

## STEPS TOWARD AN EVOLUTIONARY-BASED THEORY OF PSYCHOTHERAPY - II

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### Summary

This paper discusses a falsifiable evolutionary-based hypothesis of psychotherapy and intervention techniques and measures which might be used to test the hypothesis. The hypothesis is: psychotherapy increases the frequency of reciprocal exchanges. Particular attention is paid to the topics of hypothesis clarity and simplicity, measurement options and complications, and the consistency of the hypothesis with evolutionary theory.

It is concluded that the hypothesis can be tested and falsified and that such testing might contribute to the development of other falsifiable hypotheses.

**Key words:** Psychotherapy – Evolutionary Theory – Reciprocal Exchanges – Falsifiable Hypotheses – Hypothesis Measurement

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**Declaration of interest:** none

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### Introduction

In a companion thought paper (McGuire and Troisi 2006a) we discussed issues relating to developing an evolutionary-based theory of psychotherapy and clearly falsifiable hypotheses that are consistent with evolutionary concepts. There have been previous efforts towards this end (e.g., Sulloway 1985, Slavin and Kreigman 1992, Gilbert and Bailey 2000, Bailey 2000, Gilbert et al. 2000, Troisi and McGuire 2000). On balance, they are consistent with the direction taken here. Still, as yet, there is no compelling, robust theory or hypotheses. We believe there should be some, even many.

From this state of affairs three outcomes seem clear. At present, “It is impossible to specify a particular psychotherapy that we can call evolutionary...” (Gilbert, this volume). Evolutionary-based hypotheses are tested only sporadically. There is minimal consensus among therapists and researchers regarding which intervention or outcome variables should be studied and the methods for studying them (Bower 2005). The same lack of consensus is present regarding what therapy does and how it works. Nonetheless, there are reasonable points from which to start. One is that the aim of psychotherapy is to bring about change. Thus what can change and what does change are important. Another is that some variables are more easily measured than

others. For example, if a patient interrupts a therapist whenever the therapist starts to talk the behavior is measurable. However, if a patient reports that he is more empathetic toward others as a result of treatment, measurement is more difficult. Thus identifying which variables can be measured with reasonable accuracy is important.

*Theory and hypotheses.* This brings us to theory and hypotheses. A good theory leads to falsifiable hypotheses. And the more easily and the more clearly hypotheses can be falsified the better the theory and the better the hypotheses. Without the possibility of falsification, theories and hypotheses are only explanations. Explanations may be correct – Freud’s explanation of repression or Jung’s concept of archetypes for example – yet if they can’t be falsified they are only explanations and, often, subject to endless revisions which are no more falsifiable than their original versions. A likely reason for the decline in the influence of psychoanalytic theory is that its emphasis on difficult to measure intervening processes has made its hypotheses exceedingly hard to falsify. The same point applies to evolutionary psychology theory. Its hypotheses are formulated in ways that are not easily tested and at times conflict with traditional ethological and evolutionary concepts (see Feerman, this volume).

Adaptationist explanations of DSM (American Psychiatric Association, 1994) classified disorders invite

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similar comments. For example, despite the many attempts (ourselves included) to explain depression as an evolutionary adaptation (e.g., withdrawal from painful situations, attract support from others, etc. – see McGuire and Troisi 1998 for a review), at best, such explanations are consistent with evolutionary concepts. Yet their evolutionary relevance hasn't been demonstrated. Relevance is important. For example, there is little in evolutionary biology which suggests that one should first experience anxiety well after a frightening event or depression well after a loss or have bizarre ideas about imaginary devils. Many explanations have been offered to explain these happenings. But they seldom lead to predictions which can be tested compellingly. And there is a critical difference between taking an empirically established correlation such as "loss is followed by depression" and calling it a hypothesis compared to developing a hypothesis which predicts something that has not yet happened and then testing it to see if it does happen using a replicable methodology. This said, findings and conclusions from many studies – often ingeniously designed and interpreted – remain consistent with predictions from evolutionary theory (e.g., Daily and Wilson 1988, Allen and Badcock 2003, Gilbert and Allan 1988) and this consistency not only makes them difficult to ignore but also provides a ray of hope.

**Measurement.** What to measure is a perennially vexing problem in psychotherapy research often because what seems most relevant to measure can't be measured either easily or convincingly. It is our view that the least ambiguous outcome measure that is consistent with evolutionary theory is change in functionally defined behavior – hereafter called "functional behavior(s)". But even here there are potential complications. Functional behaviors (often discussed as "life history strategies") are age- and sex-specific. So a critical point concerns which functional behaviors psychotherapy might try to change. For example, attempts to increase the frequency of kin-investment in adults is a more relevant therapeutic objective than it is with a 4-year old. So too with survival-related behavior. Efforts to change this behavior could be a relevant therapeutic objective among risk-taking male teenagers but largely irrelevant among 85 year old great grandmothers who are physically well and aware of what is required to survive.

Further, there needs to be consequences from not enacting functional behaviors or enacting them in a compromised way. A mother of eight healthy children who reports she is no longer motivated to reproduce presents a very different intervention problem than a 25 year old single female who reports she is frightened of pregnancy. A word of caution here. To say that evolutionary-based psychotherapy should focus on changing functional behavior is not the same as suggesting that therapists should try to 'mold' patients into persons who more closely map to evolutionary models of average evolved age-sex behavior. Change in functional behaviors is relevant when there are undesirable consequences from such behaviors and patients want to alter the consequences. When these criteria are met, constraints, traits, brain quirks, and individual differences again enter the picture – not every 25 year old female who has not reproduced is suffering consequences.

A good example of a functional behavior is reciprocal exchange. Patients often report that they have difficulty developing and or maintaining these exchanges. The behavior is consistent with evolutionary concepts (Trivers 1971) and the idea that human beings seek to achieve biological goals (McGuire and Troisi 1998). Thus, for those patients who report undesirable consequences from their reciprocal exchanges and who seek change, an aim of therapy as well as a measure of its effectiveness might be an increase in the frequency of exchanges. Reciprocal exchanges have the distinct advantage that they can be measured behaviorally and in multiple other ways.

Still, there are potential complications. A behavior that appears to be easy to measure may be more difficult to measure precisely than imagined initially. Despite the many discussions in both the evolutionary and psychiatric literature about reciprocal exchanges – for example, computer simulations and the use of research subjects in tests of tit-for-tat behavior (e.g., King-Casas et. al. 2005), detailed reports of the frequency and function of reciprocation among normal and psychiatric populations (e.g., Essock-Vitale and McGuire 1985a, b; McGuire et. al. 1994), and theoretical formulations about the evolution of reciprocation (e.g., Nowak and Sigmund 2005) — what is involved in these exchanges is often far from simple. An example illustrates this point.

C asks D, a next door neighbor, to feed his dogs while he is away from home for two weeks. D agrees to do so and the dogs are fed. C returns home and has incurred a 'reciprocal debt' to D. If a few days later D asks C to purchase an ear of corn when D goes to the market and D does so, both C and D 'understand' that C's reciprocal debt has not been repaid in full. On the other hand, if D asks C to spend a day helping him clear brush from his yard and C does so, both C and D are likely to consider the debt repaid. Such exchanges are difficult to measure because of differences in the exchange "currency" (e.g., dog care versus brush cleaning).

Such exchanges raise a host of questions and it is tempting to try to answer them. For example, how do people go through the 'calculations' that determine the costs and benefits of exchanges especially when they involve different behaviors, time, expertise, life styles, ages and sex, personal preferences, outcomes, and not cash (where calculations are relatively straightforward)? Is the amount of time between a favor and its repayment important and part of the calculation? Does requested repayment differ from volunteered repayment? Are there evolved computational systems which can handle different currencies such as Tooby and Cosmides (2005) have postulated — that is, much like algebra where rules applying to the "Xs" and "Ys" remain unchanged even though their referents often change. An interesting possibility but one that is likely to elude clear falsification.

The preceding discussion suggests several conclusions. One is that real-life reciprocation goes on much like the examples above, only occasionally like the tit-for-tat demonstrations in research projects. Another is that even seemingly straightforward measures are often not as straightforward as they appear at first. A third is that explanations, however interesting and plausi-

ble, may elude measurement and falsification – a correlate of this point is that without something tangible to measure a theory of therapy is benign and lacks utility.

*Measurement complications.* There are however two far more critical measurement issues than those just discussed. First, among patients who experienced difficulties in reciprocal exchanges and wish to increase their frequency there will be individual differences in therapy's starting point, what is desired from therapy, and what is taken as an indication of intervention effectiveness. If patient S has two reciprocal exchanges per month prior to therapy and patient T has 12, they commence therapy at different starting points. In principle this need not be a problem if the measure of intervention effectiveness is limited to an increase in frequency. Similar arguments apply to what is desired – patients will differ – and what constitutes an effective intervention.

Second, a more critical issue is this: what are the criteria for distinguishing between compromised reciprocal exchange behavior that is a consequence of selection and/or individual variation and the same compromised behavior that is a consequence of adverse experiences or secondary to an illness? The clinical phenotypes may be similar, perhaps indistinguishable in some instances. This distinction is important because compromised behavior due to selection effects or secondary to an illness presupposes different intervention tactics – for example for compromised behavior that is secondary to an illness, treating the illness would be the obvious treatment target but not a target for selection-based compromised behavior.

We don't have a sure answer to the selection versus secondary-to-an-illness question. But we do have a suggestion. If a patient has a life-long history of reciprocal exchange difficulties, has not suffered from adverse experiences during development, and is otherwise free of somatic illnesses or illness features such as signs and symptoms that might interact with exchange behavior, a reasonable assumption is that the compromised behavior is a consequence of selection. Only a very small percentage of patients seeking therapy would likely meet these criteria. But this need not be a drawback if the aim is to develop and test a hypothesis.

Selecting patients for an intervention study who meet the criteria above thus might be a wise methodological step. This done, the issue of "severity" needs to be addressed. Twelve reciprocal exchanges per month is different than two exchanges per month. Again, this issue is manageable. What is required are accurate measures of the frequency of pre-therapy and post-therapy exchanges. Then, for example, if psychotherapy increases the frequency of exchange only among those patients whose pre-therapy frequency rate per month is six or more, two subpopulations of patients with generally similar phenotypes but with different responses to treatment will have been identified. The hypothesis would not have been falsified for the six-or-more exchanges group but it would have been for the less-than-six exchanges group.

*The medical and economic models – examples of what is needed.* Consider the differences in clarity when trying to explain a reduction in patients' chronic anxiety in response to the administration of a drug when

compared to the same reduction in response to psychotherapy. In the drug intervention the target of treatment (anxiety) is known, the intervention (a chemical) is known and standardized, and what is to be measured (reduction in anxiety) is known. In psychotherapy, the target of treatment (anxiety) is known and what is to be measured (reduction in anxiety) also is known. But because of differences in therapists' techniques, the intervention is difficult to standardize. Whatever one's views about the use of the "medical model" as a methodology for assessing intervention effects, research conducted using this model has the virtue of being falsifiable. For example, if drug X is predicted to reduce a specific symptom but only 10% of those individuals receiving the drug show clinical improvement the prediction is falsified. There could be many reasons for this outcome – for example, patients might differ physiologically despite having the same symptom. Further research might clarify if the symptom reduction hypothesis is relevant to a subpopulation of patients. But further research is unlikely unless a falsifiable hypothesis is tested and it produces findings which generated new research questions.

Economic models provide another example of what we are recommending. One of its models predicts that (all else equal) a reduction in the cost of an item will result in increased sales of an item. It is a simple and straightforward prediction: if A happens B will follow. It is tested daily (e.g., sales at stores) without the requirement of knowing much about the intervening events – indeed, without necessarily postulating them.

There is nothing original in these examples or our recommendations. They are already a part of evidence-based medicine. In effect, outcome research can be conducted without knowledge of intervening events or mechanisms. Again, consider an example. If drugs A and B are both postulated to reduce high blood levels of cholesterol and drug A does so 80% of the time while drug B does so only 20% of the time, what can one surmise from these findings? The obvious answer is that drug A is more effective at reducing blood levels of cholesterol compared to drug B. However, nothing certain is known about the mechanism(s) by which drug A works although such findings may provide suggestive hints particularly if drugs A and B work by different physiological mechanisms. Yet even here one must be cautious because known mechanisms might not be the mechanisms responsible for change. This has been the prevailing story of neurotransmitter research: what at first seemed to be a primary mechanism later turned out to be a secondary mechanism or even an irrelevant mechanism. While such research would not clarify possible etiological factors, at least one finding would be established: there is a strong positive correlation between the administration of drug A and the decline in cholesterol. This finding might then serve as a spring board for further research.

*Measuring reciprocal exchanges.* How might reciprocal exchanges be measured? In our view, behavioral evidence (e.g., direct observation) is superior to subjective reports and what elsewhere we have called "indirect assessment methods" (e.g., paper-and-pencil tests) (McGuire & Troisi 2006a). Subjective reports and indirect assessments are likely to be influenced by brain quirks, the tangled interpretation of

stimuli, deception, etc. (McGuire & Troisi 2006a). But in nearly all instances of therapy direct observation, except within therapy sessions, is difficult. Moreover for many patients observations outside of therapy sessions would be viewed as “unethical” and/or as infringements on their privacy. These factors are not insurmountable. Measures that are likely to be approximately accurate indicators of reciprocal exchanges include the number of others requesting favors per unit time and the number of favors done for others per unit time. Such measures are not ideal but they are measures and they are far less subjective than, say, reports about feelings.

*What interventions might work?* What might therapists do to increase the frequency of reciprocal exchanges? The list is long and includes such therapist behaviors as clarifying patients’ biological goals and the tactics used to achieve goals, instructing patients how to read others’ signals and to recognize the impact of their behavior on others, assisting patients in developing realistic views of themselves and others, instructing patients on ways to satisfy themselves, clarifying how emotions influence the way one thinks, feels, and behaves, etc.

Therapist techniques differ and often significantly. Such differences may be critically or minimally important for therapeutic outcome – we suspect that they are important or at least it should be assumed that they are until proven otherwise. Yet it doesn’t follow that these differences need to be considered when studying if therapy improves reciprocal exchanges. All possible outcome influencing factors can’t be studied if a clearly falsifiable hypothesis is to be developed and tested – recall that economic hypotheses can be tested without consideration of all possible variables – for example, stores selling clothes and others are selling food – or even measuring the same variables. Further, attempts to take account of the many possible influencing variables can easily send research into a tailspin of complexity and uncertainty and lead to idiosyncratic conclusions. Adopting a far simpler model is likely to be the better choice as a first research approach. Thus, therapist techniques along with other possible influencing factors are probably best disregarded initially. If it should turn out that 85% of therapist A’s patients increase reciprocal exchanges while only 15% of therapist B’s patients do so, therapist techniques take on greater theoretical interest and would find their way into a revised theory and new hypotheses.

*An explanatory digression.* At this point a question might be: how can the authors be so unconcerned about possible differences in therapist techniques? The quick answer to this question is that we are uncertain about the effects of different techniques, this despite the fact that therapists often identify their techniques as “supportive,” “psychodynamic,” “cognitive,” etc. The more salient answer is that differences in techniques may be far less important than the fact that therapists interact with patients in positive ways which most therapists appear to do. What is involved here is discussed in detail elsewhere (McGuire and Troisi 1987) in regulation-deregulation theory. A key hypothesis of this theory is that positive social feedback (e.g., attentive listening, responding to the content of what patients say, appropriate praise, etc.) alters peoples’ brain neurotransmitter profiles. In turn, emotions, cognition, and

behavior change. From this perspective, if therapists provide positive social feedback, even though they do so differently, it may be far more outcome-relevant than how they describe their techniques.

We are not suggesting that positive social feedback changes compromised functional behavior – this seems unlikely – only that therapist differences in technique might not be as important as often postulated. Changing selection-based functional behaviors is likely to require very specific intervention tactics and skills which remain to be specified.

## Discussion

A reasonable criticism of our suggestions is that they ‘put the cart before the horse’. Wouldn’t it be more reasonable to try and precisely characterize patients in terms of evolved constraints, traits, quirks, functional behaviors, etc. before studying intervention effects? Or, wouldn’t it be more reasonable to clarify what therapists do – i.e., precisely define the tactics and details of interventions – than to disregard therapist differences? Ideally, the answer to both of these questions is yes. But research must start somewhere and rarely does it do so by accounting to all possible influencing factors.

What might be the research outcomes of our recommendations? Two examples are considered.

*Example 1.* One-hundred patients report that they have difficulty with reciprocal exchanges — e.g., that they occur infrequently, those that occur are not gratifying, and that they seek to increase the frequency and improve the quality of their exchanges – and these patients meet the previously mentioned criteria for compromised exchange behavior that is likely due to selection effects. The frequency of exchanges is measured prior to therapy. An evolutionary-based theory of psychotherapy predicts that psychotherapy will increase the frequency of reciprocal exchanges. Therapy is provided. Say an increase in the frequency of exchanges occurs only among 15 patients. The theory has been falsified – time for a new theory and/or new hypotheses and/or for new research into the reasons why the theory fails to predict accurately. The reported ungratifying quality of exchanges might best be disregarded initially as an outcome measure because its introduction could complicate the interpretation of findings — for example, what if therapy leads to a decrease in the frequency of reciprocal exchanges but the quality of those that occur improves? Quality, not frequency, could be studied, but that would require another type of research. There are of course many possible reasons for a 15% outcome. And given this low outcome percentage they might now take on greater interest. Are capacities for reciprocal exchanges far more resistant to change than normally assumed? Is psychotherapy irrelevant to bringing about such changes? Etc.

*Example 2.* Say 90 of the 100 patients in Example 1 show an increased frequency in their reciprocal exchanges following therapy. Such a finding would be consistent with the hypothesis but would not prove it, only establish that the hypothesis has not been falsi-



fied. The study would require replication studies. And, if replicated, it would suggest, for example, that differences in therapists' techniques minimally influence outcome – something else is causing change.

*Final comments.* It seems likely that the “simplicity” of our recommendations may lead some readers to feel that we have failed to address many of the complexities of evolutionary biology and psychotherapy. We agree. We have not done so. In our view there is a trade-off: if the aim is to develop falsifiable hypotheses, initially complexity may need to be sacrificed in favor of simplicity.

There are clear limitations to what we have suggested. We have used a very narrow definition of therapy. This definition was not adopted to be restrictive. For example, we are not opposed to therapists attempting to reduce signs and symptoms which distress patients — this will go on anyway if only because patients will insist on it. But from the perspective developed here such reductions need to make sense in an evolutionary framework otherwise they are best understood in an alternative conceptual framework. There are also potential conflicts with culture. Antisocial Personality Disorder provides an example. From an evolutionary perspective it may be an evolved alternative strategy. From a cultural perspective it may be a behavior which needs to be changed.

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