2.5.3 Contributions of psychology to the understanding of psychiatric disorders

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Introduction

The contribution of psychology to psychiatry dates back to the early days of experimental psychology. Indeed, Kraepelin performed his pioneering work on psychopharmacology in Wundt's laboratory, and Jung studied the electrodermal reactions of schizophrenic patients during word association tasks that he derived from the British associationist doctrine. Since that time, the impact of psychology on psychiatry has steadily grown. A quick glance at the most influential journals in the field of psychiatry shows that many authors have their roots in experimental psychology.

Psychiatric problems are as numerous as the psychological contributions to their understanding, with contributions ranging from developmental psychology to psychopharmacology. It is not possible to cover even the essentials of the theoretical and empirical work that has been carried out in this field in this chapter. Therefore, rather than trying to summarize the theoretical or empirical contributions, we focus on three lines of research that we believe have been the most important contributions of psychology to psychiatry. The types of contributions that are addressed originate from biological psychology, from information processing frameworks, and from theories about schemas, beliefs, and intentions. The aim is to give a critical, yet balanced and non-technical, overview of the relative merits of these three approaches. In doing so, the emphasis will be on anxiety disorders and affective disorders. The justification for this is both epidemiological and theoretical; anxiety and affective disorders are the most prevalent mental health problems in modern industrial societies, with lifetime prevalences of about 25% for anxiety disorders and 20% for affective disorders. Accordingly, psychological analyses of these disorders have been extensive and have yielded results that are clinically relevant.

People try to make sense of their environment and the behaviour of fellow humans, and to the extent that this is successful, it allows for some prediction and control. When trying to understand how others behave in daily life, people tend to follow a rather specific explanatory strategy. Behaviour and emotional expressions are reduced to beliefs and intentions that are attributed to the person whose behaviour or emotions are to be explained. Consider a simple but straightforward example: if we want to explain why X is carrying an umbrella, this piece of behaviour is reduced to a combination of belief (it might rain) and intention (X does not want to become wet). This combination of belief and intention is thought to provide an adequate explanation. Thus knowledge about beliefs concerning the weather enables us to predict the behaviour of others and to change their behaviour by changing their beliefs.

Almost from its onset, academic psychology has been profoundly discontented with this type of 'intentional explanation' of behaviour. The problems that arise with such explanations are numerous, but in the present context it will suffice to mention the following points. To begin with, knowledge about beliefs and intentions critically depends on the individual's ability to give a reliable and valid introspective account. However, there is a large body of knowledge from experimental psychology indicating an asymmetry between people's readiness to verbalize their cognitive activity and their inaccuracy in this domain. Intriguing examples can be found in Nisbett and Wilson's classical paper 'Telling more than we can know'. Another case in point is provided by all sorts of memory illusions which may occur in children and adults, and which often take the form of grossly inaccurate autobiographical recollections that are described with strong subjective confidence.

A second problem with intentional explanations is perhaps even more fundamental. No doubt, beliefs and intentions can be associated with behaviour or emotions. But the type of feelings and thoughts that enter consciousness and are accessible to verbalization may be 'end-products' of deeper processes. In daily life, people may have access to these end-products and may understandably rely on them when they try to predict behaviour of others. Meanwhile, many researchers believe it to be the task of scientific psychology to move beyond end-products and to treat beliefs and intentions as phenomena to be explained rather than as explanatory constructs.

A final objection to intentional explanations is especially relevant for psychopathology. Intentional explanations assume that subjects are rational in the sense that, given their intentions and beliefs, they are thought to act in their best interests. Yet, in this sense of the word, psychiatric patients almost by definition behave irrationally. Thus, even if one would accept a role for intentional explanation in the psychology of healthy individuals, one could argue that, certainly in psychiatry, intentional explanations are doomed to fail.

If psychology has a contribution to make to the understanding of psychiatric problems, it should be able to pinpoint psychiatric problems to 'underlying' psychological processes. The success of this explanatory endeavour should be apparent from its fruits; it should produce powerful predictions of clinical phenomena and effective...
interventions, preferably successful not only in psychological laboratories but also in treatment settings. The three categories of psychological contributions to psychiatry that are the focus of the present chapter will be introduced below.

In the first category, clinical phenomena to be explained and treated are reduced to biopsychological phenomena. Cognition, emotion, and behaviour are all organized by the brain, and some authors take this to indicate that mental disorders are, in fact, disorders of the brain. This approach stands in the tradition of the 'mental diseases are brain diseases' doctrine that was successfully launched by Wilhelm Griesinger in his *Mental Pathology and Therapeutics* (for a detailed appreciation, see Arens[67]) and is still influential. In the words of one of the pioneers of modern biological psychiatry, 'abnormal behaviour presupposes disturbed cerebral functioning.' Prominent illustrations of this approach are theories and data about the neuropsychology of anxiety and its disorders.[68,69] Obviously, notions like beliefs and intentions have no place in biopsychological contributions to psychiatry. If anything, these concepts deserve explanation.

A second category of contributions circumvents intentional explanation not by focusing on central nervous system dysfunction, but rather by emphasizing aberrations in the way psychiatric patients process information. When relating information processing approaches to biopsychology, it is tempting to draw parallels with computer technology. Whereas biopsychology focuses on the neuronal 'hardware' of patients, information processing research concentrates on the 'software'. Disturbances in the filtering of stimuli, the regulation of attention, the functioning of memory, and so on are investigated as possible antecedents or maintaining factors of psychopathology. In the same way as aberrations of computer software can be analyzed without assuming 'intentions' of the machine, psychiatric problems should be explicable without invoking beliefs and intentions as explanatory notions.

Finally, there are psychological approaches that explicitly and deliberately challenge the widely held assumption, referred to above, that beliefs are but epiphenomena with little or no explanatory power. The central idea here is that many psychiatric problems can be fruitfully and validly reduced to erroneous beliefs held by patients. Like normal people's beliefs about the weather, such beliefs are assumed to be accessible to introspection and open to verbalization. Furthermore, these beliefs are thought to be far more than end-products; they are held to contribute to the origin and maintenance of the disorder.

The remainder of this chapter will be devoted to illustrations of these three types of contributions to the understanding of psychiatric disorders. To the degree that the knowledge is valid and relevant, it should enable one to predict and change psychiatric phenomena. When discussing the various contributions, we shall focus on these two criteria of validity and relevance; what insights allow for predictions and interventions that are relevant to the pragmatic science that psychiatry is by its very nature.

### Biological psychology

To a large extent, psychiatric symptoms can be interpreted in terms of radicalized temperaments and extreme emotions.[69] For example, the shyness implicated in social phobia is connected to neuroticism and, as some researchers prefer to call it, negative affectivity. Likewise, the impulsive behaviour of a psychopathic criminal is the extreme manifestation of a trait known as sensation-seeking. Phobic reactions represent exaggerations of normal fear, while the blunted affect of a schizophrenic patient indicates the breakdown of normal emotion regulation. Thus it is obvious that the study of temperament and emotions is relevant to psychiatry.

Emotion, mood, psychopathology, and temperament all refer to what Oatley and Jenkins[101] have termed the 'affective realm'. The primary difference between these constructs has to do with the time course of the affective phenomena involved, with emotional states lasting for a few seconds and temperaments lasting for years. Although it is tempting to view affective phenomena as subjective inner experiences, research shows that they can fruitfully be conceptualized as biologically based action tendencies. Indeed, the function of affective phenomena is to guide and manage our thoughts and actions in a complex world that is difficult to predict. Accordingly, severe emotional disorders undermine real-life planning. This is exactly what the work of, for instance, Damasio[102] demonstrates. Consider the following experiment. Normal subjects and patients with bilateral frontal-lobe damage are instructed to play a gambling game that involves several card decks. Some of the decks are advantageous in that they result in overall gain in the long run, while other decks are disadvantageous because they cost money in the long run. Meanwhile, it is not possible for subjects to make an exact calculation of the gains and losses associated with each deck. Therefore subjects have to sample all decks and, on the basis of this information, they gradually have to adopt a strategy in which bad decks are avoided. Normal subjects are able to develop such a strategy, but patients with bilateral frontal damage are not. They continue to sample the bad decks and consequently lose all their money. This is associated with the insensitivity of these patients to repeated losses and punishment. In more general terms, the results of this experiment accord well with the observation that, owing to their blunted affect, patients with bilateral frontal-lobe damage have great difficulties in planning ordinary life. Thus affective phenomena provide us with heuristics that guide our decisions.[111]

### Emotions and affective style

The idea that the two cerebral hemispheres make different contributions to the development of dysphoric emotions (e.g., depression, anxiety) has attracted considerable attention in the past few years. Evidence for this idea comes from two separate research lines. The first is exemplified by the experimental work of Hugdahl,[113] who directly manipulated hemisphere information processing (e.g., by confining visual stimuli to one visual field/hemisphere) in order to examine the differential involvement of the two hemispheres in emotional reactions. His studies indicate that fear-relevant stimuli (e.g., pictures of snakes) evoke a cardiac defence reaction when they are flashed to the right hemisphere (i.e. left visual field) of healthy subjects, but not when they are flashed to the left hemisphere (i.e. right visual field) of these subjects. Also, the two hemispheres of normal subjects apparently differ in their conditionability. That is, when visual stimuli flashed to either the right or the left side of the brain are followed by electric shocks, only the stimuli presented to the right hemisphere eventually elicit conditioned fear reactions. Similarly, studies by Wittling[114] indicate that emotionally provocative film clips presented
to the right hemisphere of normal subjects produce a higher increase in cortisol secretion than do left hemisphere presentations of the same film clips. This suggests that cortisol reaction to emotional stimuli is under the primary control of the right hemisphere. Taken together, these findings seem to imply that the right hemisphere is more sensitive to emotional information.

A second line of research relies on direct measures of brain activity. A good example is provided by the electroencephalography (EEG) studies of Davidson, who demonstrated that, in normal subjects, negative emotions (e.g., disgust, fear) are accompanied by a stronger right-hemisphere than left-hemisphere activation. Research findings such as these have led many authors to conclude that the right frontal areas sustain negative emotions and avoidance behavior, whereas the left frontal areas are more involved in pleasant emotions and approach behavior (for a more refined version of this theory, see Heller and Nisichke). There are good reasons to take this argument one step further and to assume that right- and left frontal over-activation are trait-like characteristics that reflect susceptibility to avoidance-related and approach-related behavior, respectively. This is, habitual over-activation of one frontal area or under-activation of the other frontal area corresponds to certain affective styles. Extroversion to this issue is a study which found that young infants with a strongly activated right frontal hemisphere tend to react with crying to subsequent maternal separation. The idea that asymmetries in frontal activation are linked to affective styles is further buttressed by the finding that heightened right hemisphere activation during rest predicts the extent to which healthy adults react with fear and disgust to emotional film clips.

Research on frontal asymmetry and affective phenomena is important because it may shed light on certain psychiatric conditions, such as poststroke depression. Depression is considered to be the most common emotional consequence of stroke, occurring in 20 to 50 percent of stroke patients. There are robust indications that poststroke depression has a negative impact on the rehabilitation of stroke patients. Additionally, damage to the left frontal area is more likely than damage to any other cortical region to be associated with depression. A plausible interpretation of this association is that left frontal lesions result in a decreased activity in this region which, in turn, would lead to a deficit of the positively valenced approach system sustained by the left frontal areas. Such an interpretation accords well with the finding that neurologically intact patients with current unipolar depression or a history of unipolar depression exhibit reduced left-frontal EEG activity.

Some authors have argued that left frontal underactivation represents a risk factor for depression because it biases individuals in favor of a negatively valenced avoidance system. Interestingly, there is tentative evidence that left frontal underactivation is accompanied by cognitive biases (see below) that are known to play a role in the etiology of depression. Future research on affective brain asymmetry may clarify the neurophysiological basis of these cognitive biases. In addition, research in this domain may provide us with clinical tools for predicting treatment outcome. A study by Buhler et al. nicely illustrates this point. These authors measured hemisphere activation by means of a dichotic listening task before depressive patients underwent cognitive-behavioral treatment. Cognitive therapy responders exhibited higher left-hemisphere activity (as indexed by larger right ear advantages for verbal materials) than non-responders. In fact, left hemisphere activation was the best predictor of a favourable response to cognitive therapy.

Temperament

Although the details are still a matter of debate, most researchers agree that only a few dimensions are needed to classify a wide variety of personality types. Students of personality accept that neuroticism (or negative affectivity, behavioural inhibition, or avoidance) corresponds to one core dimension, while extraversion (or positive affectivity or approach) refers to another core dimension. Furthermore, most researchers consider psychoticism (or impulsive sensation-seeking) to be a basic trait. The widely used Big Five taxonomy lists agreeableness and openness to experience as two additional basic traits.

As Gray and Zuckerman have pointed out, there is no one-to-one relationship between basic traits and neurohormone systems. For example, the enzyme monamine oxidase, which has an important function in dopamine breakdown, is negatively correlated with sensation-seeking. This is in line with the observation that disinhibitory forms of psychopathology (e.g., borderline personality disorder, bipolar disorder, positive schizotypic symptoms) are accompanied by low levels of monoamine oxidase. However, sensation-seeking also correlates strongly with testosterone. Likewise, the arousal component of neurotic anxiety is thought to be mediated by high levels of noradrenaline (norepinephrine), while the avoidance component has been related to low levels of paminobutyric acid. The important point to note is that personality dimensions can be decomposed into more basal behavioural tendencies which probably do correspond to discrete neurohormone systems. For example, the trait of impulsive sensation-seeking involves strong approach behaviour which is a function of dopamine and testosterone, weak inhibition which is a function of seratonin, and low arousal which is a function of noradrenaline. Thus, a high score on sensation-seeking is brought about by several neurohormone systems interacting with each other.

Clarifying the precise linkages between personality, behavioural tendencies, and neurohormones will lead to a better appreciation of studies concerned with the genetics of psychopathology. For example, in their twin study, Andrews et al. found evidence for a genetic transmission of depression and anxiety disorders, but they also concluded that this genetic component involves the general vulnerability factor of neuroticism rather than specific symptoms or diagnostic categories. However, it should be noted that, in the final analysis, it is not personality traits such as neuroticism that are inherited, but genes that code for proteins which, in turn, regulate neurohormones.

The concept of behavioural inhibition is closely linked to neuroticism and negative affectivity. It refers to the tendency of some children to interrupt ongoing behaviour and to react with vocal restraint and withdrawal when confronted with unfamiliar people or settings. Behavioural inhibition is thought to be a stable and inherited response disposition that characterizes approximately 10 to 15 percent of children. Cross-sectional and longitudinal data collected by Biederman et al. indicate that behavioural inhibition is a vulnerability factor for a broad range of anxiety disorders. As to the biological underpinnings of behavioural inhibition, relevant parameters have been identified by Schmidt et al., who noted that behaviourally inhibited children exhibit relatively high morning levels of the stress hormone
cortisol. They speculated that these high cortisol levels may sensitize subcortical arousal circuits (e.g. the amygdala) and this would make children more prone to developing serious anxiety symptoms. Interestingly, work on psychophysiological parameters that tap subcortical fear responsivity (e.g. the eye-blink startle reflex) supports this interpretation. For example, Grillon et al. measured startle reflexes in children with a parental history of an anxiety disorder (such children often meet the criteria for behavioural inhibition). They noted that these children displayed a heightened startle reflex magnitude. This is in agreement with the notion that anxiety-prone (i.e. behaviourally inhibited) children have hyperexcitable subcortical circuits that may promote fear behaviour and avoidance.

People high in positive affectivity or extraversion frequently feel joyful, optimistic, and assertive, while those low in positive affectivity are disinterested and pessimistic. Positive affectivity is a stable temperamental dimension with a substantial genetic component. There is also evidence that it is specifically linked to depression. However, the details of this link are far from clear. Some studies indicate that low positive affectivity reflects a predisposition to developing depressive symptoms, while other studies suggest that it modulates the course of depressive symptoms. Another unresolved issue concerns the precise cognitive variables that mediate the connection between positive affectivity and depression. It is well established that individuals who rely on a pessimistic explanatory style (i.e. who habitually ascribe bad life events to their own faults) run a greater risk of becoming depressed than those who rely on an optimistic explanatory style. It is also clear that explanatory style has a genetic component and is a good predictor of the long-term outcome of cognitive treatment for depression. However, solid evidence for the idea that a pessimistic explanatory style functions as a specific cognitive mediator between low positive affectivity and depression is lacking. It may well be the case that such a style predisposes individuals to both anxiety and depression.

Psychophysiological parameters

In general, the search for unique biological markers of psychiatric diagnoses has produced disappointing results. A good diagnostic marker is one that possesses sufficient sensitivity (i.e. high detection rate of a specific disease) and specificity (i.e. strong discrimination between a specific disease and other diseases). Although psychophysiological research has discovered a variety of deviations in psychiatric populations, ranging from an excess of fast EEG activity in alcoholism to abnormal eye-tracking movements in schizophrenia, none of these deviations satisfy the criteria for being good markers. Apparently, the organizing principle behind these deviations is quite different from that of the Diagnostic and Statistical Manual of Mental Disorders (DSM). This has led some authors to conclude that we should stop looking for biological markers of the nosological categories listed in the DSM. After all, there is no reason to treat the taxonomy provided by the DSM as the gold standard.

This is not to say that the psychophysiological abnormalities that have been revealed in various psychiatric populations are meaningless. Space limitations preclude a systematic review of this issue, and therefore we shall focus on a few arbitrarily chosen examples. About 40 to 60 per cent of schizophrenic patients fail to show an electrodermal orienting response to simple sensory stimuli (e.g. sounds). This electrodermal non-response is not due to hospitalization or medication effects, but reflects a profound attentional disturbance. Electrodermal non-response in schizophrenia has a number of interesting clinical correlates. For instance, electrodermal non-response has been found to be related to poor premorbid adjustment, negative and/or more severe schizophrenic symptoms (e.g. blunted affect), poor response to neuroleptics, CT scan abnormalities (e.g. increased ventricular brain ratios), and poor long-term social functioning. Thus, even though electrodermal non-response is neither a sensitive (i.e. only a subgroup of schizophrenic patients exhibit the phenomenon) nor a specific (there are other conditions such as autism and schizotypal personality disorder in which the phenomenon occurs) marker of schizophrenia, it is a clinically relevant phenomenon.

One hypothesis about the origins of hallucinations is that patients misinterpret their own subvocal speech about emotionally charged topics as voices. This idea has been tested in a number of studies where electromyographic data on vocal muscle activity were collected together with reports of hallucinations. The results of these studies were generally negative in that no clear temporal association was found between increased vocal activity and the onset of hallucinations. Although the subvocalization theory of hallucinations proved to be incorrect, it inspired a whole new avenue of research that focused on what has been termed inner speech. Indeed, neuroimaging studies of activity in the language areas of the brain (e.g. Broca's area) during hallucinations did find evidence to suggest that hallucinations are linked to inner speech. Findings such as these have been a major impetus to cognitive theories about schizophrenia. A good example is provided by Frith who argued that most schizophrenic symptoms can be interpreted in terms of self-monitoring deficits. According to this view, hallucinations occur when people attribute their own inner speech to an external source.

Some decades ago, Chapman and Chapman concluded in their classic monograph on schizophrenia that 'most clinical psychologists and psychiatrists believe, almost as an article of faith, that thought disorders and the other symptoms of schizophrenia are a response by the patient to his emotional problems'. This situation has drastically changed due to the extensive biopsychological work in this field. This work has revealed systematic relationships between schizophrenic symptoms and cognitive deficits. Although it is not clear which of these deficits are primary and central to the disorder and which are secondary reactions to the symptoms, some authors have concluded that the time is ripe for small-scale cognitive remediation programmes for schizophrenia.

Psychophysiological parameters such as electrodermal response or electromyographic activity are traditionally viewed as the peripheral ends of a chain. They may reflect central dysfunctions, but do not themselves serve as determinants of these dysfunctions. A further example of such a peripheral parameter is the eye-blink startle reflex. The central antecedents of startle reflex have been well studied because this reflex provides an objective index of defensive action tendencies. For example, patients with phobias, panic disorder, or post-traumatic stress disorder all exhibit exaggerated startle potentiation when startle reflexes are provoked in the context of threatening stimuli (e.g. pictures of phobic objects). In contrast, psychopathic individuals fail to show any startle potentiation in the presence of threatening stimuli. Precisely because startle reflexes do not depend on introspection, startle
Information processing

In the first half of the twentieth century academic psychology was predominantly behavioural in its orientation, concentrating on relations between stimuli (input) and responses (output) and largely ignoring the processes that take place between input and output. Given the ambition to develop a true science of behaviour, this reluctance to dwell upon non-observables in the 'black box' is understandable, but the approach had serious limitations. For example, early century behaviourists, especially in the United States, were largely silent about individual differences as it was assumed that all behavioural manifestations result from stimulus configurations. Evidently, however, people differ very much from each other, and these individual differences are to a large extent linked to biologically based temperaments. A psychological science that ignores individual differences would be of little use to psychiatry.

Another serious shortcoming of behaviouristic psychology was that it ignored the important issue of how the organism manages to produce a response after a stimulus has been perceived. It is clear that stimulus perception and response preparation must be involved, but some stimulus–response relations (e.g. fear conditioning) are acquired through learning processes. Does not learning involve memory? And does not the notion of memory implicate certain qualities of the black box? Likewise, intelligent behaviour requires attention, self-monitoring of motor acts, emotion, and speech, and again all these functions imply a highly specific cognitive architecture of the black box.

In the 1960s, computer sciences began to offer a powerful metaphor for conceptualizing this cognitive architecture. Within a decade, the black box became an appealing and scientifically respectable challenge, dramatically changing the landscape of psychology. This shift has sometimes been referred to as the 'cognitive revolution' in psychology. Whereas the behavioural tradition concentrated on how organisms learn, the central issue for cognitive psychology became how intelligent systems process information. The relevance to psychiatry is straightforward. Given a specific disorder, say depression, the question arises as to whether it is associated with peculiarities in processing information and whether these peculiarities play a role in the genesis or maintenance of the disorder. Before discussing some of the main findings from this research, two preliminary remarks are in order.

First, possibly because of its association with computer sciences, much of the early information processing research in psychiatry concentrated on emotionally neutral information. In memory tests, patients were asked to memorize emotionally neutral or even nonsense words. The self-relevance and 'emotional valence' of information was regarded as reflecting irrelevant 'noise'. It is now clear that this is incorrect; the valence or meaning of the information to be processed is highly relevant to the cognitive psychology of emotional disorders. Depressive patients, for instance, perform poorly on tests measuring memory for emotionally neutral information. Interestingly, the memory performance of depressives is better for emotionally negative material (e.g. words like dead, guilty, suffering) than for neutral information. This indicates that, apart from a general memory dysfunction, depressive patients exhibit a domain-specific memory bias. Another illustration is provided by patients suffering from obsessive-compulsive disorder who are sometimes extremely uncertain about their own memory performance (Did I turn the gas off or didn't I?) This uncertainty may contribute to the functioning and suggests a failure of
metamemory, i.e. the ability to judge whether or not a retrieved piece of memory relates to events that really took place. However, given the clinical picture of obsessive-compulsive disorder, it does not make sense to assume a general deficit in metamemory. Patients do trust their memory when it relates to whether or not they went to the grocery store. What these examples illustrate is that aberrations in information processing do occur in psychopathology, but that such aberrations are often domain specific and confined to the processing of information with a specific emotional valence.

A second preliminary issue is concerned with the fact that information processing takes place at separate stages that are hierarchically organized. These stages involve attention to the information to be processed, sensory registration of the information, comparison of incoming stimuli with information stored in memory, interpretation, decision about action, motor preparation and execution, etc. There is some debate about the precise number of stages that need to be distinguished and their exact sequence. We shall not enter this debate here, but will concentrate on those aspects and information processing stages that are both best studied and most relevant to psychiatry, i.e. attention, memory, and interpretation.

### Attention

We cannot possibly process all the information that is available. By allocating the limited attentional resources that we have to specific stimuli, some information is selected for further processing at the expense of other information. Aberrations in the regulation of attention occur in a wide range of psychiatric disorders.

First, severe disorders like schizophrenia or attention-deficit hyperactivity disorder are accompanied by a general deficit in attentional selection. Patients display attentional deficits that are largely independent of the semantic content of the information to be processed. A good illustration is provided by the systematic work of Shallice and colleagues who sought to identify the earliest stage at which cognitive deficits occur in schizophrenics. This work revealed that schizophrenic patients perform at a normal level on repetitive motor tasks and tasks measuring visual acuity, but display a slowing of speed when they have to react to a signalled target stimulus. This accords well with Krasnepols' clinical observation that a general attentional dysfunction is one of the core disturbances in schizophrenia.

In many other conditions, however, attentional disruptions are not general but domain specific, because they depend on the context (or 'valence' or 'meaning') of the information to be processed. As there is no reason to suppose that such an 'attentional bias' is accessible to introspection, researchers adopted paradigms from experimental psychology for documenting and unravelling the phenomenon. Typically, several groups of patients and healthy controls are asked to perform a task during which distractors, which vary in content (e.g. emotionally neutral versus emotionally negative), are presented. To the degree that attention is selectively allocated to emotionally negative material, for example, performance on the primary task will deteriorate more when negative distractors are presented than when the distractors are neutral. When patients are more distracted by, for example, specific negative distractors than by neutral distractors, relative to healthy or psychiatric controls, it is inferred that they display a domain-specific 'attentional bias'. While a wide variety of such tasks is available, by far the best studied is the so-called emotional Stroop test.

Patients are seated before a computer screen and are presented with a sequence of words varying in colour. Words are sometimes emotionally neutral and sometimes emotionally valent (e.g. related to threat). The subject's task is to ignore the word's meaning and to name its colour. Findings with this paradigm range among the most robust data in the cognitive psychology of emotional disorders. Anxious patients typically slow down when colour naming emotionally provocative words relative to neutral words. Healthy controls typically do not. Attentional bias, measured with this approach, has been documented in addiction and eating disorders, but most notably in anxiety disorders, i.e. phobias, panic disorder, social phobia, generalized anxiety disorder, post-traumatic stress disorder, and obsessive-compulsive disorder. The most intriguing aspect of this phenomenon is not that patients selectively attend to issues of personal concern, but that they are apparently unable to abstain voluntarily from selective attention to threat. After all, they do try to ignore the meaning of the distracting word content. In psychology, such involuntariness is seen as a feature of 'automatic processing', which is contrasted with 'controlled processing' which does require willful effort.

Another psychological feature distinguishing controlled from automatic processing is awareness. Whereas controlled processing (e.g. driving a car by a novice driver) requires awareness of the information to be processed and of the operations to be carried out, automatic or automatized processing (e.g. driving a car by an experienced driver) does not require such awareness. Thus the question arises as to whether anxiety-related non-volitional selective attention to threat requires consciousness of the material that is processed. The answer to this is negative. There are good reasons to believe that even when anxiety patients cannot possibly be aware of the information that is presented to them, they preferentially process information with a negative content. Evidence comes mainly from a modification of the Stroop test. Words to be colour named are presented for an ultrashort period of time, say 20 ms, after which they are replaced by a coloured mask that effectively blocks the after-image of the word, preventing conscious recognition. These masked words are emotionally neutral or negative, and the subject is asked to name the colour of the mask as quickly as possible. Again, anxious patients selectively slow down when a mask is preceded by a threat word as opposed to neutral words. This selective attention to cues that are not consciously identified has been observed with high-trait anxious subjects, with phobics, and with patients suffering from generalized anxiety disorder.

Selective attending to preconscious threat cues in anxiety not only affects ongoing low-level cognitive operations, but also seems to activate the sympathetic branch of the autonomic nervous system; fearful subjects display increased electrodermal activity when confronted with masked presentations of threat pictures, whereas controls do not.

While attentional bias has been observed in several disorders, a curious exception to this rule is depression. Depressed patients tend to selectively remember negatively valenced information ('memory bias'), but they typically do not selectively attend to threat. This may be because depressed people are preoccupied with (perceived) loss in the past and not, like anxious people, with (perceived) future harm. From a functionalistic position, it seems that selective attention is to be expected for those cues that are relevant for immediate action, like approach or avoidance. In line with this implication from general emotion theory, it was found that attentional bias also occurs for emotionally positive cues, given that the cues relate to highly desirable
immediate action. For example, subjects who fast for 24 h selectively attend to positively valenced cues related to eating.\(^{127}\)

Theoretically and clinically, a crucial question is the causal status of attentional bias: is it related to the genesis and/or maintenance of disorders or is it a result of these disorders? The empirical evidence that has accumulated favours both interpretations. Thus, several studies have found that attentional bias disappears or is reduced after successful cognitive therapy.\(^{128-133}\) The finding that cognitive behaviour therapy affects not only behaviour and self-reported complaints but also objective manifestations of information processing, while encouraging for cognitive behaviour therapy, suggests that attentional bias results from anxiety. Obviously, the relevance of attentional bias would be rather limited if there were epiphenomenal and superficial features of the disorder, appearing and disappearing with the waxing and waning of the syndrome. Still, there is more to attentional bias than this. MacLeod and Hagan,\(^{134}\) using the preconsciously operating "masked" Stroop test in a prospective study, observed that future distress could be predicted from a currently present tendency to selectively attend to threat. This was replicated in a cross-sectional study.\(^{135}\)

Of particular interest is the prediction of distress from current attentional bias was unrelated to current anxiety levels. Independent of current anxiety, attentional bias was a predictor of subsequent symptoms. This suggests that anxiety and attentional bias are reciprocally related; anxiety may foster attentional bias, but in and of itself attentional bias may be a vulnerability factor. A therapeutic implication would be that reducing attentional bias may help in reducing anxiety disorders or preventing relapse after successful treatment. While promising observations have been reported,\(^{136}\) no controlled data are yet available.

### Memory

Memory is not what lay people often think it is. It is not a hard disk from which information can be easily retrieved by a simple command. Information represented in memory can sometimes be temporarily inaccessible and can pop up unexpectedly at other times. Not all information is stored in the memory in the same way. Sometimes memories are so vivid that they seem as if one experiences the event again, but other memories are only global abstract ways of knowing what has happened. In some mood states things are remembered that would not easily be remembered in other mood states. Since memory-related disturbances often accompany psychiatric problems, even if there is no direct organic cause for these disturbances, and since memory seems to be essential for our daily functioning, the study of memory processes in different forms of psychopathology has become a major topic in psychological research.

One important issue is the influence of mood on the accessibility of specific memories. Numerous experiments indicate that a person's mood influences what memories are reported when the person is asked to retrieve a memory related to a specific cue; the cue word can be neutral (e.g. street) or highly affect laden (e.g. disappointment). Memories of negative events of unhappy life periods, and even of negative self-descriptors recently learned, are more accessible in a negative mood than in a happy mood, while the reverse is true for memories of positive events of happy periods in life and positive self-descriptors. This seems to hold for naturally fluctuating mood and experimentally induced mood, as well as for psychopathological mood states, notably clinical depression.\(^{136,137}\)

This phenomenon of mood-congruent memory seems to be rather specific for depressive patients or patients with concomitant depressive mood. Accordingly, researchers have focused on the role that it might play in the origins or maintenance of depressive disorders. Teasdale's model is probably the best known and has been very influential. This model states that depressive patients are caught in a vicious circle, such that their negative memories lead to negative interpretations of current events, which in turn maintain the depressive mood, while the depressed mood maintains the increased accessibility of negative memories and the reduced accessibility of positive memories.\(^{138}\) The precise status of the influence of mood on memory has not been fully elucidated\(^{139}\) and the findings in this field do not lead to direct therapeutic interventions, other than the type of cognitive therapeutic methods which seemed to be more related to schema theories (see section below on schemas and beliefs) than to memory theories. However, it is clear that the recall of autobiographical memories is influenced by the current mood of the individual. Thus memory functioning in depression is not disturbed in a general way, but is biased towards the emotionally laden content of the memories. As with attentional bias, memory bias is related to the content of the information that is to be processed.

Why do anxious patients display a clear attentional bias, whereas depressed patients do not display attentional bias but are characterized by a mood-congruent memory bias? One speculation is that this has to do with the original functions of the patient's emotional state. It is helpful to view anxiety disorders as dysfunctional variants of normal anxiety and depressive disorders as dysfunctional variants of normal states of grief. In 'normal' or 'functional' anxiety, there is a potential danger and the resulting anxious state is directed at immediate survival. Thus the attentional system aims at a quick (although perhaps somewhat 'clumsy') signalling of danger, thereby facilitating sufficiently early fight or flight. By definition, the perspective is on the future. In conditions of grief, however, the focus is on what has been lost, and the information processing aims at integrating the new fact (e.g. the death of a loved one) with memories of the lost object. Thus the perspective is on the past, until the new fact has been sufficiently assimilated. It seems that in dysfunctional anxiety and in depressive disorders, the information processing system displays biases directed related to the processes that have priority in the functional variants.

Research on memory functioning in depressed patients has recently focused more on the degree to which memories are specific. Williams\(^{140}\) observed that suicidal and depressed patients find it more difficult than somatic patients and healthy people to report memories of specific events, i.e. depressive patients tend to react with very global memories to cue words. For example, a depressed patient might react to the cue word 'party' with 'I've never liked parties', rather than 'I didn't like the party my neighbour organized last Sunday afternoon; there was almost nobody I know, and the music was very loud so that you couldn't talk to each other'. Although it is unclear how specific this phenomenon of 'overgeneral memory' is for depression, Williams\(^{140}\) has speculated that this deficit may play a crucial role in maintaining depressive disorders. One way in which overgeneral memory might be problematic is that if one cannot find memories of specific events, problem solving becomes very difficult. Memories of specific events can help us to find effective solutions for present problems and to avoid ineffective ways of coping with life. For example, Evans et al.\(^{141}\) reported that the failure of depressed patients to react with detailed
memories to cue words is related to their poor problem-solving capacity, and a longitudinal study by Brittlebank et al.\textsuperscript{[5,5]} showed that overgeneral recall of autobiographical memories predicts a failure to recover from depression following psychopharmacological intervention.

Again, one could speculate about the original function of overgeneral memories in depression. One possibility, related to our speculation about the mood-congruent memory bias in depressed mood, is that in grief the major task for the individual is to integrate the loss into general schematic representations of the self and the world. It does not seem helpful to integrate the loss of a child, for example, into every separate memory of the thousands of specific events one has shared with the child. Thus the observed bias again seems to be closely related to the content of the disorder. However, there are other speculations about the original functions of overgeneral memories.\textsuperscript{[4,5]} For example, it has been proposed that overgeneral memories result from overlearned strategies to avoid painful traumatic memories. Yet another speculation is that the inability to produce specific memories results from a sort of cognitive stagnation in early childhood; young children have more difficulties in producing specific memories than adults, and traumatic experiences during these periods might lead to such a stagnation.

Most recently, attention has been drawn to traumatic memories. In psychopathological conditions directly related to traumas, such as post-traumatic stress disorder, efforts to remember fully what happened seem to be less successful than expected, given the fact that emotional events are usually better remembered than neutral events.\textsuperscript{[6,4]} However, extremely vivid memories seem to pop up and can have an almost real quality; the patient sometimes smells, sees, hears, etc. what has happened during the trauma. It seems as if verbally stored memory, i.e. the conceptual meaning of the traumatic event, is poorly developed, whereas memory on a sensory level is highly developed. Apparently, these 'sensory-coded' memories are poorly accessible for voluntary attempts to remember. According to one psychological memory theory,\textsuperscript{[5,5]} this may be related to how the experience was encoded; experiences encoded on the level of sensory data are poorly accessible to explicit attempts to remember, but easily accessible when cues resembling the original data are used (implicit memory). However, when people process an experience by giving it a conceptual meaning, the opposite seems to occur—explicit memory is relatively good, but implicit memory is relatively poor. What may happen during highly traumatic experiences is that the often unexpected experience, which is difficult to understand conceptually, is processed on a low level (i.e. on the sensory data level) and not at a conceptual level. High stress levels and uncontrollability may also play a role in paralysing higher cognitive functions, so that there is poor conceptual processing of the trauma. This hypothesis explains how lively intrusions and poor explicit memory are both parts of psychopathological conditions that can follow a traumatic experience. Moreover, there is a direct therapeutic implication; by helping patients to give better organized conceptual meanings to the traumatic experience, the memories on the sensory data level are transformed to a conceptual level and memory-related complaints should reduce. In the coming decades, psychological studies of memory will undoubtedly further clarify how memory-related problems following a traumatic experience can be understood and how treatment could help patients recover from its aftermath.

**Interpretation**

Research on attention and memory emphasizes biased processing of relatively isolated pieces of emotionally charged information. At a level of processing that is typically characterized as 'higher', such pieces of information from memory and the outside world are integrated to create a more or less coherent representation of what is going on. For convenience, we refer to this integration of information by the generic term 'interpretation'. Meanwhile, it should be acknowledged that interpretation, thus defined, covers cognitive activity that ranges from the immediate efforts of imposition of a Gestalt on an ambiguous visual stimulus configuration (e.g. the famous Rubin illusion that can be seen as either a vase or two faces) to the rather effortful logical deduction of a valid conclusion from a set of premises.

The present focus will be on three related types of interpretation biases: disambiguation bias, the inference of correlations that are illusory, and using emotional responses as a source of information about the safety or danger of the immediate environment.

The degree to which situations are positive or negative, dangerous or safe, and so on is often quite ambiguous, and assessing the meaning of such situations requires interpretation or 'disambiguation'. Psychologists have studied how affects is related to the interpretation of ambiguous material. In such studies, anxious or depressed people and normal subjects are invited to disambiguate inherently ambiguous stimuli. For instance, subjects are asked to write down orally presented homophones like 'killed' and 'kill', or 'dye' and 'die'. Relative to non-anxious people, anxious subjects tend to favour the negative version of an ambiguous word over the neutral version.\textsuperscript{[9,6]} Likewise, clinically anxious patients, unlike recovered patients and non-patients, tend to negatively disambiguate sentences like 'They discussed the priest's convictions' or 'The doctor examined little Emma's growth'.\textsuperscript{[4,5]} Similarly, anxious people tend to overestimate the chances that unfortunate events will happen to them,\textsuperscript{[5,5]} and they take less time to understand negative turns in narratives but are slower to understand positive turns of the story.\textsuperscript{[6,5]}

The fact that disambiguation bias in anxiety disappears after successful treatment\textsuperscript{[5,4]} suggests that the bias follows from anxiety. This idea is strengthened by the observation that experimentally lowering the mood in healthy subjects is attended by the emergence of a negativistic interpretation bias.\textsuperscript{[5,4]} On the other hand, it seems obvious that disambiguating the world in a negative way may contribute to the maintenance of negative affect. Thus it appears that interpretation bias and negative affect are reciprocally related—negative affect fosters interpretation bias and vice versa.

Research on disambiguation biases has at least two limitations. First, the stimulus configurations to be interpreted are presented simultaneously rather than sequentially. It cannot be established whether the interpretations relate to causal sequences that are assumed by patients but not by normal subjects. Furthermore, the validity of the interpretations given by patients and normal subjects cannot be independently established; patients may be more negativistic than normal controls, but there is no gold standard which can be used to decide whether patients are overly negativistic or whether non-patients are unduly optimistic.

Research on interpretation bias that does not suffer from these problems was initiated by Tomarken et al.\textsuperscript{[5,6]} In the first study, subjects with high and low anxiety were presented a series of slides from three categories, two neutral and one fear-relevant. Each slide was
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Immediately followed by one of three outcomes: a tone, nothing, or shock. The conditional probability of any outcome given any preceding slide was exactly one-third. After exposure to dozens of slide-outcome combinations, subjects were asked to rate the probability of a given outcome provided that it was preceded by a given slide. Highly anxious subjects systematically overestimated the covariation between fear-relevant slides and shock. In an extended replication, de Jong et al. showed spider slides, weapon slides, and neutral slides to severe spider phobics. Slides were randomly paired with shock, tone, or nothing. Again, spider phobics tended to overestimate highly the association between spider slides and shock. This suggests that anxiety is associated with a tendency to overestimate the association between a feared cue and personal harm.

Selective processing of spider-shock combinations was manifest not only from 'illusory correlations', as reported by the subjects, but also from electrodermal response. Subjects not only reacted with stronger electrodermal responses to spider slides, but also reacted with stronger electrodermal responses to shocks that were preceded by spider slides rather than control slides. As to the question of causality, the reasoning of de Jong and coworkers was similar to that employed in the above studies of selective attention. If illusory correlations between cue and harm result from anxiety, reductions in anxiety should be followed by reductions in illusory correlations. However, if illusory correlations act as an antecedent anxiety, they should predict future anxiety, independent of present anxiety, and inducing fearful expectation in normal subjects should make them confirmation biased and suffer from an illusory correlation.

In line with the notion that anxiety causes illusory correlations, it was reported that, using the paradigm of Tornmen et al., untreated phobics show larger illusory correlations than treated phobics not previously tested. It should be noted that, although illusory correlations after therapy tend to be low, there is substantial interindividual variation. Likewise, although long-term follow-up of behaviour therapy for spider phobias tends to be good, here too the variance is considerable. By testing whether residual illusory correlation after therapy predicts relapse, one may obtain a first impression of an anxiogenic role of illusory correlation. The correlation between residual illusory correlation after therapy and the increase in phobia severity from posttest to follow-up after 2 years was found to be 0.61. This correlation remained unchanged, even when post-treatment severity of complaints was partialed out. This indicates that the tendency to overestimate the association between phobic cue and harm, and by itself, independent of anxiety, may contribute to the (re)occurrence of fear.

If illusory correlations in and by themselves contribute to the maintenance of anxiety, normal subjects should become resistant to disconfirmation after an illusory correlation between cue and harm is induced. In an experiment that specifically addressed this issue, normal subjects were shown a random series of two neutral slides (circles and crosses), each of which was sometimes paired with shock and sometimes not. During the initial trials, suggestive combinations were given with the target slide being paired with shock in 70 per cent of the presentations and the control slide in 30 per cent of the presentations. During the remainder of the trials, target slides were gradually paired less frequently with shock whereas control slides were paired more frequently with shock so that, finally, both target and control slides had been paired with shock in exactly 50 per cent of the trials. Throughout the experiment, probability estimates of the connection between slides and shocks were obtained after each slide. In the suggestive phase, subjects reported that they expected the target slide to be specifically related to shock. After the suggestive series, even though the true target-shock association dropped below 50 per cent and below the association of control slide and shock, subjects remained convinced of the target-shock association and there was no sign that the disconfirmatory evidence resulted in extinction of this belief. Thus it is quite easy to induce an illusory correlation between cue and harm in healthy subjects. The striking observation was that, once established, the illusory correlation remained unchanged, even in the face of disconfirming evidence.

Anxious subjects report that they think fear cues are predictive of harm. The present experiments indicate that, if such an association is established, it becomes self-supporting. Confirmatory evidence is overvalued, disconfirmatory evidence is ignored, and fearful expectations remain intact.

Research on illusory correlations is interesting but seems to suffer from a lack of clinical or 'ecological' validity. Under laboratory conditions, patients may see unrealistic associations between cues and feared consequences, but outside the laboratory the association between feared cues and anticipated disasters is virtually absent; touching door knobs does not result in HIV infection, palpitations do not predict cardiac arrest, and so on. Although feared and avoided cues do not predict objective harm, they reliably predict something else—the occurrence of an anxiety response. Could the very occurrence of an output phenomenon like an emotional response influence the interpretation of the stimulus that served as the input? There is strong evidence that it can. To a large extent, people derive the meaning and valence of a situation from their responses to that situation. This is illustrated by the social psychology work of Valins, who showed, for example, that when male students received false feedback about cardio-acceleration when seeing a particular erotic picture, the subjective attractiveness of the picture increased. There is a wealth of data from psychopathology indicating that panic patients in particular take the 'output phenomenon' of anxiety responses as predictors of serious harm. In line with this, Ehlers et al. using set-ups not unlike the one mentioned above, showed that once panic patients are provided with false feedback about cardio-acceleration, subjective fear increases just like heart rate and blood pressure.

The tendency to interpret fear responses as indicators of danger has been labelled 'emotional reasoning'. In the realm of anxiety and threat, such emotional reasoning appears to be a prominent characteristic not only of panic patients, but of anxious patients in general. Groups of 41 panic patients, 20 spider phobics, 58 social phobics, other anxiety patients, and normal subjects were given written scenarios that related to panic, social interaction, confrontation with a spider, and a non-disorder relevant situation. Four subtypes were constructed and administered for each scenario: information about objective danger was either given or not given; similarly, information about the occurrence of a fear response was given or not given. Subjects were asked to rate each of the four subtypes of the scenarios in terms of objective danger.

Normal subjects were barely influenced by response information. Their danger ratings simply followed danger information provided by the scenarios, but scores were not affected by fear information. The pattern in the patient groups was strikingly different. Compared with controls, patients generally gave higher danger ratings, even when object safety information was provided. More important was that
patients, but not normal controls, engaged in ‘emotional reasoning’; the dangerousness of a situation was inferred from the fact that anxious responses were included in the scenarios. Remarkably, this ‘emotional reasoning’ of patients was not specific, i.e. all groups of patients displayed the reasoning pattern to the same degree in all domains that were studied (panic relevant, socially relevant, and spider relevant).  

Schemas and beliefs

Reducing emotions and behaviour to a person’s beliefs and goals appears to be closely associated with the idea of the human as rational actor. This notion may have some explanatory virtue in non-clinical areas, but it may be hard to see how beliefs and intentions may account for psychiatric problems.

An initial solution was proposed by psychoanalytic theory, which stated that intentions (wishes, desires) unknown to rational consciousness direct the emotions and behaviour of patients with emotional problems. The main problem with this view is that there is no corpus of scientific data to support it.  

That is, many psychological processes like memory or attention do not require consciousness (see the discussion of attention above), but these ‘unconscious’ or ‘preconscious’ processes are typically rather diffuse and non-specific, or ‘quick and dirty’ and far less elaborate than beliefs.

A second way of applying intentional explanations to psychopathology is bluntly to assume that the emotions and behaviour of patients and healthy people are guided to the same degree by beliefs. The crucial difference between clinical and non-clinical groups may be the content of their beliefs. In cognitive theories of the type discussed here, it is assumed that various forms of psychopathology are associated with specific beliefs about the self, others, or the outside world. According to cognitive psychology, such basic beliefs are organized in schemas. Schema is a highly theoretical term denoting a hypothetical knowledge structure in memory. Schemas are assumed to organize selective attention, interpretation of events, and strategies for survival. In other words, schemas underlie information processing, conscious thoughts, emotions, and behaviour. Although much of the content of the knowledge represented in these schemas is not necessarily accessible to direct introspection, it is possible to reconstruct it in verbal terms. Such reconstructions are usually called assumptions or beliefs, and it is assumed that, in psychiatric patients, such ‘pathogenic’ beliefs are unrealistic or dysfunctional, resistant to disconfirmation by corrective information, and play a crucial role in the genesis and/or maintenance of the disorder.  

In the remainder of this section, we first discuss evidence relating to the existence and nature of such unrealistic ideas, and then consider the rather important issue of the causal status of unrealistic/dysfunctional beliefs in psychiatric disorders.

Presence and subjective credibility of ‘pathogenic’ beliefs

Traditionally, it has been believed that ‘neurotic’ symptoms are experienced by the patient as egodystonic and that patients recognize the irrational character of their emotions or behaviour. Recent data show that this widely held idea needs to be qualified. The common way of gaining access to someone’s beliefs is to ask the person. A number of studies have followed this approach and yielded findings that do not support the hypothesis of egodystonicity of neurotic problems. One could counter that when, for example, an obsessive-compulsive patient claims that she really believes that not washing her hands after turning a doorknob will result in catching a disease, this may be a rationalization. That is, confronted with her own irrational washing behaviour, the formulated belief may be a post hoc explanation intended to justify the behaviour to herself and/or to others. However, it is far from obvious that it is socially more desirable to express beliefs that others regard as highly irrational (touching doorknobs results in disease) than to ‘admit’ that one endorses the view that the behaviour is irrational. Widely held impressions about egodystony, obtained in the consulting room, may be caused by patients denying beliefs that they expect the clinician to find untenable.

Irrational beliefs about the outside world are common in anxiety disorders. Especially interesting are people with monosymptomatic phobia who tend, in contrast to other anxiety patients, not to suffer from comorbid pathology and who present the clearest case of irrational emotions and behaviour in otherwise healthy people. While egodystony in specific phobias is even a diagnostic criterion of DSM-IV (‘The person recognizes that the fear is excessive or unreasonable’), systematic questioning using a paper-and-pencil task, without the social pressure of an interviewer, reveals that spider phobics tend to endorse highly irrational beliefs about the dangerousness of spiders. The credibility of these frightening ideas is especially high in the presence of the phobic cue. Social phobics appear to have negative beliefs about their own social performance and about others, whom they believe to be more critical and rejecting than they actually are. Furthermore, prominent other-centred problem-related convictions are found in patients suffering from borderline personality disorder who tend to believe that other people are malevolent, and will abuse, punish, or abandon the patient when the relationship becomes intimate.

Apart from beliefs about the outside world and others, a number of psychiatric disorders appear to be characterized by problematic beliefs about the self in general or about specific internal events. Generalized beliefs about the self being vulnerable and worthless are found in depression and borderline personality disorder. Panic patients firmly believe that specific benign bodily sensations such as palpitations predict imminent catastrophes (e.g. cardiac infarction). Interestingly, the paroxysmal occurrence of the sensations feared by panic patients is highly prevalent in the general population. The crucial difference between such non-clinical and clinical panic is that non-clinical panickers are far less inclined to believe, during the attacks, that they may die from suffocation, have a cardiac arrest, lose consciousness, and so on. Remarkably, phobics appear to expect similar catastrophic consequences of experiencing fear and related bodily sensations during confrontation with the phobic object.

Other internal events that can be subject to distorted beliefs are cognitive processes. Examples are the belief, found in many elderly people, that one’s memory is failing although objective memory performance is, by all standards, normal. Just as the bodily sensations that panic patients fear are quite common, so are the types of intrusions that obsessive patients report. The content of the clinical intrusions is no different from that of non-clinical intrusions. The latter also circle around themes like sex, aggression, blasphemy, and illness. What is different, however, is the appraisal of the intrusions. While obsessive-compulsive patients regard them as highly aversive
and try to resist them. Healthy people do not. Relatedly, negative intrusions are common after loss and trauma. Post-trauma intrusions can be interpreted as normal and adaptive responses to extremely aversive events or, alternatively, as indications that one is losing control, that one can never concentrate or enjoy life anymore, etc. Victims suffering from post-traumatic stress disorder report far more negative ideas about intrusions than do victims without post-traumatic stress disorder. Worry is a hallmark of generalized anxiety disorder, in which patients appear to hold a wide range of beliefs about the pros and cons of worrying (so called "meta-cognitions") which are hypothesized to fuel the worry process. Hearing voices, curious as it may sound, is not a pathognomonic sign of schizophrenia. This phenomenon occurs in approximately 10 per cent of the general population and is related to conditions like sensory deprivation, sleep difficulties, intense imagery, and so on. How people cope with voices might be relevant when considering to what extent hallucinations might develop into pathological phenomena.

Thus there is good evidence that patients with a variety of disorders maintain problem-related beliefs that are highly unrealistic or dysfunctional. Two questions arise. First, given that there are no formal thought disorders, why do patients not give up such beliefs in the light of disconfirming evidence? Second, are there arguments that these beliefs are causally related to the problem? If so, what are the clinical implications?

Immunity against disconfirmation

It is perfectly possible that a large number of people develop irrational beliefs of the types discussed above, but the vast majority are quick to reject them and do not present for mental health care. However, that would make the question even more compelling for those who do seek help. Why do these patients fail to reject problem-related beliefs that are logically and/or empirically untenable or extremely dysfunctional?

Behavioural processes seem to be involved in irrational fear. The conviction that a particular situation predicts harm motivates avoidance and escape of that situation. This deprives the patient of the opportunity to experience the fact that the feared situation is innocuous. In traditional behavioural accounts of fears and phobias, avoidance and escape were also seen as maintaining factors. Escape and avoidance were held to reduce anxiety immediately, and this anxiety reduction was thought to reinforce avoidance. From the present cognitive stance, this view appears not to be entirely correct. Patients do not avoid because otherwise they will become anxious, but because they believe that harm will be inflicted if they do not avoid. Thus avoidance maintains anxiety disorders because it prevents disconfirmation of fear-related beliefs. Likewise, depressed patients seem to behave in ways that tend to confirm their dysfunctional beliefs and they do not engage in activities that might yield disconfirming information. Depressed patients who believe that they are unlovable tend to isolate themselves, and the lack of social interaction is likely to be interpreted as further evidence for their belief.

Feared situations can be avoided but, as indicated above, some beliefs relate to internal events. Some patients fear internal events like bodily sensations (e.g. panic patients or hypochondriacs) or intrusions (e.g. obsessive-compulsive patients), and such internal events cannot be avoided. Panic patients have experienced hundreds of attacks attended by feared sensations but not followed by the catastrophe that they believe to be predicted by these sensations. Likewise, obsessive patients have typically experienced hundreds or thousands of intrusions without the feared outcome materializing. Why do these patients not give up their beliefs if they believe them to be so clear and personally experienced as contradicting evidences?

The phenomenon of safety behaviour, which appears to be functionally equivalent to avoidance and escape, may be relevant here. Once the feared situation is encountered and the patient believes harm is about to occur, he or she may try to prevent this. For instance, panic patients may sit down and try to relax in order to prevent cardiac failure. The non-occurrence of a heart problem may then be attributed not to the harmlessness of palpitations, but to the effectiveness of sitting down and relaxing. The rituals by which obsessive-compulsive patients respond to intrusions seem to serve the same function: predicted harm is prevented by some safety operation.

Apart from avoidance, escape, or safety behaviours that immunize against disconfirmation, the cognitive biases discussed earlier may, of course, be relevant here. The phenomenon of emotional reasoning appears to be especially relevant to our understanding of hypochondriasis. The feared sensations are not followed by any catastrophe, but they are followed by intense anxiety. Given that anxious patients take the occurrence of anxiety as evidence for the presence of danger, beliefs may be strengthened by the very fear to which the beliefs give rise. Other cognitive biases may also serve to maintain the disorder. Selective attention to threat reinforces the experience of living in a threatening environment and selectively remembering negative experiences may foster hopelessness. Interpretation biases may provide subjective evidence that the disturbing beliefs are valid.

Causality and clinical relevance

The issue of causality of beliefs to behaviour and emotions can be treated not only as a philosophical topic, but also as a straightforward empirical issue. If beliefs are causal to pathology, normal subjects should show pathology-related behaviour and emotions once they are experimentally made to endorse the beliefs held by patients. In contrast, reducing the credibility of irrational beliefs in patients should result in a reduction of symptomatology. If the beliefs are rejected altogether, pathology should disappear. There is some evidence that allows for an evaluation of the causal status and (hence) clinical relevance of problem-related beliefs in neurotic disorders.

When healthy subjects are made to believe, as panic patients do, that particular bodily sensations predict harm, they immediately become anxious upon experiencing these sensations. Likewise, normal subjects tend not to become anxious during lactate infusion, whereas panic patients do so. But once normal subjects are made to believe that lactate infusion is a highly aversive experience, they become extremely upset during the infusion. Some authors have pointed out that a certain type of appraisal bias known as thought-action fusion underlies the etiology of pathological obsessions. Briefly, thought-action fusion refers to an overevaluation of intrusive thoughts, such that unwanted thoughts are appraised as equivalents of unwanted actions. An example would be the belief that unacceptable thoughts are as bad as the actual actions they describe. It is easy to see how thought-action fusion could contribute to an inflated sense of responsibility and, eventually, suppression and neutralization attempts. A recent study explored the effects of experimentally induced thought-action fusion. Students underwent a bogus
EEG recording session. The experimental group were informed that the apparatus was able to pick up the word 'apple' and that thinking of that word could result in the administration of electrical shocks to a person in an adjacent room. The control group were only told that the EEG equipment was sensitive to 'reading' simple words such as 'apple'. After having spent 15 min in the EEG laboratory, students completed a short questionnaire about the characteristics (e.g. frequency, aversiveness) of the target word (i.e. apple). Results showed that students in the experimental group reported a higher frequency of target thoughts, more discomfort, and a greater urge to suppress compared with control students. These findings are consistent with the idea that thought-action fusion promotes intrusive thinking.\(^{120}\)

Even more relevant may be the numerous experiments indicating that reducing the credibility of catastrophic misconceptions greatly reduces the panicogenic effects of so-called pharmacological challenge tests in panic disorder. Pharmacological interventions such as lactate infusion or CO\(_2\) inhalation induce salient bodily sensations in both normal subjects and panic patients. Panic patients believe that these sensations predict harm, while normal subjects do not. If, prior to the challenge, panic patients are reassured about the procedure by a careful explanation of the nature of the sensations to be experienced, by giving them the illusion that there is in control, or by greatly increasing the predictability of what will happen, the challenge become far less frightening and panic attacks are blocked.\(^{121,122}\)

Yet another demonstration of the causal role of beliefs in psychopathology is provided by an experiment by Lopatka and Rachman.\(^{123}\) Obsessive-compulsive patients are known to believe that they are excessively responsible for the catastrophes that may happen and if they make a mistake in a specific area, which varies from patient to patient. They feel uncomfortable if they are not very sure that they undertook every possible action to reduce their responsibility and compulsively check, wash, or perform other rituals to ensure that they did not play a role in bringing about any misfortune to others or to themselves. Lopatka and Rachman\(^{123}\) asked obsessive-compulsive patients to sign contracts that stated that the experimenter took all responsibility for possible harmful consequences following a specific action of the patients which usually elicited discomfort and compulsive behaviours. Compared with those who had signed neutral contracts, not only the feelings of responsibility, but also the urge to check, discomfort, and related variables, were significantly lower when the patient had signed the contract in which the experimenter took all responsibility. When the contract stated that all responsibility was given to the patient, perceived responsibility increased compared with those who had signed neutral contracts, and obsessive-compulsive complaints also tended to increase. This experiment demonstrated that obsessive-compulsive complaints can be increased or decreased by experimentally manipulating responsibility beliefs.

If reducing the credibility of negative beliefs results in a reduction of pathology-related phenomena in the laboratory, the theoretical relevance may be clear: apparently, the manipulated beliefs play some role in the origin or maintenance of the pathology. Clinically much more interesting are the effects of reducing catastrophic beliefs outside the laboratory and in treatment settings. These efforts typically take the form of cognitive therapy for specific disorders. These interventions target specific beliefs assumed to be the cognitive nucleus of a certain disorder. The therapist identifies the specific content that the supposed pathogenic belief has for a certain patient, while the tactical aim of the therapist is to have these beliefs replaced by less irrational and less invalidating ones. From the assumed causal status of such beliefs it follows that cognitive therapy should be uniquely effective.

Of course it would be incorrect to infer etiology from treatment effects. However positive, it can never be ruled out that the effectiveness of cognitive therapy, or any other biological or psychological treatment, may result from other processes than the ones that were intended to target. What can be said however, is that failures of cognitive therapy would have posed huge problems for the underlying theory. So far, the literature shows that cognitive theories rank among the most effective treatment for psychopathological problems.\(^{124-128}\)

**Summary**

This chapter has been organized around three psychological perspectives on psychiatric problems: biological psychology, information processing, and the cognitive psychology of beliefs. These perspectives reflect three hierarchically related levels on which psychiatric problems can be studied. Human information processing presupposes neural hardware, and beliefs presuppose information processing. When psychiatric phenomena are to be explained in psychological terms, they should be reduced to underlying psychological processes. It is often assumed that the further this reduction is taken, the more respectable and fundamental theories become. On this view, theories focusing on beliefs would be relatively superficial, while theories about neural hardware would be relatively fundamental, if not 'the real thing'; We shall not enter the somewhat philosophical discussion about the merits and necessity of reduction, but reiterate that we do not share the view that the quality of explanations critically depend on the degree of reduction. Rather, it is suggested that the optimal degree of reduction is pragmatic, i.e. the degree of reduction should be dictated by what scientific stance offers the best opportunities for prediction and experimental manipulation of psychopathological phenomena. After all, it would be hard to defend that a certain psychological perspective is superficial when it allows for effective prediction and intervention. The other way round, it would be problematic to claim that perspectives that do not result in precise prediction and intervention nevertheless offer profound understanding.

The merits of neuroscientific contributions to psychiatry are extensively discussed elsewhere in this volume. Technological developments in the field of neuroimaging offer tantalizing prospects of relating disorder to its neural hardware. It will be particularly interesting to find out what neurophysiological patient-control differences reflect emotions and cognitions of patients, and what brain characteristics represent true pathophysiology. In obsessive-compulsive disorder, for instance, positron emission tomography (PET) scans reveal distinctive brain changes. These changes disappear after successful treatment of obsessive-compulsive disorder, no matter whether the treatment is pharmacological or behavioural.\(^{129}\) Of course, this suggests that the observed PET scan deviations reflect, rather than cause, obsessive-compulsive pathology. The implication is that normal subjects who are, experimentally, made to feel, think, and act like obsessive-compulsive patients will display the same abnormalities found in patients. Studies like these will no doubt be undertaken and most probably a differentiated picture will occur: just as differences between patients and controls in beliefs may or may not be epiphenomena, visualized differences in brain function may or may not be a mere reflection, rather than a biological cause, of the disorder. Hap-
ply, these issues are empirical and can and will be solved by obtaining controlled data.

Theories and paradigms from information-processing approaches have been applied to a wide range of psychopathological phenomena. The emphasis in this chapter was on the selective processing of emotionally valent information. Distinguishing between processing of neutral and negatively valenced information proved to be fruitful; a number of robust processing biases, related to the emotional valence of the information, were identified. There are some indications that such processing biases serve to maintain psychiatric disorders. For instance, relapse after behaviour therapy could be predicted from the tendency of successfully treated patients to see illusory correlations between phobic cues and harm. Also, the occurrence of anxious and depressive problems after severe life stress could be predicted from a tendency to attend selectively to threatening material that was unconsciously perceived.

While explanation of neurotic disorders in terms of beliefs is at odds with popular views about egodystonic, the unreliability of introspection, and the epiphenomenal status of beliefs, such 'intentional' explanations have proved remarkably successful. Not only did they generate successful laboratory research, but their clinical implications are straightforward and have made effective treatments available for bulimia, anxiety disorders, and depression, for example. Cognitive therapies have recently been developed even for notoriously difficult disorders such as schizophrenia, and results from controlled trials are quite encouraging.

In clinical psychology, a distinction is commonly made between causal processes in the origin of disorders (aetiology and pathogenesis) and causal factors involved in their maintenance. As to the aetiology, progress in behavioural genetics and in the identification and measurement of stable individual differences in temperament have indicated traits that act as vulnerability factors for the development of psychopathology. In the area of anxiety and affective disorders, so-called 'negative affectivity' is of particular relevance. Meanwhile, although negative affectivity, or 'neuroticism' contributes to the development of pathology and while patients with anxiety or affective disorders almost invariably score in the extremes of negative affectivity, the vast majority of people high in negative affectivity do not suffer from Axis I disorders. There is very little data to indicate under what conditions some people at risk develop maladaptive and negative schemas about the self, others, and the outside world. For more is known about how such schemas or beliefs, once established, are maintained. Processing biases and avoidance, escape, and safety behaviour may act in concert to maintain neurotic disorders. No doubt this picture is very incomplete, but it covers much of the existing data, it has a good record when it comes to experimental validation, and it has therapeutic implications that are encouragingly effective.

References


