No blue genes?

Because depression is known to run in families, scientists have searched hard for evidence of hereditary transmission. One of the most encouraging leads came two years ago when University of Rochester geneticist Lowell R. Weitkamp reported that susceptibility to depression was linked to variation in a particular component of the body's immune system known to be encoded on chromosome six (SN: 12/5/81, p. 356). Now, however, two independent research groups have reported that those early findings are not holding up.

Weitkamp, working with University of Toronto psychiatrist Harvey C. Stancer, reported that in families with two depressed offspring, the depressed siblings tended to share a particular type of HLA (human leucocyte antigen). They interpreted this to mean that the genes predisposing people to depression are physically linked to (and are therefore transmitted in tandem with) the HLA genes in question. But Weitkamp's methods have been subject to criticism since the time of the original report, most notably by National Institute of Mental Health researchers Lynn Goldin and Elliot Gershon. Specifically, the government scientists argue, Weitkamp found a linkage between HLA type and depression only in families with two depressed offspring; when families with three or more depressed siblings were included in the analysis, the distribution of HLA types was predictable by chance. Weitkamp's reasoning was that in families in which only two of the offspring were depressed, the parents carried fewer depression genes—and that as a result the increased chances of sharing a HLA type would be more easily detectable. This reasoning, Goldin and Gershon argue, is fallacious. Nevertheless, when they did their own linkage study they tried it both ways: In neither analysis did the distribution of HLA type appear to exceed chance.

Most recently, researchers at The Jewish Hospital of St. Louis have reported that they, too, have failed to replicate Weitkamp's findings. Brian K. Suarez and Theodore Reich, writing in the January Archives of General Psychiatry, report that even in a patient sample "purified" by removing manic depressives (perhaps a genetically distinct subtype of depression) no significant HLA linkage could be found.

Weitkamp defends his reasoning in limiting the study to families with two depressed siblings, arguing that families with more than two are most likely carrying additional depression genes unrelated to HLA. In fact, he says, it might be more appropriate to study depressed cousins rather than siblings; cousins sharing other hereditary diseases, such as multiple sclerosis and Hodgkin's disease, have been shown to share HLA type (a small number of depressed cousins in his original study also did, he notes). He also points to several studies of another kind that have shown an association (in unrelated subjects) between depression and particular HLA antigens; each of these studies has been dismissed as statistically insignificant, he notes, but collectively they point to what might still be a significant association. "There's still a lot of smoke there," Weitkamp says. "I'm not backing off—not yet. We're pursuing this vigorously."

Changing ideas in depression

A leading theory about depression says that serious mood disturbance has it roots in distorted thinking—and that depressed patients can be taught not to generalize one unhappy experience (a personal rejection, for example) into a general sense of worthlessness. Such teaching — known as cognitive therapy — has been used with tremendous success, and recently it has been championed as an alternative to psychiatric drugs, which presumably act directly on the mood centers of the brain. Recent research suggests, however, that cognitive therapy and drug therapy may have more in common than previously thought: Drugs alone appear to be just as potent as teaching and discussion in straightening out unrealistic ideas.

Psychologist Anne D. Simons and her colleagues at Washington University in St. Louis studied two groups of depressed patients, one receiving psychotherapy twice weekly, the other receiving daily doses of anti-depressant drugs. As they report in the January Archives of General Psychiatry, both treatments were effective in alleviating depression. But surprisingly, the patients receiving medication showed nearly identical intellectual changes as well — healthier ways of thinking about themselves and the world — even though they had never discussed such views with a therapist. In addition, patients whose depression did not lift — whether they were taking drugs or receiving psychotherapy — showed no cognitive changes — suggesting, Simons says, that intellectual change is an aspect of recovery rather than a cause. Depression, Simons concludes, should be thought of as a loose fusion of psychological and physiological processes to which therapy can gain entry in various ways. Improvement in any one process, she says, pulls the related processes along.

How does one choose, then, between drugs and cognitive therapy? Psychotherapy takes time and costs more money; drugs sometimes have unpleasant side effects. But there is also evidence, Simons says, that only certain personalities are suitable for cognitive therapy; specifically, patients must enter therapy with a sense of self-mastery — a sense that they can bring about changes in themselves. For those patients who do choose to talk through their thoughts and attitudes, there may be an additional benefit: Preliminary evidence, Simons told Science News, indicates that a successful course of cognitive therapy may be more effective than drugs in preventing relapse.

Low blood sugar no cause for panic

A spate of popular books has in recent years convinced many of the harmful effects of sugar, but it has also had an untoward side effect: Many have also become convinced that a whole range of symptoms — including many psychiatric symptoms — are caused by low blood sugar, or hypoglycemia. Panic attacks—uncontrollable rushes of anxiety — are often blamed on low blood sugar, especially when they occur (as they often do) while waiting in line in supermarkets or restaurants. But panic victims have never been studied to see if their blood sugar is actually low when the panic attacks occur.

Psychiatrist Jack M. Gorman and co-workers at the New York State Psychiatric Institute gave 10 known panic victims infusions of sodium lactate, a substance that is known to trigger panic attacks, and at the same time took blood samples. All of the subjects experienced panic attacks but, as the researchers report in the January American Journal of Psychiatry, none had a blood glucose measurement even close to hypoglycemic level. Hypoglycemia is known to provoke a rapid increase in adrenaline (which causes the lightheadedness, nausea and anxiety), and it may be that in some cases this kind of stress interacts with an underlying brain disorder to cause panic. But low blood sugar itself, the researchers conclude, is not the source of panic.

Pardes departs

Herbert Pardes, director of the National Institute of Mental Health (NIMH) since 1979, announced his resignation this month. He will become chairman of the department of psychiatry at Columbia University and director of the New York State Psychiatric Institute and the Columbia-Presbyterian Hospital psychiatric service in New York. Pardes has been widely praised for strengthening basic research in biological psychiatry during his tenure; he has been just as widely criticized for diminishing NIMH support for social science research.