and they might be responsible for some positional instabilities of the atoms.

However, one should also consider the effect these charges in the specimen might have on the trajectories of subsequent incoming electrons. They would most certainly distort the optics on a local level. Furthermore, as the charges build up and dissipate — by whatever mechanism(s) — the fluctuating local electric fields cause fluctuating local distortions in the microscope's optics (A.P.K. *Ultramicroscopy* **7**, 351–370; 1982). The net result would be somewhat like looking at a photograph lying (face up) on the bottom of a pool of water whose surface is inundated by random ripples. Is it the painting that is undergoing 'convulsions', or is it just its image?

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HOWIE REPLIES—Specimen charging effects certainly can disrupt the imaging process in the electron microscope, giving rise for example to sudden and large image displacements. It seems unlikely, however, that this can account for the majority of the observed apparent changes of structure, particularly changes from single crystal to multiple twin and back again.

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Legal problems of Huntington's chorea tests

SIR—Recent correspondence on the ethics of using a recombinant DNA probe to diagnose Huntington's chorea has not mentioned counsellors' potential legal obligation to provide such a test, regardless of their personal ethical judgement. That the G8 probe does not provide accurate diagnosis is irrelevant here, as all medical diagnoses provide only evidence for disease states, not proof of them. An established genetic screen, fetal karyotyping to detect Downs syndrome, can give good evidence that a fetus is normal, but can also give false negatives for a mosaic fetus[1] or for one carrying a Downs-causing translocation[2], as well as through sampling or laboratory errors. Counselling about the test for at-risk mothers includes mention of these risks, as will analagous counselling for the G8 probe[3].

The expectation that mothers over 35 years old will receive fetal karyotyping, with attendant counselling, has recently acquired a legal standing, with the award of £35,000 damages to Mrs Ayten Yagiz, who sued the City and Hackney Health Authority after giving birth to a Downs syndrome daughter when she claimed not to have been offered fetal karyotyping[4]. This would appear to establish a precedent for saying that doctors have a legal liability to provide established genetic tests for at-risk mothers. At 40 years old, Mrs Yagiz daughter's risk of Downs was ≃1% (ref. 5), far less than that facing most parents seeking counselling for Huntington's chorea, cystic fibrosis or other inherited diseases for which cloned probes are becoming available. Other analagous cases are currently awaiting trial. Once cloned probes join fetal karyotyping as an established genetic screen, genetic counsellors may find they have an obligation to use them beyond their present research facilities' ability to do so. This may be a constraint on putting such probes into clinical use.

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Is cannibalism all in the mind?

SIR—Behrensmeyer *et al.* show that trampling of bones in sand can produce marks mimicking cutmarks made by flake tools. They comment that the evidence for meat-eating among early hominids is called into question by this finding.

Putative cutmarks on human bones found at various historical sites, notably at Knossos[2], have been used to advance the hypothesis that cannibalism was practised in these cultures. The finding of Behrensmeyer *et al.* raises the possibility that this hypothesis, too, is unfounded and strengthens Arens'[3] contention that apparent evidence for ritual human cannibalism is in fact a psychological phenomenon of anthropologists rather than a dietary phenomenon of 'barbaric' civilizations.

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Linkage between the nation states

SIR—The 'self-thinning' rule of plant ecology[1,2] states that as communities of plants mature, increases in the mean mass per individual plant (W) tend to be accompanied by decreases in the number of plants per unit area (N), and in particular that

$$\log W = A + B \log N$$

where A and B are constants. If B took the value -1 then decreases in N due to self-thinning would be exactly matched by increases in W and the mass of standing crop per unit area ($=W.N$) would be constant ($=A$). In practice[3,4], B tends to take values close to $-3/2$, so that

$$\log W = A - 3/2 \log N$$

Hence

$$\log W = 3 \log(W.N). - 2A$$

and the standing crop increases with W. Denness[5] suggests that the '$-3/2$ rule' applies to the distribution of size and density between the states of Europe; and hence he argues that if the total population is to expand the number of nation states in a given area must decrease. The evidence rests on a log–log graph in which W was equated with nation size (population) and N with 1/(national area) (that is, with number of nations per unit area)[6]. The graph resembled a scatter plot, but the addition of a grid of lines of slope $-3/2$ yielded some apparently meaningful associations between nations[6]. I suggest that any resemblance with the '$-3/2$ rule' of plant ecology is fortuitous.

The figures for population density of

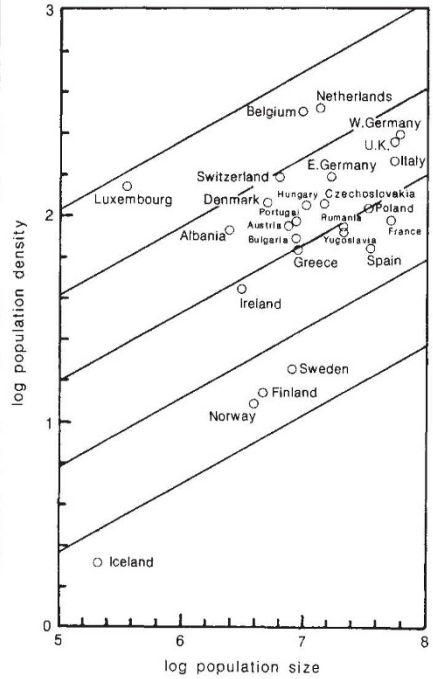

Fig. 1 A plot of $\log_{10}$ population density (people per km^2) against $\log_{10}$ population size (people per nation). The added lines have a slope 1/3.

different nations conceal substantial fluctuations; in cities the number of people per square kilometre may be greater than in rural areas by several orders of magnitude. It seems reasonable to suppose that bigger nations will support bigger conurbations than small, and that hence the mean population density for a large nation may be greater than for smaller ones. If this were so, then nations of similar character might fall into alignment on a graph of log population density on log nation size (Fig.1). They would also give apparently meaningful alignments in graphs of log nation size (W) on log area ($= 1/N$) (ref. 6). In particular, if the slope of the graph of log population density ($=W.N$) on log nation size ($=W$) is approximately 1/3, then the slope of the graph of log nation size (W) on log area ($= 1/N$) will be approximately 3/2; but this does not mean that people obey the same 'laws' of nature as plants. The alignments, I suggest, are attributable to the factors that control the aggregation of people in cities and not to any inherent tendency for the nation states within a continent to undergo 'self-thinning'.

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Is Classic Coca-Cola the real thing?

SIR—During 1985, the Coca-Cola Company discontinued production of its "old" Cola-Cola soft drink and introduced the New Coca-Cola. In response to public demand for the "old" Coke, the Coca-Cola Company began marketing the Classic Coca-Cola. Consumer complaints about difference in taste between the Classic and "old" version were supported by claims from both the Sugar Association, Inc.[1] and *Newsweek*[2] that the Coca-Cola Company had discontinued the use of sucrose (table sugar) in 1984. Since the Classic Coca-Cola container bears the words "Original Formula" and the major ingredient other than carbonated water in both the Classic and the New Coca-Cola is "high fructose corn syrup and/or sucrose", while that of "old" Coca-Cola was "sugar", we determined the sugar content of the newly introduced drinks to compare them with our previous analyses of "old" Coca-Cola.

Sugar analyses, using a gas-liquid chromatographic procedure[3], confirm claims[1,2] that neither Classic nor New Coca-Cola contain sucrose, whereas in 1983 we found that "old" Coca-Cola contained 4.7% sucrose (Table 1). There was also no sucrose in what we refer to as "transition" Coke since it was produced just prior to the introduction of the New Coca-Cola on 23 April 1985. Total sugar content of "transition", Classic and "old" Coca-Cola was very similar, but the new version contains about 10% more total sugars than any of the others.

As noted above, the Coca-Cola labels now read "high fructose corn syrup and/or sucrose", thus eliminating the use of the word "sugar". Recently the cereal industry made a similar move by eliminating the word "sugar" from the name of cereals and from its advertising even though the product and the sugar content remained unchanged[4]. Why discontinue the use of the word "sugar" which most consumers understand to be table sugar? Because health conscious consumers often associate sugar with obesity, diabetes mellitus, heart disease and dental caries[5].

Sucrose (table sugar) has long been considered the mortal enemy of the teeth by virtue of the amount ingested and its pattern of use[6]. What impact upon dental health should one expect from the replacement of sucrose with fructose and glucose in soft drinks? Such an assessment is impossible at the present time because there are no definitive studies which demonstrate clearly the contribution of sucrose, fructose, or glucose in soft drinks to incidence of dental caries.

If sucrose is the arch criminal of dental caries as has been claimed[7], one might expect a decrease in the incidence of dental caries due to the elimination of sucrose from the soft drinks. However, if fructose and glucose are as cariogenic as sucrose in soft drinks, one might expect an increase in the incidence of dental caries due to an increased amount of total sugar as seen in the New Coca-Cola. What effect, if any, the changes will have on consumer demand and/or health remains to be seen.

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Table 1 Sugar analysis of Coca-Cola by gas-liquid chromatography

Product (code)	Date of production	Date of analysis	Sugar content (%)			
			Fructose	Glucose	Sucrose	Total
"Old" (032483289)	24 Mar. '83	12 Apr. '83	3.3±0.1	2.9±0.3	4.7±0.3	10.9
"Transition" (no code)	Early 1985 before 23 Apr. '85	13 Sept. '85	6.3±0.1	4.5±0.1	0	10.8
New Coca-Cola (061485189)	14 June '85	13 Sept. '85	7.0±0.1	4.9±0.1	0	11.9
Classic Coca-Cola (K8H22)	22 Aug. '85	13 Sept. '85	6.3±0.1	4.6±0.1	0	10.9

Sugar contents are mean ± standard deviation of triplicate determinations.

Are we all out of Africa?

SIR—Additional information on world population distributions of human marker genes, such as that provided by Wainscoat *et al.*[1] for five closely linked polymorphic restriction enzyme sites in the β-globin gene cluster, is always welcome. One must agree, on the basis of the restriction enzyme evidence provided, that there is a clear distinction between African and Eurasian populations, an opinion in accord with at least some other genetic studies. While Wainscoat *et al.* are nominally cautious about a second proposition, stating that their data are "consistent with" the notion that anatomically modern man (*Homo sapiens sapiens*) arose in Africa and subsequently spread to Eurasia and the Americas, it is difficult to deny that their results will be taken as "new evidence that the origin of modern man lies in Africa"[2].

A close scrutiny of this new evidence is especially important because of its potential value when the prime archaeological rationale for asserting great antiquity for South African *H. s. sapiens* at Klasies River Mouth and Border Cave is not conclusive[3,4], has been challenged[5], and the *H. s. sapiens* status of the Omo specimens from Ethiopia has been questioned[6].

On the basis of a sample perhaps more limited than the authors concede, Wainscoat *et al.* note that three haplotypes (the specific sequence of appearance [+] or