

alyz[ing] the activities of our laboratories.”

And many researchers say they do not believe the proposed merger is necessary. “I do not see a reason to upset everything,” says André Rougé, a particle physicist at the Ecole Polytechnique in Palaiseau, outside Paris. “There are already collaborations and joint labs. If the structures become too complex, it could stifle new initiatives.” Researchers are particularly concerned that a rapprochement between IN2P3 and DAPNIA might be a first step to IN2P3 being swallowed up by the CEA. With responsibility for research into nuclear energy and atomic weapons, the CEA is seen by many scientists as having different priorities from the basic science mission of the CNRS. “We will get lost in the CEA’s objectives, even in basic research,” says physicist Harry Bernas of the University of Paris at Orsay, a former director of the IN2P3 laboratory on that campus. Bernas adds that such a development would present a “great danger,” especially in politically sensitive areas such as nuclear waste research, where the “CNRS provides the only independent evaluation” of government policy.

Courtillot counters, however, that CNRS researchers’ fears about the CEA’s nuclear priorities are “not warranted,” arguing that DAPNIA has long had a reputation for doing independent fundamental research on its own. The question should be settled sometime in the next month or two, when Allègre is expected to take action on the Aubert report’s recommendations. Says Brézin: “France cannot have two research strategies in this domain. There must be one French policy.”

—MICHAEL BALTER

GENETICS

Discovery of ‘Gay Gene’ Questioned

Six years ago, molecular geneticist Dean Hamer and his colleagues at the National Cancer Institute (NCI) announced to great fanfare that they had found a genetic link to male homosexuality. Their work indicated, they said, that an as yet unidentified gene on the X chromosome influences who develops the trait (*Science*, 16 July 1993, p. 321). Researchers were excited by the possibility of one day learning the biological basis for sexual orientation but also wary, given that initial reports of genetic linkages for other complex traits, such as manic depression and schizophrenia, had fallen apart under further scrutiny. Now the “gay gene” linkage may be suffering a similar fate.

On page 665, clinical neurologists George Rice and George Ebers at the University of Western Ontario in London and their colleagues report failing to find a link between male homosexuality and Xq28, the

chromosomal segment implicated by the NCI team’s study. In addition, unpublished work from a group led by psychiatrist Alan Sanders at the University of Chicago does not provide strong support for a linkage. Taken together, Rice says, all the results “would suggest that if there is a linkage it’s so weak that it’s not important.” He adds that genetics may still contribute to homosexuality, but researchers should be looking elsewhere for the genes.

Hamer disagrees that the Xq28 linkage is weak, citing possible problems with how Rice’s team selected their study subjects. And other observers say that the jury is still



The challengers. From left to right are Western Ontario team members Keith Cousins, George Ebers, Holly Armstrong, George Rice, and Harriet Margalies.

out. Elliot Gershon, a psychiatric geneticist at the University of Chicago, calls the Ontario team’s finding “interesting and important” but cautions that more data are needed. “Failure to find linkage in this study does not mean it doesn’t exist,” he says.

That genes may contribute to homosexuality in males became clear in 1991 when psychologist Michael Bailey of Northwestern University in Evanston, Illinois, found that fully 52% of the identical twins of gay men were also gay, compared to just 22% percent for fraternal twins. Then in 1993, Hamer’s team pointed to a place where a putative “gay gene” might reside.

They homed in on the X chromosome, which males inherit only from their mothers, because they noticed a preponderance of gay relatives on the maternal side of the families of the gay men they studied. When the researchers took a closer look at the X chromosomes of 40 pairs of gay brothers from the families with maternal gay relatives, they saw that the brothers were far more likely to share certain DNA signposts, or markers, on the Xq28 region of the chromosome than would be expected by chance. The team confirmed the linkage in a second study of 33 new families with gay brothers, published in *Nature Genetics* in 1995. In this X chromosome snippet, the researchers concluded, lay a gene that

could nudge males toward homosexuality.

Meanwhile, intrigued by the initial report, Rice and Ebers undertook their own study to see if the result would hold up. They recruited families with two or more gay brothers through ads in Canadian gay news magazines. The families responding to the ads included 52 pairs of brothers willing to donate blood, which the researchers examined for the presence of four markers in region Xq28, using methods similar to those employed by Hamer’s group.

But the Ontario team found that gay brothers were no more likely to share the Xq28 markers than would be expected by chance.

And although a statistical analysis of the data could not rule out the existence of a gene in this region with a small influence on the trait, it could exclude the possibility of any gene in Xq28 with a major genetic influence, say, doubling a male’s chances of being gay. Ebers interprets all these results to mean that the X linkage is all but dead. “What is troubling is that there is no hint or trend in the direction of the initial observation,” he says.

Hamer, however, thinks that the way the Ontario researchers selected the families would tend to hide the Xq28 contribution.

He always said, he points out, that the gene does not influence all cases of male homosexuality but only those that are transmitted maternally. And in contrast to his group, Hamer says, the Ontario team did not select families based on the presence of maternal transmission. “Maybe there was an X chromosomal linkage in some families, but those families weren’t analyzed,” Hamer says.

Ebers says they didn’t select their families based on maternal transmission because they found no convincing evidence for such transmission in the family pedigrees. What’s more, even after his group removed two families that might wash out an X chromosome effect because there were signs of the trait in females or in the father, the results remained the same. Nor was the effect evident in a study led by Sanders, which he reported last June at a meeting of the American Psychiatric Association. His team had found only a weak hint—that wasn’t statistically significant—of an Xq28 linkage among 54 gay brother pairs.

A much larger study, using, say, 200 gay brother pairs, could probably resolve the issue, researchers say, but funding for such a project has been hard to obtain. So could any successful efforts to pluck out a gene in Xq28, something Hamer’s group is pursuing. But the Ontario team doubts that route will pay off. “We’re looking for a link on other chromosomes,” Rice says.

—INGRID WICKELGREN