

BEHAVIOR

Questioning laid-back sex

Impotence and frigidity are believed to be associated in some cases with anxiety, so sex therapists often counsel patients to approach sex with a more relaxed outlook and not to worry about performance. But recent studies at Brown University indicate that anxiety often can *contribute* to sexual arousal, reports John P. Wincze, a Brown professor and director of the sexual performance laboratory at the Providence (R.I.) Veterans Administration Hospital.

Wincze first shows an anxiety-provoking film — such as an Alfred Hitchcock thriller or a tape of a violent auto accident — to some of his subjects, and a “neutral” film to others. All subjects are then immediately shown a sexually arousing film. Their heart and respiration rates and other responses are recorded, as well as a “rating” of their own arousal. Preliminary findings suggest that people respond more readily to erotica following an anxiety-producing film than after a neutral one.

“Of course,” Wincze says, “it might be that they are so relieved to be away from the anxiety-producing film that they evaluate the erotic experience more highly. But an alternative explanation is that the anxiety has physically primed them for sexual response. Some of the physiological reactions to anxiety ... are similar to the cues that signal sexual arousal and, under some circumstances, may ‘prime’ the body for sexual activity.”

Lithium toxicity

Although lithium appears to help in the treatment of many manic-depressives, cases continue to accumulate that document the toxic effects of the drug in some patients (SN: 5/26/79, p. 345). Such adverse reactions usually consist of various combinations of tremor, drowsiness, confusion, disorientation, delirium, and even seizures, coma or death.

Among the latest reports is an account of five apparent cases of “severe neurotoxicity during lithium treatment” in which patients were receiving relatively standard doses of the drug. The results indicate that certain types of patients may be more prone than others to toxic reactions to lithium, report A. Preston West of the Neuropsychiatric Institute at the University of California at Los Angeles and Herbert Y. Meltzer of the University of Chicago Pritzker School of Medicine.

Patients who exhibit relatively high levels of psychotic symptoms and anxiety appear to be more susceptible “to the development of severe lithium neurotoxicity,” the researchers report in the July AMERICAN JOURNAL OF PSYCHIATRY. “The identification of psychological factors ... that are associated with lithium neurotoxicity may help us understand the biology of this puzzling toxic syndrome.”

Brains: The younger the better

Children’s brains are generally considered to be less developed but at the same time more “plastic” and better able to learn and adapt than adult brains. Now, from the University of Chicago comes confirmation that humans appear to have 50 percent more brain cell interconnections, or synapses, when they are infants than when they grow to adulthood. The results are reported by researcher Peter Huttenlocher, who used an electron microscope to analyze the frontal cortex cells of 21 human brains, of varying ages, after death. He found synaptic density at its peak at about two years of age, after which it declined until 16 years of age; after that, it remained fairly constant until age 72. The “initial overproduction of the synapses” may account for a child’s ability to recover speech following a brain injury, or to readily learn to speak second languages without an accent, says Huttenlocher, a pediatrics and neurology professor.

BIOLOGY

Julie Ann Miller reports from Bar Harbor, Maine, at the 50th Anniversary Symposium of the Jackson Laboratory

The mapping of a mouse

Jackson Laboratory biologists “would rather fight than switch,” Eva Eicher said in summarizing the laboratory’s past and continuing research on mice and their genes. While other geneticists flocked to microorganisms, insects and even parasitic worms, in Bar Harbor, Maine, the mouse remained king. That single-mindedness has paid off in the accumulation of mice with a wide variety of characterized genetic defects and in the collection of copious information about their genes. Eicher apologizes for the mouse genetic map being a blur of too much information. Currently, 423 genes appear on the diagram and another 300 are awaiting assignment. Genes have been discovered on each of the mouse chromosomes including, recently, the Y chromosome. Just this year, Eicher points out, a large group of genes had to be relocated from one chromosome to another. Eicher and Janan T. Eppig are now developing a computer program that will help map genes and make that map a more useful tool.

The map of mouse genes reflects genetic relationships among the hundreds of traits examined, but it does not correspond precisely to distances along the chromosomes. “We want to anchor the genes to the chromosomes,” Eicher says. To do this, she and co-workers are using mouse ovarian tumors in which the cells do not divide normally. Abnormalities of the chromosomes viewed microscopically can be correlated with the genes expressed. In the future, Eicher expects scientists to do less exact mapping, but simply assign genes to sections of chromosomes. She predicts a series of easily detected traits will be chosen as “core genes” and a few carefully planned matings will determine the relative position of a gene being studied.

“Comparative mapping of man and mouse are not as far apart as we previously thought,” Eicher says. Already there are a few instances of genes grouped together in both species. Eicher predicts that eventually scientists will be able to rearrange a diagram of the human genes into the mouse genetic map.

Viral clues to cancer biochemistry

Whether or not viruses cause human cancers, studies on cancer-causing animal viruses are expected to point out genetic changes that contribute to the malignancies. Researchers working on the avian sarcoma virus recently demonstrated that it can use a normal cellular gene to initiate cancer (SN: 5/26/79, p. 344). Now, David Baltimore of Massachusetts Institute of Technology reports a similar situation for another cancer-causing virus.

The chromosome of Abelson mouse leukemia virus contains the end segments necessary for the virus to function (segments shared by other RNA viruses) and a center portion homologous to a portion of DNA in normal mouse cells. In infected cells, the viral gene makes a protein similar to a normal cell protein, but in amounts up to 100 times greater than normal. In addition, the normal cell product is made predominantly in thymus cells, whereas the disease affects lymphocytes not made in the thymus. Baltimore suggests that the virus causes cancer by producing too much of a natural cell product or by producing it in cells of a type not equipped to handle it.

The protein made by the Abelson virus gene, and also that of the avian sarcoma virus gene, is an enzyme that attaches phosphate groups to specific proteins. However, the specificities of the enzymes coded by the two viruses are different.

Baltimore finds the Abelson virus-coded protein in the outer membranes of mouse cells. Previously, investigators had been confident that cancer-causing agents work primarily in the cell nucleus. Baltimore suggests that, in this case, an altered membrane component might induce cancer by continually importing molecules that keep the cell growing or by acting as a receptor to make the cell abnormally responsive to a growth factor.