Neurosciences in Washington, D.C., was established in order to increase the effectiveness of Fidia's research and development efforts. The Fidia Research Foundation sponsored top-level scientific meetings with prestigious lecturers, published authoritative books and journals, instituted awards and scholarships, and created a postgraduate International School for the Neurosciences. These initiatives were expressions of our wish to be as near as possible to the heart of scientific progress and were not even remotely thought of as an improbable means to "corrupt" scientists or to enhance obscure research data.

All of the above initiatives are now defunct, because of the unfortunate reversal Fidia took after I left the company in 1991, but the strategic concept is still very much alive. For more than 4 years, a select group of researchers has been working with me in a new health-care concern, Lifegroup, in intensified interaction with academic centers of excellence, foremost among them the group led by Levi-Montalcini. Her keen intuition about the role of nerve growth factor in the homeostatic interplay between the neurons and immune systems (*3*) led us to discover surprising new leads in immunomodulation and inflammation control (*4*).

As such successful industry-academic collaborations may come under scrutiny, I should like to invite debate about this critical issue. There appear to be no general rules or guidelines, either for academic researchers or for industrialists, about how to perceive and how to live with these interactions. What are the limits beyond which research sponsorship becomes too lavish? What should the independent researcher accept or reject in terms of collaboration with industry? Through public discussion of these questions, we may be able to reach a consensus, guaranteeing the kind of mutually rewarding interaction that both industry and academy pursue.

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Aggression in Mice and Men?

The title of the report "Aggressive behavior and altered amounts of brain serotonin and norepinephrine in mice lacking MAOA" by Olivier Cases *et al.* (23 June, p. 1763) implies a correlation between aggressive behavior and the absence of monoamine oxidase A (MAOA) in mice, and its first sentences go on to draw a parallel with an hypothesized association between MAOA deficiency and "aggressive behavior" in men in a Dutch lineage (*1*). Yet Cases *et al.* describe phenotypic deficiencies in their mice ranging from "head nodding" and "trembling" to "moving backward," "frantic running," hunching, sleep abnormalities, and other developmental problems. Adults show abnormal swimming, hunched posture, and almost parenthetically we learn that they carry a retinal degeneration gene and are blind. Among all these massive and diverse deficits, Cases *et al.* draw attention to one, that resident males attack intruders faster than do mice with MAOA. This then becomes the focus of their statement that "aggression" in mice and men may be directly correlated to a specific gene deficit. Drawing causal or even correlative connections between complex socially defined behaviors and particular cellular and molecu-

lar processes is notoriously problematic. In a field fraught with implications for those concerned with the roots of violence in human society, caution is particularly required (*2*). Neither the title of the report nor its conclusions are adequately sustained by the complex behavioral data presented, and yet these have already been seized upon by the media—at least in the United Kingdom—and are likely to prove grist to the mill of ideologues and some politicians as supportive of biologically determinist views.

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Response: In our report we demonstrated that aggressive behavior and alterations in the cerebral cortex of the transgenic mouse line Tg8 resulted from the genetic loss of MAOA. The primary index of aggression was the bite wounds of Tg8 males housed in groups. Male mice of the C3H strain, similarly grouped, did not show such wounds. We emphasized the correlation between aggressive behavior and the absence of MAOA in our title and in the conclusion of our report to answer those who have suggested that aggression in MAOA-deficient men is merely a result of the "frustration" of being "in the middle of families of unaffected people" (Peter Breggin, as quoted by Charles C. Mann, Special Report: Genes and Behavior, 17 June 1994, p. 1689).

When listing several of the behavior traits of mouse pups, Rose omits mentioning that Tg8 pups bit the experimenter while C3H pups did not. With regard to the beam-walking test, the fact that Tg8 *and* C3H mice carry a retinal degeneration gene was stated in our report; blindness may be a factor contributing to the poor performance of the Tg8 mice. Thus Rose's rejection of our conclusions would appear to be less founded on our experimental results than on his own opinion (*1*, p. 381) that to describe dramatic changes in neurotransmitters "as if they were the cause of particular behaviours is to mistake correlation or even consequence for cause." Does Rose really believe that the "distress" of Tg8 pups causes the excess of serotonin (a ninefold excess in the brain at birth)? We maintain that this excess of serotonin is a causative factor of the pups' altered behavior.

Rose has also criticized (*1*, p. 380) the 1993 report by Brunner *et al.* (*2*) about abnormal behavior in men who are deficient in MAOA. Such men display retarded motor development, outbursts of threatening words and gestures, awkward sexual behavior, difficulties in task planning, and disrupted sleep. Much more rarely, excessive acts such as arson or attempted rape have been committed. Tg8 mice display abnormal motor development and disrupted sleep, aggressive behavior, altered sexual behavior, and altered reactions in new situations. We agree that experimental results must be examined scrupulously, but do not agree that such comparisons are in essence misleading.

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Women, Math, and Test Scores

The article "Sex differences in mental test scores, variability, and numbers of high-scoring individuals" by L. V. Hedges and A. Nowell (7 July, p. 41) does not adequately address the question of mental test validity in predicting youth's performance in science and math courses or related careers. Studies have shown that standardized test scores tend to underpredict college grades for women and may overpredict for men (News & Comment, 17 Feb. 1989, p. 885); performance in school involves more than the ability to do well on standardized tests. In addition, the fairness of these tests for all populations, especially for girls and minority students, must be seriously questioned.

We find particularly disturbing the statement by Hedges and Nowell (p. 45) that "differences in the representation of the sexes in the tails of ability distributions are likely to figure increasingly in policy discussions about salary equity." Using standardized test scores as an argument for justifying salary inequity would be silly. It is job performance, not test scores, that should determine salary and advancement. This performance depends on many factors, including ability, education, training, work environment, and such personal qualities as motivation, commitment, and creativity. Test results do not predict on-the-job performance for the teacher, physician, lawyer, or scientist.

Finally, it concerns us that Hedges and Nowell suggest intervention for boys to raise their low literacy scores, but do not recommend interventions known to help girls reach their full potential in math and the sciences. Does this omission imply that girls cannot be helped? As women in science, we find this a dangerous assumption. Let us not exclude one-half of the population in our quest for math and science literacy.

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