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Psychotherapy outcome research: Implications of a new clinical taxonomy

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Abstract

Since the publication of DSM-III in 1980, the scientist-practitioner gap in clinical psychology has expanded, as almost all outcome research in clinical psychology has been on diagnosed mental disorders within a medical model using drug trial methodologies, whereas most practising clinicians undertake functional analyses and case formulations of clinical psychological problems (CPPs) and then apply tailored interventions within an ongoing hypothesis-testing methodology. But comparatively reliable assessment and generalizable conclusions in psychotherapy outcome research require a comprehensive theory-derived conception or operational definition of 'CPPs', standardized functional analyses, and a taxonomy of CPPs comparable to DSM's listings of mental disorders. An alternative conception and taxonomy of CPPs have recently been proposed, offering improvements in the reliability and generalizability of case formulation-based psychotherapy outcome research. It conceives of CPPs as instances of the formation and operation of self-sustaining problem-maintaining circles (PMCs) of psychological-level causal elements—that is, at the level of cognitions, behaviours, emotions, and events or situations (stimuli). The paper describes this new conception of CPPs, a subsequent nascent taxonomy of evidence-based PMCs which standardizes the underlying mechanisms that maintain CPPs, and ensuing benefits to research (as well as to practice) in clinical psychology. These benefits include being able to encompass all treatment-worthy CPPs, not just diagnosable mental disorders; to assess theory-derived intervention strategies, not just arbitrary therapy bundles; and to directly feed back into psychological theories, not just expand an atheoretical list of patented "evidence supported therapies."

KEYWORDS
case formulation, clinical psychological problems, outcome research, problem-maintaining circles, taxonomy, transdiagnostic

1 | INTRODUCTION

The overwhelming dominance of the "weak medical model" of clinical psychological problems (CPPs; W.C. Follette & Houts, 1996) imposed by psychiatry's *Diagnostic and Statistical Manual of Mental Disorders* (APA, 2013) has not, on balance, been good for clinical psychology's standing, independence, or its clinical or theoretical contribution (DCP, 2013; Hofmann, 2014; Johnstone, 2014). A minor balm to this criticism has been the observation that, in practice, few clinical psychologists devote much time to psychiatric diagnosis (Bieling & Kuyken, 2003; D. Dobson & Dobson, 2009). Training and clinical practice are much more focused on psychological-level case formulation (Carey & Pilgrim, 2010; J.B. Persons, 2008).

However, the same cannot be said of *research* in clinical psychology, which is still—and is increasingly—conceptually dominated by questions surrounding the treatment of diagnosed mental disorders

(Roth & Fonagy, 2005; W.B. Stiles & Shapiro, 1989, 1994; D. Westen et al., 2004).

The resultant scientist-practitioner gap (Dozois, 2013) is often attributed to clinicians who resist or ignore the evidence-based dictates of research scientists (Baker et al., 2008). However, perhaps most clinical psychology practitioners have not abandoned the scientist-practitioner model in the slightest. Perhaps, instead, the gap can be attributed to researchers in clinical psychology who, since the introduction of DSM-III in 1980, have diverged to study another discipline's subject matter—psychiatry's mental disorders—in preference to their own, CPPs.

If this is the case, then an alternative solution would be to realign the conceptual models and methodological procedures of researchers away from the (fiscally and administratively imposed) research questions of an adjacent discipline and back to clinical psychology's own original mission: to discern "*what* treatment, by *whom*, is most effective for *this* individual with *that* specific problem [note, 'problem', not 'diagnosis'], and under *which* set of circumstances" (Paul, 1967, p. 111). That is, to resume research on the treatment of CPPs—the case-formulated problems that most clinical psychologists instinctively and pragmatically target—rather than on the treatment of diagnosed psychiatric mental disorders.

On the other hand, the greatest advantage of DSM diagnosis in the research setting has been its reliability (N.C. Andreasen, 2007; T.A. Brown et al., 2001), even at an admitted sacrifice to its validity (Clark et al., 1995; Hyman, 2010; Kendell & Jablensky, 2003). Case formulation is, in contrast, notoriously variable and difficult to systematize (Bissett & Hayes, 1999; S.C. Hayes et al., 1996), a factor critical to the generalizability of research results (Dozois, 2013). Clinical psychologists have been found to be more reliable and consistent in the descriptive aspects of case formulation than in its inferential aspects (W. Kuyken et al., 2005).

Until recently, no alternative conception and subsequent systematic codifiable taxonomy of CPPs that could replace psychiatric diagnosis in both clinical and research settings have been available (Andersson & Ghaderi, 2006; T.A. Brown & Barlow, 2009). One such has now been advanced (G.M. Bakker, 2008a, 2008b, 2019, 2021), that, if widely adopted, can restore the critical feedback loop between clinical psychological theory, research, and practice (Mansell et al., 2009; Neale & Liebert, 1986; Salkovskis, 2002).

CPPs involving problematic levels of anxiety, depression, alcohol consumption, obsessive–compulsive behaviour, and so forth have historically been conceived of as instances of moral weakness, or demonic possession, or deeply rooted psychodynamic pathologies, or maladaptive behaviour patterns (N. Haslam & Ernst, 2002). The most dominant current model—the medical model embodied in DSM diagnosis—sees CPPs as biologically based internal "mental disorders."

However, the new conception in development sees the essential difference between a normal, adaptive grief reaction, and a depression CPP that warrants clinical intervention, or between an understandable anxious state and a CPP involving problematic anxiety, or between fussiness and an obsessive–compulsive CPP, in the formation and operation of one or more undesired, self-maintaining, psychological-level causal cycles, involving people's thoughts, feelings, behaviour, and situations or stimuli (G.M. Bakker, 2019).

Such a maintenance cycle is labelled a "problem-maintaining circle" (PMC), and the body of empirical evidence-based theory supporting and elaborating this conception of CPPs as "PMC Theory."

Several examples of such PMCs are illustrated in Figure 1.

The origins, justifications, and benefits to clinical *practice* of this new conception of CPPs and its ensuing CPP taxonomy have been outlined elsewhere (see especially G.M. Bakker, 2019). In this paper, the justifications and benefits of PMC Theory in the *research* context are outlined.

Key Practitioner Message

- Not all clinical psychological problems (CPPs) are diagnosable mental disorders.
- Clinically relevant research needs to focus on the treatment of case-formulated CPPs.
- This requires a new DSM-equivalent conception and taxonomy of CPPs, which has recently been introduced.
- Psychological-level "problem-maintaining circles" (PMCs) can explain all CPPs that incorporate cyclic maintenance processes.
- Research on CPPs in PMC theory is more theory-based and treatment-relevant than research on mental disorders.

2 | DSM'S DOMINANCE

The dominance of the medical model of mental disorders within clinical psychology since the publication of DSM-III (APA, 1980) has been thoroughly described (e.g., G.M. Bakker, 2019; T.A. Brown & Barlow, 2002; W.C. Follette & Houts, 1996; Waterman, 2007; M. Wilson, 1993). This has occurred conceptually, administratively, and, to a lesser but equally unfortunate degree, clinically (W.C. Follette, 1996; Waterman, 2007). Adverse conceptual and clinical effects of this inappropriate medical model dominance have been publicly bemoaned in the UK by the British Psychological Society's Division of Clinical Psychology in its *Position Statement on the Classification of Behaviour and Experience in Relation to Functional Psychiatric Diagnosis: Time For a Paradigm Shift* (DCP, 2013) and in the US by a petition signed by over 15,000 people and over 50 organizations (Open Letter to the DSM-5, ipetitions, n.d.). However, the deleterious effect on *research* in clinical psychology has not been adequately recognized and continues unabated.

In the 1980s, at around the same time as cognitive behaviour therapy (CBT) emerged from radical behaviourism—potentially enabling

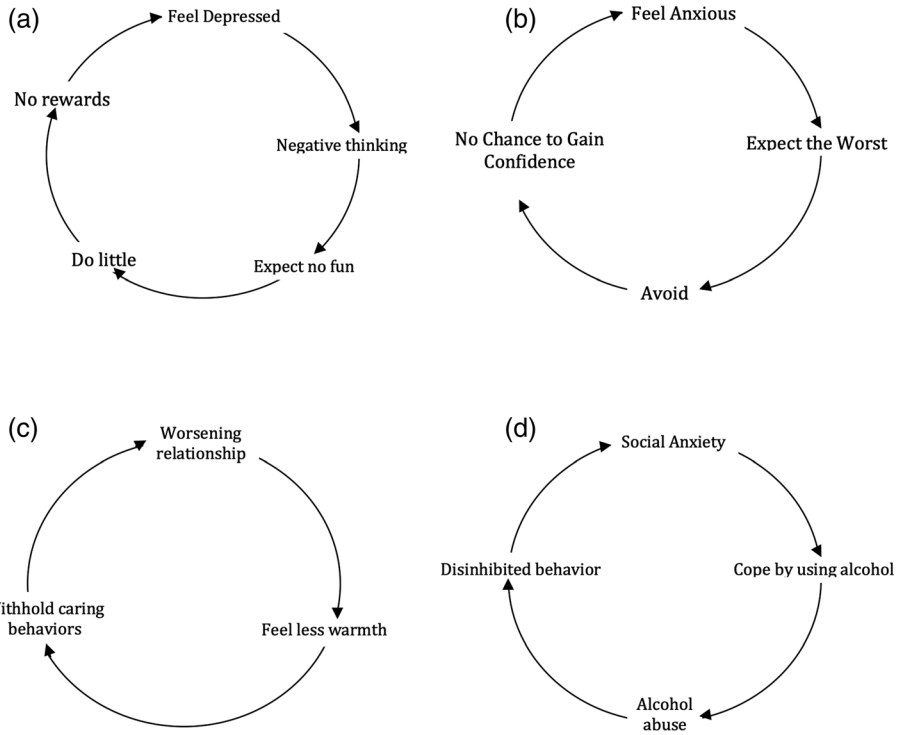

FIGURE 1 Examples of problem-maintaining circles (PMCs) which underlie CPPs

comprehensive case formulations with high face validity and high clinical utility—there was a coincidental, concurrent shift following the publication of DSM-III from outcome studies of the use of therapy manuals to address specific target problems—CPPs such as low assertiveness or examination anxiety—to clinical trials of manualized therapy packages targeting DSM-diagnosed mental disorders, using the concepts and methodology of drug trials (M.R. Goldfried & Wolfe, 1998). "Diagnostically homogeneous samples have become increasingly normative … It has become harder for researchers to obtain funding for samples based on other criteria" (Roth & Fonagy, 2005, p. 488).

Clinical psychology has been profoundly shaped by the biomedical model and associated drug trial methodology. For example, in the US, NIMH funding goes almost exclusively to randomized controlled trials (RCTs) testing "the efficacy of standardized (i.e. manualized) psychological treatments in reducing the symptoms of *DSM*-defined psychiatric diagnoses" (Deacon, 2013, p. 853). As a result, between the two most dominant disciplines in mental health research, clinical psychology journals cite psychiatry journals at a much higher rate than the reverse (N. Haslam & Lusher, 2011).

Psychotherapy outcome research has concurrently moved from assessing our ability to resolve identified CPPs to measuring intervention effects on the level of symptoms of psychiatric mental disorders (G.M. Bakker, 2019). The resultant efficacy research (Chambless & Hollon, 1998) produced high levels of methodological rigour (Anand & Malhi, 2011), but low generalization to clinical reality and low external validity (M.R. Goldfried & Wolfe, 1998; Kendell & Jablensky, 2003). Most DSM diagnoses have achieved moderate reliability coefficients, their acknowledged raison d'etre (N.C. Andreasen, 2001; Hyman, 2010), but are of persistently dubious validity (N.C. Andreasen, 2007; Kendell & Jablensky, 2003; Smith et al., 2009) for many reasons, including an absence of biological markers, vague dimensional criteria, and especially their circular definitions (Carey & Pilgrim, 2010; Hickey, 1998; Stein et al., 2010). How useful is it to attribute a person's social anxiety and avoidance to the presence of the putative mental disorder "Social Anxiety Disorder," which is known to be present by observation of their social anxiety and avoidance? The unsupported claim that some latent, underlying, real condition exists "causing" these symptoms become known as the reification of mental disorders (Hyman, 2010).

Although there were initially some gains as CBT progressed within a disorder-specific approach (Mansell et al., 2009), "the focus on DSM diagnostic disorders … constrains how we think about, and the kinds of questions we ask about, clinical problems" (M.R. Goldfried & Wolfe, 1998, p. 145). Despite the fact that clinicians have largely persisted in tailoring treatments to their evidence-based case formulations, researchers in clinical psychology, who are required to DSM-define their targets and manualize their treatments so as to be eligible for research grants, find it hard to even imagine they could do otherwise (Hyman, 2010, p. 157). The medical model has thoroughly dominated conceptually in research settings.

Two of the critical and radical sources of restraint upon advancement in clinical psychological research resulting from the imposition of this conceptual mismatch have been (a) the reification of mental disorders and (b) the testing of bulk prescribed manualized treatment packages.

2.1 | Problems due to the reification of mental disorders

When Mental Disorder A is defined by levels of a collection of symptoms, and improvement is operationalized as an overall reduction in

those symptom levels, then the validity of a research conclusion and subsequent treatment guideline that Treatment Package X is statistically more or less efficacious that Treatment Package Y for Mental Disorder A relies upon whether that collection of symptoms consistently indicates a valid homogeneous underlying disorder. However, the "naive-realist or objectivist position that mental disorders are essence-based, classically definable, objectively grounded, and discovered by carving psychiatric nature at its joints has generally taken a beating" (N. Haslam, 2002, p. 203).

It is easy to forget that DSM diagnoses are created by committee consensus, not by purely empirical data (Flanagan & Blashfield, 2010; D. Westen et al., 2004). For example, it is still uncertain even as to whether Major Depressive Disorder is a distinct disorder or the severe end of a continuum (Flett et al., 1997; R.C. Kessler et al., 2003; D. Westen et al., 2004). "Many have come to believe that we are dealing with clear and discrete disorders rather than arbitrary symptom clusters" (Tucker, 1998, p. 159). This unjustified assumption is termed the reification of mental disorders (W.C. Follette, 1996; W.C. Follette & Houts, 1996; Hickey, 1998; Hyman, 2010; Kendell & Jablensky, 2003).

Among the reasons to doubt whether most DSM diagnoses exist in the real world, as opposed to being handy administrative categories, is that DSM's disorders are far from discrete. Comorbidities can range from 50% to 90% (R.C. Kessler et al., 1996, 1999; Newman et al., 1998). In field trials of DSM-5, Regier et al. (2013) found nine of 23 diagnoses had reliability coefficients that fell in the "poor" range, including Major Depressive Disorder (MDD) and Generalized Anxiety Disorder (GAD). Debates continue as to whether the high comorbidity between MDD and GAD is due to symptoms (criteria) in common, or a cause or mechanism in common (D. Westen et al., 2004).

"We make attempts at understanding so-called comorbidity between anxiety and depression, rather than considering the possibility that anxiety may inhibit competent functioning that results in a diminished view of self and dysphoric mood" (M.R. Goldfried & Wolfe, 1998, p. 145). That is, we ignore case formulation-generating clinical psychological theory, which attends to mechanisms or processes rather than alleged underlying disease states or conditions. We forget that the idea that a person who is persistently sad has an internally residing illness, disease, or disorder is a *model*, a metaphor, that can be useful and fit well, or be misleading and misapply.

DSM diagnosis-based research relies on the drug metaphor which "has served as a conceptual lowest common denominator—a simple model that proponents of all the schools … can use" (W.B. Stiles & Shapiro, 1989, p. 537). In order to be administratively usable by clinicians with a range of theoretical orientations, this atheoretical, phenomenological approach ignores underlying mechanisms (Kim & Ahn, 2002).

In this sense, DSM represents a prescientific stage of development in attempts to bring taxonomic order to aberrant behavioural phenomena (Carson, 1997). Being atheoretical (T.A. Brown & Barlow, 2009; Clark et al., 1995), it stunts the desired circular processes between research and theory development (G.M. Bakker, 2019; Mansell et al., 2009, p. 8; Neale & Liebert, 1986) and between researchers and clinicians (Mansell et al., 2009, p. 8; A.E. Kazdin, 2008) that scientific progress requires.

The assumption that, in order to be scientific, clinical psychology's theories need to be reduced or related to biological-level theories is a clear example of "greedy reductionism" (Dennett, 1995). Each level of analysis has its own relevance, usefulness, and importance (G.M. Bakker, 2019; Satel & Lilienfeld, 2013). "No portion of the biopsychosocial model has a monopoly on the truth" (Deacon, 2013, p. 856). "Psychological realism" is the position that regards the psychological level of analysis and intervention as equally as valid as the neurological level or any other level (sociological, biochemical, etc.; Bortolotti & Broome, 2009).

To claim that ADHD or anorexia nervosa has a biological basis is in one sense a tautology (K.S. Kendler, 2005)—*all* psychological experience occurs in the brain—and in another sense is an assertion requiring justification. Is biology a *useful* level of analysis here? A correlation between psychological events and biological events does not make psychological events into biological events (Miller, 2010) and "for the time being, psychology appears comfortably safe from replacement by neuroscience and molecular biology" (Deacon, 2013, p. 856). With CPPs, and even with most mental disorders, the "disorder" or problem mechanism is at the *person* level, not the biological level (Banner, 2013, p. 510). It is identified and treated in clinical psychology at the level of the person's thoughts, feelings, and behaviours (Banner, 2013, p. 511).

The drug metaphor has been explicit in all the major comparative psychotherapy outcome studies (W.B. Stiles & Shapiro, 1989, 1994). It, and DSM's "socio-political medical disease metaphor" (Carson, 1997), have come to dominate, despite conceptual misalignment and a lack of clinical utility, because "the example of medicine is a socially powerful, economically compelling one for psychotherapy" (W.B. Stiles & Shapiro, 1989, p. 537), and due to a lack of serious taxonomic competition (Andersson & Ghaderi, 2006; Carson, 1997).

So research on the treatment of Major Depressive Disorder (MDD), for example, has assumed MDD to be a homogeneous underlying condition and has invariably ignored the breadth of underlying psychological mechanisms likely represented in the sample group and potentially revealable by theory-based case formulation. This has resulted in disjointed, atheoretical, "cookbooks" of treatment guidelines and recommendations, with little growth in the theoretical rationales behind them.

What is needed is outcome research on groups of subjects defined by a conceptualization of CPPs that is overtly psychological-level, theory-rich, and preferably involves mechanisms and processes that are uncoverable through case formulation.

2.2 | Problems with manualized treatment packages

Another consequence of the drug metaphor and DSM's dominance in clinical psychological research has been the "small revolution

in research and practice" represented by psychotherapy manuals (Luborsky & DeRubeis, 1984). While diagnosis specifies treatment targets (symptoms), manualization specifies treatment ingredients (W.B. Stiles & Shapiro, 1989, p. 522).

The methodological arguments for manualization of treatments in research settings are clear. Some degree of standardization and treatment fidelity is necessary to turn correlational data into causal experimental data. "One cannot test experimental manipulations one cannot operationalize" (D. Westen et al., 2004, p. 637). So manuals attempt to minimize the role of clinical judgement—unnaturalistic as this may be. Treatments were manualized to standardize them for research, not for clinical efficacy (Luborsky & DeRubeis, 1984; Truijens et al., 2019) Reliability is increased (G.T. Wilson, 1996) and the training and supervision of, at least beginning, therapists is facilitated (G.T. Wilson, 1998).

However, from the start, serious limitations of this approach when applied to mental disorders were noted (M.E. Addis & Krasnow, 2000; D.H. Barlow et al., 1999; S.L. Garfield, 1996). Manual-based treatments are not developed from the information given by a diagnosis per se (Andersson & Ghaderi, 2006, p. 75) nor are they initially designed to serve as the foundation of either practice or training (D. Westen, 2002; D. Westen et al., 2004). They are developed often from accumulated research with functional analyses, case formulations, and the psychotherapeutic models that ensue from these (Andersson & Ghaderi, 2006, p. 75) "as attempts to ensure independence from other approaches under test" (Roth & Fonagy, 2005, p. 495). So manuals outlining theory-based programs are then applied to purely descriptive, atheoretical diagnoses.

Also, "most clinical practice is much more heterogeneous than any manual would credit" (Roth & Fonagy, 2005, p. 495). D. Westen (2002) has claimed that highly specific symptoms explainable by narrow theory may benefit from treatment manualisation, but not complex generalized problems such as depression (p. 418). The fixed number of sessions specified by manualized therapy bears little resemblance to clinical practice (M.E. Addis et al., 1999).

Further, the manualized approach requires a new manual for every significant variation in presenting problem, such as cultural background, physical handicap, poverty, and sexual orientation (J.B. Persons, 2013, p. 451). Differing underlying mechanisms are likely in such cases. For example, D. Westen et al. (2004) have pointed out that it is unlikely that the same techniques will help a depressed person with acute feelings of inadequacy after a job loss, versus chronic feelings of inadequacy, versus unacknowledged homosexuality, or a background of childhood sexual abuse.

Research with blunderbuss treatment manuals gives little indication as to precise mechanism or effective ingredients for change, and "without the resulting knowledge of critical processes, treatment manuals are likely to include all sorts of ingredients that make little difference and underemphasize those that do" (A.E. Kazdin, 2001, p. 145). So when outcome studies of treatment packages fail to indicate exactly *what* has worked, the "theorist-practitioner gap" may grow even wider (J.B. Persons, 1991, p. 102).

3 | SUBSEQUENT LIMITATIONS OF EMPIRICALLY SUPPORTED THERAPIES

Empirically supported therapies or treatments (ESTs) as currently formulated are disorder-focused and diagnosis-based (J.B. Persons, 2013). In psychotherapy outcome research following the drug metaphor, the diagnostic selection of subjects and the treatment phase are separate processes, often undertaken by different people (T.D. Eells, 2013b). As a part of blinding, the results of the initial assessment may even be kept from the therapist (J.B. Persons, 1991, p. 100). There is no idiographic case conceptualization or tailored explanation of a subject's problems. Assessment is descriptive and atheoretical. Treatment is standardized and manualized. All subjects are viewed as having the same problem—the same mental disorder—differing only in severity.

So whether depression, for example, appears to be related to a decline in positive reinforcers, or to a distorted self-schema, or to a perception of helplessness, all subjects receive all components of the package (J.B. Persons, 1991, p. 101). "By definition, diagnostic homogeneity assures a uniformity of symptom presentation, but the problems that underpin patients' presentations [their CPPs] are likely to be diverse … Different life experiences can result in the same diagnostic end point … [and] exposure to the same event can lead to different diagnostic outcomes" (Roth & Fonagy, 2005, p. 487). These phenomena have been termed DSM's problems with multifinality (Cicchetti & Rogosch, 1996) and divergent trajectories (Nolen-Hoeksema & Watkins, 2011).

Such efficacy research is meant to test the effect of a treatment approach on levels of diagnostic symptoms in standard conditions, replicable across independent investigators (Lopez & Valdivia, 2007, p. 358). However, it barely tells us *what* works, let alone, *why* and *how*, which is what we actually need to be discovering at this tertiary stage of clinical psychological research (A.E. Kazdin, 2001). Despite masses of RCTs conducted along drug trial lines, precious little is known about the mechanisms through which ESTs work (Murphy et al., 2009).

The criteria for the establishment of ESTs include comparison of an experimental (treatment package) group with a wait list control group (Nasser, 2013). Among the many problems with such an overly lenient methodology is the admission of placebo effects, and the lack of control over irrelevant components in the package. Hence, "purple hat therapies" (Rosen & Davison, 2003) with irrelevant—even bizarre—distinctive components, such as eye movement desensitization and reprocessing (EMDR) (Arkowitz & Lilienfeld, 2012) and Emotional Freedom Techniques (EFT) G.M. Bakker (2013, 2014), can easily acquire EST status.

Clinicians frequently regard ESTs as having poor external validity to inform real-world practice (D. Westen et al., 2004). Therefore, it is hardly surprising that so many clinicians appear to ignore guidelines (Baker et al., 2008; Carroll & Rounsaville, 2008) produced through the assembly of efficacy studies by groups such as the Chambless Task Force (Chambless & Hollon, 1998).

When psychotherapy outcome research has been based on an inappropriate medical model of DSM-diagnosed mental disorders and a clunky and shallow drug metaphor, it is not surprising that the

results of comparative clinical trials have been unclear, inconsistent, or disappointing, and that the most prominent reviews of psychotherapy outcome conclude that "we should be cautious in interpreting and implementing the findings of research based exclusively on DSM" (Roth & Fonagy, 2005, p. 498).

Important comparative studies such as cognitive therapy versus behavioural activation for depression problems (e.g., Cuijpers et al., 2008; K.S. Dobson et al., 2008), or exposure therapy versus cognitive therapy for anxiety problems (e.g., Powers et al., 2008), have not produced clear guidelines. This is unsurprising, though, when subjects are DSM-diagnosed and then randomly allocated to groups, as though they all have the same CPP. This is an example of theory governing the nature of research (W.C. Follette & Houts, 1996). Clinicians rarely follow this model (J.B. Persons, 2008). They are more likely to administer several concurrent interventions, and to base this on the individual case formulation that has been developed, on the assumption that each intervention or homework is targeting a different CPP, or part of a CPP. If cognitive therapy and behavioural activation, for example, are treating different things—different depression-related CPPs—and if the subjects in an experimental group have undetermined amounts of these different things, then effectiveness will be randomly distributed.

This problem has been thoroughly outlined by Smith et al. (2009), who have pointed out that factor analytic and other studies indicate that depression, for example, is not a coherent, homogeneous psychological construct, and assessing the effect of cognitive therapy versus behavioural activation on a DSM-defined "Depression" group is an example of assessing the relationship of a construct with another multidimensional construct (such as PTSD or Neuroticism) which has *multiple* (diagnostic) criteria. The resultant composite correlation will be an average of the correlations with each of the dimensions or criteria, each of which could correlate quite weakly with the others.

"Psychotherapy process-outcome research's task is to establish what is therapeutic about psychotherapy. In general, its yield has been disappointing … despite great advances in methodological and conceptual sophistication" (W.B. Stiles & Shapiro, 1989, p. 521) because the "sophistication" has been within the wrong model. When the presence of a problem (in this case of a mental disorder) is defined by levels of some symptoms, and "cure" is defined by a reduction in these symptoms, then the therapist's goal is to make the patient feel, think, or behave differently, not necessarily to remove or alter the underlying psychological mechanism involved. Such an emphasis on symptom reduction "prevents clinicians and researchers from connecting the basic research on psychological processes to the development of psychological treatments" (Lopez & Valdivia, 2007, p. 349).

In place of developing a list of proprietary ESTs through DSM-based efficacy studies on lumps of manualized therapy, it is now clear that clinical psychological research and practice would progress immeasurably more by seeking "change principles with empirical support" (Rosen & Davison, 2003) or change strategies and theories (D. Westen et al., 2004) that can be applied within evidence-based and standardized clinical case formulations. A case formulation approach to psychotherapy outcome research would reflect underlying theory better than DSM diagnosis-based research does (J.B. Persons, 1991) and stands a better chance of finding differences between treatments in comparative outcome studies (A.E. Kazdin, 1986), because a case formulation or conceptualization is explicitly developed to "explain the origins of a problem, account for the maintenance of the current problem, and make predictions about prognosis, [and] prescribe treatment options" (Page et al., 2008, pp. 89–90).

"One can only speculate how fruitful psychological research would prove to be were decades of the financial and headspace resources devoted to biological research … available to psychology" (Miller, 2010, p. 738). In a similar vein, the development of clinical psychology's own problem taxonomy, relevant to case formulations, warrants an effort comparable to the millions of person-hours that have been devoted to the assembly of the DSMs over the decades (G.M. Bakker, 2019).

It is not RCT methodology that is at issue here. This is not an argument for reversion to tentative, hypothesis-generating, qualitative, or single-case research. Psychological theory is well advanced, and there is no substitute for the gold standard of the RCTs' tight experimental control, operational definitions, random assignment, precise measurement, and significance tests (Dozois, 2013). However, such methodology should be applied to the testing of theory-based, case formulation-identified mechanisms of change (Dozois, 2013; A.E. Kazdin, 2001) or intervention strategies and processes (D. Westen et al., 2004) rather than treatment packages thrown at symptom clusters. This can better show us *how* treatment works and thus better build on the psychological knowledge base (A.E. Kazdin, 2008).

The weakest link, however, in such an RCT methodology is standardization of assessment in the absence of moderately reliable (but clinically and theoretically irrelevant) symptom-based DSM diagnosis. No one has yet standardized the case formulations of CPPs to a comparable degree.

4 | THE BENEFITS OF FORMULATION AND TAILORING

It is an empirical question as to whether individualized case formulation-based treatment selection and tailoring will produce therapy outcomes potentially superior to those of standardized therapy packages applied to diagnosed subject groups, based on the claim that a formulation is explanatory, and not just descriptive (Zachar & Kendler, 2010). This can be tested.

Some empirical comparisons between manualized and individualized *behavioural* treatments have been undertaken, as, in behaviour therapy, a functional analysis is considered essential to the development of an intervention plan (S.N. Haynes & Williams, 2003). But at the same time as CBT case formulation arose out of the functional analyses of the behaviour therapists, DSM diagnosis was becoming dominant in psychotherapy outcome research designs. So cognitive-behavioural researchers have been slow to carry out similar studies (T.D. Eells, 2013b, p. 427; J.B. Persons, 1991, p. 103), likely due to difficulty in acquiring funding.

However, in the face of heavy pressure toward diagnostic uniformity in outcome research and against tailoring and case formulation, some research has been undertaken comparing the effectiveness of the two approaches. In these comparisons, case formulation approaches have been handicapped by the lack of uniformity and standardization of functional analyses and formulations—something that the new PMC-based conception and subsequent taxonomy is intended to remedy.

(So it is ironic that a PMC has formed holding back research on [PMC-inclusive] case formulation: studies of individualized case formulation and treatment have been very scarce, and when [even rarer] comparisons are made with standardized treatment protocols applied to diagnosed groups, tailored treatments are handicapped by a lack of standardization of formulations.)

But even before a reliable formulation-based taxonomy of CPPs has been disseminated, there are indications as to the overall benefits of tailoring treatments for individual clients according to an evidence-based behavioural functional analysis or CBT case formulation (A. Ghaderi, 2011).

For example, Jacobson et al. (1989) found that standardized research-structured marital therapy was less successful, though not immediately, but at 6 month follow-up, than a clinically flexible treatment. Schneider and Byrne (1987) found individualized training was superior, on one of two dependent variables, to standardized training in increasing cooperative play among children.

Treatment of self-injurious behaviour has benefited from the functional analysis of its underlying mechanisms of maintenance among adults (Iwata et al., 1994; Repp et al., 1988) and among children (Durand & Crimmins, 1988), as has depression treatment (McKnight et al., 1984), and children's disruptive behaviour (Carr & Durand, 1985). Kearney and Silverman (1999) found that in the treatment of school refusal behaviour outcome was better when therapy was based on an individual assessment producing a functional analytic model. They attributed this to the "heterogeneous population" involved (which can be interpreted as implying a variety of PMCs involved).

Results with CBT-based conceptualisations and treatments have been similar. For example, B.F. Chorpita and Weisz (2005) developed a Modular Approach to Therapy for Children with anxiety, depression, or conduct problems (MATCH) comprising a group of intervention modules guided by decision flowcharts which functioned as simplified and standardized case formulations. They subsequently found this program to be more effective than standard EST-guided treatment using evidence-based treatment manuals (J.R. Weisz et al., 2012). They concluded that "intervention procedures developed and tested over the decades in randomized controlled trials do have value for clinical practice but that a systematic restructuring of those procedures [a taxonomy] may enhance their benefits" (J.R. Weisz et al., 2012, p. 281). A 2-year follow-up by B.F. Chorpita et al. (2013), however, found no overall difference between modular care and standardized treatment.

A. Ghaderi (2006) also obtained better results treating bulimia patients with a flexible evidence-based CBT program than with a rigid manual-based one. J.B. Persons et al. (2006) achieved similar outcomes using a case formulation approach with mixed depressed and/or anxious patients in a private practice setting to those achieved in DSM-based RCTs, despite multiple comorbidities, multiple therapies, and unstandardized formulations.

Lundkvist-Houndoumadi et al. (2016) were able to help non-responders to a manualized group CBT program for anxious youths by moving them on to individualized family CBT.

B.C. Chu and Kendall (2009) discovered that therapist flexibility in selecting and administering CBT treatment processes increased client involvement and engagement, which in turn predicted improvement. Anderson and Strupp (1996) also found that the most effective clinicians were those that occasionally violated the manualized treatment guidelines. Hence, it has been suggested that idiographic case formulations may have most clinical utility with complex or severe problems (D. Dobson & Dobson, 2009, p. 16; T.D. Eells, 2013b, p. 439; J.B. Persons, 2013, p. 452).

Many clinicians would argue that almost *all* of their clients are "complex" in this way. For example, Johansson et al. (2012) found that individually tailored internet-based CBT for depression and related problems was more effective than standardized treatment among subjects with higher levels of depression at baseline and more comorbid problems. Vernmark et al. (2010), on the other hand, did not find such a difference between individualized and standardized. But tailoring internet-based therapy to individual clients has also been found to increase effectiveness when working with anxiety problems (Carlbring et al., 2011; Nordgren et al., 2014).

With substance abuse problems, Conrod et al. (2000) compared matching interventions to subjects' personality profiles with deliberately mismatching them. They found no difference between groups on follow up, but personality matching is a very crude form of case formulation, and outcome was assessed 6 months after only one 90-min therapy session.

This time with alcohol dependence problems, Litt et al. (2009) compared outcome after a personalized functional analysis led to tailored treatment to a manualised treatment. They found differences on four of six post-treatment measures. When S.N. Haynes et al. (1997) introduced a "functional analytic clinical case model" which visually represents variables and their functional relationships to summarize a functional analysis/case conceptualization, they reviewed single case studies showing improved outcome following functional analyses.

In parallel with such findings, Easden and Fletcher (2018) showed that case formulation competence predicted outcome for CBT. Therefore, any case formulation-based taxonomy of CPPs that standardizes formulations should support treatment outcomes even further.

Formulation-based therapy selection is more flexible and varied than diagnosis-based manualized therapy because many variations in formulation subsequent to a functional analysis are possible within the one diagnosis. So Rapee et al. (2009), for example, found that CBT designed to maximize improvement in identifying mediating cognitive processes in social phobia was more effective than standard CBT.

Effectiveness has been assessed, as well as efficacy. Several studies have found that when CBT therapists use case conceptualization

in the real world, outcomes in terms of effect size are similar to those produced in RCTs (W. Kuyken et al., 2001; J.B. Persons et al., 2006).

When treatment addresses processes rather than symptoms, as in the unified protocols of transdiagnostic CBT group treatments, outcomes have also been found to be as or more effective (P.M. McEvoy & Nathan, 2007). In general, transdiagnostic treatment protocols targeting common *maintaining* factors have been shown to work well, and to help with comorbid problems, and result in high levels of client satisfaction and therapeutic alliance (P.M. McEvoy et al., 2009).

Occasionally, manual-based treatment has produced comparable or superior results. For example, Schulte et al. (1992) found this with a group of subjects with specific phobias. A careful reanalysis of their results (Page et al., 2008) showed that the clinicians in Schulte et al.'s (1992) study who tailored their treatment had worse results *if they omitted exposure as a part of treatment*, suggesting that case formulation is superior if, and only if, empirically supported treatments are administered subsequent to an accurate reliable case formulation (Page et al., 2008). The need for standardization of such formulations—a taxonomy—is again implied. There remains a need to improve inter-rater reliability of case formulations (Macneil et al., 2012; J.B. Persons et al., 1995).

Another exception came from Emmelkamp et al. (1994), who compared the outcome of tailor-made cognitive behavioural therapy of 11 OCD subjects with standardized in vivo exposure therapy of 11 others. No significant differences resulted between the two groups. This finding is not surprising, though, if there is little variability of active PMCs among the 22 subjects, and the tailoring was therefore largely irrelevant, minimal, or superficial. In *Practical CBT* (G.M. Bakker, 2021), 23 Depression PMCs are illustrated, 12 Chronic Pain ones, 13 Alcohol abuse/dependence ones, and 21 Anxiety-related ones. But there is only one OCD PMC figure illustrating only two maintaining processes. They involve an over-reaction to an intrusive thought or urge which leads to recurrences and generalization of responses, and the reinforcing effect of tension reduction when a ritual is performed. These two PMCs correspond to the two most evidence-based effective treatment components in OCD treatment—cognitive therapy, and exposure and response prevention. Of course, details vary from individual to individual, but it is no surprise if tailoring general treatment selection to an individual with an OCD problem is not critical, as long as these two PMCs are addressed. It is recognized that simple categorical diagnosis is often enough for treatment selection with very homogeneous disorders (Macneil et al., 2012)—implying homogeneity as to the *PMCs* involved.

At the very least, any claim that manualization is necessary to guarantee efficacy in the light of guidelines and ESTs having been derived from diagnosis-based RCTs can be dismissed, as comprehensive reviews have not supported this; nor that manual adherence is necessary (Truijens et al., 2019). In examining manual adherence, Hauke et al. (2014) found that individualizing the treatment of Panic Disorder assisted in patients' motivation for exposure therapy and hence improved outcome. In the area of couple therapy, the best effects on marital distress have occurred in studies in which less structured protocols have been administered by more experienced therapists, with less pre-therapy training (Wright et al., 2007).

A recent review of many RCTs on the question (J.B. Persons & Hong, 2016), which also included several uncontrolled trials and six studies covering hundreds of single case studies, concluded that (even with unstandardized case formulations) overall, the RCTs show the outcome of treatment guided by a case formulation to be superior to standardized treatment.

Farmer and Nelson-Gray (1999) expounded "the need for a theoretically-based classification system to facilitate clinical science" (p. 390) and added that, on top of the clinical utility purposes of functional analytic classification, "Classification systems of behavior can provide a framework for testing hypotheses, serve as nomenclature or language base that defines the elements of a field or study, and provide a communication function that, among other functions, promotes research on clinical phenomena" (Farmer & Nelson-Gray, 1999, p. 390). In short, such a system can supplant all of DSM's current functions.

So, although attitudes to and the comparative results of case formulation approaches are mixed, the overall picture is positive and is yet to benefit from better standardization of, and more training toward, formulations (W. Kuyken et al., 2005; Mumma & Smith, 2001). Others have attempted this in the past. For example, W. Kuyken et al. (2008) have presented a model/metaphor of standardized case conceptualization, with an example illustrated by a PMC diagram of PTSD *maintenance* (fig. 2, p. 763).

5 | RESEARCHING WHAT CLINICIANS DO NOT DO

Should clinicians practise what researchers recommend, or should researchers study what clinicians practise? The consensus of philosophers of science is that both need to occur (Neale & Liebert, 1986). "We need more science in practice … We also need more practice in science" (Dozois, 2013, p. 8).

That this does not occur enough has been referred to as "the scientist-practitioner gap" (Dozois, 2013; T.D. Eells, 2013b; M.R. Goldfried, 2010; J.B. Persons, 1991, 2013). Currently, treatments are required to fit the needs of research methods rather than vice versa (Agras et al., 2000; D. Westen et al., 2004), and clinicians are criticized for ignoring the research (M.E. Addis et al., 1999; G.T. Wilson, 1998) and for resisting the adoption of ESTs into their practices (Baker et al., 2008; Carroll & Rounsaville, 2008; Strupp & Anderson, 1997; J.R. Weisz et al., 2012).

On the other hand, among the arguments advanced by clinicians for the irrelevance of research trials as they are currently devised is that the trials typically treat highly selected groups with a single diagnosis, while in clinical practice patients have many comorbidities and atypical symptom profiles (Clark et al., 1995; D. Dobson & Dobson, 2009, p. 3; J.B. Persons, 2008; D. Westen et al., 2004). Clinicians are more likely to apply evidence-based transdiagnostic or principle-based treatments, which allow more flexibility and hence

fidelity, than diagnosis-prescribed treatments (Kendall et al., 2008; McHugh, Murray, & Barlow, 2009). They also point out that doing therapy "by the book'" can affect the therapeutic alliance (Castonguay et al., 1996), result in unmet client needs, reduce treatment credibility (M.E. Addis et al., 1999), and include contraindicated or even harmful elements or lead to dropouts (J.B. Persons, 1991).

However, perhaps all of these mismatches reflect one common core difference: that practising clinicians are assessing and addressing CPPs, while most research examines diagnosed mental disorders. Almost all models of psychotherapy require therapists to devise and carry out an individualized treatment based on the results of an individualized assessment, but controlled outcome studies evaluate psychotherapies in which assessment is purely descriptive and atheoretical (J.B. Persons, 1991, p. 100), both assessment and treatment are standardized, and in which assessment and treatment are rigidly separated (J.B. Persons, 1991, p. 99; D. Westen et al., 2004). For many years, clinicians have argued that clinical trials of treatments should be based on individual case formulations (Tarrier & Calam, 2002).

Further, the disease model overemphasizes symptom reduction as the primary outcome measure, even though Chambless and Hollon (1998) cautioned against this (M.R. Goldfried & Wolfe, 1998, p. 145). Clinical psychologists in general do not aim for immediate symptom reduction. This is an appeasement policy and can interfere with exposure programs, grief work, exposure and response prevention, and other therapies that address *underlying mechanisms* rather than overt symptomatology. In fact, some effective treatments can even exacerbate symptomatology, at least in the short run. Management of children's temper tantrums would be an example of this.

There are many indications that the targets of practising clinical psychologists—CPPs—are not identical to the objects of most current research—mental disorders (G.M. Bakker, 2019).

First, 30–50% of all people who seek psychological help cannot be given a clear diagnosis because their problems do not fit criteria and categories neatly (Messer, 2001). They may be "subthreshold" (Howard et al., 1996), or "subclinical" (D. Westen et al., 2004), or situation-specific (such as being evidenced only at work). But they may evidence high levels of distress (Roth & Fonagy, 2005), and one or two symptoms may occur strongly, when three or more are required for a diagnosis (D. Dobson & Dobson, 2009). Most trials in clinical psychology exclude subthreshold presentations (Roth & Fonagy, 2005) despite a recognition of the arbitrariness of thresholds (Preisig et al., 2001; Sullivan et al., 1998), when the very conception of "mental disorder" is unclear (Frances et al., 1995), and despite the subsequent inevitable debates over the dangers of over-diagnosing and over-medicalizing versus missing problems or not preventing them (R.C. Kessler et al., 2003).

Limiting psychotherapy outcome research to DSM-based RCTs in order to develop ESTs has meant we have derived very few guidelines to help with adjustment disorders, dysthymia, NOS diagnoses (S.W. Stirman et al., 2003), subthreshold mental disorder symptoms, or diagnostic comorbidities (S.W. Stirman et al., 2005)—that is, for most presenting problems (Deacon, 2013).

A second indication that mental disorders and CPPs are very different things is the fact that relationship problems cannot be conceived as mental disorders (Sperry et al., 2005; D. Westen et al., 2004). In DSM, these are relegated to a few peripheral nonclinical "V" codes. But clinical psychologists address relationship problems with the same assessment tools and therapeutic models and methods as they apply to intrapersonal psychological problems (G.M. Bakker, 2019). Relationship problems are regarded as just another category of CPP.

Finally, whereas comorbidity rates are a conceptual and practical problem under the medical model, most clinical psychologists are trained, and expected, to work with a "Problem List" because most clients want help with multiple problems (D. Dobson & Dobson, 2009, p. 3), which may be interrelated, subthreshold, or include *inter*personal issues.

M.R. Goldfried (2000) has observed that when funding bodies require a focus on DSM-defined psychiatric conditions, this has dramatically decreased research on CPPs such as public speaking anxiety, interpersonal problems, and anxiety- and depression-related problems such as poor self-esteem.

However, perhaps the widest scientist-practitioner gap in clinical psychology lies not with research into therapeutic procedures and diagnosis-based ESTs, but with a massive under-focus on assessment processes (Hunsley & Mash, 2007), and an associated lack of research recognition of the ubiquity of case formulation (J.B. Persons, 2013, p. 448). Research on the "treatment utility of assessment" (S.C. Hayes et al., 1987) has been sparse ever since DSM diagnosis became the necessary and sufficient form of assessment.

In practice, most clinical psychologists do very little diagnosing (Carey & Pilgrim, 2010) but much case formulating (J.B. Persons & Tompkins, 1997) to determine treatment strategies (Roth & Fonagy, 2005, p. 61). The associated Problem List will often not even include a formal diagnosis (D. Dobson & Dobson, 2009, p. 40). Problems simple enough to be adequately served by a diagnosis-based EST model (D. Westen et al., 2004) tend to be rare in clinical practice (J.R. Weisz et al., 2012, p. 275). Psychotherapy outcome research that ignores or excludes the case formulation step then bears little relevance to clinical practice. "Practitioners report finding little in current psychotherapy research to guide them in moment-by-moment decisions they must make within sessions" (W.B. Stiles & Shapiro, 1989, p. 522). ESTs and treatment manuals do not "teach problem-solving processes to clinicians for treatment planning" (Beutler, 2000, p. 1002). Nor are theoretical implications readily derivable (A.E. Kazdin, 2008), as the feedback loop between empirical research and theory development is blunted (Neale & Liebert, 1986).

6 | RESEARCHING WHAT CLINICIANS ACTUALLY DO: THE ALTERNATIVES

6.1 | Epistemic iteration

Some have claimed that our understanding of mental disorders is in its infancy (Tucker, 1998), and rather than abandoning the model, we

should persist with the process of "epistemic iteration" (K.S. Kendler, 2009) and continue to research along current lines until all becomes clearer. But even large sectors of psychiatry itself have lost patience with this approach (Sanislow et al., 2010), noting that DSM-5 is proving to be no advance on DSM-IV in terms of the "epistemic blinders" and "administrative strictures" of its "fictive diagnostic categories" (Casey et al., 2013, p. 811). "If to obtain a grant or to publish a paper, one has to select study populations according to a system that is a poor mirror of nature, it is very hard to advance our understanding of psychiatric disorders" (Casey et al., 2013, p. 810).

Hence, the US National Institute for Mental Health's Research Domain Criteria (RDoC) project (NIMH, 2011) aims "to bring mental health research in line with other areas of medicine that base diagnostic symptoms on underlying biology and not just clinical presentation of symptoms" (B.C. Chu & Ehrenreich-May, 2014, p. 429). The lack of progress in psychiatric research has been attributed to DSM being based on patients' or clinicians' subjective impressions rather than objective measures, and the subsequent heterogeneous symptom profiles (Allsopp et al., 2019) mean that research on major depression, for example, uses subjects with different presentations, and these likely encompass a large number of biologically distinct entities (Casey et al., 2013). Clinical psychologists are even more concerned that these heterogeneous groups represent a large number of *psychologically* distinct entities (i.e., CPPs). What of CPPs that do not have a critical "underlying biology" but instead have a critical underlying *psychological* dysfunction?

The RDoC project is psychiatry's response to the crisis of DSM's inadequacy even for psychiatry itself, and even in respect to those problems more likely to be amenable to biological-level explanation and intervention, such as schizophrenia and bipolar affective disorder. It is a deliberate redoubling of the biological focus and insistence with regard to these disorders.

Therefore, two reasons that the RDoC project is an unsatisfactory response for clinical psychology are that (a) clinical psychology is much closer to uncovering the essence of CPPs than psychiatry is to defining or explaining mental disorders (G.M. Bakker, 2019). Forty years of endeavour since the publication of DSM-III has discovered not one biological marker for a mental disorder (Kupfer et al., 2002, p. xviii; Stein et al., 2010). But second, (b) "NIMH is clearly prioritizing biological, genetic, and neural processes over behavioural and interpersonal processes" (B.C. Chu & Ehrenreich-May, 2014, p. 430) as explanatory mechanisms. This is not clinical psychology. This is psychiatry. Such will not elucidate CPPs and their underlying psychological mechanisms.

6.2 | Transdiagnostic research

The transdiagnostic movement in clinical psychological research and treatment (Barlow, Allen, & Choate, 2004; Harvey et al., 2004; Kring & Sloan, 2010) has sought to identify fundamental processes underlying multiple, often comorbid, psychopathologies (Krueger, 1999; Nolen-Hoeksema & Watkins, 2011), and to advance transdiagnostic treatment modules that target processes rather than individual diagnoses (P.M. McEvoy et al., 2009). This movement has restored some research focus on psychological theory, processes, and subsequent treatment of CPPs (Mansell et al., 2009).

However, it has had limited success to date, especially because it has not *replaced* the medical model but rather has added a complicating dimensional layer to the assessment process (T.A. Brown & Barlow, 2009). It accepts fully the conception of CPPs as mental disorders and that the new DSM-5 "defines the diagnostic categories by which we organize multidisorder work" (B.C. Chu & Ehrenreich-May, 2014, p. 429).

Instead of producing (EST-like) guidelines such as "Treatment X can be helpful with Mental Disorder A," transdiagnostic research results in advice of the form "Transdiagnostic Treatment Y can be helpful with Mental Disorders B and C, when Transdiagnostic Process Z is involved in both." Such logic recognizes the critical relevance of Transdiagnostic Process Z (whether it be rumination, or avoidance, or neuroticism) and sees it as relevant to, but not equivalent to, or fully explaining, Mental Disorders B or C. Transdiagnostic research studies the "emotional dysregulation" (Gross, 2007) involved in mental disorders but does not offer an alternative conception of CPPs or define "emotional regulation" (ER) any more precisely than "mental disorder" is defined. "ER refers to attempts to change subjective experiential, cognitive, behavioural, or physiological emotional responses in oneself or others" (Payne et al., 2014, p. 393). It is a dimensional complication added to a categorical mental disorder model.

The step yet to be taken is to *define* CPPs in ER/psychological terms and to develop a subsequent taxonomy of such "dysregulations."

6.3 | Single case studies

Already, over 20 years ago, M.R. Goldfried and Wolfe (1998) proposed "a new outcome research paradigm that involves an active collaboration between researcher and practicing clinician" (M.R. Goldfried & Wolfe, 1996, p. 1007) that "individualizes the intervention on the basis of an initial assessment and case formulation" (p. 1013). They promoted the study of theoretically integrated treatments and recommended less of a focus on heterogeneous, dimensionalized, diagnosed clinical problems (M.R. Goldfried & Wolfe, 1998).

Unfortunately, in the absence of standardization of case formulation as an alternative to (psychologically heterogeneous) diagnoses, this recommendation can currently only result in single case designs, which represent a pre-DSM and pre-CBT stage of development in clinical psychological research (Hersen & Barlow, 1976), but are still promoted today in the light of DSM's failures (Arco, 2011) and the clinical irrelevance of ESTs (Stewart & Chambless, 2010). M.R. Goldfried and Wolfe (1998) themselves described the futility of comparing different treatments using random assignment of diagnosed subjects when diagnoses are heterogeneous with regard to specific CPPs (Allsopp et al., 2019), as when some depressed subjects may especially hold unrealistically self-critical views of themselves, while others may have experienced negative reactions from significant others in their life (M.R. Goldfried & Wolfe, 1998, pp. 145–146).

Different underlying psychological-level mechanisms within the diagnosed samples will represent different CPPs, likely responsive to different specific treatments. These need to be pared out by individualisation of case formulations.

But RCTs of treatments targeting standardized CPPs are much preferable to reversion to much weaker single case designs. In the balance required between individualization and standardization—and hence replicability (J.B. Persons, 1991)—of treatment, the sticking point to date has surrounded standardization of diagnoses versus of CPPs, that is, the use of a taxonomy of CPPs in the assessment phase.

6.4 | The case formulation approach

In response to the questionable external validity of traditional efficacy research, J.B. Persons (1991) and J.B. Persons & Silberschatz (1998) proposed a "case formulation approach" to psychotherapy research which requires the development of "assessment-plus-treatment protocols" based directly on the therapeutic model being assessed. In this approach, assessment and treatment planning are connected, individualized, theory-driven, and all conducted by the therapist. Critical elements of RCT methodology, such as random assignment of subjects to conditions, are retained. But the scientist-practitioner gap can be narrowed, and differential efficacy of different treatments for different CPPs is more likely to be detected (T.D. Eells, 2013b; J.B. Persons, 2013).

The case formulation research strategy is therefore neutral with regard to the theoretical orientation of the psychotherapy being studied (J.B. Persons, 1991, p. 99). Subjects qualify according to an assessment and formulation within any robust model of CPPs and subsequent psychotherapy approach. This enables much more naturalistic and powerful comparisons between therapies—even derived from different models or orientations.

Such an approach, unlike diagnosis-based research, allows theory-driven psychological assessment of problems and links assessment and treatment. But it currently involves individualization rather than standardization of interventions, and a "pragmatic approach to psychiatric assessment, allowing for recognition of individual experience" (Allsopp et al., 2019).

Therefore, significant limitations to this approach have been identified. The accuracy and reliability of case formulations is difficult to determine when no standardization of them—no taxonomy of CPPs—is available (J.B. Persons, 1991, p. 103). Formulations and the treatments that flow from them can be very idiosyncratic to a particular therapist (S.B. Messer, 1991; Schacht, 1991). Classical functional analysis involving behavioural principles, reinforcement technologies, stimulus control, and so forth can be complex, vague, and idiographic (S.C. Hayes et al., 1996, p. 1153). "The development of functional diagnostic categories, which would naturally emerge from the repetition of individual functional analyses" (Bissett & Hayes, 1999, p. 382) requires that functional analysis be sufficiently systematized first (Mace, 1994), but it has remained "neither specific nor replicable" (Bissett & Hayes, 1999, p. 381).

Individualization of formulation and treatment in outcome research bars meaningful generalization in terms of theory development or evidence-based treatment guidelines (S.L. Garfield, 1991). Standardization is necessary "to permit other investigators to identify similar patients or problems" (Herbert & Mueser, 1991), as DSM allows.

Therefore, J.B. Persons' (1991, 2013) approach retains diagnosis as one part of the formulation (T.D. Eells, 2013b, p. 430), recommends the use of ESTs derived by the conventional method (T.D. Eells, 2013b, p. 431), and sees case-formulation-guided therapy research as mostly single case research within a "mixed methods psychotherapy research agenda that includes an evidence-based case formulation approach as an addition to the randomised clinical trial" (T.D. Eells, 2013a, p. 458). "While the RCT is well suited to the goals of an efficacy study, the case formulation approach is well suited to the tasks of effectiveness and dissemination research" (T.D. Eells, 2013b, p. 436).

Once again, this involves a serious compromise due to the requirement to standardize and thus to select a homogeneous sample (A.E. Kazdin, 2001, p. 148). However, if RCT-level psychotherapy research is based instead on groups that are homogeneous with regard to a reliable taxonomy of CPPs, rather that psychologically heterogeneous diagnosed mental disorders, then it will have more clinical utility, more implications for psychological theory, be just as reliable, more valid, and be just as much about treatment *efficacy*. A reversion to single case studies will not be necessary.

Kazdin (2001, p. 146) has provided several examples of how RCTs based on case conceptualizations can tell us much more about mechanisms, moderators, and generality, as well as effectiveness. A case formulation approach will bring us much nearer to discovering "empirically supported principles" (ESPs) of change (Rosen & Davison, 2003), rather than ESTs. Unfortunately, "the biomedical approach to psychotherapy research is not intended to identify ESPs" (Deacon, 2013, p. 854). A case formulation-based research program seeking ESPs could systematically assess in vivo exposure for situational fears, imaginal exposure for mental stimuli such as traumatic memories or obsessional thoughts, and behavioural activation for depressive inertia (Deacon, 2013). Such theory-based RCT programming has been advocated since the rise of behaviour therapy (Eifert, 1996).

J.B. Persons (2013) has come close to this position when advocating for the development and study of smaller "kernels" of intervention (Embry & Biglan, 2008) taken from the complete manualised EST protocols of therapy—the "ears" (J.R. Weisz et al., 2011). What is needed to follow this through, however, is a system of standardization of the kernels of CPPs implicating those kernels of therapy.

7 | A NEW CONCEPTION AND TAXONOMY

If CPPs are not regarded as merely mental disorders, but rather as psychological-level processes or mechanisms, then what class of mechanisms can underlie and explain *all* CPPs?

A hint has come from Salkovskis (2002). He observed that RCTs of therapy packages guide treatment development less than do experimental studies of "factors likely to be involved in the *maintenance* of a specific disorder, symptoms and clinical presentations" (p. 6) [italics added]. He advocated the development of Empirically Grounded Clinical Interventions (parallel to Rosen & Davison's, 2003 Empirically Supported Principles of Change [ESPs]) so that "treatment *principles* are based on a scientifically framed and evaluated theory of *maintenance* of the specific problem" (italics added; note: "problem," not "diagnosis"). Assessment in this framework results in a case formulation which includes "the conceptualization of dysfunctional systems in the *maintenance* of clinical problems" (Tarrier & Calam, 2002, p.311; italics added).

The universality and critical role of maintenance mechanisms in CPPs involving problematic behaviours, cognitions, or affect, and hence in their treatment, can be seen in the fact that feedback or cyclic processes are ubiquitous in evidence-based models of every CPP. For example, Westbrook et al.'s (2011) *An Introduction to Cognitive Behaviour Therapy* illustrates a generic CPP model (p. 13), and problems with depression (p. 253), anxiety (p. 281), phobias (p. 295), panic (p. 297), health anxiety (p. 300), social anxiety (p. 302), GAD (p. 305), OCD (p. 308), PTSD (p. 311), eating disorders (p. 320), perfectionism (p. 321), and many more, *all* incorporating cyclic maintenance processes.

This universality and connection with treatment selection have resulted in an entire textbook (Abramowitz & Blakey, 2020) dealing with the maintenance processes in fear and anxiety problems and the treatment mechanisms they imply or indicate.

This ubiquity and treatment-relevance has led to the proposal (G.M. Bakker, 2019) that the critical, necessary, and sufficient difference between being anxious and experiencing an anxiety-related CPP warranting clinical psychological intervention, or between a chocolate binge and a bulimic CPP, or between a tiff and a relationship problem, is the formation and operation of any undesired, self-maintaining, psychological-level causal cycle that involves people's thoughts, feelings, behaviour, and situations.

This new conception, and a subsequent nascent taxonomy, of CPPs has recently been advanced (G.M. Bakker, 2008a, 2008b, 2019, 2021) as an attempt to offer the paradigm shift which has been called for (DCP, 2013) and to distinguish CPPs from psychiatric mental disorders. It conceives the defining essence of CPPs as residing in active, self-sustaining circles of psychological-level causal elements (thoughts, feelings, situations, or behaviours) that maintain undesired or negative psychological states, thoughts, attitudes, moods, sensitivities, behaviours, or habits. Several examples of these PMCs at the core of CPPs have been illustrated in Figure 1.

The PMC model of CPPs has emerged naturally from the confluence of (a) an empirically bolstered emphasis on problem *maintainers* over precipitants in clinical case formulation and treatment planning (G.M. Bakker, 2008a, 2008b, 2019, 2021); (b) the rise of transdiagnostic research, models, and treatments for CPPs (D.H. Barlow et al., 2004); and (c) research on network approaches to psychopathology which see disorders as resulting from the causal interplay between symptoms, possibly involving feedback loops, and how this model can explain the limited success of traditional research strategies (Borsboom & Cramer, 2013).

Case formulations, especially within the CBT tradition, are very often represented visually by a "functional analytic clinical case model"—a vector-graphic representation of critical variables and their functional relationships (S.N. Haynes et al., 1997). The generalized form of such diagrams, comprising elements connected by causal arrows, can be seen in the many evidence-based models of numerous CPPs to be found in CBT texts. Almost all of these visually presented models explicitly incorporate causal feedback loops or cycles, evidencing the core role that maintaining (vs. precipitating) factors play in the cognitive-behavioural understanding of CPPs (G.M. Bakker, 2019).

An evidence-based taxonomy of CPPs derived from these maintenance-focused, psychological-level models, and the large research effort that has produced them can list the smallest core causal loops that can exist alone in evidence-based models of psychopathology or in the individualized case formulations based upon them. They can be so listed under clinically useful and intuitive problem area headings, such as "Depression," "Anxiety," and "Relationship Problems." This taxonomy can guide and standardize the definition, detection, assessment, codification, and communication of CPPs.

Bakker (2019, appendix) has proposed a coding of PMCs in the form "PMC a.b.c," where a numeral in position "a" indicates the general problem area, such as (1) *anxiety* or (6) *chronic pain*; numeral "b" indicates the general type of maintenance mechanism, such as (1) *avoidance* or (4) *sleep disturbance*; and numeral "c" the specific maintenance mechanism, such as (1) *cognitive avoidance*, (2) *emotional avoidance*, or (3) *behavioural avoidance*. In the case of (4) *sleep disturbance*, the numeral at "c" could be 1 for low energy levels (maintaining a depression problem for example), or 2 for the effects of subsequent poor concentration from poor sleep, or 3 if nightmares are adding to general daytime distress.

For example, the PMCs illustrated in Figure 1 would code as (a) PMC 2.2.3 (a depression problem maintained by negative thinking, which may benefit from Cognitive Therapy); (b) PMC 6.1.3 (a persistent pain problem maintained by behavioural avoidance); (c) PMC 3.5.3 (a relationship problem reinforced by ongoing mutual dislike/disrespect.); and (d) PMC 13.6.1 (an interactive problem in which coping by using alcohol maintains a tendency to social anxiety).

It must be noted that (as discussed in G.M. Bakker, 2008a, 2019), while the specific target of an intervention is indicated by a discovered operating PMC, the *mode* of intervention is not strictly indicated. So, for example, a cognitive element such as negative thinking in a depression problem can be addressed through Cognitive Therapy, or at a behavioural, situational, or affective level of this is deemed more practical.

When presented in easier-to-understand graphic form, rather than tables of codes, part of a taxonomic listing of PMCs is exemplified in Figure 2. This is just one page of the Depression PMCs illustrated in G.M. Bakker (2008a, 2008b).

The conception of CPPs as instances of the operation of one or more psychological-level PMCs is, unlike the reified notion of mental disorders, a conception that is, for clinical psychology, empirically derived, theory-based, treatment-relevant, categorical (not just

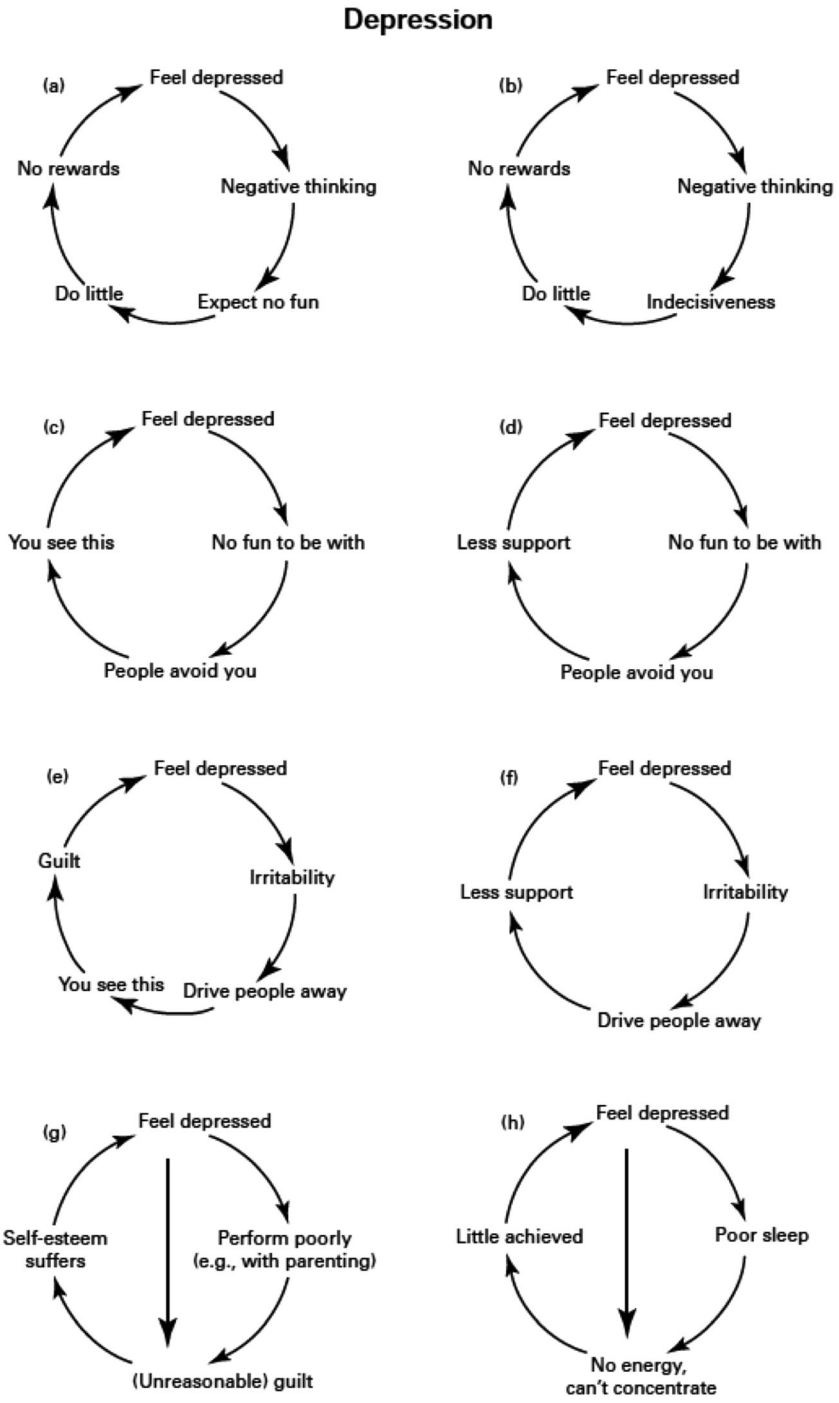

FIGURE 2 Sample of a depression PMCs taxonomic listing

dimensional), essentialist, parsimonious, and much less stigmatizing (G.M. Bakker, 2019). Treatment indications are directly implicated by the causal elements within such PMCs. For example, an Avoidance PMC (such as Figure 1b) suggests especially that some form of exposure treatment is warranted.

An embryonic taxonomy of CPPs, in the form of PMCs, has been developed by G.M. Bakker (2008a, 2008b, 2019, 2021) in the hope of a further development effort of some meaningful fraction of that devoted over the past half century to the various incarnations of DSM. Its clinical utility as an aid to clinical assessment, formulation, and therapy selection have been promoted (G.M. Bakker, 2019). However, the potential advantages of this conception and taxonomy in clinical psychological *research* and theory development are as great or greater.

8 | UPSETS, CLINICAL PSYCHOLOGICAL PROBLEMS, AND PSYCHIATRIC MENTAL DISORDERS

This new conception enables a distinction between mundane, transient, common, little-maintained (nonclinical) psychological problems (e.g., being upset) which do not involve or produce a PMC and do not

TABLE 1 "Kinds" of psychological problems

Type I General psychological problems	Type II Clinical psychological problems (CPPs)	Type III Psychiatric conditions ("mental disorders")
Normal "linear" psychological reactions	Persistent undesired psychological reactions	Diagnosable psychopathologic conditions
No PMCs identified (yet?)	PMCs have formed	Breaking psychological PMCs may not be enough. Biological PMCs may also be operating
Refer for counselling?	Refer for clinical psychological therapy	Refer for psychiatric (and psychological?) therapy
■ Grief reactions ■ Acute stress disorder ■ Anxiety states ■ Life event stress ■ Life transitions ■ Adjustment disorders ■ Personality difficulty ■ Burnout	■ Anxiety disorders (panic disorder, specific phobias, …) ■ Depression ■ Feeding or eating disorders ■ Obsessive–compulsive or related disorders ■ Sexual problems ■ Posttraumatic stress disorders ■ Relationship problems ■ Substance abuse/dependence ■ Gambling disorder ■ Sleep problems ■ Personality disorders ■ Prolonged grief disorder	■ Schizophrenia ■ Schizoaffective disorder ■ Bipolar affective disorder ■ Dementia ■ Delusional disorder ■ Autism disorder ■ Seasonal affective disorder ■ Post-viral depression ■ "Third-day blues" ■ Sleep–wake disorders

warrant intensive clinical intervention, and CPPs in which one or more PMCs have commenced to function (e.g., hypervigilance), and which are likely to perpetuate or even worsen (spiral) without clinical psychological intervention (see Table 1).

A further distinction can then also be made between mundane psychological-level problems, CPPs, and "psychiatric problems" (see Table 1), which may be usefully termed "mental disorders" (e.g., schizophrenia), and which current evidence suggests involve more than psychological-level PMCs, such that PMC-breaking will not effect a thorough "cure." It is assumed for now that such problems reflect an underlying dysfunctional *biological*-level mechanism and are likely to be eventually best addressed using biological-level treatments such as pharmacotherapy.

Table 1 calls these three categories of psychological problem Type I (general psychological problems), Type II (CPPs), and Type III (psychiatric conditions or "mental disorders") psychological problems and suggests that different types of response or intervention are usually implied or indicated. General psychological problems, such as the stress of life transitions or normal grieving, warrant at least supportive "counselling." CPPs by definition deserve clinical psychological therapeutic intervention. And mental disorders may often respond to psychiatric/psychopharmacological therapy as first-line treatment.

Of course, people and their problems are not always this simply and neatly categorical. There are at least two equally useful and valid criteria for allocating problems under Table 1's Types II and III headings, and they do not always align. They are as follows: (a) is the core dysfunction or PMC involved (the "pathology") at a psychological or a non-psychological level, such as at the biological level? And (b) is intervention at a non-psychological level (medication, ECT, light therapy, etc.) or a non-psychological level current best practice? Criterion (a) does not always determine criterion (b) for two reasons: it is well known that levels of analysis are intercausal and that biological interventions have psychological effects and that psychological interventions alter people's biology. For example, changes in the brain PET scan images of sufferers of OCD show the same effects after successful CBT treatment as after successful pharmacotherapy (Apostolova et al., 2010; O'Neill et al., 2013). Increases in testosterone levels can increase aggressive behaviour, but inducing aggressive behaviour can increase testosterone levels (Mazur & Booth, 1998). Childhood emotional maltreatment has been associated with a decrease in medial prefrontal cortex volume years later (van Hamelen et al., 2010). But in general and in the long run, it is reasonable to expect that biological level dysfunctions will most often be best addressed biologically and the psychological psychologically.

The second reason that level of dysfunction and level of intervention may not coincide is simply the stage of development of theory and intervention technology that we have currently achieved. For example, although organic factors are likely to be eventually discovered to underlie Premenstrual Dysphoric Disorder, it is currently better ameliorated by CBT (Blake et al., 1998; Christensen & Oei, 1995; Lustyk et al., 2009) than by pharmacotherapy (M.S. Hunter et al., 2002; Morse et al., 1991). The same is true for menopausal symptoms (M. Hunter, 2003; M.S. Hunter et al., 2004), and for Chronic Fatigue Syndrome (Burgess et al., 2012; Vercoulen et al., 1998; Wiborg et al., 2012).

Again, though, the distinction is a useful one, as it can guide us in the direction of our research in the future. For example, as neural mechanisms underlying OCD are uncovered (Albelda & Joel, 2012), it would be no surprise were it to move from the Type II column to Type III in the future. But for now, subsequent treatments are yet to ensue, and the level-of-treatment criterion puts it clearly still in the Type II domain. Even when its neurology is fully understood, it still may be most effectively treated at a psychological level.

Meantime, it is critical that clinicians distinguish clients who are diagnosable as suffering bipolar affective disorder, or a post-viral depression, or seasonal affective disorder, or "maternity blues" (Type III problems) from someone trapped in several psychological-level Depression PMCs (Type II problems), who the evidence indicates will be best helped with psychological-level interventions such as CBT or Interpersonal Psychotherapy.

9 | A NEW RESEARCH PARADIGM

Such an evidence-based taxonomy can provide an assemblage point for "kernels" of clinical problem (PMCs) derived from the research literature, which in turn can indicate kernels of therapeutic intervention and which can then be outcome-assessed on subject groups deemed to be experiencing those PMCs, rather than on groups defined by theoretically-heterogeneous diagnoses (Allsopp et al., 2019).

For example, one of the PMCs linking sleep disturbance with feelings of depression (for there are several possible reciprocal causal mechanisms connecting these phenomena) can be validated by undertaking a specified sleep improvement program with subjects assessed as experiencing this PMC, and noting any subsequent improvement in ongoing depression report. Hence, some degree of manualization (of kernels, not of comprehensive exhaustive therapy programs) would occur for the sake of standardization of intervention (such is exemplified in the homeworks in *Practical CBT*; G.M. Bakker, 2008b, 2021).

A full RCT design can be applied to such a test of an intervention's efficacy. Measurement and case formulation can be standardized, codified, and communicated. But subsequent EST guidelines will be PMC-based, not psychiatric mental disorder diagnosis-based. In fact, whereas EST-establishing RCTs have to date been regarded as the "gold standard" in research methodology, a more useful and more powerful research design—a "platinum standard"—has been identified in undertaking RCTs involving *mechanisms of action* (Bunge, 2013).

The essential difference between a diagnosis-based RCT research design and a PMC-based one in this example would be that, instead of subjects in the experimental and control groups being first formally diagnosed as suffering a Major Depressive Disorder (which would include people with undetermined heterogeneous degrees of aggravating sleep disturbance), they would all be assessed in interview by an experienced clinician as to whether they are currently enmeshed in one of the depression/sleep disturbance PMCs. For example, the study may seek to examine treatments targeting the following PMC: "Depression → Poor sleep → Reduced activity → Fewer pleasures and less social support → Depression."

As occurs naturalistically in real-world case formulation (most clinical psychologists spend more time case formulating than diagnosing), evidence of all elements of this PMC would be necessary for admission to the study, and perhaps timing of onset of each element may add weight to the evidence for its current causal relevance, for example, "Since I've been sleeping so badly, I've been too tired to drive out to my friend's place to visit him."

Then, the treatment group(s) can be administered an intervention that is known or suspected of addressing an element in this PMC—perhaps the sleep disturbance element. If only suspected to be efficacious, then comparison on the outcome (on Depression) between the treatment and the control groups can tell us whether this intervention can help depressed people by improving their (depression-affected) sleep. *This* is the critical clinical question for the clinician. Or, if known-effective treatments are applied, then having more than one treatment group may assess the relative efficacy of intervening in this PMC at different points, such as at the "Poor sleep" versus the "Reduced activity" element (via a form of behavioural activation, perhaps), or even at the "pleasures" or "social support" point of the PMC. Such designs can be as simple or complex as the PMCs being addressed. But they are orders of magnitude more clinically relevant and nuanced than current blunderbuss research designs with groups of mixed CPPs/PMCs being given intervention batteries.

Research into the treatment of CPPs in new areas, such as gambling problems, need not now be handicapped by the requirement to conceptually contort the field within a medical model by defining "symptoms" of a new "illness" or "mental disorder" called Gambling Disorder, and the development and assessment of a monolithic therapy package to cure this "sickness" (Dickerson, 1989; Hyman, 2010; Rosecrance, 1985).

Instead, fast PMCs (see G.M. Bakker, 2019, regarding "fast" and "slow" PMCs) such as the escalation of gambling behaviour by intermittent reinforcement through occasional wins, and slow PMCs such as the loss of alternative recreations and social activities through the neglect by the gambler of the rest of his life, can be addressed individually, subsequent to an assessment and case formulation tailored to finding each individual's active PMCs.

Treatments have been developed which putatively address both types of PMC. With the "fast PMC" described, cessation of the intermittent reinforcement may be attempted by a person formally self-barring from gambling venues, or through sessions of (known-effective) imaginal desensitization. If a problem gambler is assessed as significantly experiencing the "slow PMC" described as well or instead, then other interventions may be (researched and) attempted, such as lifestyle and social network changes. This would likely involve (re)developing alternative activities, involvements, recreations, and social supports. But it would most likely be effective with people assessed in case formulation as recently reduced in their alternative recreations.

How well these interventions can work, and with exactly which PMC-defined CPPs, will thus be much more finely addressed with correspondingly greater real world clinical relevance. For example, people experiencing problematic health anxiety or an illness phobia are sometimes entrapped in an avoidance PMC and have not seen their doctor in years. Others can be caught up in a hypervigilance PMC and be visiting a medical practitioner twice a week. The treatments to research in these two circumstances are not only different but are likely to be *opposite*.

Results of RCTs researching PMC-focused interventions such as these will have direct theoretical implications. They will relate to case formulations, which clinicians favour anyway and so will have more clinical utility. This can help narrow the scientist-practitioner gap. ESTs and similar guidelines will be extractable from meta-analyses, as results will be generalized due to standardization of assessments, formulations, and treatment implications, and because *efficacy* is being assessed. But these ESTs and treatment guidelines will be more consumable by clinicians.

Specific treatments will be more logically linked to problem processes (PMCs) rather than blindly, almost randomly, linked to mental disorders by purely empirical discovery, this being still prevalent in

psychiatric drug research, which is more often a trial and error process than one guided by biological theory.

Assessment and treatment will be intimately linked and blended, a phenomenon already embraced by clinicians. For example, it has already been noted that when case formulations guide treatment, *opposite* interventions can be indicated within the same diagnosis (J.B. Persons, 1991, p. 101), as can occur with problematic health anxiety. As described earlier, some are avoiders who can benefit from exposure treatment that includes, for example, periodic colonoscopies. Others are hypervigilant and need coaching in thought-stopping and distraction (G.M. Bakker, 2008b, 2009, 2021). Their case formulations will indicate whether an avoidance PMC and/or a hypervigilance or excessive checking PMC is operating. Then, an RCT can assess the efficacy of Treatment X (e.g., thought stopping training) on PMC Y (e.g., hypervigilant illness phobia) with sufficient standardization *and* individualisation.

And finally, because both constructs—the problem and the therapy package—will be less multidimensional (Smith et al., 2009), therefore, the chances of discovering differential efficacy between treatments for specified problems (PMCs) will be greater than now occurs with psychiatric diagnoses, which are heterogeneous with respect to underlying psychological mechanism, and with therapy packages which do not attempt to target specific CPPs.

The most obvious drawback of PMC-based psychotherapy outcome research on the treatment of CPPs as compared to psychiatric diagnosis-based research on the treatment of mental disorders is that current DSM-based diagnosis is at least moderately reliable (if not acceptably valid) in some psychological problem areas. Clinical case formulation has been to date much more idiosyncratic and variable (Bissett & Hayes, 1999; S.C. Hayes et al., 1996). This observation bears all the more on the need, especially in research contexts, for the increased reliability that a PMC-based taxonomy of CPPs can offer.

CONFLICT OF INTEREST

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