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A Research Pathway for Experimental Psychopathology: The Role of External Validity Criteria

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Abstract

This paper outlines a putative pathway for experimental psychopathology research developing psychological models of clinical disorders. The pathway uses established external validity criteria to define the pathway and clarifies the important role that research conducted on healthy participants can play in our understanding of clinical disorders. Defining a research pathway for experimental psychopathology in this way has a number of benefits It would (1) make explicit the need to address the external validity of developed models, (2) provide a clear set of criteria that would be required to extend research on healthy individuals to diagnostic populations, and (3) recommend using general psychological knowledge when developing models of psychopathology.

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Keywords: Experimental psychopathology; external validity; psychological models; healthy participants.

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Introduction

In its broadest sense experimental psychopathology is the study of psychopathology processes under highly controlled conditions for the purpose of developing detailed models of how psychopathology is acquired and can be treated. This research is often carried out on nonhuman animals or on healthy human participants in order to create models of psychopathology that mimic or predict the processes found in individuals diagnosed with mental health

problems (e.g. Abramson & Seligman, 1977; Vredenburg, Flett, & Krames, 1993). There is no doubt that experimental psychopathology as a research approach has made significant contributions to understanding psychopathology across many domains, including depression and anxiety (Steimer, 2011; Vervliet & Raes, 2013), schizophrenia (Jones, Watson, & Fone, 2011), neuropsychiatric disorders (Nestler & Hyman, 2010), substance abuse and addiction (Lynch, Nicholson, Dance, Morgan, & Foley, 2010), and pharmacological treatments of psychological disorders (van Gestel, Kostrzewa, Adan, & Janhunen, 2014) to name just a few.

This article will focus on some specific, and perhaps neglected issues in experimental psychopathology that relate most significantly to the development of *psychological models* of psychopathology. A full understanding of the processes that contribute to mental health problems will require the development of both biological and psychological models of a disorder, and these different explanatory paradigms will complement each other in many ways. For example, both paradigms will reveal details of processes that will need to be accounted for in the models developed in the other paradigm. In addition, the importance of psychological models should not be underestimated, given that such approaches offer a broader perspective than biological models by providing a means of integrating cognitive, behavioural and social factors into unified models of aetiology and intervention (Davey, 2015).

However, the elaboration of psychological models of psychopathology has often developed chaotically rather than via an accepted and validated research pathway (Davey, 2003). First, clinical psychology research is frequently driven by clinical experience and the practitioner's need to develop more effective and efficient interventions (Dawes, 1994). As a result many clinical psychology researchers develop models of disorders that are specific to their own clinical experience, researched in ways that often lack required levels of empirical rigour, and are built around esoteric constructs that do not match simply onto accepted and basic psychological operations related to perception, cognition and action, and in many cases may simply be redescriptions of the symptoms that it is often claimed they explain (Davey, 2003, 2013a).

Second, it is often difficult for experimental psychopathologists developing psychological models to place their research in established clinical psychology and psychiatry journals – especially if their models have been developed on healthy individuals. Given that many relevant journals have a publication requirement that research must be carried out on clinical populations with a diagnostic label, this has not only made it more difficult for experimental psychopathology researchers to find suitable outlets for their research, it also means that such research may become scattered across a wider range of either secondary journals or mainstream psychology journals rather than being published in frontline clinical psychology or psychiatry journals. This is likely to give the erroneous impression that such research does not belong to a body of research that represents a significant and alternative unified approach to understanding psychopathology[1].

Thirdly, those using experimental psychopathology methods to develop psychological models of psychiatric disorders have often been negligent in ensuring the validity of their research to clinical populations – especially if it is psychopathology-relevant research conducted on healthy individuals. Whilst external validity has been an important issue in other forms of experimental psychopathology (especially using animal models) (e.g. Belzung & Lemoine, 2011; Stewart & Kalueff, 2015), it has only recently been discussed as a significant issue in the development of psychological models using healthy human participants (Vervliet & Raes, 2013). In the case of the experimental study of psychological models conducted on healthy individuals, lack of attention to external validity can seriously undermine the value of a piece of experimental psychopathology research. Researchers may need to indicate more clearly where the research fits into a putative research pathway from basic models developed on healthy individuals to researching processes that are proven to be unique to the relevant clinical population –a validation process that has been much more transparent and fully argued in the case of nonhuman animal models of psychopathology (cf. Vervliet & Raes, 2013)

Given these background issues in the use of experimental psychopathology to develop psychological models of mental health problems, the main purpose of this article is to suggest a possible research pathway along which the development and application of such models can progress. This would have a number of benefits: it would (1) make

1 Recent changes in scope to Behaviour Research & Therapy and the launch of Journal of Experimental Psychopathology will improve the available outlets for experimental psychopathology research conducted on healthy individuals.

explicit the need to address the external validity of developed models, i.e. to provide a fuller set of arguments as to why experimental psychopathology studies can be generalized to clinical populations and diagnostic categories, (2) provide a clear program of research that would be recommended when extending research on healthy individuals to diagnostic populations, and (3) recommend using general psychological knowledge when developing models of psychopathology.

The External Validity of Psychological Models of Human Psychopathology

When experimental psychopathologists have received a full and proper training in scientific and experimental methods there should be no need to question their empirical skills. What is at issue is the need to apply principles of external validity to that research to justify the relevance of the laboratory studies to the clinical populations to which those developed models are applied. This is not an issue that is specific to experimental psychopathologists developing psychological models of disorders, it is an issue that is highly relevant to all translational research. Understanding the different forms and types of external validity is a first step to developing a research pathway that will bridge the translational gap, so it is worth looking at some of the forms of external validity that have been postulated as relevant to translational research in general.

External validity criteria vary considerably in their strength and relevance. Table 1 provides a list of validity criteria that can be found in the broader experimental psychopathology literature (e.g. animal models of psychiatric disorders) and whose definitions are adapted to be relevant to the development of psychological models of psychopathology (adapted from Belzung & Lemoine, 2011; Geyer & Markou, 1995; Stewart & Kalueff, 2015; Vervliet & Raes, 2013).

Table 1: A list of validity criteria taken from the broader experimental psychopathology literature and adapted to the development of psychological models of psychopathology.

Validity Criterion	Definition
Face Validity	The phenomenological similarity between the behaviour in the laboratory model and the symptoms of the disorder.
Predictive Validity	Performance in the laboratory model predicts performance in the disorder.
Construct Validity	The model developed in the laboratory can be compared favourably with existing clinical models of the disorder. The processes described in the laboratory model parallel the clinical processes of interest.
Aetiological Validity	It can be shown that the aetiologies in the laboratory model and the disorder are identical.
Convergent Validity	The degree to which outcomes from the laboratory model correlate with measures/outcomes from other models of the same disorder/construct.
Discriminant Validity	A model differs from other models of the same disorder to the extent that it's outcomes are different to those predicted by other models.
Diagnostic Validity	Demonstrating that the laboratory model taps into processes that are unique to the clinical population exhibiting the disorder.

Definitions adapted from Belzung & Lemoine (2011), Geyer & Markou (1995), Stewart & Kalueff (2015), and Vervliet & Raes (2013).

Face validity is the weakest of these criteria, but is usually the starting point for many researchers creating laboratory models of psychopathology. This criterion stresses there should be a formalistic or phenomenological similarity between the behaviour in the laboratory model and the symptoms in the disorder. Thus, a fear conditioning procedure conducted in the laboratory might have face validity as a model of specific phobias because the physiological measures of anxiety conditioned to the CS+ formalistically approximate to the fear elicited by phobic stimuli in phobic populations (e.g. Davey, 1992; Delgado, Olsson, & Phelps, 2006; Powers et al., 2005). Other examples might include the use of mood induction procedures to generate anxious or depressed moods in healthy participants to examine how these moods might affect performance on a selection of disorder-relevant tasks (e.g. Davey, Bickerstaffe, & MacDonald, 2006; Grant, Stewart, & Birch, 2007; Hepburn, Barnhofer, & Williams, 2006; Teasdale & Fogarty, 1979). Alternatively, some researchers have used healthy participants who score high on the relevant psychopathology (e.g. depression) but are at sub-clinical levels (e.g. Vredenburg et al., 1993).

Predictive validity is the first step to demonstrating that your model and the processes defined within it do have some relevance to the clinical disorder. The requirement here is that your laboratory model predicts behaviour in the disorder. For example, manipulating appraisals of 'inflated responsibility' (the belief that one has power to bring about or prevent crucial negative outcomes) in healthy individuals may be found to have a direct effect on perseverative activities such as compulsive checking (Bouchard, Rheame, & Ladouceur, 1999; Lopatka & Rachman, 1995), and so such a manipulation predicts behaviour found in certain forms of obsessive-compulsive disorder.

What you do not know from simply applying a predictive validity criterion is whether the behaviour in your model and the behaviour in the disorder are generated by the same underlying mechanisms or processes. When adopting predictive validity you know that the outcome of your model matches the outcomes expected in the disorder, but you do not have evidence of whether the outcomes in the laboratory model and the clinical case are achieved via the same mechanisms. To establish whether the mechanisms of the laboratory model match the mechanisms operating in the clinical case requires a more stringent criterion and can be accomplished by applying criteria provided either by construct validity or aetiological validity. *Construct validity* requires that the laboratory model compares favourably with a clinical model of the disorder (and that this favourable comparison can be established at the level of at least some of the mechanisms within the laboratory model and the clinical model). This can be achieved in a number of ways. First, it should be possible to demonstrate that the various causal processes in your model can also be identified when those processes are examined in the relevant clinical population. For example, while a model of perseverative depressive rumination can be developed in the laboratory in healthy individuals (Hawksley & Davey, 2010), experimental studies can then be undertaken to identify whether the variables important in the model are generating depressive rumination in a clinical population (Chan, Davey, & Brewin, 2013). Secondly, the processes relevant to the laboratory model can also be identified in clinical populations through the collection and examination of patient aetiological case histories. For example, a laboratory conditioning model may demonstrate that fear or anxiety elicited by a stimulus (CS) can be generated by a combination of sensory preconditioning (the association of two innocuous but contiguous stimuli – e.g. a geometric shape presented on a computer screen followed by a nonaversive 60dB 'beep') and a subsequent process which inflates the aversiveness of the second of the two contiguous stimuli (the nominal UCS – such as increasing the loudness of the 'beep' to an aversive 100dB). This UCS inflation then has the effect of generating fear to the first of the associated stimuli (White & Davey, 1989). This process gives the impression that the fear-eliciting stimulus has never actually been contiguously paired with an aversive UCS. However, a search of relevant case histories in patients with anxiety disorders does provide many examples of where this two-process model has been active in the aetiology of their anxiety problems (Davey, de Jong, & Tallis, 1993), providing some good evidence for the construct validity of the laboratory conditioning model. Thirdly, if the laboratory model has already been constructed from an established aetiological model of the clinical disorder, then construct validity is a given. However, there are further theoretical advantages to the use of the laboratory model to test the clinical model. For instance, if the clinical model is based on principles derived from a broader, well-established theoretical approach, then the laboratory model can be used to explore the further implications of that theoretical approach for the disorder. One example is the use of conditioning theory to help explain the aetiology of disorders such as specific phobias and panic disorder (Bouton, Mineka, & Barlow, 2001; Davey, 1997), PTSD (Keane, Zimering, & Caddell, 1985), and substance use disorders (O'Brien, Childress, Ehrman, & Robbins, 1998), and to understand therapeutic processes such as the roles of inhibition and extinction during exposure therapy (Craske, Liao, Brown, & Vervliet, 2012). The broad existing knowledge base around conditioning theory can then be used to test out new hypotheses about how associative or learning processes might be involved in the aetiology of the disorder (e.g. Davey, 1992), and thus enhance our understanding of the clinical model. *Aetiological validity* is a slightly stronger version of construct validity in that it specifies that evidence should show that the laboratory and clinical models are identical. By it's very definition, aetiological validity implies that the laboratory model is a faithful reconstruction of the clinical model, but this may be very difficult to achieve in practice, and perhaps can only be achieved in circumstances where the laboratory model has been built explicitly on knowledge of the clinical model. In contrast, construct validity merely implies that the laboratory model can be compared favourably with the clinical model (i.e. has at least some important similarities) and that the laboratory model may merely mimic the processes important in the clinical model. In many cases it may be sufficient to mimic the processes in the clinical model because of the difficulty in establishing exactly how causal processes work in the

clinical model, and this is true of network models of brain processes involved in psychopathology when the details of the actual brain processes involved in the psychopathology are only poorly understood (e.g. Menon, 2011).

Convergent and Discriminant validity are criteria by which a researcher would attempt to demonstrate the degree to which their laboratory model is either similar to or different from other models of the disorder. *Convergent validity* will be helpful to a researcher when they are attempting to develop a new laboratory model of a disorder and showing that it predicts similar outcomes to more established and proven models. *Discriminant validity* is applicable when the researcher is attempting to test between laboratory models to demonstrate that one model has more validity than another. Both types of validity are important in each of these distinct roles.

Finally, *diagnostic validity* is a criterion described by Vervliet & Raes (2013) as one of particular importance for experimental psychopathologists developing psychological models. Over and above showing that the laboratory model compares favourably with the detail of the mechanisms underlying the clinical model (construct validity), diagnostic validity requires in addition that the researcher should be able to demonstrate that the laboratory model taps into processes or characteristics that are unique to the clinical population exhibiting the disorder. For example, if a researcher has developed their laboratory model of a disorder on a group of healthy participants, why is it that only a relative minority of the population acquires the disorder? One implication of this question is that the clinical population may possess characteristics that make them highly vulnerable to the critical variables and causal processes in the laboratory model, and diagnostic validity will be achieved when it can be demonstrated that there are unique features of the clinical population that do make them vulnerable to these important processes within the model. For example, laboratory models of 'jumping to conclusions' (the process of making a decision about the meaning or importance of an event on the basis of insufficient evidence) have been central to recent cognitive models of paranoia in psychosis (Savulich, Shergill, & Yiend, 2012), but why doesn't everyone 'jump to conclusions' and so consequently develop paranoia? The answer may lie in the fact that jumping to conclusions is significantly related to deficits in cognitive functions such as working memory, verbal memory and cognitive processing speed (Ochoa et al., 2014), and these are deficits that are regularly found during, and even prior to, the onset of psychotic symptoms (Carrion et al., 2015), and so will make those individuals vulnerable to the development of paranoia through the model's important process of 'jumping to conclusions'. Another example of diagnostic validity is one from our own lab. We have developed a laboratory model of perseverative worrying (a cardinal diagnostic characteristic of Generalized Anxiety Disorder) based on the mood-as-input model (Davey, 2006; Meeten & Davey, 2011). One feature of this model is that worriers use their concurrent mood to evaluate their progress at the worry task, and if they are in a negative mood (which is common in pathological worriers), that negativity informs them that they haven't yet succeeded in dealing with the worry and so must continue worrying. Not everyone is a pathological worrier, so what is it about pathological worriers that makes them particularly vulnerable to the processes within this model of worry? The answer seems to be that they possess a number of characteristics that make them especially vulnerable to using their negative mood as information during a worry bout, and so are much more likely to perseverate when worrying in a negative mood (Meeten & Davey, 2011, pp1266-69). The fact that diagnostic validity makes the researcher think carefully about how their model relates to the unique characteristics of clinical populations adds another dimension to validating the model, and may even extend the model to providing psychological markers of vulnerability for psychopathology.

A Research Pathway for Experimental Psychopathology

It's quite easy to see that the external validity criteria listed in the previous section begin with rather weak formalistic criteria and develop onwards to significantly stronger criteria that attempt to equate the laboratory model with the clinical phenomenon on a range of different levels. The important questions these criteria ask are sequentially: (1) Do the outcomes of your laboratory model superficially look like the clinical phenomenon? (2) Does your model predict what happens in the clinical phenomenon? (3) Can you show that the processes in your model closely resemble the processes in the clinical phenomenon? And (4) does your model explain why the relevant clinical population is differentially vulnerable to the clinical disorder?

This sequence of criteria lends itself to the construction of a virtual research pathway for experimental psychopathology in which an initial basic laboratory model is developed in stages using a succession of empirical

methodologies to answer the questions posed by each set of more rigorous validity criteria. This research pathway is illustrated in Table 2.

Table 2: A putative three-stage research pathway for experimental psychopathology (see text for further elaboration).

	Stages/Validity Criteria		
	Stage 1	**Stage 2**	**Stage 3**
	Proof of Concept	**Clinical Validity**	**Diagnostic Validity**
Validity Questions	*"Does your model produce measurable behavioural/physiological/cognitive outcomes that resemble the clinical phenomenon?"*	*"Does your model predict what happens in the clinical phenomenon?"* *"Can you show that the processes in your model closely resemble the processes in the clinical phenomenon?"*	*Does your model explain why the relevant clinical population is differentially vulnerable to the clinical disorder?"*
Purpose	• To establish models of psychopathology with formalistic similarity to their corresponding disorder. • Proof of Concept Studies. • Developing laboratory models of psychopathology symptoms (e.g. for testing intervention efficacy). • Adaptations of core psychological knowledge to mimic psychopathology processes.	• To establish that your laboratory model predicts behaviour in the disorder. • To apply a well-developed pre-existing theoretical model to a disorder to expand an understanding of the disorder.	• To demonstrate that your laboratory model taps into processes that are unique to the relevant clinical population. • To establish why the relevant clinical population is vulnerable to the processes in your model (and which give rise to the disorder).
Methodology	• Experiments that provide evidence of causal relations between events that allow theory building. • Inferential experimental studies used to develop laboratory models.	• Experimental studies demonstrating that novel predictions from the model/theory apply to the disorder. • Case histories – demonstrating that processes specified in the model can be identified in the aetiology of clinical cases	• Literature & systematic reviews. • Meta-analyses of studies exploring processes relevant to your model. • Experimental studies generating characteristics typical of the clinical population in healthy participants (to test the relevance of these characteristics to symptom generation).
Participant Type	• Mainly healthy participants.	• Healthy participants or clinical populations.	• Clinical populations or healthy participants.

Stage 1 – Proof of Concept

This proof of concept stage is where the researcher constructs their model and demonstrates its feasibility and potential for use in explaining the clinical phenomenon. This process may involve the adaptation of core knowledge from other areas of psychology to help explain the clinical phenomenon (e.g. the use of existing knowledge of perception, memory, and decision-making processes to construct models explaining the attentional and interpretational biases that underlie anxious responding). But more commonly, this stage will involve the use of basic

experiments under highly controlled conditions for the purpose of identifying causal relations between events. It is the identification of important causal relations using experimental procedures that is one of the defining features of experimental psychopathology, and the detection of causal relations is the very basis of effective theory building. However, experimental procedures are not just useful for detecting causal relations, they can also be used to infer the existence of processes that cannot be directly observed, directly manipulated or directly measured. Inferential methodologies have been a significant feature of experimental psychology over the years, and in particular were an important methodology in the development of cognitive models of animal learning where self-report methods are not an option (Dickinson, 1980; Rescorla, 1980), and in the construction of models of human memory. Inferential techniques are a useful way of developing models of the cognitive processes that underlie clinical disorders, and some specific examples of how this type of methodology has been used to elucidate cognitive processes in psychopathology include (1) identifying some of the associative processes that underlie learned fear and anxiety (Davey, 1992; Davey & McKenna, 1983; White & Davey, 1989), (2) describing the detailed cognitive processes that contribute to attentional and interpretation biases in anxiety and mood disorders (Blanchette & Richards, 2010), and (3) clarifying the role of working memory in the efficacy of eye movement desensitization and reprocessing (EMDR) interventions (van den Hout & Engelhard, 2012). These are just three of many examples, but in each of these cases respectively, the associative processes, attentional processes, and role of working memory cannot be directly observed and had to be inferred from manipulations conducted in elegantly designed experiments.

Stage 2 – Clinical Validity

At this stage the predictions of the model need to be tested against knowledge of what actually happens in the clinical phenomenon. Clinical participants can be tested experimentally to determine whether the important variables in the model are relevant in causing or maintaining the significant symptoms (e.g. Chan et al., 2013). However, in at least some cases, this approach may be problematic because of the additional distress it may cause to already distressed individuals (e.g. subjecting these individuals to variables that may increase symptoms such as anxiety or depression). Alternative methods include examining case histories for evidence of the processes described by the model in the aetiology of clinical patients (e.g. Davey et al., 1993), or investigating the aetiologies of clinical patients using self-report questionnaires and surveys (e.g. Öst & Hugdahl, 1981; Merckelbach, Arntz, & de Jong, 1991).

Stage 3 – Diagnostic Validity

This stage represents the final link up between the psychological model developed in Stage 1 and the relevant clinical phenomenon. Because not everyone acquires a particular psychopathology, we need to ask what it is about the relevant clinical population that makes them differentially vulnerable to the disorder, and then clarify how the model explains this. Some progress towards this goal can be made using literature reviews and meta-analyses to demonstrate that the clinical population has characteristics that make them vulnerable to the important variables and processes in the model. But arguably more convincing evidence for diagnostic validity would come from experimental studies showing that members of the clinical population are significantly more reactive or sensitive to the model's processes than healthy control participants. For example, the reasoning bias of 'jumping to conclusions' is a critical variable in cognitive models of paranoid delusions, and as predicted by the model experimental tests show that a higher percentage of participants with paranoid delusions jump to conclusions compared to non-clinical control participants (Fine, Gardner, Craigie, & Gold, 2007; Garety, Hemsley, & Wessely, 1991; Savulich et al., 2012). This provides some validation for the cognitive model in that jumping to conclusions is a critical variable in the model that is significantly more prominent in the relevant clinical population. However, as argued below, it may not be necessary to make explicit comparisons between clinical populations and healthy individuals in this final stage – diagnostic validity might also be addressed in other ways.

One example is by comparing healthy individuals with participants who score high on measures of the disorder but are at sub-clinical levels (e.g. Vredenburg et al., 1993). This approach is likely to be helpful and valid for disorders that are known to be dimensional rather than categorical in nature (Krueger & Piasecki, 2002), and examples of disorders with known dimensional latent structures include Generalized Anxiety Disorder (Niles, Lebeau, Liao, Glenn, & Craske, 2012), Obsessive-Compulsive Disorder (Olatunji, Williams, Haslam, Abramowitz & Tolin, 2007), and at least some psychotic experiences (Johns & van Os, 2001). The fact that the symptoms of some disorders appear to

be dimensional rather than categorical may initially appear contradictory to establishing diagnostic validity by identifying characteristics that specifically define clinical populations and make them differentially vulnerable to the model being tested. But there is no reason to suppose that these differential vulnerability factors are not themselves dimensional. The vulnerability factors themselves may be specific diagnostic identifiers (e.g. in the examples we have used so far, characteristics such as jumping to conclusions, potential to use mood as information, etc.), but once triggered may facilitate symptoms in a non-linear or exponential way.

A second example is to test healthy individuals and to manipulate the critical variables in the model known to cause the disorder so that they approximate the levels of those characteristics in the clinical population. This is only possible, of course, if the researcher has prior knowledge that the clinical population possesses these characteristics, and an example will be provided in the next section.

You can see from this discussion that there is no one single method to conclusively demonstrate diagnostic validity, and it is likely to be a convergent process bringing together existing knowledge of the characteristics of the clinical population, experimental studies comparing clinical and healthy populations, and even studies of the model's critical variables conducted on healthy individuals.

Some Implications of the Research Pathway

Clearly, a research programme can be constructed across all three stages of the pathway, addressing each of the validity criteria in turn, but this is likely to be a lengthy and burdensome process and would seem appropriate only for the development of entirely new or novel psychological models. Alternatively, research can be conducted at any individual stage if the knowledge required by earlier stages is already available. For example, research can commence directly at Stage 2 if well-developed psychological models exist and convincingly address the validity questions posed at Stage 1. One clear example of this is when conditioning models are applied to psychopathology. The conditioning models have been well-developed elsewhere (e.g. in the animal learning literature), have a formalistic resemblance to the psychopathology (e.g. anxiety/fear learning), and so can be used to determine whether the processes defined by the model predict behaviour in the clinical disorder (e.g. Davey, 1989, 1992; Davey et al., 1993). What is relatively unclear in this stage-based model is what would constitute sufficient evidence to be able to move confidently from one stage of the research pathway to the next. There may be no objective criteria by which to judge this, although the potential impact of the model on theory and/or practice may require more or less convincing validity evidence depending on the importance of the research questions being addressed.

It may also be the case that certain types of research could be prioritized at particular stages of the pathway. For example, it may be risky to start at Stage 1 by spending excessive time and funding on research that turns out not to be clinically relevant. In contrast, the higher costs of intervention research may be better placed in the later stages of the pathway – especially if there is evidence that the intervention may already be viable, and to focus on diagnostic validity as a means of determining the effectiveness of the intervention in relation to the diagnostic characteristics of the relevant clinical population.

Models of psychopathology that postulate new mechanisms of aetiology may have to progress through all three stages of the pathway, beginning with proof of concept in Stage 1, through clinical validity in Stage 2, and ending with diagnostic validity in Stage 3. One interesting feature of this full pathway is that it would in principle be possible to answer validity questions at all three stages using studies conducted entirely on healthy individuals. It would require that certain relevant information about the pertinent clinical population was available in the literature, but studies conducted on healthy individuals could be sufficient to address all validity questions, including those posed by diagnostic validity. For example, it would be quite normal practice to construct the clinical model in Stage 1 on healthy participants to demonstrate its feasibility and potential for use in explaining the clinical phenomenon. Secondly, if information is available in the clinical literature about some of the basic processes through which clinical populations develop their disorder (e.g. that some specific phobias are acquired by contiguous experiences with aversive events), the predictive validity of the model could be tested on healthy individuals by seeing if the model produces outcomes that are consistent with aetiological knowledge in the clinical literature (e.g. Alvarez, Biggs, Chen, Pine, & Grillon, 2008; Davey & Matchett, 1994; White & Davey, 1989). At Stage 3, the model should be in a well-developed state that enables the researcher to identify the significant variables that generate the disorder via the model. If these

variables represent known characteristics of the clinical population, then the researcher may be able to manipulate these variables in healthy participants and predict outcomes representative of the disorder (providing some evidence for diagnostic validity). For example, the mood-as-input hypothesis of perseverative worrying argues that pathological worriers use their negative mood as information that they have not achieved the goals of their worry and so persevere with their worrying (Meeten & Davey, 2011). But not everyone in a negative mood perseveres when worrying, so what makes pathological worriers different? A search of the social psychology literature on mood reveals that when people are engaged in a task (such as worrying) they will only use their moods as information under certain conditions (e.g. when they lack expertise in the task, only when they know their mood is relevant to the task in some way, or when they are experiencing high cognitive load) (Ottati & Isbell, 1996; Schwarz & Clore, 1983; Siemer & Reisenzein, 1998). Interestingly, all these factors are active in individuals identified as pathological worriers. Pathological worriers have poor problem-solving confidence, and so lack confidence in their expertise to deal with the worry problem (Davey, 1994; Laugesen & Dugas, 2000), are actively attempting to repair negative mood, and so their mood is very relevant to the task (Clark & Isen, 1982), and anxiety is known to increase cognitive load and reduce working memory capacity, and thus be more likely to render mood relevant as information (Hayes, Hirsch & Mathews, 2008; Eysenck & Calvo, 1992). Given this knowledge of the relevant clinical population, the experimental psychopathology researcher can now test the diagnostic validity of the mood-as-input model by experimentally manipulating these factors in healthy individuals to simulate conditions that are characteristic of the clinical population.

Conducting experimental psychopathology research fastidiously via this three-stage virtual programme of research will be a lengthy process. But research at each level is valid in its own right. A consequence of this is that journal editors and funding bodies should not be asking researchers at Stage 1 to provide concrete evidence that their research or model is relevant to a clinical population. They also should not be insisting that the research is only clinically valid if it is done on clinical populations. Indeed, as I've argued above, it is entirely possible for all three stages of the research to be conducted on healthy individuals and still be perfectly clinically relevant and clinically valid if linked to the relevant clinical literature.

Conclusions

Over the past decade there have been many challenges facing the experimental psychopathologist studying psychological models of clinical disorders. These have ranged from clinical research on healthy participants being excluded from clinical journals, to intensive competition for funds with popular alternative explanatory approaches such as neuroscience and genetics (Davey, 2015). One way in which experimental psychopathology can begin to compete with these alternative approaches is to be very clear about what constitutes valid experimental psychopathology research and the pathway by which such research needs to progress and be validated. This paper outlines a putative pathway for experimental psychopathology research developing psychological models of clinical disorders, and will hopefully encourage researchers to specify in the introduction to their papers the research framework in which their models have been developed and the validation processes they have adopted to justify their models. This will communicate to the broader clinical research community that psychological models derived from experimental psychopathology methodologies are a valid and coherent contribution to understanding and treating mental health problems, and provide a valuable alternative perspective to neuroscience and genetic approaches.

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References

Abramson, L. & Seligman, M. (1977). Modeling psychopathology in the laboratory : history and rationale. In Psychopathology: Experimental Models (ed. J. Maser and M. Seligman), pp. 1–26. W. H. Freeman and Co. : San Francisco.

Alvarez, R.P., Biggs, A., Chen, G., Pine, D.S., & Grillon, C. (2008) Contextual fear conditioning in humans: Cortical-hippocampal and amygdala contributions. *Journal of Neuroscience*, 11, 6211-6219. https://doi.org/10.1523/JNEUROSCI.1246-08.2008

Belzung, C. & Lemoine, M. (2011) Criteria of validity for animal models of psychiatric disorders: Focus on anxiety disorders and depression. *Biology of Mood & Anxiety Disorders*, 1, 9. https://doi.org/10.1186/2045-5380-1-9

Blanchette, I. & Richards, A. (2010) The influence of affect on higher level cognition: A review of research on interpretation, judgment, decision making and reasoning. *Cognition & Emotion*, 24, 561-595. https://doi.org/10.1080/02699930903132496

Bouchard, C., Rheaume, J., & Ladouceur, R. (1999). Responsibility and perfectionism in OCD: an experimental study. *Behaviour Research and Therapy*, 37(3), 239– 248. https://doi.org/10.1016/S0005-7967(98)00141-7

Bouton, M.E., Mineka, S., & Barlow, D.H. (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychological Review*, 108, 4–32. https://doi.org/10.1037/0033-295X.108.1.4

Carrion, R.E., McLaughlin, D., Auther, A.M., Olsen, R., Correl, C.U., & Cornblatt, B.A. (2015) The impact of psychosis on the course of cognition: A prospective, nested case-control study in individuals at clinical high-risk for psychosis. *Psychological Medicine*, Psychological Medicine, 45, 3341-3354. https://doi.org/10.1017/S0033291715001233

Chan, J.C.S., Davey, G.C.L., & Brewin, C.R. (2013) Understanding depressive rumination from a mood-as-input perspective. *Behaviour Research & Therapy*, 51, 300-306. https://doi.org/10.1016/j.brat.2013.02.007

Clark, M. S. & Isen, A. M. (1982). Toward understanding the relationship between feeling states and social behavior. In A.H. Hastrof, & A.M. Isen (Eds.), *Cognitive social psychology* (pp. 73-108). Elsevier: New York.

Craske, M.G., Liao, B., Brown, L., & Vervliet, B. (2012) Role in inhibition in exposure therapy. *Journal of Experimental Psychopathology*, 3, 322-345. https://doi.org/10.5127/jep.026511

Davey, G.C.L. (1989) UCS revaluation and conditioning models of acquired fears. *Behaviour Research & Therapy*, 27, 521-528. https://doi.org/10.1016/0005-7967(89)90086-7

Davey, G.C.L. (1992) Classical conditioning and the acquisition of human fears and phobias: review and synthesis of the literature. *Advances in Behaviour Research & Therapy*, 14, 29-66. https://doi.org/10.1016/0146-6402(92)90010-L

Davey, G.C.L. (1997) A conditioning model of phobias. In G.C.L. Davey (Ed) Phobias: A handbook of theory, research and assessment. Chichester: Wiley.

Davey, G.C.L. (2003) Doing clinical psychology research: What is interesting isn't always useful. *The Psychologist*, 16, 412-416.

Davey, G.C.L. (2006) A mood-as-input account of perseverative worrying. In G.C.L. Davey & A. Wells (Eds) Worry and its Psychological Disorders. John Wiley. https://doi.org/10.1002/9780470713143.ch13

Davey, G.C.L. & Matchett, G. (1994) UCS rehearsal and the enhancement and retention of differential 'fear' conditioning: Effects of trait and state anxiety. *Journal of Abnormal Psychology*, 103, 708-718. https://doi.org/10.1037/0021-843X.103.4.708

Davey, G.C.L., de Jong, P.J., & Tallis, F. (1993) UCS inflation in the aetiology of a variety of anxiety disorders: Some case histories. *Behaviour Research & Therapy*, 31, 495-498. https://doi.org/10.1016/0005-7967(93)90130-M

Davey, G.C.L. (2013a) Does a menu make a restaurant? – Clinical constructs as potential category mistakes. http://www.papersfromsidcup.com/graham-daveys-blog/does-a-menu-explain-a-restaurant-clinical-constructs-as-potential-category-mistakes.

Davey, G.C.L. (2015) The funding of mental health research in the UK – A biased and flawed system? http://www.papersfromsidcup.com/graham-daveys-blog/-the-funding-of-mental-health-research-in-the-uk-a-biased-and-flawed-system.

Davey, G.C.L., Bickerstaffe, S., & MacDonald, B. (2006) Experienced disgust causes a negative interpretation bias: A causal role for disgust in anxious psychopathology. *Behaviour Research & Therapy*, 44, 1375-1384. https://doi.org/10.1016/j.brat.2005.10.006

Davey, G.C.L. (1994). Worrying, social problem-solving abilities, and social problem-solving confidence. *Behaviour Research and Therapy*, 32 (3), 327-320. https://doi.org/10.1016/0005-7967(94)90130-9

Dawes (1994) House of cards: Psychology and psychotherapy built on a myth. New York: Free Press.

Delgado, M.R., Olsson, A., & Phelps, E.A. (2006). Extending animal models of fear conditioning to humans. *Biological Psychology*. 73: 39–48. https://doi.org/10.1016/j.biopsycho.2006.01.006

Dickinson, A. (1980) Contemporary animal learning theory. Cambridge: Cambridge University Press.

Eysenck, M.W. & Calvo, M.G. (1992). Anxiety and performance: The processing efficiency theory. *Cognition and Emotion*, 6, 409–434. https://doi.org/10.1080/02699939208409696

Fine, C., Gardner, M., Craigie, J., & Gold, I. (2007). Hopping, skipping or jumping to conclusions? Clarifying the role of the JTC bias in delusions. *Cognitive Neuropsychiatry*, 12, 46-77. https://doi.org/10.1080/13546800600750597

Garety, P.A., Hemsley, D., & Wessely, S. (1991). Reasoning in deluded schizophrenic and paranoid patients: Biases in performance on a probabilistic inference task. *Journal of Nervous and Mental Disease*, 179, 194-201. https://doi.org/10.1097/00005053-199104000-00003

Geyer, M. & Markou, A. (1995) Animal Models of psychiatric disorders. In: Bloom F, Kupfer D, editors. Psychopharmacology: the fourth generation of progress. New York: Raven Press; pp. 787–798.

Grant, V.V., Stewart, S.H., & Birch, C.D. (2007) Impact of positive and anxious mood on implicit alcohol-related cognitions in internally motivated undergraduate drinkers. *Addictive Behaviors*, 32, 2226-2237. https://doi.org/10.1016/j.addbeh.2007.02.012

Hawksley, J. & Davey, G.C.L. (2010) Mood-as-input and depressive rumination. *Behaviour Research & Therapy*, 48, 134-140. https://doi.org/10.1016/j.brat.2009.10.004

Hayes, S., Hirsch, C.R., & Mathews, A. (2008) Restriction of working memory capacity during worry. *Journal of Abnormal Psychology*, 117, 712-717. https://doi.org/10.1037/a0012908

Hepburn, S.R., Barnhofer, T., & Williams, J.M.G. (2006) Effects of mood on how future events are generated and perceived. *Personality and Individual Differences*. 41:801–811. https://doi.org/10.1016/j.paid.2006.03.022

Johns, L.C. & van Os, J. (2001) The continuity of psychotic experiences in the general population. *Clinical Psychology Review*, 21, 125-41. https://doi.org/10.1016/S0272-7358(01)00103-9

Jones, C.A., Watson, D.J.G., & Fone, K.C.F. (2011) Animal models of schizophrenia. *British Journal of Pharmacology*, 164, 1162-1194. https://doi.org/10.1111/j.1476-5381.2011.01386.x

Keane, T.M., Zimering, R.T., & Caddell, J. (1985). A behavioural formulation of posttraumatic stress disorder in Vietnam veterans. *The Behavior Therapist*, 8, 9–12.

Krueger, R.F. & Piasecki, T.M. (2002). Toward a dimensional and psychometrically informed approach to conceptualising psychopathology. *Behaviour Research & Therapy*, 40, 485–499. https://doi.org/10.1016/S0005-7967(02)00016-5

Laugesen, N. & Dugas, M. (2000). Worry in adolescence: Relationship with problem orientation and problem solving skills. 'State-of-the-Art Lectures, Invited Symposia, Keynote Addresses, Poster session', International *Journal of Psychology*, 35(3), 272 – 369.

Lopatka, C. & Rachman, S. (1995). Perceived responsibility and compulsive checking: An experimental analysis. *Behaviour Research and Therapy* 33(6), 673-684. https://doi.org/10.1016/0005-7967(94)00089-3

Lynch, W.J., Nicholson, K.L., Dance, M.E., Morgan, R.W., & Foley, P.L. (2010) Animal models of substance abuse and addiction: Implications for science, animal welfare, and society. *Comprehensive Medicine*, 60, 177-88.

Meeten, F. & Davey, G.C.L. (2011) Mood-as-input hypothesis and perseverative psychopathologies. *Clinical Psychology Review*, 31, 1259-1275. https://doi.org/10.1016/j.cpr.2011.08.002

Menon, V. (2011) Large-scale brain networks and psychopathology: A unifying triple network model. *Trends in Cognitive Science*, 15, 483-506. https://doi.org/10.1016/j.tics.2011.08.003

Merckelbach, H., Arntz, A. & de Jong, P.J. (1991) Conditioning experiences in spider phobics. *Behaviour Research & Therapy*, 29, 333-335. https://doi.org/10.1016/0005-7967(91)90068-E

Nestler, E.J. & Hyman, S.E. (2010) Animal models of neuropsychiatric disorders. *Nature Neuroscience*, 13, 1161-1169. https://doi.org/10.1038/nn.2647

Niles, A.N., Lebeau, R.T., Liao, B., Glenn, D.E., & Craske, M.G. (2012). Dimensional indicators of generalized anxiety disorder severity for DSM-V. *Journal of Anxiety Disorders*, 26(2), 279–286. https://doi.org/10.1016/j.janxdis.2011.12.007

O'Brien, C.P., Childress, A.R., Ehrman, R., & Robbins, S.J. (1998) Conditioning factors in drug abuse: Can they explain compulsion? *Journal of Psychopharmacology*, 12, 15-22. https://doi.org/10.1177/026988119801200103

Ochoa, S., Haro, J.M., Huerta-Ramos, E., Cuevas-Esteban, J., Stephan-Otto, C., Usall, J., Nieto, L., & Brebion, G. (2014) Relation between jumping to conclusions and cognitive functioning in people with schizophrenia in contrast with healthy participants. *Schizophrenia Research*, 159, 211-217. https://doi.org/10.1016/j.schres.2014.07.026

Olatunji, B.O., Williams, B.J., Haslam, N., Abramowitz, J.S., & Tolin, D.F. (2007) The latent structure of obsessive-compulsive symptoms: A taxometric study. *Depression & Anxiety*, 25, 956-968. https://doi.org/10.1002/da.20387

Öst, L.G. & Hugdahl, K. (1981) Acquisition of phobias and anxiety response patterns in clinical patients. *Behaviour Research & Therapy*, 19, 439-447. https://doi.org/10.1016/0005-7967(81)90134-0

Ottati, V. & Isbell, L. (1996). Effects of mood during exposure to target information on subsequently reported judgments: An on-line model of misattribution and correction. *Journal of Personality and Social Psychology*, 71, 39-53. https://doi.org/10.1037/0022-3514.71.1.39

Powers, A.S., McClure, E.B., Phelps, E.A., Woldehawariat, G., Grillon, C., & Pine, D.S. (2005) Classical fear conditioning in the anxiety disorders: A meta-analysis. *Behaviour Research & Therapy*, 43, 1391-1424. https://doi.org/10.1016/j.brat.2004.10.007

Rescorla, R.A. (1980) Pavlovian second-order conditioning. New Jersey: Lawrence Erlbaum Assoc.

Savulich, G., Shergill, S., & Yiend, J. (2012). Biased cognition in psychosis. *Journal of Experimental Psychopathology*, 3, 514–536. https://doi.org/10.5127/jep.016711

Schwarz, N., & Clore, G. L. (1983). Mood, misattribution, and judgments of well-being. Informative and directive functions of affective states. *Journal of Personality and Social Psychology*, 45(3), 513-523. https://doi.org/10.1037/0022-3514.45.3.513

Siemer, M., & Reisenzein, R. (1998). Effects of mood on evaluative judgements: Influence of reduced processing capacity and mood salience. *Cognition and Emotion*, 12, 783-805. https://doi.org/10.1080/026999398379439

Steimer, T. (2011) Animal models of anxiety disorders in rats and mice: Some conceptual issues. *Dialogues in Clinical Neuroscience*, 13, 495-506.

Stewart, A.M. & Kalueff, A.V. (2015) Developing better and more valid animal models of brain disorders. *Behavioural Brain Research*, 276, 28-31. https://doi.org/10.1016/j.bbr.2013.12.024

Teasdale, J.D. & Fogarty, S.J. (1979) Differential effects of induced mood on retrieval of pleasant and unpleasant events from episodic memory. *Journal of Abnormal Psychology*, 88, 248-257. https://doi.org/10.1037/0021-843X.88.3.248

Van den Hout, M.A. & Engelhard, I.M. (2012) How does EMDR work? *Journal of Experimental Psychopathology*, 3, 724-738. https://doi.org/10.5127/jep.028212

van Gestel, M.A., Kostrzewa, E., Adan, R.A.H., & Janhunen S.K. (2014) Pharmacological manipulations in animal models of anorexia and binge eating in relation to humans. *British Journal of Pharmacology*, 171, 4767-4784. https://doi.org/10.1111/bph.12789

Vervliet, B. & Raes, F. (2013) Criteria of validity in experimental psychopathology: Application to models of anxiety and depression. *Psychological Medicine*, 43, 2241-2244. https://doi.org/10.1017/S0033291712002267

Vredenburg, K., Flett, G.L., & Krames, L. (1993). Analog versus clinical depression – A critical reappraisal. *Psychological Bulletin*, 113, 327–344 https://doi.org/10.1037/0033-2909.113.2.327

White, K. & Davey, G.C.L. (1989) Sensory preconditioning and UCS inflation in human 'fear' conditioning. *Behaviour Research & Therapy*, 27, 161-66. https://doi.org/10.1016/0005-7967(89)90074-0