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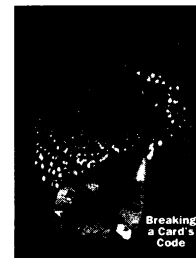
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Cover: Encryption of digital information offers assurance that transactions involving smart cards are secure. However, physical tampering to cause encryption errors creates patterns that can be exploited to reveal confidential information. The colorful swirls in the background graphically represent patterns that emerge when encrypted data no longer appear random. (Image: Cliff Pickover/IBM)



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Letters

Recognizing complex cancer causes

As the environment-versus-gene pendulum has swung toward the genome as the determinant of cancer risk, SCIENCE NEWS has swung along with it ("Gene variations sway prostate cancer risk," SN: 11/9/96, p. 295). I do not follow some of the accompanying logic, however.

I agree that environmental and behavioral risk factors for prostate cancer have not been well identified and that family history is an important factor. I agree that susceptibility genes may soon be characterized. But how do you explain your juxtaposition of

CORRECTION

Sexual Orientation, a book cited in "Animals' Fancies" (SN: 1/4/97, p. 8), was edited by Lee Ellis and Linda Ebertz. Anne Perkins and James A. Fitzgerald contributed a chapter to it.

two facts early in the article: that prostate cancer remains rare in Africa and that black U.S. men have one of the highest incidences in the world, higher than that of white U.S. men? How can these facts be consistent with the statement that genetic variation may account for much, perhaps even most, of the differences in prostate cancer risk?

Geographic variation in cancer rates within ethnic groups (as seen in migrant studies) has long been accepted by cancer epidemiologists as a key argument for the existence of environmental risk factors, even when they remain unidentified.

The new emphasis on genetic determinism when discussing diseases with complex causal pathways is disturbing. People are concerned that their future will be "read" in the DNA, whereas in fact, risk estimates for sporadic cancers remain vague.

At its worst, overemphasis on genetic susceptibility can reduce the study of multifactorial diseases to a hunt for the single big

gene. Such oversimplification even appears now in the study of the tobacco-related malignancy, to the possible detriment of public health efforts.

Let's skip the fad and stick to the reliable and inevitable middle position, with genes and environment interacting to a greater or lesser extent for each malignancy, but always interacting.

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