What is interesting isn’t always useful

WHAT is clinical psychology research? Did you do a practical class in it during your undergraduate degree? The chances are that you didn’t. Is it a core content area of the curriculum? Usually not. The scientist-practitioner model of clinical psychology research is rarely encountered at undergraduate level, yet applied psychology generally and clinical psychology specifically represent some of the most common reasons that applicants give for wanting to take a psychology degree. The intense competition for postgraduate clinical training places in many countries, including the UK and the US, attests to the continued enthusiasm for this branch of psychology after graduation.

Arguably, clinical psychology has a central importance as both a profession and a discipline. In many countries it is a valued profession with a high profile, and in any discipline understanding how processes that contribute to normal functioning can also lead to dysfunction represents an important and necessary part of the body of knowledge.

In the June 2002 issue of The Psychologist, Thomas et al. wrote an article entitled ‘Clinical psychology under threat’. It outlined the difficulties of conducting research in clinical psychology in the UK, and asked why training clinical psychologists to doctoral standards had not prevented a decline in clinical research. There are some obvious reasons for this:

- Clinical psychology research requires a wide range of research skills (many of which are not taught in a relevant context at undergraduate level) and a clear understanding of theory and explanation.
- Clinical populations represent only a small proportion of the population, and access to such populations is often difficult to obtain.
- Many forms of research are either difficult to conduct with clinical populations (because of the severity of the symptoms observed in psychopathology) or raise important ethical issues (e.g. conducting experimental manipulations that might precipitate clinical symptoms or interfere with ongoing treatment).
- Having to split their time between the provision of clinical services and research means that clinical psychologists often do research as an afterthought, without the necessary skills development required to do it properly.

It is this last issue that I would like to spend the rest of this article pursuing. I am not clinically trained, but came to psychopathology research as an experimental psychologist with an interest in understanding how cognitive and psychological processes contribute to the development and maintenance of anxiety-based disorders. To this extent, I may well be seen by clinicians as an outsider preaching heathen philosophy. But from the standpoint that I have come from, clinical psychology research in the last 10 years or so has developed some sloppy methodological habits, is often unclear about what constitutes an explanation of a clinical phenomenon, and, in some areas, has become obsessed with ‘clinical
experience’ and diagnostic categories to the detriment of understanding the psychological processes involved in psychopathology. The following sections describe some of these issues, and are illustrated in the context of some of the psychopathology research carried out by the Clinical Experimental Psychopathology Research Group at the University of Sussex over the past 10 years.

‘Processes’ not categories A significant majority of clinical psychology research (and psychiatric research) is carried out around diagnostic categories and on populations with specific diagnostically defined symptoms. In 2002 both the British Journal of Clinical Psychology and, arguably the most evidence-based and experimentally oriented of psychopathology journals, Behaviour Research and Therapy, published around 60 per cent of their articles on research dedicated to DSM diagnostic categories or their direct analogues. Through their training, the clinical psychologist’s understanding of psychopathology is based on the diagnosis and categorisation of disorders as described in the most recent version of the DSM. But this may not be a good starting point for undertaking research that is attempting to understand aetiology and its associated psychological mechanisms. In a recent article Krueger and Piatecki (2002) note:

The typical approach to research on psychopathology involves defining ‘pure groups’ of individuals (persons who meet criteria for a specific DSM disorder, but not for other disorders) or ‘impure groups’ of individuals (persons who meet criteria for a specific DSM disorder, regardless of the other disorders for which they might meet criteria) and contrasting them with ‘comparison groups’ (persons who do not meet criteria for psychopathology, or who meet criteria for a putatively distinct form of psychopathology). (p.489)

There are at least two immediate difficulties with this typical approach. First, comorbidity among DSM-defined psychopathology is the rule and not the exception (Kessler et al., 1994). This not only challenges DSM’s categorical conception of psychopathology, but suggests that a full understanding of psychopathology will be dependent on describing underlying aetiological processes that either give rise to cross-categorical symptoms, or may give rise to quite different symptoms under different circumstances. For example, research that we have recently conducted suggests that in some of those disorders characterised by perseverative activities (e.g. pathological worrying, compulsive checking, perseverative ruminations), perseveration can at least in part be explained across all these symptom types by the contribution of a single psychological mechanism (Davey et al., in press; Startup & Davey, 2001). Understanding these types of perseverative disorders will probably require intensive study of this common mechanism rather than a blinkered focus on the individual diagnostic categories. This will at least help us to understand, rather than ignore, the importance of comorbidity, and to develop treatments that are soundly based on the cognitive and psychological mechanisms that develop and maintain the critical features of psychopathology.

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Another consequence of researching by diagnostic category is that there is often an implicit tendency to view a diagnostic category as representing a ‘disorder’ of a normal psychological process. Generalised anxiety disorder (GAD), for example, is often referred to as a ‘disorder of worrying’ – as though there is some normal, functional worry mechanism that has broken down and needs mending. Not only does this have the effect of stigmatising clinical patients as somehow qualitatively ‘different’ to everyone else (e.g. Hayes & Follette, 1992), it also seems to imply that psychopathologies exist in discrete categories that have quite different dynamics to any ‘normal’ processes that they might resemble. Recent research using psychometric technologies such as item response modelling is beginning to indicate that psychopathology can be conceptualised as continuous and dimensional, ranging from relatively ‘normal’ functional characteristics at one end to the more severe pathological symptoms at the other (e.g. Cooke & Michie, 1997; Santor & Coyne, 2001). These findings are perhaps not so surprising to those of us who have been doing analogue research on psychopathology for many years now, and who can quite easily see that those cognitive processes that underlie ‘normal’ functioning activities (subclinical worrying and checking) may subsequently come to maintain extreme forms of these activities (e.g. pathological worrying in GAD and compulsive checking in obsessive compulsive disorder).

Beware constructs derived from ‘clinical experience’ Clinical researchers are very attached to their ‘clinical experiences’ and often attempt to bring these experiences into clinical research by creating constructs from them. Such constructs are not directly underpinned by observable actions, behaviours or symptoms, but have a validity through the fact that the clinician commonly experiences a set of characteristics within a particular clinical population. The usual practice is then to develop a tool or instrument to measure the construct, and subsequently to identify how the construct relates to clinical symptoms – all in the belief that this will enable an understanding of the processes that develop and maintain those symptoms.

Unfortunately, more often than not, these constructs can detract from attempting to elucidate the cognitive and psychological mechanisms that underlie the disorder, confuse descriptions of symptoms with causes and consequences of the symptoms, and generally set off unproductive chains of research.

Even in issues of Behaviour Research and Therapy since January 2002 the reader will find articles espousing the virtues of the following constructs: ‘not just right experiences’ (Coles et al., 2003), over-valued ideas (Veale, 2002), intolerance of uncertainty (Buhr & Dugas, 2002), interpersonal sensitivity (Harb et al., 2002), thought–action fusion (Zucker et al., 2002), and inflated responsibility (Barrett & Healy, 2002). All are used in contexts where the construct is assumed to give a better insight into the psychological characteristics experienced in certain disorders (e.g. not just right experiences) or have some explanatory power (e.g. thought–action fusion), or both (e.g. intolerance of uncertainty).

It is tempting to say that clinical psychology researchers avidly develop such constructs because they are consumed by their clinical experiences and have little time (or training) for maintaining a knowledge and understanding of how other relevant areas of pure psychological
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Mechanisms underlying the psychopathology of the symptoms or understanding of the dispositional features of its sufferers, when also run the risk of becoming reified into DSM-diagnosed disorder, such constructs contribute to the psychopathology? Psychological processes and mechanisms hinder us in understanding how established psychopathology such constructs reveal that NJREs are correlated with OCD features but not with features of other domains of psychopathology, that NJREs may represent a specific form of perfectionism, and that compulsions may represent attempts to relieve the anxiety caused by NJREs. Quite possibly. But in creating constructs such as this, researchers need to ask a series of very important questions. Does the construct merely redescribe the symptomatology of OCD? Is the construct (and its experience by the client) simply a trivial, epiphenomenal consequence of the mechanisms underlying OCD? And does the construct help or hinder us in understanding how established psychological processes and mechanisms contribute to the psychopathology?

Because they are derived mainly from clinical observation of patients with a DSM-diagnosed disorder, such constructs also run the risk of becoming reified into characteristics of the DSM category and dispositional features of its sufferers, when such constructs provide little explanation of the symptoms or understanding of the mechanisms underlying the psychopathology. More on this next.

**Explain how the arrow works**

Deriving constructs from clinical experience and then attempting to understand how these constructs relate to symptoms can have implications for how the psychopathology is understood and explained. For example, box and arrow schematic models are a popular feature of psychological explanation generally, and clinical psychology research is no exception. But we have to remember that in very many cases these models do not represent the mechanisms underlying psychopathology. They represent a convenient way of describing the relationships between the variables that have been identified, or, quite regularly, created from clinical experience. But often such models leave the really interesting and informative features of the psychopathology unexplained.

Figure 1 represents an illustrative schematic model of how some of the variables thought to be importantly involved in OCD interact to cause symptoms (there are several similar models in the literature – e.g. Rachman, 2002; Salkovskis, 1999 – this example draws some common features from these separate models). On the face of it, such models look compelling and instructive, and there is no lack of evidence suggesting that the arrowed links between boxes exist in some form or other. For example, there is evidence from both survey studies (e.g. Foa et al., 2002) and studies that have involved experimental manipulation (Ladouceur et al., 1995; Lopatka & Rachman, 1995) that there is a link between inflated responsibility and compulsive checking. However, such models tell us little, if anything, about the psychological or cognitive mechanisms involved in OCD. In particular, how do the arrows in Figure 1 do what they do? How do they respectively convert negative mood into inflated responsibility, and cause inflated responsibility and negative mood to trigger compulsive actions and rituals? Arguably, these are the most interesting aspects of the psychopathology! The arrows represent the mechanisms by which variables interact, and a case can be made that clinical psychology researchers have often neglected to address how these mechanisms work, and have neglected to look elsewhere in the psychology literature for what might be suitable mechanisms to explain how their variables interact (see Startup & Davey, 2001).

Furthermore, when arrows are included in schematic models they are often seductive enough to convince that they are unidirectional processes with linear effects (rather than representative of what might be quite complex mechanisms); this can lead to other assumptions about the model that may not be true. For example, one of the challenges for psychopathology research is to explain the severity of the symptoms that the sufferer experiences, and clinical psychology researchers have a tendency to do this by inventing ‘vicious cycles’ within their models.

An early example of this is David Clark’s (1986) famous catastrophic misinterpretation model of panic disorder (see Figure 2), which postulates that a triggering stimulus gives rise to anxious apprehension, which in turn causes ambiguous bodily sensations, which are interpreted catastrophically and leads to further anxious apprehension, and so on. This sequence is repeated until a panic attack occurs. (But note that the outcome, a ‘panic attack’, doesn’t constitute part of the model!) Arrows that viciously recycle can be found in a substantial number of schematic psychopathology models, and often attempt to explain symptom severity (examples include OCD, social phobia and depression, to mention just a few). However, such accounts assume that severity can only be achieved by repeatedly recycling the same arrows around a system that builds severity in a simple cumulative way.

**Let’s do some correlations**

Because of its very nature, clinical psychology lends itself to research that emphasises the relationships between symptoms, psychopathology constructs, and psychological processes. Correlational studies are a means of getting a handle on whether predicted relationships between psychopathology measures and psychological processes exist. They are
almost a methodology of choice for many clinical researchers for whom experimental methodologies are too invasive or raise complicated ethical issues when studying a clinical population. This may be very well if correlational studies are planned and executed with clear forethought about how the results can be interpreted – but very often they are not.

A good example of how poor planning can cloud understanding rather than enhance it comes from the study of the role of the disgust emotion in anxious psychopathology. For the past 10 years, research on the role of disgust has burgeoned to the point where in 2002 there were 28 papers published in clinically related research journals with ‘disgust’ in the title – only six of these involved an experimental manipulation. Recent review articles have proclaimed disgust as the ‘forgotten emotion of psychiatry’ (Phillips et al., 1998), and studies have linked the disgust emotion to animal phobias, OCD, blood-inoculation-injury phobia, and eating disorders. To be sure, disgust is certainly experienced in these disorders, but 10 years of research has so far failed to provide real convincing evidence that disgust has any precipitating or causal function.

We all know that correlation does not imply causation, but the issue here is that the design of many disgust correlational studies blurs rather than sharpens our understanding of the disgust–psychopathology relationship. First, many studies have failed to use properly balanced designs in which predicted disgust-relevant psychopathologies are compared with suspected disgust-irrelevant psychopathologies (e.g. de Jong et al., 1997; Sawchuk et al., 2000). Because anxiety and disgust are closely related emotions, studies that do not include disgust-irrelevant control comparisons do not provide differential predictions that would rule out the implication of anxiety as a mediating variable – thus rendering the findings relatively trivial. When studies have included disgust-irrelevant psychopathologies, some have begun to reveal significant relationships between disgust and those psychopathologies that would not have been predicted at the outset (e.g. between disgust and situational phobias; Muris et al., 1999). Furthermore, many studies have not taken any measure of trait or state anxiety. Thus they will inevitably be unable to determine whether any relationship between disgust and psychopathology measures is independent of levels of anxiety (e.g. Arrindell et al., 1999). It is quite clearly possible that elevated disgust levels are a consequence of the effects of anxiety raising levels of all negative emotions.

This lack of foresight in planning such studies – but with a willingness to publish them anyway – has created a literature that is difficult to unravel and that encourages the belief that disgust is involved in the aetiology of a range of anxious psychopathologies, when there is insufficient evidence to come to this conclusion. It is also indicative that clinical psychology research has spent over 10 years nurturing this literature without generating a single explanatory model or describing a single mechanism that implicates disgust in the aetiology of anxious psychopathology.

**Conclusions**

I suspect many readers think I may have been too harsh – and, OK, relatively selective in the examples and areas of
psychopathology that I have dipped into. However, there are many other examples of
naive and ill-thought-through contributions to research in clinical psychology and
psychopathology that space does not permit me to mention. But understanding
psychopathology is an honourable goal that will contribute significantly to alleviating
suffering in the world. As such, it deserves to be done properly, with clinicians at all
levels making informed and valuable contributions. In the UK at least, this is
not yet as it should be (see Thomas et al., 2002). In particular, clinical psychologists
not only need to be involved more regularly in research, they need to
● have research skills as a significant component of their continuing
professional development;
● have had the opportunity to learn directly about clinical psychology
research methods in their undergraduate careers; and
● have a good knowledge of ‘pure’ psychology research that is relevant to
their clinical research specialities.

If psychologists do not regularly, efficiently and effectively become involved in
psychopathology research, then other disciplines – notably psychiatry – are ready
and waiting to hijack this subject matter.

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