# Letters

## When Kin Correlations Are Not Squared

In response to a number of inquiries concerning the proportion of genetic variance in IQ explained by the MZA [monozygotic] correlation, we have prepared the following explanation (Articles, 12 Oct., p. 223).

It is a common misunderstanding that the intraclass correlation is squared to estimate the proportion of variance explained by genetic factors. Familial correlations represent components of variance; they are not squared (*1*).

The reason that the intraclass correlation is not squared in our application is that the quantity to be estimated is the proportion of variance in twin A's IQ that is associated with twin A's genotype, and not the proportion of variance in twin A's IQ associated with twins B's IQ. In the latter case, an observed intraclass of 0.70 would be squared to yield an estimate of 0.49 for the proportion of IQ variance shared by the two twins. In the former case, however, the observed phenotypes are imperfect indicators of the underlying genotypes, so that the correlation itself provides a direct estimate of the proportion of IQ variance shared with the unobserved genotype. The situation is analogous to the estimation of reliability in psychometrics whereby the correlation between two parallel forms of a test provides a direct estimate of the proportion of observed test score variance associated with unobserved true score variance (that is, the reliability of the test) (*2*).

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REFERENCES AND NOTES

1. This result is best explicated by the use of a path diagram; see R. Plomin, J. C. DeFries, G. E. McClearn, *Behavioral Genetics: A Primer* (Freeman, New York, 1990), pp. 238–239.
2. This issue is also explicated by A. Jensen [*Psychol. Bull.* **75**, 223 (1971)] from the point of view of reliability theory and the common elements formula for correlation. See, also, the reply to Jensen by J. K. Miller and D. Levine, *ibid.* **79**, 142 (1973).

## Frazil Ice

In the News & Comment article "Zebra mussel invasion threatens U.S. waters" by Leslie Roberts (21 Sept., p. 1371), reference is made to "frazzle" ice. "Frazil" is the correct spelling for the type of ice that blocked the Monroe, Michigan, water intake. This word, of French-Canadian origin, describes ice formed in turbulent, supercooled water. The term, from an Old French word meaning coal cinders (*fraisil*) apparently came into use because of the appearance of the ice.

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## Imprisoned in Sudan

Moneim Attia, an eminent environmental physiologist from Khartoum, returned to Sudan some years ago after training and experience in Germany and Kuwait. His goal was to develop research on the problems raised by the local climatic challenges of his country. He was arrested in his home on the night of 13 January 1990. He has been detained without trial or accusation since then. We understand that his treatment has been inhumane in several ways, such as being kept without communication with his family, being frequently beaten, and being kept blindfolded day and night for long periods. His arrest was ordered by Lieutenant General Omar Hassan Al-Bashir, head of the Revolutionary Command Council for National Salvation, Khartoum, Sudan.

We the undersigned environmental physiologists urge our colleagues from all fields to write to Lieutenant General Al-Bashir, as well as to the ambassadors of Sudan in their countries, saying that they are aware of the bad treatment received by Moneim Attia and that this treatment (absence of trial or accusation, torture) violates several international conventions: (i) the Convention against Torture and Other Cruel, Inhuman or Degrading Treatment or Punishment; (ii) the Covenant of Civil and Political Rights; and (iii) the U.N. Body of Principles.

We understand that several other scientists are similarly detained in Sudan. What we do to defend Moneim Attia will have the general effect of helping protect all scientists who choose to help their countries.

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## Carcinogenesis Debate

In her News & Comment article discussing our papers on carcinogens (9 Nov., p. 743), Jean L. Marx says that our position is, "Below the toxic dose, carcinogenesis would not be a problem . . . because there would be no increased cell proliferation," that is, thresholds are the general case. That is not our view, as is clear from our papers. It is reasonable to assume that low levels of mutagens might add a small increment to our enormous endogenous level of DNA adducts coming from oxidant by-products of normal metabolism. However, the risk should be considerably lower than predicted by linear extrapolation from high dose tests because increases in mitogenesis can be unique to high doses and inducible general defense systems act as a buffer at low doses. The risk from nonmutagens at low doses may be zero (for example, in the case of saccharin). Our view, as can be seen in our papers, is not that mitogenesis is a single-factor explanation for carcinogenesis. Rather our view is that you cannot understand mutagenesis (and therefore carcinogenesis) without taking mitogenesis into account and that at high doses chronic mitogenesis can be the dominant factor. This is also the view of S. M. Cohen and L. B. Ellwein and is supported by their work (Articles, 31 Aug., p. 1007).