Causation and

…a decade ago we used to have to go round talking about punitive toilet training... Now we truly understand that OCD [obsessive compulsive disorder] is a brain disorder. (Susan Swedo quoted in Brown, 1997, p.31)

JANE HERLIHY *and* **JOHN GANDY**

argue that neurological explanations do not make psychology redundant.

HOW do we feel as psychologists as we read a quote like this? It seems that we can study and explain mental phenomena for a long time, but when a neurobiological explanation is offered the work is suddenly proclaimed complete. A researcher who has spotted the brain activity, or the gene that accompanies a behaviour, is lauded as having found the cause – the 'real' explanation. So are psychologists redundant? Shall we move on to the next disorder, in the hope that the neurologists and geneticists won't catch up too quickly?

Surely, we feel, this cannot be right. It can't be that simple. And yet many psychologists subscribe to theories of psychiatric disorders that show that, since drug X acts on neurotransmitter Y and the symptoms reduce, thus neurotransmitter Y is the cause of the disorder. Maybe the clinical psychologist's role is to stick to trying to alleviate any secondary, psychological consequences of the 'illness'.

The issue here is 'neurological reductionism'. We define this as the tendency to think that neurological theories show cause, while others show mere correlation, or to assume that this level of explanation is inherently superior to the others. Most psychologists will, whenever possible, publicly disavow this kind of thinking. Nevertheless it remains a tangible trend in a lot of what is said in the discipline. Perhaps more importantly, the general public is inclined to think that neurological explanations are the psychologist's ultimate goal. Even if we reject it, it is not healthy that we should be so distant from what the public thinks we think.

In this article we offer a critique of how we think in psychology, which might offer a way out of the confusion. Not only will we question the assumed superiority of neurological explanations, but by examining the way in which causality is misunderstood in our explanations of disorder, we hope to show that such comparisons between different levels of explanation are simply spurious.

All in the mind?

Neurological reductionism is a widespread and enduring phenomenon. Its prevalence has a number of causes. Ussher (1991) argues that by adopting the positivistic principles of science, medicine and psychiatry came to command the respect and high esteem that it still holds today – to the envy of many psychologists. Unfortunately those same principles encourage a rather immature form of materialism – one that tacitly assumes that to rely on anything but the tangible, like brain matter, somehow implies unworldly and mystical thinking.

The very existence of scientific psychology should have freed us from this. But neurological reductionism leaves us with a persistent mind–body duality. Clinical psychologists struggle to convince patients that problems that are 'all in the mind' are just as valid as diseases with physiological aetiology. The debate is particularly important in the approach taken to conditions such as chronic fatigue syndrome (see e.g. Spencer, 1998). There seems to be genuine confusion among clinicians about how to choose between the poles: collude in the accepted superiority of the medical model, or educate sufferers in the psychological explanations of such conditions.

Neurological reductionism may have become so ingrained in popular consciousness because it offers comfort to those suffering from a disorder. Receiving a diagnosis of a specific physical illness after perhaps months of unexplained symptoms comes as a relief for many (Chadwick, 1995). Similarly, Swedo's work on the neurology of OCD has been enthusiastically embraced by at least one support group in America (Brown, 1997). It seems that no one wants to have a 'mental illness'; a brain disorder or physical disease seems so much more worthy of sympathy. This dualism allows us to be responsible for our minds but the victims of our bodies.

Health psychology: The end of mind–body dualism?

The development of health psychology as a discipline claims to transcend this mind–body dualism. Health psychology, it is true, has introduced a psychological level of explanation to 'medical conditions' such as diabetes or heart disease. However, the divergence of health and clinical psychology as separate disciplines gives the lie to the very aims of this new area. Now there is a new dualism. 'Health psychology' style interventions targeting health behaviour change are generally based on social cognition models of intentions and actions. 'Clinical psychology' style interventions targeting mental health problems are based on diagnostic categories, such as the model of panic disorder, the model of social phobia, and so on. But if we are really to understand the relationship between the mental and the physical, then adopting different models is not the way: we need to examine the basis of our explanations – is everything to be reduced to material causes? Are we just waiting for magnetic resonance imaging and genetic research to provide the truth?

We contend that reductionism in all its forms is a symptom of a philosophical deficit, a result of our failure to resolve the problem of causation. So what does psychology have to offer?

Levels of explanation

The field of visual perception provides us with a good and well-known example of

explanation

neurological reductionism and its antithesis. Consider this quotation from Barlow (1972, p.380): 'A description of that activity of a single nerve cell which is transmitted to and influences other nerve cells and of a nerve cell's response to such influences from other cells, is a complete enough description for functional understanding of the nervous system. There is nothing else "looking at" or controlling this activity, which must therefore provide a basis for understanding how *the brain controls behaviour* [italics added].'

Barlow's argument is misleading with regard to causation. In her public lectures on the development of the brain, Susan Greenfield stressed how behaviour and perception are the interaction of the body with its environment. The feeding of qualitatively varied information from this interaction into the brain determines what the brain does (Greenfield, 2000). So it is equally true to say that behaviour controls the brain as it is to say that the brain controls behaviour. Hence it makes more sense to say neither, but to understand that behavioural and neurological descriptions are two ways of describing the same phenomenon.

Nonetheless, this quotation from Barlow exemplifies the persistent belief that all phenomena can and will eventually be reduced to neurological explanation. Unfortunately reductionism of this type always seems to lead to a final caveat, in which there is an extra ingredient – a ghost in the machine; free will; 'qualia' which have to be programmed in, *post hoc*, to the computational model.

It was this kind of thinking that prompted Marr (1982) to explain the importance of different levels of description. Marr distinguished between three perspectives:

- that of the 'plain man', or the experiential;
- that of brain scientists, physiologists and anatomists, or the physical embodiment; and
- that of experimental psychologists, or the information-processing.

Marr did not pursue a general philosophical point. He was simply introducing his preferred method for his substantive work on vision. In this respect Marr distinguished between three levels of theory. Firstly, there is the 'computational', which concerns goal, strategy and constraints – what the system does with what its got. This is equivalent to the intentions, motivations and behaviour of the 'plain man'. Secondly, there is the description of the 'hardware', how the process is physically realised. And thirdly, there is the 'representation and algorithm' level, which concerns the form of input and output and the rules of processing. This is the 'information-processing' level. To achieve a comprehensive analysis of vision it is absolutely necessary to address all three levels.

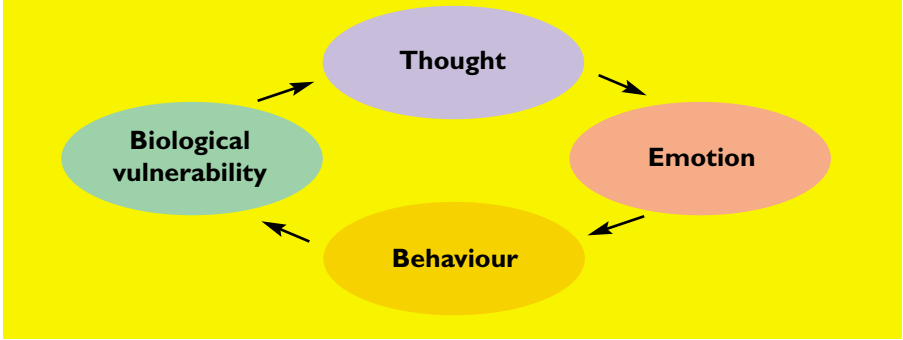
This has understandably become a commonly used convention in cognitive psychology. But we are not absolutely sure how these levels relate causally. Marr says: 'Each of the three levels of description will have its place in the eventual understanding of perceptual information processing, and of course they are logically and causally related' (1982, p.25). But how? Marr continues: 'The choice of an algorithm is influenced for example, by what it has to do and by the hardware in which it must run'. But the nature of this influence cannot be causal. If the hardware 'caused' the algorithm then we would be right back to Barlow and the idea that the 'brain controls behaviour'.

Steven Rose (1991) has the beginnings of an answer: There is no causal relationship between levels of explanation and it makes no sense to speak of one. We can take one single phenomenon and describe it at different levels – the biologist might specify the movement of hormones about the body, the English psychologist the feeling of love and the French psychologist *l'amour*. According to Rose, each explanation is a translation of the others. Thus the problem, for Rose, is not explaining the flow of causality between these explanations, but a mapping exercise between levels. 'All behaviours of the individual organism must in principle translate into biochemistry (though not all translations will be useful). But not all biochemistry translates into behaviour' (p.101). Thus the task becomes isolating the useful translations and looking at the mapping relationship between the levels of explanation – what are all of the components of the event?

Now we have an answer to the article quoted at the beginning. To recap, it has been found that a particular pattern of brain activity is concurrent with obsessive-compulsive behaviours and cognitions. Should we be surprised by this? Of course not. We can safely predict that particular patterns of brain activity will accompany every particular pattern of behaviour. There simply couldn't be any behaviour or cognition that didn't have corresponding brain activity. In Rose's language – we have made a translation from 'psychologese' into 'neurologese'.

We are not suggesting that the neurological level is the wrong mode of description for OCD. Such a description is vital for a comprehensive understanding of the condition and is necessary for the development of drug treatments. Our

FIGURE 1 Cognitive behavioural model



objection is to the prior assumption that neurology has the only 'key'.

The mistake arises when we start dealing with causation. The neurological processes did not *cause* the behaviours and cognitions, and it is even misleading to speak of correlation. The biology and psychology are only separable in the abstract, as it is possible to separate light from its brightness. They are two pieces of information about the same event. So where can we look for causes?

Causation

For any particular event to occur exactly as it did (concretely not abstractly) the whole universe needed to be arranged precisely as it was immediately before the event. We could say, therefore, that the event has only one cause. On the other hand we can divide the universe up in a (potentially infinite) number of different ways, by describing it on different levels, so we could say that the event had a (potentially infinite) number of causes.

So we must be clear. Whenever we identify a cause or causes for an event we are choosing from a number of possibilities. Our actual choice depends on salience – the cause which we identify depends on which question we are asking.

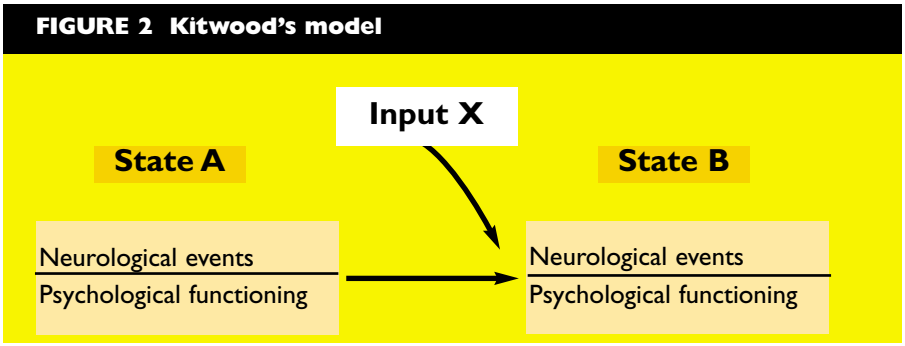
The cognitive-behavioural model

Many clinical psychologists make use of the cognitive-behavioural model (see Figure 1). This cycle is used to describe the processes associated with emotional disorders such as anxiety.

The cycle can be broken by therapy at any point, and different kinds of therapy are designed to tackle different parts of the process. Drugs deal with the biological event, behavioural therapy with the behaviour, and cognitive therapy with the thoughts and emotions. Therapy is effective if it blocks the flow and thereby halts the process. And yet it has been found that therapy is more effective when the cognitive is combined with the behavioural. It is even more effective, at least in the short term, when it is combined with pharmacotherapy. Why should this be – shouldn't breaking the flow be enough? It is possible to argue that intervening at different points is weakening those links and this is why combination therapies should work.

However, if we remove any sense of causal flow from the model we can make better sense of the interactions of these therapies. Consider instead three aspects (or translations) of a single event – thought, emotion, and biological vulnerability. The components are modes of description, and it does not make sense to speak of causation within the triangle.

FIGURE 2 Kitwood's model



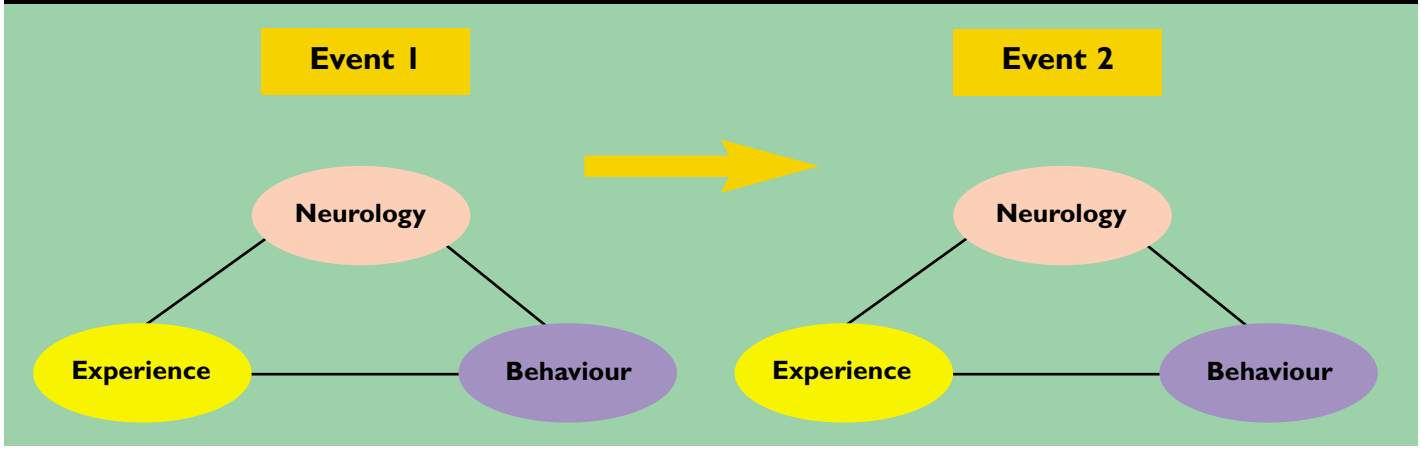
An alternative model Another theorist who has used this way of thinking is Tom Kitwood, in his description of the development of dementia. He uses the model in Figure 2 to explain how dementia might develop in an individual. In this model a person moves from one state to the next, with each state involving a psychological event, and a corresponding biological event. He clearly describes the relationship between the two: 'It is not that the psychological experience is causing the brain activity or vice versa; it is simply that some aspect of the true reality is being described in two different ways' (Kitwood, 1997, p.17).

The next thing to occur to the individual in State A might be a deterioration in certain neural pathways, or it might be the loss of a loved partner. It is the combination, the dialectical process that takes place when State A is combined with input X, that leads to State B.

The model we are proposing (in Figure 3) allows us to distinguish more effectively between symptom and cause as a guide to therapy. To explain, if patterns of brain activation are matched with a particular disorder this would not show that those brain activations are causal. If brain activation is interfered with resulting in successful treatment of the disorder, this too would not show that those brain activations were causal. Another causal event must lie outside the event triangle.

Looking at the model above we can see how, in theory, therapy that targets any, some or all of the three aspects can be effective. Changing any one will change the preceding event and therefore what follows it, or, its effect. That is why, in

FIGURE 3 Causation



theory at least, cognitive therapy might, for example, be effective against disorders with biochemical causes. Similarly, psychoactive drugs might be effective against disorders that had cognitive causes.

The perils of misunderstanding causality From the point of view of a patient or a therapist we might suppose that it doesn't strictly matter what caused a disorder providing the therapy works. However, theory is important for more than academic reasons; it has practical consequences. A large study conducted in London and Toronto provided an excellent demonstration of the perils of the continued popular misunderstanding of causality in mental illness (Basoglu *et al*., 1994).

The primary aim of the study was to measure the effectiveness of cognitive-behaviour therapy (CBT) alone and in combination with drug therapy (alprazolam) in the treatment of panic disorder. CBT was indeed found to be helpful as a treatment, and even more so in combination with alprazolam. Control groups receiving no psychological treatment and placebo drugs attested to the efficacy of the two active treatments. However, the researchers continued to take measurements as the participants were taken off the drug and placebo treatments. At this point there was a considerable amount of relapse and rebound anxiety (higher levels of anxiety than prior to the intervention). This was not only in the drug therapy group, as might be explained by biological processes involved in withdrawal, but also in the placebo group. This finding was explored in an associated investigation which found that those individuals who attributed their improvement to drugs had a far poorer outcome once the drugs were withdrawn, than those who, although taking the drugs, attributed their improvement to their own efforts. The authors of this study concluded that 'overemphasis on the value of medication in an effort to enhance compliance may be less than helpful' (p.658). We are back to the chronic fatigue syndrome debate – do we tell patients it's physical or psychological?

The flow of causation

Our conclusion is that we, psychologists, do not need to make this choice. We should not elevate the neurological over the behavioural or cognitive, or foster this idea among the general public. We should strive to develop and understand a new model in which a human being moves from one psychophysical state to another, describable on different levels by different specialists. Causation flows between these states and not between the levels of description.

■ *Dr Jane Herlihy is a clinical psychologist at Barnet, Enfield and Haringey Mental Health NHS Trust. E-mail: jane@herlihyi.freeserve.co.uk.*

■ *John Gandy is a freelance writer and works at Hill House School for autistic spectrum disorder in Lymington. E-mail: gandy_j@yahoo.co.uk.*

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