Slings and arrows

Depression and life events

KEITH OATLEY *describes how even in an age of genomics, neuroimaging and Prozac, our understanding of emotional disorders continues to involve the ups and downs of everyday experience.*

WITH the availability of more and more drugs that affect the mind, it is tempting to think that the most common kind of emotional disorder – depression – is like an infection for which one can take antibiotics. A difficulty with this formulation is that it veers too close to the idea that physical pain is due to insufficient aspirin in the bloodstream. Depression can be thought of as a disturbance of brain metabolism, a kind of illness, and indeed in some people it takes on those characteristics, but its most usual occurrences are not independent of life circumstances, plans, or relationships.

In the new era of genomics and psychopharmacology, psychologists of all different persuasions need to recognise that adversities – in psychology's terms, life events or stressors – are as important now as they were found to be 30 years ago.

Depression and adversity

A major depressive episode can be diagnosed if, for at least two weeks, a person is unbearably sad or has lost pleasure in most things, together with four other symptoms that include sleep disturbance, lack of energy, slowing of action, inability to concentrate, feelings of worthlessness, and thoughts of suicide. In Western industrialised countries about one in six people suffer during their lives from depression at this level, which would justify them being seen in a psychiatric outpatients clinic. Depression can be a terrible scourge, and antidepressant drugs including selective serotonin reuptake inhibitors (SSRIs) such as Prozac have been a boon for many. But we are still some way from Aldous Huxley's (1932) Brave New World of pharmaceutical insulation from events.

The research on depression that made the biggest impact in recent times was conducted by sociologist George Brown and psychologist Tirril Harris, as described in their 1978 book. They found that in its first onset depression usually happens for a reason, a serious adversity. They interviewed 458 women in South London, and found that 37 of them (8 per cent of the total) had become clinically depressed in the previous year. Of the 37 women who became depressed, 33 (almost 90 per cent) had suffered an adverse life event (such as a bereavement) or a serious difficulty (such as being in a relationship with an abusive husband). This compared with only 30 per cent of the women who did not become depressed suffering such an adversity. In only four of the 37 women who became depressed was their onset of depression unrelated to any adversity. If, when a serious life event or difficulty struck, the women in Brown and Harris's study lacked a protective factor such as social support from an intimate relationship in their life, their risk of suffering a breakdown was much increased.

Brown and Harris's work is a fine example of a piece of social science research that was done really well, and that had an important effect. The finding that most people did not get depressed because there was something wrong with their personality, but because there was something wrong in their lives, transformed how depression was seen by general practitioners and psychiatrists in Britain.

Winifred Bolton and I took Brown and Harris's work as the starting point of a theoretical article (Oatley & Bolton, 1985) addressed to psychologists, to show how mind and world are connected. Most of the serious life events that cause depression are losses, either of important relationships or

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Most serious life events which cause depression are losses of one kind or another

DISCUSS AND DEBATE

How might psychological therapy or pharmacotherapy be affected by the finding that a person may become more vulnerable to depression after having had a first episode?

What are the implications of depression and normal sadness sharing some common neural mechanisms?

If it became relatively easy to assess whether children had some particular gene, such as the 5-HTT promoter gene in its less effective form, what might the implications be?

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roles or of life projects that were fundamental to people's identities. Protective factors such as social support described by Brown and Harris were parts of people's lives that enabled them to feel themselves even when substantial losses occurred. For people who were protected in this way, a serious adversity could cause sadness or anger, but seldom the hopeless and disabling despair that put them at risk of abandoning children and spouses in suicide attempts.

Kindling: In the aftermath of a first episode

Although for some people depression becomes chronic, for most it resolves, so that after several months they can get on with their lives. But what happens then? Is the person who has been depressed more liable to become depressed again? Unfortunately the answer is yes.

The phenomenon has been called kindling, as described for instance by Kenneth Kendler and his colleagues (e.g. Kendler *et al*., 2000). They found that although a first episode of depression is indeed usually caused by a severe adversity, the experience of one depressive episode makes a second more likely, the second makes a third yet more likely, and so on.

Scott Monroe and Kate Harkness (2005) have distinguished two ways in which kindling might work. One is that the depressive disposition might become autonomous, so that processes that trigger depression come to be internal rather than external. This corresponds to the older idea of depressive personality. The alternative is that kindling is a sensitisation process in which, with increasing experience of depression, progressively less severe adversities can trigger each succeeding episode. Sensitisation can be thought of as like the formation of mental habits that become more and more easy to fall into. These two explanations have not yet been fully distinguished, but for both it seems clear that therapy that could prevent a recurrence after a first episode would be especially important.

The effects of serotonin

It is known that genetic factors play a part in depression. But how do they work, and do they run counter to the idea that life events are important? One answer, recently found by Avshalom Caspi and his colleagues (2003), is that genes do increase the risk of depression, but they do so in conjunction with adverse life events.

Caspi and his team studied a cohort of 1032 people (males and females) who were followed up at approximately two-year intervals from age three to age 26 in the Dunedin Multidisciplinary Health and Development Study, in New Zealand. The participants were tested for the 5-HTT transporter gene, which promotes the transmitter substance serotonin. The gene is polymorphic: it occurs in two forms, one short and the other long. The long form is more efficient at promoting serotonin. Each person has two such genes, on paired chromosomes. In the cohort, 17 per cent of participants had two shorts, 51 per cent had a short and a long, and 31 per cent had two longs. Participants were also assessed for adverse life events between their 21st and 26th birthdays.

The results were striking. For participants who suffered an adverse life event, those who had gene pairs that included at least one short form of the gene were more likely to become depressed than those who had two long forms. The risk of becoming depressed was greatest for people with two short forms of the gene. Where people had at least one short form of the gene but no adverse event, depression did not occur. Having two long forms of the gene, and hence increased serotonin, was a protective factor for depression in response to adversities, similar to the effect of social support. The study of genomics – understanding the effects of specific genes – is clearly going to become more and more important. But genes are not always destiny. Often, perhaps usually, they work in conjunction with environmental factors in development and, in the case of depression, in conjunction with events such as adversities.

What has the new technology of neuroimaging told us? Helen *et al*. (1999) used Positron Emission Tomography (PET) to study how drugs that affect serotonin work. First they asked normal women volunteers to write autobiographically about two events that made them sad. When the women read and thought about these passages, they became sad. During this time, as compared with during neutral moods, PET-measured activity in a limbic brain region called the subgenual cingulate was substantially increased, while activity in the prefrontal cortex was decreased. These results on normal women were then compared with results from a different set of participants who had been clinically depressed and treated for six weeks with an SSRI drug. Among the depressed participants whose mood improved on this drug that increased brain serotonin, PET neuroimaging showed decreased activity in the subgenual cingulate region and increased activity in the prefrontal cortex:

a pattern opposite to the one found during sadness. This study indicates that depression is related to normal sadness, which in turn is related to losses. The same brain regions are involved in each case. Effects of serotonin and effects of adversity are not competing explanations, they work on the same underlying neural mechanisms.

Attachment style

Following discovery of the system of infants's attachment to their caregivers, Mary Ainsworth and her colleagues (1978) devised a test for one-year-olds in which their mothers left them in a strange room for a few minutes and then rejoined them. Based on how they reacted emotionally to the departure and reunion, infants were classified as having one of three styles of attachment: secure, ambivalent, or avoidant. Do these styles persist?

It turns out that life events are again critical. Everett Waters and colleagues (2000) studied 60 white middle class people who had their attachment style assessed at age one by the test in a strange room, and who at the age of 21 were given an adult attachment interview. The continuity of attachment style was striking: 72 per cent of people retained their style of attachment from infancy to adulthood. However, of those whose style changed, there was a significant association with an adverse life event such as physical or sexual abuse during childhood, loss of a parent, parental divorce, or parental mental illness.

In another study, Judith Crowell and colleagues (2002) assessed adult attachment in 157 couples three months before they were married, and then again 15 months after they were married. Most participants (78 per cent) retained their attachment style between the pre-married and married state. Before marriage, 50 participants reported unresolved loss of, or abuse by, an earlier attachment figure. Alongside their attachment style, they were therefore described as 'unresolved'. Of these, 46 per cent remained 'unresolved' after marriage, but the stability of participants' 'resolved'/'unresolved' status from before to after marriage was associated with severe life events.

The responsive person

We psychologists have tended to like conceptualisations such as that of personality traits that persist in the individual despite changes in circumstances. We have also been drawn to genetics, perhaps for a similar reason: genetic explanations point to how and why this person is different from that one.

There need, of course, be nothing wrong with such explanations. But most psychology takes place elsewhere: in the flux of our interactions with others, in the aspirations, progressions and reverses that occur as we make our way through the world. A core of our being does persist through the welter of events: it would be hard to recognise ourselves or our friends if we changed too much. But the soul, or as we might now say the psyche, is not immutable. We are affected by experience.

Brown and Harris's (1978) study was critical to psychiatry because it was the first substantial epidemiological study of depression. Epidemiology is fundamental in medicine because it shows the circumstances under which people become ill. Thus it was important in discovering that germs cause infectious diseases, that smoking increases the risk of several kinds of cancer, and that human immunodeficiency virus (HIV) is spread by sexual contact and hypodermic needles. Brown and Harris's epidemiology was important for the recognition that life events and difficulties provoke onsets of depression.

For psychologists, the effect goes deeper. Brown and Harris's work was a model of the investigation of interactions of mind and world. It was base on a new methodology: a semi-structured interview that would yield brief pieces of biography each of which described a particular event or a particular difficulty, that could be read to a research team, who then made independent judgments of its severity. Other kinds of event-related research now take place in the study of emotions (see Oatley *et al*., 2006) where, for instance, participants can be asked to keep diaries of emotions and relate them to events that elicited them. In emotions and emotional attachments, and in the more menacing relatives of such states, emotional disorders such as depression, we see responsiveness to experience. Mind and brain are influenced by the world, especially by the social world. Studies of such influences are taking their place in psychology alongside studies of persisting abilities and dispositions.

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