

Anxiety: Fear Grows Up

If the 1960s and 1970s signaled the dawning of the Age of Anxiety, the 1980s may be the decade when, scientifically, anxiety comes of age. As with the more serious psychiatric afflictions — schizophrenia and severe depression — researchers are beginning to find biochemical and possible genetic factors associated with anxiety. Breaking from previous psychiatric thinking, behavioral scientists now believe that “intrapyschic conflict is not sufficient to explain anxiety states,” says Donald F. Klein, director of psychiatric research at the New York State Psychiatric Institute.

The emerging picture of anxiety is similar to those of certain other disabilities: Psychological factors do exist, but primarily against a backdrop of “susceptibility” among persons who go on to develop full-blown symptoms of anxiety — defined by the American Psychiatric Association as “apprehension, tension or uneasiness that stems from the anticipation of danger, the source of which is largely unknown or unrecognized.”

Such feelings are, of course, understandable and “normal” in the face of real danger from a perceived source. Normality yields to neurosis, however, when the fear becomes pervasive and generalized; it often blossoms into phobias — illogical fears of specific objects or situations — punctuated by dreaded panic attacks. Anxiety may be technically less serious than a psychotic break with reality, but it can incapacitate a person for years, or perhaps the better part of a lifetime.

Klein was among the first to demonstrate possible neurochemical causes in one type of severe anxiety — agoraphobia, which can be narrowly defined as a fear of open spaces but frequently escalates to a fear of almost anything outside one’s own home. Klein, also a professor of psychiatry at Columbia University, has demonstrated that the panic attacks accompanying agoraphobia may be controlled with an antidepressant drug, imipramine (SN: 5/26/79, p. 340).

Just why a drug for depression would help a phobic has been unclear. But insight into this and other aspects of the changing concept of anxiety were discussed last week in Washington at a meeting of the American Psychopathological Association. Klein’s treatment may have been successful because agoraphobia “is a psychosomatic form of depression,” according to George Gardos of the Institute of Research and Rehabilitation in Boston. In a study of 57 outpatients he has treated, Gardos reports that while agoraphobics develop significantly more psychosomatic symptoms (including respiratory, gastrointestinal, endocrine and allergic prob-



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Surrogate mothers aid in anxiety studies.

lems) than depressed persons, the two disorders contain “a number of common features between them.” Both groups display similar rates of alcoholism and, Gardos notes, agoraphobia and depression often occur in the same person — frequently following a loss of or separation from a loved one. Or, he says, agoraphobia may develop in a person trying to avoid depression following such a loss.

Premature separation from a parent has long been documented as an anxiety-provoking condition among monkeys. Results presented at the meeting suggest not only that such forced separation may trigger significant chemical changes in the body but that differences in individual monkeys’ reactions to this and other stresses might have a genetic basis.

Christopher Coe of Stanford University reports that pituitary-adrenal levels in young monkeys rise significantly 30 minutes after they have been separated from their mother and placed in either an adjacent cage (where they can still see her) or in a separate room. A similar rise is seen when the monkeys are removed and placed in a social group — indicating that the acute anxiety relates specifically to the mother-loss and not simply to isolation (although over the long term, such monkeys do appear less anxious in social groups than in isolation).

Coe and colleague Seymour Levine obtained similar results in 3- to 5-month-old monkeys after they were removed from their cages containing surrogate mothers — cloth-covered paintbrush rollers to whom monkeys have been found to develop close attachments. Interestingly,

though, the adrenal levels rose only when the young monkeys, as opposed to the cloth surrogates, were removed from the cage; monkeys raised by real mothers, however, displayed elevated adrenal levels in both experimental conditions — suggesting, Coe says, that the surrogate-reared monkey may be at least as attached to his environment as to his substitute mother.

In another project at the University of Wisconsin, Stephen Suomi has found that the magnitude of heart rate changes in 1-month-old monkeys under stress is “a good predictor” of the animals’ response to stress in later life. In one experiment, monkeys would hear a specific tone just prior to a 105-decibel blast of noise; the extent of the animals’ change in heart rate during the preliminary tone (which they learned was a prelude to the blast) correlated with the amount of anxious behavior (self-clutching, etc.) the monkeys would exhibit under separation and different stresses later in life.

Moreover, Suomi notes the similarity of heart rate changes and other stress responses among half-siblings upon separation from mother, as well as the tendency of “some monkeys [to] consistently produce individuals that appear to be at high risk for high stress reactions.” These findings point to a possible genetic factor in anxiety, Suomi says, and eventually may “be able to aid our understanding of anxiety at the human level.”

Genetic links have also been implicated in a study of 51 pairs of human twins in which one twin suffered from a type of agoraphobia or obsessional behavior. Gregory Carey of the University of Minnesota reports that in each disorder, the chances that *both* twins would be affected was close to 90 percent in identical twins — about twice the rate found in non-identical twins.

Recently, the most spectacular evidence of brain chemistry involvement in anxiety has been the discovery that specific receptor cells in the brain appear to seek out benzodiazepines, or tranquilizing drugs, such as Valium — suggesting that the human brain manufactures its own anti-anxiety substances or “natural Valium” (SN: 11/10/79, p. 325). Steven Paul, chief of preclinical pharmacology at the National Institute of Mental Health, says that although such a substance — if it exists — has not been definitely pinpointed, he and others have identified several “good candidates.” Two purines, inosine and hypoxanthine, head the list. But Paul told SCIENCE NEWS his group has found another substance (unnamed) with a molecular weight of 2,000 to 3,000 that constitutes another possibility. In laboratory tests, all three chemicals bind more weakly than Valium to receptor cells — a prerequisite for any such natural tranquilizer. “It may be that there are more than one of these substances” in the brain, he says. □