Behavior

Depressed to the bone. . .

Hormonal changes that accompany recurring bouts of severe depression reduce bone density in the lower back and increase the likelihood of developing osteoporosis, a bone-thinning disease, a new study finds.

"Depression is by no means only a 'mental' disorder," a team of German psychiatrists concludes in the November AMERICAN JOURNAL OF PSYCHIATRY. "This study identifies [it as] a new clinical risk factor for osteoporosis."

Many depressed people exhibit high concentrations of cortisol in their blood. Excess amounts of this hormone in other conditions, including anorexia nervosa, have been linked to osteoporosis.

Ulrich Schweiger and his colleagues at the Max Planck Institute of Psychiatry in Munich used a computerized tomography (CT) scanner to measure the density of the three lowest bones in the back. They recruited 53 women and 27 men diagnosed with major depression, as well as 27 women and 30 men free of mental and medical disorders. Participants averaged about 60 years old, and none was younger than 40.

Overall, bone density in depressed volunteers fell about 15 percent below that in the healthy group. Depressed women displayed the lowest average bone density. This pattern held when the researchers controlled statistically for other influences on bone density, such as weight, height, prior medical problems, cigarette smoking, and estrogen treatment in the female volunteers.

"These are intriguing findings that need to be confirmed in further studies," remarks Dan G. Blazer, a psychiatrist at Duke University Medical Center in Durham, N.C. "The results are statistically significant but not dramatic."

Future studies should consider whether major depression fosters poor eating habits, which can undermine bone density, Blazer asserts. The long-term effects of estrogen use on mood, hormones, and bone density in depressed women also require closer examination, he adds.

. . . and aided by a shocking paradox

When antidepressant drugs and psychotherapy fail to lift the dark veil of severe depression — especially if suicide seems likely — electroconvulsive therapy (ECT) becomes a treatment option. Psychiatrists generally view ECT as an effective way to pull someone out of a depression temporarily, even though the technique elicits intense controversy outside their profession.

A clear picture of the types of brain changes ECT produces to relieve depression has yet to emerge. But researchers now report that successful ECT involves a biological paradox. Several studies have found reduced blood flow in the brains of untreated depressed people, compared to healthy adults; but cerebral blood flow declines even more in people whose depression eases after ECT.

A total of 54 individuals diagnosed with major depression completed the study. Mitchell S. Nobler, a psychiatrist at Columbia University, and his coworkers tracked cerebral blood flow with sensors placed on the scalp to measure the escape of minute amounts of a radioactive substance inhaled by participants.

The 28 participants whose condition improved after a session of ECT showed drops in cerebral blood flow about 1 hour after treatment. Those who responded to ECT displayed further decreases in blood flow over the course of treatment, which averaged about nine sessions, the researchers assert in the November ARCHIVES OF GENERAL PSYCHIATRY.

Any explanation of how this effect eases depression remains speculative, Nobler's group notes. ECT's anticonvulsant properties may quell the activity of the few brain areas that become overactive during depression, they theorize (SN: 9/12/92, p.165).

Biomedicine

Kathy A. Fackelmann reports from Reston, Va., at the Conference on Advances in AIDS Vaccine Development

Bacterium guards against HIV

It's not just the stuff of bad cheese anymore.

Two years ago, investigators identified the bacterium *Listeria monocytogenes,* which had fouled soft cheeses, as the cause of many cases of a flu-like illness in several states.

Now, researchers at the University of Pennsylvania School of Medicine in Philadelphia are trying to transform this troublesome bug into a vaccine for HIV, the AIDS virus.

Yvonne Paterson and her team began their experiment by inserting a specific HIV gene into the chromosomes of *L. monocytogenes.* They found that, when grown in culture, these genetically engineered bacteria use the information encoded by the HIV gene to manufacture one of the virus' protein products.

Paterson's team decided to immunize mice with the newly transformed bacterium suspended in a solution. After giving the animals an intramuscular injection of this vaccine, the researchers found several encouraging signs of protection.

White cells called T lymphocytes taken from the mice secreted large amounts of gamma interferon, a substance thought to keep HIV at bay, the scientists discovered. In addition, they found, such mice showed long-term evidence of killer T cells directed against HIV.

Once *L. monocytogenes* has set up shop inside a cell, the researchers believe, the altered bacterium starts to produce the HIV protein. The cell's machinery recognizes that viral protein as foreign and escorts it to the surface of the cell — where it flags the attention of killer T cells.

The researchers hope that such a process will yield protective immunity against HIV. In the event HIV does get into the bloodstream of an immunized animal, the T cells will be primed to attack, Paterson says.

L. monocytogenes can cause illness in people with impaired immune systems who eat contaminated food. Yet people with HIV appear relatively resistant to this bug. "You'd expect AIDS patients to be full of *Listeria,* but they're not," Paterson says. Still, the safety and efficacy of this approach remains to be proved, she adds.

One HIV strain defends against another

Researchers know that infection with a mild strain of a virus often shields against a future bout with a related strain. Would chimps previously infected with one strain of HIV show protection against another?

Malcolm Martin of the National Institute of Allergy and Infectious Diseases in Bethesda, Md., and his colleagues decided to see. To find a novel isolate of HIV, they began by screening blood taken from AIDS patients. The researchers eventually discovered a new strain of HIV, dubbed DH12, which appears to infect chimp cells as easily as human cells. Next, they looked at two chimpanzees that had been infected with a laboratory strain of HIV many years ago. The team then gave those chimps varying doses of DH12.

The researchers could find no sign of this new virus in the blood of the chimps. "As far as we could tell, there was no evidence of DH12 infection," Martin says.

In contrast, control chimps that had never been exposed to any type of HIV also received injections of DH12. The controls developed a rash and swollen lymph nodes. Chimps generally develop an early infection with HIV but do not develop AIDS.

Are the two chimps protected from DH12? Nobody knows for sure, but preliminary evidence from Martin's laboratory looks promising. The chimps developed "sky-high" concentrations of antibodies against DH12, Martin reports.

Ultimately, such research aims to develop a human vaccine for HIV — perhaps a weakened virus that could provoke a mild infection but never develop into the panoply of illnesses that kill people with AIDS.