Malnutrition damages early nerve cells

MIT scientists have found direct biochemical evidence of damage to brain neurons (nerve cells) due to malnutrition during the few weeks before and after birth.

In the MIT studies, the brains of rats that were undernourished from mid-gestation and killed at weaning contained about 20% less norepinephrine than the brains of adequately fed control animals. The chemical, norepinephrine, is a neurotransmitter—secreted in the brain to use it to communicate with one another.

Signal process

Signals travel electrically through the long body of a neuron until they reach a synapse, the connecting gap between the endings of two neurons. When the signal across one neuron reaches the synapse, it causes a chemical neurotransmitter to be released into the small space between the cells. The neurotransmitter chemically triggers the next neuron. If there is an inadequate supply of neurotransmitter for the neuron to release, then the normal passage of signals cannot take place.

Writing in the current issue of the journal Science, William J. Shoemaker, a graduate student, and Dr. Richard J. Wurtman, professor of endocrinology and metabolism in MIT's Department of Nutrition and Food Science, report what Wurtman calls a "first small step in understanding how protein malnutrition affects brain neurons.

There has been mounting evidence that inadequate protein early in life—people—really determines the development of the brain, with the ability to learn, and with behavior. Several investigators, notably Dr. Myron Winick and his associates at the Cornell University Medical School, have discovered chemical changes in the brains of malnourished animals and children. However, these changes—in the amounts of DNA and fatty substances—might reflect disturbances in the supporting cells of the brain or in the myelin membranes that insulate the nerves.

Different kinds of neurons use different kinds of chemicals as their neurotransmitters. Those which release norepinephrine are known to have a role in the control of mood, in the learning process, and in the control of blood pressure, heart rate, blood sugar, and glandular function. Another neurotransmitter, dopamine, also was found to be deficient in undernourished rats in the MIT studies, but this effect was not nearly as dramatic as the effect on norepinephrine. Low brain dopamine levels previously have been noted in Parkinson's disease.

In the work of Shoemaker and Wurtman may be applicable to the study of human diseases. Kwashiorkor, for example, is a condition of acute protein starvation which appears among children one to three years old in economically undeveloped areas. Even when an afflicted child grows to adulthood, Wurtman explains, his behavior and learning ability may be impaired.

Preliminary results of research on kwashiorkor being conducted by MIT scientists in Guatemala suggest that similar changes in norepinephrine metabolism take place in the human brain under conditions of inadequate protein early in life. Wurtman cautions, however, that the preliminary results for humans do not constitute conclusive evidence.

Dr. Wurtman does not yet know if the norepinephrine deficiency in rats results from fewer norepinephrine-containing neurons, or because there is less neurotransmitter present in each of a normal number of neurons. If the number of neurons is normal, then the disorder might be reversible by correcting the dietary protein deficiency or by drugs. However, if the number of norepinephrine neurons is depressed, the disorder may be incurable, since brain neurons lose the ability to divide long before the rest of the body is mature. Future studies with rats and primates may provide insights into the reversibility of these changes in brain norepinephrine.

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