But his own cinematic contributions attest that while films do not reflect reality, they do reflect our psychic aspirations and terrors and that, in doing so, they should not be devalued or ignored, any more than they should be presented as literally true. Any art form that has people of Crichton's caliber working in it should be taken a lot more seriously than he himself seems inclined to take it.

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Rammal Medal Selection

Certain passages of Michael Balter's article "Physics prize falls foul of Middle East politics" (News of the Week, 5 Mar., p. 1422) might lead the reader to believe that French authorities possibly exerted influence on the deliberations of the prize jury responsible for the awarding of the 1998 Rammal Medal for physics.

I would like to clarify that no intervention was made by the services of the Embassy of France in Beirut to sway, in any way, the decisions of the French Physical Society and the Ecole Normale Supérieure Foundation, the institutions responsible for the adjudication of the Rammal Medal. As the spokesperson of the French Foreign Ministry announced as early as 8 March 1999, these organizations acted autonomously in both the nomination and the determination of conditions for ratification of this year's medal recipient.

The French Embassy in Beirut's role in the controversy was limited to reporting on the adverse reaction in Lebanon to the nomination of an Israeli for the prize, in response to inquiries that it had received on the subject. The Embassy of France did not participate in either the nomination or the review of the 1998 Rammal Medal candidates.

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Response

I reported that the scientific attaché of the French embassy in Beirut contacted the president of the Rammal Medal jury to complain that the jury's decision to award the medal to an Israeli was causing strains in Franco-Lebanese relations. This is supported by e-mail exchanges made available to *Science* and interviews with officials involved. The attaché told me that he acted in a "personal capacity." The French Physical Society (SFP) was still considering the jury's decision at that point, but as I also reported, SFP officials themselves are divided on whether outside pressure, including the views of embassy officials, had any influence on their decision not to award the prize.

—Michael Balter

Mitochondrial Recombination?

In her article "Can mitochondrial clocks keep time?" (News Focus, 5 Mar., p. 1435), Evelyn Strauss discusses a paper by Adam Eyre-Walker *et al.* (*1*) questioning the strict maternal inheritance of human mitochondria. Given that the large body of vertebrate phylogeny (as well as population studies using mitochondrial genetic information) rests on the assumption that the vertebrate mitochondria are clonal in their mode of inheritance, evidence of recombination between maternally and paternally derived mitochondria would seri-

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ously question a large set of conclusions, including clock inferences, "the mitochondrial Eve," and countless phylogenies.

Eyre-Walker *et al.*'s conclusion rests on one observation, that a "phylogenetic tree constructed using human mitochondrial sequences contains a large number of homoplasies." The paper concludes that this high number of homoplasies must be the result of recombination between maternal and paternal lineages.

However, a fundamental principle of their tree construction algorithm is that hypothetical ancestral types at nodes of the tree all are extinct. There is no reason to believe that this is a valid assumption for populations in general, and specifically in the case of humans, where the time since a common ancestor probably is only around 200,000 years. The likely presence of haplotypes ancestral to other recorded haplotypes in the analyzed population sample will automatically lower the number of inferred homoplasies and could even remove them all.

The high number of homoplasies claimed by the authors might well be an artifact of the analysis applied. An answer to the important question of mitochondrial recombination is likely to stem from the application of more appropriate analytical tools to the now very large database of human mitochondrial sequences.

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Are Centrosomes or Aneuploidy the Key to Cancer?

A News of the Week article by Elizabeth Pennisi about centrosome replication (5 Feb., p. 770) points out that centrosomes "might be a key to cancer." Indeed, abnormal structures and numbers, that is, more than two, of centrosomes have already been found in nearly all cancers tested (*1, 2*). The idea that centrosomes might be a key to cancer originated with Theodor Boveri, who proposed almost a century ago that multipolar mitosis causes aneuploidy, an abnormal balance of chromosomes, and that aneuploidy causes cancer (*3*).

However, as Pennisi says, Boveri's "idea got lost, as researchers concentrated on understanding the specific gene malfunctions that lead to cancer." Accordingly, gene mutations are now investigated to explain the abnormal centrosomes and the aneuploidy of cancer cells. For example, mutant p53 (*4*) and an overexpressed centrosome-associated kinase SKT15 (*5*) are thought to destabilize centrosome replication, and a defective mitotic checkpoint gene is thought to cause chromosome nondisjunction (*6*).

But these mutations are consistently found in only a minority of cancers. The p53 mutation is found in fewer than half of aneuploid and genetically unstable colon cancers (*7*); only 12% of primary breast cancers contained the mutant kinase (*5*); and only two of 19 aneuploid colon cancers contained the mutated, putative checkpoint gene (*6*). In contrast, nearly all cancers are aneuploid (*8*) and have abnormal centrosomes (*1, 2*). In addition, there is as yet no functional proof that the mutations cause aneuploidy.

I therefore suggest that both the abnormal structures and numbers of centrosomes in cancer cells are caused by aneuploidy (*9–11*). Because aneuploidy unbalances huge numbers of genes, it can readily explain, by abnormal dosages of normal genes and thus independent of gene mutation, not only abnormal centrosomes, but also the many other dominant, cancer-specific phenotypes, including dedifferentiation, neoantigens, metastasis, altered growth, morphology, and metabolism (*9, 11*). All these complex phenotypes are determined in vivo by assembly lines of many kinetically linked enzymes (*12*). It is for this reason that aneuploidy can change cellular phenotypes much better than gene mutation. Aneuploidy changes cellular phenotypes like a car factory changes its output, by altering the number of assembly lines. Balanced changes produce more or fewer normal cars, but random changes (aneuploidy) produce abnormal cars (cancer). By contrast, negative gene mutations are typically recessive; even rare positive mutations are buffered by up- and downstream enzymes of an assembly line (*12*) and thus are unlikely to generate the dominant phenotypes of cancer cells (*13*). In support of this view, some researchers interviewed by Pennisi propose that aneuploidy causes cancer by altering the dosage of apparently unmutated "oncogenes" and "tumor suppressor genes." But they do not suggest that changing the dosage of one gene by means of the chromosome number simultaneously changes that of thousands of others.

Indeed, aneuploidy can even explain the "genetic instability" of cancer independent of gene mutation, because aneuploidy generates nonparental karyotypes autocatalytically (*10, 11*). [This hypothesis does not exclude that mutation of mitosis genes also may cause aneuploidy as, for example, in Bloom's syndrome (*14*)]. My hypothesis further predicts that carcinogens function as aneuploidogens, which has been demon-