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CHAPTER 14

EXPERIMENTAL RESEARCH IN CLINICAL PSYCHOLOGY

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Psychopathology, as the branch of psychology concerned with abnormalities in mental functioning, consists of three main branches. Descriptive psychopathology seeks to characterize and organize the symptoms and syndromes of mental illness. Clinical psychopathology attempts to devise effective techniques for their diagnosis, treatment, and prevention. Experimental psychopathology applies the methods and principles of psychological science to understanding their nature and origins. Obviously, the three enterprises are closely related. Without good description, experimentalists do not know what to investigate; and systematic research is predicated on the existence of a reliable and valid diagnostic system, which in turn is based on systematic description. Basic research on the nature and origins of psychopathology will inevitably contribute to a refined diagnostic system, as it uncovers subtle differences among superficially similar syndromes and features that unite categories that seem different on the surface. It should also

yield a new generation of assessment devices, more firmly grounded in the paradigms and procedures of modern experimental psychology (Kihlstrom & Nasby, 1981; Nasby & Kihlstrom, 1986). Ultimately, of course, an important aim of experimental psychopathology is to lead to advances in treatment and prevention analogous to those achieved elsewhere in scientific medicine.

Experimental psychopathology has two main components: Laboratory models of psychopathology develop simulations of the symptoms and syndromes of mental illness in subjects who are not otherwise at risk for mental illness, and in nonhuman animals; research on psychological deficits studies defects in the functioning of basic psychological processes that presumably account for abnormal and maladaptive patterns of experience, thought, and action. The purpose of the present chapter is to give a brief overview of both lines of experimental research on psychopathology and to suggest directions for future activity.

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LABORATORY MODELS OF PSYCHOPATHOLOGY

Laboratory models of psychopathology attempt to create, in otherwise normal individuals, conditions resembling various psychopathological states. These models are rarely exact replicas of naturally occurring mental illness, but rather represent a laboratory phenomenon, or cluster of phenomena, that mimics a particular syndrome or symptom. In a sense, a laboratory model is a formal theory of mental illness, in that it assumes that the causal agents manipulated in the laboratory somehow parallel those that operate in the real world; and that the laboratory phenomenon can be analyzed to suggest hypotheses about previously unobserved features of the clinical syndrome, and employed to test proposals for treatment and prevention.

In an important paper, Abramson and Seligman (1977) listed the standards against which any laboratory model of psychopathology should be evaluated. Such models should, first and foremost, preserve the essential features of the symptom or syndrome observed in the clinic. That is, a proper laboratory model of phobia, depression, or schizophrenia would produce symptoms in normal subjects that parallel those observed in diagnosed patients.

This requirement presents a serious problem for the erstwhile modeler, because it is not always clear what the essential symptoms are. Psychiatric diagnoses traditionally have been construed in terms of the classical view of categorization: that a diagnostic category may be defined by the singly necessary and jointly sufficient features that define a proper set (Cantor & Genero, 1986; Cantor, Smith, French, & Mezzich, 1980). Thus, a patient might have to present with each of Bleuler's (1911/1950) "four As" (associative disturbance, anhedonia, autism, and ambivalence) in order to be labeled schizophrenic; and any patient presenting these four symptoms would be labeled schizophrenic, regardless of any other presenting symptoms. In contrast, Cantor and her colleagues (1980) argued cogently that the diagnostic categories are fuzzy sets of features that are correlated with, but not singly necessary or jointly sufficient for, category membership. The principal result of this situation is considerable heterogeneity among category members, such that they are related by family resemblance more than any set of common defining features. That is, Schizophrenic A may have some symptoms in common with Schizophrenic B, and B may have some symptoms in common with Schizophrenic C, but A and C might have no features in common.

Heterogeneity within syndromes is a fact of diagnostic life, at least for the major psychoses such as schizophrenia and affective disorder, and it wreaks havoc with the laboratory modeling enterprise. Unless we know the essential features of a syndrome, we can hardly try to reproduce those features in the laboratory. One solution to this problem is to forswear any attempt to model syndromes of psychopathology, but to model specific symptoms such as thought disorder or hallucinations instead (Costello, 1970). This solves the problem of within-category heterogeneity of features but raises the question of the degree to which superficially similar symptoms are in fact identical across syndromes. For example, hallucinations are common features in schizophrenia, depression, and organic brain syndrome, but it is not at all clear that an adequate laboratory model of one would serve as a satisfactory explanation of the other.

Whether the investigator is modeling single symptoms or whole syndromes, Abramson and Seligman (1977) argued further that a proper laboratory model of psychopathology must also demonstrate commonalities with the clinical phenomenon in terms of causes, cures, and preventative strategies. For example, the experimental manipulations known to produce the laboratory model should have their counterparts in the life histories of clinical patients. If one is to adopt a classical conditioning model of phobia, one had better find evidence of aversive conditioning experiences in the lives of phobics. Similarly, effective treatment regimens ought to be predicted by experimental manipulations that successfully reverse the laboratory model. Finally, there should be similarities in terms of underlying biological structures and processes. If certain genetic or hormonal factors mediate the response of experimental subjects to the independent variables, the same factors should be involved in the vulnerability of patients at risk for true psychopathology.

Few laboratory models, if any, would meet all of these standards. In fact, Abramson and Seligman (1977) argued cogently that many of the classic laboratory models—such as Masserman's (1943) and Maier's (1949) studies of frustration and conflict, Watson and Rayner's (1920) case of Little Albert, Osmond and Smithies's (1952) proposed mescaline models of psychosis—are not at all compelling when subjected to close scrutiny. Aside from the limitations of any particular research program, Abramson and Seligman (1977) argued that the success of the laboratory modeling enterprise in general is constrained by three considerations: (a) Ethical considerations, especially (though not exclusively) as applied to models developed in human subjects, may prevent the pro-

duction of full-blown symptoms and syndromes in otherwise undisturbed individuals; (b) even given an exhaustive description of some symptom or syndrome of psychopathology, the experimenter may not have any idea how to produce it in the laboratory; and (c) most laboratory models, as will be seen, are developed on nonhuman animal subjects—thus effectively precluding the expression through language of the cognitive and experiential symptoms that are some of the most central features of psychopathology.

Nevertheless, this state of affairs has not prevented a large number of investigators from pursuing the laboratory modeling approach in the study of a wide variety of conditions, ranging from phobia to schizophrenia (for authoritative reviews of many major lines of investigation, see Maser & Seligman, 1977). Laboratory models have the advantage over other experimental approaches to psychopathology because, at least in principle, they can go beyond the description of the psychological differences between patients and nonpatients and get at issues of cause. In this section, we provide brief sketches of some of the more salient of these attempts.

Experimental Neurosis

In a historical sense, perhaps the most prominent laboratory model of psychopathology is the work on experimental neurosis initiated in Pavlov's (1927) laboratory and continued by others (for overviews, see Mineka & Kihlstrom, 1978; Thomas & Dewald, 1977). Following Pavlov's (1927) classic report, other experimenters successfully induced neurotic behavioral disorders in a wide variety of animals (sheep, goats, pigs, and cats as well as dogs) by means of a wide variety of procedures: difficult discriminations, variable intervals, and punishment of appetitive responses. These and other procedures (see Mineka & Kihlstrom, 1978, for fuller descriptions) generally led to the replacement of some previously adaptive response, innate or acquired, by some uncharacteristic behavior (hypersensitivity, howling, rapid respiration, piloerection, muscular tension, mydriasis; or passivity, lethargy, and anorexia), often accompanied by signs of autonomic arousal. In other words, the behavioral consequences of experimental neurosis roughly paralleled the symptoms of anxiety and depression.

While the various procedures used to induce experimental neurosis have a number of consequences in common, it has proved difficult to find any common thread that runs through these demonstrations that could explain the effects. Mineka and Kihlstrom (1978) proposed that in each case, environmental

events of vital importance to the organism (e.g., obtaining food or escaping shock) become unpredictable, uncontrollable, or both. Their reanalysis of the classic demonstrations of experimental neurosis showed that many of the phenomena of experimental neurosis are mirrored in the behavioral effects of unpredictable and/or uncontrollable shock (see also Thomas & Dewald, 1977), and that important elements of unpredictability and/or uncontrollability could be found in the procedures employed in the classic investigations. Interestingly, anxiety has often been related to the occurrence of unpredictable events, while depression has been related to learned helplessness and the lack of control (Alloy, Kelly, Mineka & Clements, 1989; Mineka & Kelly, 1989). Thus, although the commonalities between experimental and human neurosis are far from perfect at the level of symptoms, there are salient parallels between the apparent sources of experimental neuroses and factors presumed to be of causative significance in the clinical syndromes.

Phobias, Obsessions, Compulsions, and Anxiety

Another historically prominent modeling enterprise has been concerned with fears, phobias, and other aspects of anxiety (Marks, 1969, 1987; Rachman, 1978; Rachman & Hodgson, 1980; Wolpe, 1958). This line of research has its origins in the work on experimental neurosis just described and also in the classic case of Little Albert described by Watson and Rayner (1920; for critiques of both the case and subsequent accounts of it, see Harris, 1979; Samelson, 1980). Conventional thought in this area equated phobias with classically conditioned fear responses and compulsions with avoidance responses, while anxiety states were attributed to generalization of fear conditioning. The model was extremely heuristic—it led directly to the development of systematic desensitization and flooding (implosion) therapies (for example, Yates, 1970), but recent work has added several layers of theoretical sophistication (Mineka, 1985a, 1985b, 1987).

Mineka (1985a, 1987) provided the most thorough statement and analysis of the animal models of phobias, obsessive-compulsive disorders, and generalized anxiety states; for reasons of space, we limit our discussion to phobias. Mineka (1985a) argued, at the outset, that any model of anxiety-based disorders must confront the apparent fact that fear, whether normal or pathological, has three aspects—cognitive, behavioral, and psychophysiological—that are at least partly dissociable (Lang, 1968; Rachman, 1978).

Nevertheless, she cogently argued that recent work on fear conditioning and avoidance in rats and monkeys forms the basis for an animal model of phobia that successfully encompasses symptomatology, etiology, maintenance, and therapy.

To begin with, Mineka (1985a) noted that simple classical conditioning models of phobia—in which a phobic object, such as an animal, serves as a conditioned stimulus (CS) for an aversive unconditioned stimulus (US), such as an animal bite—fail because of the simple fact that phobias are notoriously difficult to extinguish, whereas most classically conditioned fears extinguish over a moderate number of trials. On this ground alone, avoidance learning would seem to fare somewhat better. Unfortunately, as Seligman (1971) noted, phobics avoid the CS, while animals in avoidance situations avoid the US, and there have been few if any successful attempts to train them to avoid the CS. These and other considerations have prompted a return to a fear-conditioning model of phobia, but with two important revisions. First, the selectivity of phobias is accounted for by the concept of preparedness (Seligman, 1971), in which the most common phobic objects are seen as representing a class of stimuli that were dangerous to our evolutionary forebears. Finally, the fact that many phobics apparently lack a history of traumatic experience with their phobic objects—perhaps the most serious difficulty with conditioning models (Jacobs & Nadel, 1985)—is accounted for in terms of observational and vicarious conditioning.

Mineka's (1985a, 1985b, 1987) own work on fear conditioning provides the best example of the model in action. She begins with the fact that fear of snakes in wild-reared rhesus monkeys is intense, persistent, and difficult to modify. Although such a fear would seem to be natural and thus innate, rhesus monkeys reared under ordinary laboratory conditions show no significant fear of snakes: Thus, snake fear appears to be acquired through experience. Of course, it would not be too difficult to show that monkeys that are bitten by vipers or squeezed by pythons fear snakes subsequently. Much more interestingly, in an elegant series of studies, Mineka (1987) and her colleagues showed that fear of snakes can be acquired quickly by laboratory monkeys solely on the basis of observing a wild-reared counterpart react fearfully to an objectively harmless snake. Moreover, when the lab-reared animals were given 12 sessions of implosion therapy, they quickly became able to reach past the snake to grasp food. Despite this reduction in one component of fear, however, the animals continued to show other signs of gross behavioral disturbance.

Thus, animal models of phobic disorder seem to be

a particularly promising experimental vehicle for asking questions about the origins, maintenance, and treatment of psychopathology. There are unresolved questions about social phobias and agoraphobia, but overall the line of research has proved productive. Unfortunately, as Mineka (1985a) noted, analogous models for the remaining anxiety-based disorders have not received much attention (Beck & Emery, 1985; Dollard & Miller, 1950; Eysenck, 1979; Eysenck & Rachman, 1965). Avoidance appears to provide a good start for obsessive-compulsive behavior (Carr, 1974; Teasdale, 1974; but for a more thoroughly cognitive approach see Reed, 1985), while the experimental neurosis literature lays the basis for an analysis of anxiety disorder (Mineka & Kelly, 1989), but work in both areas has only just begun.

Hysteria and Hypnosis

Laboratory models of psychopathology are not limited to experiments involving nonhuman animals. For example, another historically important laboratory model of psychopathology has been offered by hypnosis (Kihlstrom, 1979). Hypnosis is a social interaction in which one person (the subject) responds to suggestions offered by another person (the hypnotist) for various kinds of imaginative experiences. In the classical instance, as represented by the responses of highly hypnotizable individuals, these experiences are accompanied by a degree of subjective conviction bordering on delusion (Kihlstrom & Hoyt, 1988) and feelings of involuntariness bordering on compulsion. Thus, two of the cardinal phenomena of hypnosis are also important symptoms of psychopathology—thus creating the rationale for hypnosis as a laboratory model of certain forms of mental illness.

In the late 19th and early 20th centuries, clinical and experimental interest in hypnosis was largely motivated by the apparent similarities between its phenomena and the symptoms of hysteria (for a detailed history, see Ellenberger, 1970; Perry & Laurence, 1984). Both states involve subjectively compelling disruptions and anomalies in conscious awareness and control—blindness, deafness, amnesia, paralysis, compulsive automatisms, and the like—in the absence of any evidence of brain insult, injury, or disease. The fact that phenomena closely resembling hysterical symptomatology could be induced in (selected) normal subjects, simply by means of the hypnotist's spoken word, led Janet, Freud, and others to propose psychogenic theories of hysteria as correctives to the somatogenic view that had prevailed up to the time of Charcot, and to develop a wide variety of "talking cures" for its treatment.

More recently, hypnosis has been employed as a vehicle for experimental research on divisions in consciousness and the relations between conscious and nonconscious mental processes that seem to be involved in hysteria (Hilgard, 1977; Kihlstrom, 1984, 1987, 1989, 1990a, 1990b). For example, hysterical patients frequently give evidence of intact perception and memory, in apparent contradiction to their presenting complaints. Thus, a patient might claim to be blind yet navigate successfully around an unfamiliar room (Sackeim, Nordlie, & Gur, 1979) or claim a loss of identity while simultaneously displaying personal knowledge. Similar contradictions occur in hypnosis. For example, subjects experiencing hypnotic analgesia nevertheless may show unaltered psychophysiological responses to painful stimuli, and amnesic subjects commonly show intact priming effects of their forgotten experiences. While such paradoxes have sometimes been taken as evidence that both hysterical and hypnotic subjects are engaged in role enactment (Sarbin & Coe, 1979), more recent experimental analyses have linked both states to a broad range of phenomena of implicit cognition, in which certain aspects of perception, memory, thought, learning, and action are dissociated from phenomenal awareness (Kihlstrom, 1987, 1989, 1990b). This experimental research, in turn, suggests a common mechanism underlying functional disorders of memory, sensory-perceptual, and motor function—a suggestion which, if proved correct, would indicate that the conversion disorders classified by *The Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised* (DSM-III-R) as somatoform in nature are more properly regarded as a subcategory of dissociative disorder (Kihlstrom, 1990a).

PSYCHOLOGICAL DEFICIT IN PSYCHOPATHOLOGY

The study of psychological deficits has a long tradition, beginning with the studies by Jung on word associations, Kraepelin on continuous performance, Shakow on attention, and Goldstein on concept attainment. The classic review of studies of psychological deficit by Hunt and Cofer (1944) indicates that by midcentury a great variety of procedures familiar to academic experimental psychologists had been applied to clinical patients, covering such diverse topics as sensory thresholds, perceptual processes reaction time, reflex functions, eye movements, motor performance, word associations, startle response, conditioning, memory, language, and thought. This tradition continued after the war, and the vast amount of research in this area was authoritatively summarized

by Buss (1966), Maher (1966), Costello (1970), and Yates (1966, 1970). With the exception of a few systematic programs of inquiry (e.g., Eysenck, 1955; Lindsley & Skinner, 1954; Wishner, 1955), however, it seems fair to say that experimental psychopathology in that era was guided more by phenomena than by theory. Investigators were concerned with differences between patients and nonpatients in performance on psychological tasks, but these tasks seem to have been selected almost arbitrarily, with little theoretical motivation and little effort expended in linking the positive findings in one domain with those in others.

What was needed was an overarching theoretical conception of psychological functions that would link a variety of findings and direct research toward fundamental rather than peripheral topics. This goal came into view with the formal emergence of cognitive psychology, which for the first time brought diverse studies of attention, perception, learning, memory, thought, and language under a single rubric—the information-processing approach (e.g., Atkinson & Shiffrin, 1968; Newell & Simon, 1972). Within a few years, from 1967 to 1973, this model became widely accepted within academic experimental psychology and began to filter down to clinical psychology to guide the work of experimental psychopathologists.

Emblematic of this transition is the review of psychological deficits in schizophrenia by Chapman and Chapman (1973a), which devoted most of its pages to classical approaches but included a chapter on information processing. Only 6 years later, a whole book appeared on the latter topic, in which the earlier approaches received hardly a mention (Matthysse, Spring & Sugarman, 1979). A similar shift is apparent in research on depression. Before 1967, depression was generally considered to be a purely emotional disorder, involving as it does dysphoria as its primary symptom, and studies of cognitive deficits were largely nonexistent (Miller, 1975). However, the work of Beck (1967) and Seligman (1975) drew attention to the cognitive deficits characteristic of depressed patients, opening up a whole new line of inquiry on perception and cognition in the affective disorders (Ingram & Reed, 1986). For a further treatment of developments in information-processing theory and their implications for experimental psychopathology, see Kihlstrom (1983) and Ingram (1986).

Schizophrenia

As suggested earlier, the longest and most vigorously pursued line of research on psychological deficit has focused on schizophrenia (for reviews, see Chapman & Chapman, 1973a; Magaro, 1980; Matthysse et

al., 1979; Neale & Oltmanns, 1980; Nuechterlein & Dawson, 1984; Oltmanns & Neale, 1982; Saccuzzo, 1986; Schwartz, 1978, 1982; Spaulding & Cole, 1984; Widlocher & Hardy-Doyle, 1989). Much of this research has focused on problems of language and communication, since that is how schizophrenic thought disorder typically manifests itself (Kasanin, 1944). Using tasks derived from experimental psycholinguistics, the nature of schizophrenic language might be analyzed for various hypothesized phonological, syntactic, and semantic deficits (for reviews, see Maher, 1972; Schwartz, 1978, 1982). Thus, for example, schizophrenics might experience difficulties in the organization and production of speech, in the application of grammatical rules, in the network of associations making up semantic memory, or in the use of context to comprehend ambiguous utterances. Although it is the universal experience of clinicians that it is difficult to communicate with schizophrenics—Kraepelin (1896) referred to their language problem as “schizophrenia”—the results of formal studies have largely failed to reveal any uniform deficits in language processing (e.g., Schwartz, 1982).

This is not to suggest that everything is all right with schizophrenic cognition. However, Schwartz (1982) and many other investigators have proposed that the locus of psychological deficit in schizophrenia lies at some other aspect of the cognitive system. Such a deficit, it is argued, would in turn account for the manifest disorders in thinking and language observed in schizophrenia. Much of this research on schizophrenic information processing has been inspired by McGhie and Chapman's (1961) classic interview study, which underscored the problems experienced by schizophrenics in deploying and maintaining selective attention and has been more or less explicitly based on a generic multistore model of information processing. In this model, information flows between various storage structures (sensory registers, primary memory, secondary memory), in the process of which it is subject to various manipulations and transformations subjected by various control processes (e.g., pattern recognition, attention, rehearsal). Thus, appropriately designed experimental tasks could, in principle, detect selective deficits in the operation of one or more storage structures and control processes.

A good example of this sort of research is an experiment by Saccuzzo, Hirt, and Spencer (1974) employing a backward-masking paradigm to study information-processing speed (for a review, see Saccuzzo, 1986). In their experiment, subjects were presented with a visual array of 16 letters and then

asked to report which of two targets had been presented in the matrix. Presentation of the array was followed by a meaningless masking stimulus, used to control the amount of time a representation of the array was held in iconic memory. By varying the interval between array and mask, Saccuzzo et al. (1974) were able to show that schizophrenics read information out of iconic memory and into primary memory more slowly than controls. Other results from their study indicated that schizophrenics' iconic memories may be degraded to begin with and persist longer than those of normals.

Subsequent research by Knight and his colleagues, using similar sorts of paradigms, clarified the role of schizophrenic subtype (e.g., good vs. poor premorbid adjustment) in the experiment of Saccuzzo et al. (1974) and identified a further problem at the stage of primary memory (Knight, Elliott, & Freedman, 1985; for a review, see Knight, 1984). They showed that poor premorbid schizophrenics gave similar responses to pattern and cognitive masks, while normals and good premorbid schizophrenics responded differently to them (depending on the interval between array and mask). The entire pattern of results indicates that the principal difficulty in schizophrenics' visual information processing is not at the level of the icon per se, or even in readout from the icon, but rather in certain automatic perceptual processes interposed between iconic and short-term visual memory. This deficit in perceptual organization may, in turn, be responsible for some of the problems apparent in schizophrenic speech and language (Knight & Sims-Knight, 1982); somewhat paradoxically, it also leads to superior performance by schizophrenics on tasks (e.g., judgments of numerosity) where the operation of these organizational processes (e.g., gestalt principles of similarity or proximity) cause poor performance in normals (Place & Gilmore, 1980).

Affective Disorder

In contrast to the long history of experimental studies of schizophrenia, the affective disorders have been much less frequently studied from the perspective of psychological deficit. In the earliest review of this literature, Miller (1975) attributed this state of affairs to a general tendency to construe depression as a disorder of emotion rather than cognition and a corresponding belief that depressives would show few performance deficits on the kinds of affectively neutral perceptual, cognitive, and motor tasks studied in schizophrenia. Nevertheless, Miller (1975) did find

evidence of psychological deficit in severe, mild, and subclinical forms of affective disorder.

The wide acceptance of Schacter and Singer's (1962) cognitive-attributional theory of emotion, coupled with the publication of Beck's (1967) seminal monograph on depression, changed this situation considerably. Schacter and Singer argued that emotional states were cognitively constructed, while Beck argued that specific modes of thought predisposed individuals to depressive forms of mental illness. The next decade saw the progressive evolution of helplessness and hopelessness theories of depression in the hands of Seligman, Abramson, Alloy, and their associates (e.g., Abramson & Sackeim, 1977; Abramson, Seligman, & Teasdale, 1978; Abramson, Metalsky, & Alloy, 1988; Mineka, 1982; Seligman, 1975), plus a growing appreciation of the reciprocal effects of depression on cognitive processes in memory and thought (e.g., Bower, 1981; Ellis & Ashbrook, 1988; Isen, 1984; Kuiken, 1989; Mayer, 1986; Singer & Salovey, 1988; Tobias, Schacter, & Kihlstrom, 1990). Thus, either because cognitive processes influenced mood, or mood influenced cognitive processes, or both, it has made sense to search for psychological deficits among patients with affective disorder (Alloy, 1988; Blaney, 1977; Coyne & Gotlib, 1983; Ingram & Reed, 1986; Miller, 1975).

In contrast to most work in schizophrenia, much recent research has focused on identifying various cognitive styles, as opposed to deficits per se, that may be characteristic of depression. For example, Abramson, Alloy, Seligman, and their colleagues have identified a tendency for depressives, or normals at risk for depression, to make global, stable attributions concerning the causes of important negative life events, despite the actual pattern of consensus, consistency, and distinctiveness information available to them (e.g., Metalsky, Halberstadt, & Abramson, 1987; Peterson & Seligman, 1984). Such a pattern is depressogenic in that it predisposes the person to react in a depressive manner to the occurrence of negative life events or the absence of positive ones: It is the diathesis, which when combined with a negative event stressor leads to depression. When the person is also disposed to make internal causal attributions, the depression is accompanied by lowered self-esteem. Of course, nondepressives also show attributional biases, as evidenced by the fundamental attribution error (Nisbett & Ross, 1980; Ross, 1977), so it is not proper to consider depressives' errors as evidence of a psychological deficit.

Moreover, some research has identified aspects of performance in which depressives are, at least in some

sense, superior to normals. A good example is the assessment of covariation between events (Crocker, 1981). Alloy and Abramson (1979) compared depressed and nondepressive subjects on a task in which button-pressing behavior sometimes, but not always, led to the illumination of a light. Both Beck's (1967) cognitive theory and the original learned-helplessness theory of depression predict that depressives would underestimate the covariation between act and outcome. In fact, depressives proved to be more accurate than nondepressives. The finding has been replicated under a number of different conditions (Alloy & Tabachnik, 1984) and has given rise to the concept of depressive realism: While normal individuals may be subject to an illusion of predictability and control, depressives accurately perceive when events are random and uncontrollable—thus, in some sense depressives are sadder but wiser than their nondepressed counterparts.

No fully satisfactory theoretical account of depressive realism has yet been proposed (Alloy & Abramson, 1988; Kayne & Alloy, 1988). The phenomenon may reflect merely an inability of current laboratory paradigms to capture the cognitive distortions and errors suffered by depressives. Alternatively, depressives, far from possessing specific depressogenic schemata (Beck, 1967), may lack certain organized cognitive structures that promote the illusion of control and predictability in normals and that protect normals from depression. In any event, psychological deficit in depression appears to pose a paradox: Whereas schizophrenics appear to be more vulnerable than normals to certain distortions and errors, depressives are less prone to them. In other words, the psychological deficit in depressives may be that they lack precisely the biases and distortions that keep the rest of us sane.

The development of cognitive models of depression, and the corresponding revival of inquiry on psychological deficits in the affective disorders, has stimulated new developments in treatment (e.g., Beck, 1976; Mahoney, 1974; Meichenbaum, 1977). Moreover, as with laboratory models of hysteria, research on psychological deficits in depression has laid the foundation for revisions in diagnosis. Thus, Abramson et al. (1988) have postulated the existence of a "hopelessness subtype" (p. 43) of depression. Thus, feelings of hopelessness are held to be sufficient, but not necessary, to cause depression; but when such feelings occur, they will necessarily lead to a specific form of depression whose characteristic symptoms, prognosis, and preferred treatment may be quite different from other forms of depression that

may have other proximal causes. The reasoning is not tautological, any more than it is to assert that hypothyroidism is a necessary but not sufficient cause of mental retardation and that when hypothyroidism occurs it will lead to the specific syndrome of cretinism.

Linking Laboratory Models with Psychological Deficits: The Case of Psychopathy

Research on psychopathy, or antisocial personality disorder, illustrates how the development of a convincing laboratory model of psychopathology can breathe new life into studies of psychological deficit and how studies of psychological deficit can influence both the classification of psychopathology and our understanding of normal mental processes. The enduring tendency of psychopaths to engage in antisocial behavior, and their persistent failure to respond to punishment, led to extensive research comparing psychopaths and controls on measures of response to aversive stimulation, avoidance learning and punishment, psychophysiological arousal, and the like (for reviews, see Hare, 1970; Hare & Schalling, 1978). All of this work was in the classic pattern of psychological deficit research and seemed to implicate some sort of deficit in the arousal system—either underarousal or an enhanced ability to modulate arousal.

Somewhat later, Gorenstein and Newman (1980) noted the strong similarities between the behavior of psychopaths and that of rats with lesions in the septal area. Thus, animals with septal lesions do not freeze when punished; although they do not show a generalized learning deficit, they are rather poor at passive avoidance and delay of gratification; they show a steep temporal gradient of fear arousal and have difficulty acquiring conditioned fear responses. In short, these animals show performance deficits where they are required to suppress or alter habitual responses in order to avoid aversive consequences: They are impulsive. Without specifically proposing that psychopaths suffer brain damage in the limbic system, these investigators used the septal animal model to generate both an alternative theoretical explanation of psychopathic personality and an innovative line of empirical research.

The parallels between psychopathic humans and septal animals offer two alternative possibilities for research: testing psychopaths for behavioral patterns characteristic of septal animals, and testing septal animals for patterns characteristic of psychopaths. Newman and his associates have pursued the former

line of inquiry in an extensive series of studies (for a review, see Patterson & Newman, 1987). This research shows that the characteristic behavior of psychopathic individuals results not from any lack of arousal, but rather from an emotional response to frustration, especially in mixed-motive situations. When the appetitive behavior of nonpsychopathic individuals is interrupted by an aversive event, their most likely response is to pause and collect information about the changed situation. By contrast, psychopathic individuals respond to the changed circumstances with increased effort, which effectively prohibits such reflectivity. One prediction is that psychopaths can learn to modulate their behavior if forced to pause and reflect on its consequences, and research by Newman's group indicates that this is so.

Based on this approach, Patterson and Newman (1987; see also Newman, 1987) proposed that the entries in DSM-III-R may hide a heretofore unrecognized group of syndromes of disinhibition, just as other clusters of syndromes are organized around themes of schizophrenia, depression, and anxiety. Such a category might include, in addition to psychopathy, borderline personality disorder, attention deficit disorder, histrionic personality disorder, somatization disorders, early onset alcoholism, and the current miscellaneous category of disorders of impulse control not otherwise classified (e.g., pathological gambling and kleptomania). These syndromes of psychopathology are linked to normal personality features such as Type A personality and extraversion by an underlying proneness to disinhibition. Evidence in support of this proposal comes from studies showing that normal extraverts and hyperactive children respond in a manner similar to psychopaths on a variety of experimental tasks—tasks derived, at the outset, from behavioral studies of rats with septal lesions. Thus, work in experimental psychopathology, drawing on studies of both laboratory models and psychological deficits, has had an impact on the organization of descriptive and clinical psychopathology.

Problem of Differential Deficit

One problem that stands in the way of progress in investigating psychological deficits in psychopathology is the diagnostic system itself. Despite advances in the rigor and reliability of the diagnostic process, the nosological categories remain heterogeneous. There is little at the level of the observable symptom that unites one schizophrenic or manic depressive with another (Cantor et al., 1980; Cantor & Genero, 1986).

Without a diagnostic system for reliably segregating cases of psychopathology into distinct, relatively homogeneous categories, there will be too much noise in the data to permit firm conclusions about the nature of the disorders in mental functioning implicated in clinical psychopathology.

Even with fairly homogeneous diagnostic categories, however, and reliable procedures for sorting patients into them, the study of psychological deficit is plagued by a subtle but thorny problem: the discriminating power of tests of psychological deficit (Chapman & Chapman, 1973b, 1978). As Chapman and Chapman (1973b) noted, the favored paradigm in studies of psychological deficit involves documenting differential deficits (this is sometimes called the logic of dissociation; see Schacter & Tulving, 1982). In the simplest exemplar of this logic, two groups—for example, schizophrenics and normals—are compared on two different tests of mental performance, such as digit span forward and backward. Assume that the two groups are found to differ on digits backward but not on digits forward—a standard two-way interaction. Under ordinary circumstances, the temptation would be to conclude that schizophrenics suffer a deficit in whatever underlying psychological process is known or presumed to differentiate between the two tasks. Thus, it might be concluded that schizophrenics have a deficit in attention but not in short-term memory capacity.

The problem with this logic is that differences in task performance depend not only on differences between groups in mental ability but also on differences between tests in discriminating power. The discriminating power of mental tests, in turn, is determined by such factors as their mean item difficulties, the dispersion of item difficulties around the mean, the number of items, and their average covariance. If the two tests in question are not matched on these psychometric properties, any group differences observed may well be artifactual. This is because patients with major psychopathology generally show generalized cognitive deficits—that is, they score lower than nonpatients on almost any cognitive test. Such subjects must show greater performance deficits on the more discriminating tests, regardless of the actual presence of differential psychological deficits, even if they are actually equivalent on the abilities measured by the tests in question. In principle, any finding of differential deficit (such as the foregoing comparison between digits forward and digits backward) could be reversed simply by altering the properties of the tests in question.

A similar problem arises in a variant on the differ-

ential deficit design, in which two groups are administered two tests, each under two conditions manipulated by the experimenter. Thus, to extend the preceding example, tests of digit span forward and backward might be administered under two levels of ambient noise level. If so, a triple interaction might be found such that schizophrenics and normals differ on digits forward under high- but not low-noise conditions, and that the group difference on digits backward is exacerbated by high noise levels. Unfortunately, the manipulation of any experimental variable that affects accuracy will yield a greater performance deficit on the more discriminating test, even though there is no effect on the specific mental ability presumably tapped by the test.

Chapman and Chapman (1973b) argued that these problems cannot be corrected by any obvious statistical means, such as analysis of covariance or the use of residualized error scores. Nor can the direction of the artifact be predicted in advance, such that conservative tests could be devised. The only guaranteed solution to the problem is extensive pretesting, using a standardization sample of normal subjects with widely varying abilities, to match the tests in question in terms of reliability, difficulty, and number of items. Given tests that are equivalent on these psychometric properties, the possibility of statistical artifact may be ruled out, and any group differences in test scores or effects of experimental manipulations can be safely attributed to differential psychological deficits.

The problem of discriminating power, and its solution, is nicely illustrated in a classic study by Oltmanns and Neale (1975) on digit span. It was already known that schizophrenics perform worse than normals on such short-term memory tasks, and the deficit is especially severe under distracting conditions. However, almost by definition, a task performed under conditions of distraction is more difficult than the same task performed alone—so the interaction is difficult to interpret in terms of schizophrenics' differential vulnerability to distraction rather than overall cognitive inefficiency. Oltmanns and Neale (1975) found through pretesting with normals that a five-digit distractor task and a six-digit control task were equally difficult, as were a six-digit distractor task and seven- or eight-digit control tasks (recall that the capacity of short-term memory is approximately seven items). Comparing schizophrenics and nonschizophrenics on the six-digit control and distractor tasks, they found the usual interaction. However, the interaction disappeared when the six-digit neutral task was compared to the difficulty-matched five-digit distractor task, raising the problem of differential difficulty. Evidence

of differential deficit was obtained only when the six-digit distractor task was compared to the seven- and eight-digit neutral task. The overall pattern of results confirmed a differential vulnerability to distraction among schizophrenics, but the conditions under which the interaction was obtained indicated that the locus of the attentional difficulty was at the stage of memory encoding rather than sensory-perceptual processing. In the present context, the point is that neither of these conclusions would have been legitimate without careful matching of experimental and control tasks.

Unfortunately, more than a decade after Chapman and Chapman (1973b) drew attention to this problem, many investigators in the field of psychopathology, mental retardation, child development, aging, and neuropsychology continue to draw conclusions concerning differential deficits based on tests that have not been properly equated for discriminating power. The appropriate corrective measures are expensive and time consuming, but failure to employ them poses a serious threat to the validity of conclusions from experimental results.

COGNITIVE NEUROPSYCHOLOGY AND PSYCHOPATHOLOGY

Cognitive neuropsychology represents the merging of two previously distinct subdisciplines within psychology, cognitive psychology and neuropsychology, and has flourished as a scientific enterprise in its own right over the past 15 years. According to Ellis and Young (1988), the two main goals of cognitive neuropsychology are (a) to account for patterns of impaired and preserved cognitive performance in terms of disruption to particular components of an information processing theory or model; and (b) to increase our knowledge of normal cognitive processes based on the patterns of cognitive deficits and intact abilities observed in brain-damaged patients. We propose that the conceptual framework of cognitive neuropsychology may serve as a useful model for investigating and interpreting the psychological deficits observed in various psychopathological populations. In this section we will briefly review the basic concepts and assumptions of cognitive neuropsychology, describe examples from the cognitive neuropsychological literature, and discuss potential applications of this model to the study of psychological deficit in psychopathology.

An important concept in cognitive neuropsychology is the dissociation of a specific process or function from another. For example, if a brain-injured patient

has severe difficulty reading words but exhibits intact writing abilities, we may conclude that there is a dissociation between reading and writing. In other words, there appear to be different cognitive processes involved in reading and writing words that permit differential disruption of the two functions. An even more convincing argument for this kind of conclusion is derived from a double dissociation. That is, if in addition to observing cases where reading is impaired but writing is intact, we find individuals who exhibit preserved reading ability in the presence of impaired writing, we would have a much stronger case for concluding that the two tasks involve different cognitive processes.

Dissociations are generally interpreted within a modular framework for the mind (see Fodor, 1983; Marr, 1982). This view of cognitive organization suggests that multiple cognitive processors or modules operate relatively independently; hence, any particular module can theoretically malfunction in isolation without affecting other modules in the total cognitive system. Furthermore, modules are considered to be impenetrable by higher order influences such as beliefs, motivations, and expectations. This property of modules has been referred to by Fodor (1983) as informational encapsulation. For example, there may be a group of modules responsible for different aspects of object recognition, another group involved in facial recognition, and so on. Disruption of particular perceptual modules involved in these functions may result in specific kinds of recognition impairments. Modules are also assumed to be domain specific (Fodor, 1983); that is, each module can only process one kind of input such as visual, auditory, or tactile information. In contrast to the modular structure of input processors, Fodor (1983) proposed that more complex mental operations involving higher order processing of output from individual modules are driven by central processes that do not follow the principles of modularity and are not amenable to investigation. The important difference between modules and central processes in Fodor's model is that modules are restricted to a particular type of information and are resistant to top-down cognitive influences, whereas central processes can receive and integrate information across many domains and from many different directions, which makes them difficult to study. However, Ellis and Young (1988) argued that by examining the kinds of errors made by patients with disorders of higher mental functions and the types of difficulties they experience on various tasks, we may be able to elucidate the ways in which these complex operations can go awry as well as learn

something about how these higher mental processes normally operate.

Recently, Moscovitch and Umiltà (in press) proposed that the study of higher order disturbances in cognition provides critical evidence for the issue of informational encapsulation. They argued that if a particular function remains intact despite generalized intellectual deterioration, one can assume that it is informationally encapsulated since the malfunctioning of a central cognitive system has no effect on that function. For example, patients with Alzheimer's disease exhibit widespread intellectual deficits, yet they retain the ability to read, to repeat, and correct grammatically incorrect sentences (Schwartz, Marin, & Saffran, 1979). Thus, specific domains of preserved function in the context of generalized intellectual deterioration provide support for the notion of informational encapsulation (i.e., modular organization of those spared functions).

Specific examples of how dissociations are interpreted within the modular framework of cognitive neuropsychology illustrate the value of this approach for understanding the nature of deficits in brain-damaged patients as well as for increasing our knowledge of normal cognitive functioning.

Recognition of Faces and Objects

Several striking dissociations have been reported in the literature concerned with prosopagnosia, referring to disorders of face recognition, and object agnosia or impaired object recognition. Prosopagnosic patients are unable to recognize any familiar faces including famous faces, friends, family, and their own faces when seen in a mirror (Hecaen & Angelergues, 1962). Interestingly, however, prosopagnosia may occur in the absence of any obvious difficulty with recognizing objects and, conversely, severe object agnosia has been observed in patients without prosopagnosia (Ellis & Young, 1988). This double dissociation between face recognition and object recognition suggests that the two tasks involve different cognitive and perceptual processes such that disruption of one function can occur in isolation from the other.

Dissociable impairments within each of these categories of recognition disorders have also been reported. For example, neuropsychological findings of face processing disorders suggest that impairments involving familiar face recognition can be doubly dissociated from impairments involving identification of facial expression or unfamiliar face matching (Bruce & Young, 1986). These kinds of dissociations imply that different functional components are in-

involved in various aspects of face processing and consequently can malfunction independently of one another. These findings, combined with results from studies of normal subjects, have played an integral role in the development of functional models of face processing (e.g., Bruce & Young, 1986).

Anosognosia and Unilateral Neglect

Damage to the posterior right cerebral hemisphere of the brain frequently produces a constellation of neuropsychological symptoms. Patients sustaining injury to this region may exhibit hemiplegia (paralysis of the left side of the body), unilateral neglect (inattention to the left side of space), hemianopia (visual field defect), and somatosensory impairment on the left side. In addition, these patients often appear entirely unaware of one or more of these deficits. Unawareness of a neuropsychological deficit is referred to as anosognosia and may occur in relation to any or all of the aforementioned defects. Some investigators have suggested that this group of behavioral changes reflects disruption of a common underlying mechanism, or at least disruption of closely related mechanisms (for review see McGlynn & Schacter, 1989); however, little systematic research has been devoted to this issue.

An important study by Bisiach and his colleagues (Bisiach, Vallar, Perani, Papagno, & Berti, 1986) investigated the relation between anosognosia for hemiplegia, anosognosia for hemianopia, unilateral neglect, and various other neurological disturbances in patients with damage to the right hemisphere. Results revealed a double dissociation between unawareness of motor impairment and unilateral neglect. Some patients who completely ignore the affected side of the body may be fully aware of their motor defect. Others who continue to deny their hemiplegia, even when confronted with evidence to the contrary, may attend normally to the left side. Another striking finding was a double dissociation between unawareness of hemiplegia and somatosensory impairment, suggesting that a somatosensory disturbance is not an essential condition for the development of unawareness of hemiplegia. Bisiach, Merigalli, and Berti (1985) developed a cognitive model of the awareness disorder, viewing unawareness phenomena as modality-specific disorders of thought resulting from disruption of specific mechanisms that normally monitor the output of individual perceptual and cognitive modules. Interpretation of the former dissociations within an information processing model has heuristic value, in that we are now in a position to

investigate systematically, based on the model, specific hypotheses regarding organization of function.

Amnesic Syndromes

Amnesic syndromes occur as a consequence of various types of neurological impairment involving lesions to medial temporal or diencephalic brain regions (Schacter & Tulving, 1982; Squire, 1986). Amnesic patients typically have normal or near-normal intellectual, linguistic, and perceptual function but are unable to remember recent events and learn many types of new information. Graf and Schacter (1985, 1987; Schacter & Graf, 1986a, 1986b) distinguished between two kinds of memory, referred to as explicit and implicit memory. The former type is what we generally think of as remembering and involves conscious recollection of a recent event. However, memory can also be expressed implicitly, as a facilitation of task performance without conscious recollection. Although amnesic individuals fail to demonstrate explicit recall or recognition of recent episodes, their performance on tests of implicit memory indicates that some new information and skills can be acquired without conscious awareness of the prior learning episode(s) (e.g., Graf & Schacter, 1985; McAndrews, Glisky, & Schacter, 1987; Schacter, McAndrews, & Moscovitch, 1988). For example, the well-studied patient H. M. could acquire motor skills such as pursuit rotor and mirror tracing, even though he did not remember explicitly that he had previously performed the task (Milner, 1962; Milner, Corkin, & Teuber, 1968). Similarly, amnesic patients can learn and retain complex computer knowledge and a variety of computer operations without any recollection of encountering a computer previously (e.g., Glisky & Schacter, 1986). These kinds of findings with amnesic patients have had a significant impact on current theoretical accounts of memory organization and function (for review see Schacter, 1987) and stimulated a large body of cognitive research with normal subjects.

The striking dissociations between implicit and explicit memory in amnesic patients have been paralleled in cognitive research with normal subjects. For example, several studies have shown that variations in level or type of study processing have differential effects on implicit and explicit memory for familiar words or word pairs (e.g., Jacoby & Dallas, 1981; Schacter & McGlynn, 1989). Explicit memory is influenced by type of study processing, with better recall or recognition performance following elaborative study tasks than nonelaborative study tasks. In

contrast, implicit memory is unaffected by the study task manipulation. A variety of paradigms, such as the levels of processing manipulation, used to study dissociations between implicit and explicit memory in normal subjects can easily be adopted for examining the nature of memory impairment in amnesic patients.

The cognitive and neuropsychological literature concerned with dissociations in memory provides an excellent illustration of how the study of neurological patients can facilitate our understanding of normal information processing and how the paradigms and theories of cognitive psychology can be applied to the study of neuropsychological impairments.

Cognitive Neuropsychology as a Model for Studying Psychological Deficit

Of central importance in the former demonstrations is the attempt to interpret the patterns of impaired and intact cognitive abilities in terms of disrupted components of an information-processing model and to infer from these disturbances how normal cognitive processes are organized. The general concepts and methodology of cognitive neuropsychology may be fruitfully applied to the study of psychological deficit in so-called functional disorders. Consider, for example, how the delusions of schizophrenia may be investigated based on the assumption of modular structure of cognitive functions. Several investigators (e.g., Kihlstrom & Hoyt, 1988; Maher & Ross, 1984) view delusions as the product of disordered perceptual and attentional processes. By this account, schizophrenic patients engage in relatively normal, intact information processing to explain their anomalous perceptual experiences, suggesting that the disruption occurs at the level of specific perceptual modules. If this is a viable account, it follows that schizophrenics should exhibit reasonably intact higher order mental functions or central processes involved in judgment, inference, and problem-solving abilities. In order to firmly establish that the particular processes disrupted in delusions are modular in nature, one would want to follow the principle discussed by Moscovitch and Umlilt (in press) and obtain evidence that the converse case also exists; that is, patients with gross impairment of judgment, inference, and problem solving in conjunction with intact perceptual modules. Interestingly, two subtypes of schizophrenics have been identified that exhibit dramatically different patterns of psychological deficit (Andreasen & Olsen, 1982). One group exhibits primarily positive symptoms consisting of delusions, hallucinations, formal

thought disorder, and bizarre behavior. The other group is characterized by negative symptoms such as affective flattening, alogia, avolition, anhedonia, and attentional impairment. These two groups may provide a useful context for investigating dissociations and drawing conclusions about where in the model of normal cognitive functioning specific breakdowns are occurring.

In addition to postulating dysfunction of particular perceptual modules in schizophrenia, there is evidence to suggest that certain higher order or central processes may be disturbed in this population. Several investigators have suggested that some aspects of the schizophrenic syndrome could be explained by a self-monitoring deficit, whereby patients fail to use feedback to alter their cognitive performance (Brown, 1980; Cohen, 1978; Feinberg, 1978; Goldberg, Weinberger, Berman, Pliskin, & Podd, 1987). In addition to being unable to monitor their own cognitive behavior, schizophrenics appear to be deficient in the ability to monitor and correct ongoing motor behavior on the basis of internal, self-generated cues (Malenka, Angel, Hampton, & Berger, 1982). Interestingly, Malenka et al. (1982) found that schizophrenics were no different than normal subjects and alcoholics in terms of their ability to initiate correct responses on a complex tracing task that prevented the use of the exteroceptive cues. However, the schizophrenic patients differed from the other two groups in an important respect: They were significantly less likely to recognize and reverse false responses. The authors ruled out the possible contribution of nonspecific factors to the performance of schizophrenics and concluded that schizophrenic subjects exhibit an impaired ability to monitor ongoing motor behavior on the basis of internal cues.

From a cognitive neuropsychological perspective, one might hypothesize that a central process, in this case some kind of executive function, may be disrupted in schizophrenia such that a wide variety of behaviors (e.g., motor, cognitive, social) are performed without an adequate error-detecting and error-correcting mechanism in operation. The important dissociation to be examined in this context would be between the specific modules involved in the motor or cognitive functions of interest and the central process responsible for monitoring the output of these modules. For example, Malenka et al. (1982) indicated that schizophrenics initiated correct motor responses equally as often as normal subjects, suggesting that the relevant modules involved in producing the correct response were intact, whereas patients were impaired in their ability to correct inaccurate responses which

may rely on a higher order process. The application of a cognitive neuropsychological framework to this kind of problem stimulates a number of interesting questions that could be pursued and offers a variety of methodological techniques for investigating the relevant dissociations.

From a practical perspective, increased understanding of how the cognitive system of schizophrenics is receiving and processing information will prove crucial for developing effective rehabilitation programs. Recent approaches to rehabilitation have focused on alleviating social skill and community support deficits among the chronically mentally ill. However, Erickson and Binder (1986) argued that many of these patients suffer from subtle cognitive deficits that prevent them from learning and generalizing social and vocational skills to the real world. The cognitive limitations of these patients need to be taken into account when developing intervention programs, and the goal of treatment should be to maximize the preserved abilities and minimize the impact of disrupted functions.

Although we have focused on the psychological deficits of schizophrenia in this section, the principles of cognitive neuropsychology as a model for studying psychological deficit could just as easily be imposed on other types of psychopathology be it affective disorders, eating disorders, or anxiety disorders. In all of these categories, there exists some degree of disturbed information processing along with many intact cognitive components. By viewing the pattern of psychological deficits and preserved functions within a modular framework, research may progress in a more organized and theoretically important direction. The practical implications in terms of developing more effective treatment and rehabilitation programs for patients with various kinds of disturbances may be far-reaching.

PROSPECTS FOR EXPERIMENTAL PSYCHOPATHOLOGY

The present review of the literature on experimental psychopathology has been highly selective. Due to necessary constraints on space, we have attempted to give the reader only the flavor of recent trends in the area. Many syndromes and paradigms have been omitted—substance abuse and infantile autism, for example, and computer simulation and psychophysiology. Nevertheless, it seems clear that after more than a half century of sustained activity, the experimental study of psychopathology appears to be alive and well. New generations of laboratory models,

involving both nonhuman and human subjects, have offered new perspectives on the origins, maintenance, and treatment of phobia, anxiety, depression, psychopathy, and the dissociative disorders. The adoption of contemporary information-processing paradigms has shed new light on the cognitive, emotional, and motivational deficits that underlie the syndromes of psychopathy, depression, and schizophrenia.

One salutary aspect of the field today is the apparent convergence of its two main constituents, laboratory models and psychological deficit. Early signs of this are to be found in the literature on depression, where the learned helplessness model, derived directly from animal research (Maier, Seligman, & Solomon, 1969), led to the concept of hopelessness and empirical work on depressogenic attributional style and depressive realism. The links are even more tightly developed in the area of psychopathy, where a virtual renaissance in psychological deficit research has been instigated by the septal rat model. A further convergence is between psychological deficit in psychopathology and the neuropsychological study of brain-damaged patients. Similarly, we detect the beginnings of an injection of neuropsychological methods into the study of a wide variety of symptoms in schizophrenia and the potential of the neuropsychological model for the study of language disorders in that syndrome. The septal model suggests that the neuropsychological study of psychopathy and other syndromes of disinhibition might well be productive.

Another positive development is the rapid adoption by experimental psychopathologists of new concepts and paradigms, as they have been developed in research on normal mental life (Kihlstrom, 1983). For example, laboratory models of phobias and other anxiety-based disorders have kept abreast of trends within conditioning theory, as evidenced both by their emphasis on cognitive concepts of predictability and controllability and their use of evolutionary and psychobiological concepts related to preparedness and other biological boundaries on learning. While the information-processing view began to be widely adopted within cognitive psychology about the time of Neisser's (1967) seminal monograph and was well consolidated 5 years later (Newell & Simon, 1972), it made its entrance into the study of psychological deficit between 1973 and 1979.

However, other developments have been slow to catch on. Research on language in schizophrenia, for example, has yet to take full advantage of the vast panoply of concepts and methods offered by experimental psycholinguistics. We wonder what would be produced, for example, if experimental psychopathol-

ogists would take seriously the implications of Kraepelin's (1896) notion of schizophasia and approach schizophrenic speech and language in the manner that cognitive neuropsychologists have studied the communication disorders in brain-damaged patients. Similarly, we note that classical models of judgment and decision making based on formal, logical reasoning are threatened by the judgment heuristics approach (for reviews, see Hastie, 1983; Kahneman, Slovic, & Tversky, 1982; Kahneman & Tversky, 1984) that emphasizes the limitations of the human information-processing system and people's reliance on a set of cognitive shortcuts that permit judgment under uncertainty but also increase the probability of judgmental error. Our impression is that these theoretical developments have not been systematically incorporated into studies of thought disorder in schizophrenia, although this situation may be beginning to change (e.g., Chapman & Chapman, 1988; Kihlstrom & Hoyt, 1988; Maher, 1988).

In this regard, investigators of psychological deficit should be aware of recent trends within cognitive psychology that mark the progressive abandonment of the traditional multistore models of human information processing and their possible replacement by variants known collectively as connectionism or parallel distributed processing (PDP; McClelland & Rummelhart, 1986; Rummelhart & McClelland, 1986). By postulating the existence of a large number of domain-specific processing units, or modules, working in parallel to perform various cognitive operations and passing information back and forth between them, PDP models undercut the discrete-stage, serial-processing models that have traditionally offered the best hope of determining the locus of psychological deficit in syndromes such as schizophrenia (Kihlstrom, 1983). Although connectionist models of human information processing remain controversial (Pinker & Mehler, 1988; Smolensky, 1988), issues of connectionism and modularity have already made their way into cognitive neuropsychology, and experimental psychopathologists would do well to consider their implications sooner rather than later.

Despite the difficulties posed by actual and potential paradigm shifts, experimental psychopathology continues to offer a unique and valuable perspective on the nature of mental illness. Historically, human science has progressed along two parallel tracks, constituting two different levels of explanation. At the cultural level, events are explained in terms of social, historical, and economic forces. Thus, the incidence of mental illness may be related to socioeconomic status, or the content of delusions to the culture in

which the patient lives. At the physical level, the same events are explained in terms of biological and chemical processes. Thus, individuals may be at risk for depression by virtue of the genetic endowment, and imbalances among neurotransmitters may be linked to schizophrenia.

Psychology in general, and experimental psychopathology in particular, attempts to explain these same events at a third, intermediate level of individual mental processes—of the person's percepts, memories, beliefs, thoughts, emotions, and motives. Each level of explanation is perfectly appropriate—sociologists are not obligated to explain thoughts and action in biological terms, and psychology is not something to do until the neurochemist comes—and each, when applied properly, relies on and informs the others. Experimental psychopathology at its best, combining the three approaches of laboratory modeling, psychological deficit, and cognitive neuropsychology, provides a way of linking the symptoms of individual mental patients, or classes of patients, with both their biological endowment and the sociocultural context in which they live.

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