Coercion Justified?-Evaluating the Training in Community Living Model (The Original Assertive Community Treatment Model) A Dissertation

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B.S. (New York University) 1970
M.S.W. (New York University) 1986

A dissertation submitted in partial satisfaction of the requirements for the degree of

Doctor of Philosophy

in

Social Welfare

in the

GRADUATE DIVISION

of the

UNIVERSITY OF CALIFORNIA, BERKELEY

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Spring 1998
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Abstract


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This dissertation examines the research and theory offered for Programs of Assertive Community Treatment, the model that is supported by Institutional Psychiatry as the most well validated and best model of intervention applicable to the psychiatric population labeled the Severely and Persistently Mentally Ill.

Although this program has been researched for over 25 yrs. and the extensive literature on this model claims to have established it’s efficacy on both the systems and patient level, the findings of my critical review dispute these claims based on the examination of the empirical evidence of all available randomized controlled trials of this intervention as well as through a close conceptual analysis of the theoretical framework and a situational analysis of the individuals who invented this model.

I explicate the faulty view of science (Justification) which drives the findings, by applying a view I argue is more correct (K. Popper’s Fallibilism). My analysis demonstrates that no superior efficacy can be associated with PACT methodology when compared to no treatment or standard treatment control groups. Further, any statistically significant impact putatively favoring PACT is either a tautological outcome based on administrative rules differentially applied to PACT and CONTROL groups, or are misattributions of worker activity as client outcome, or are based only on data supporting various outcomes and the ignoring or minimizing of negative results which contradict such claims, or are based on manipulation of data to indicate significance for variables which are not supported by the data (by for example collapsing various outcome variables some of which are statistically significant, but are tautological, like number of
hospital stays, and some which are not statistically significant but empirically important like less homelessness, or less time spent incarcerated, and suggesting that the significance found (derived from the tautological components) indicates treatment effectiveness for the non tautological components).

Finally the conceptual analysis of this model demonstrates that this model is coercive and may lead to harm (excessive suicide among its treatment population for example).
To my life partner, Francine M. Gomory, without whom this dissertation could not have been attempted much less completed.
Acknowledgements

Introduction

Chapter 1
Conceptual Critique of Mental Illness

Chapter 2
TCL treatment population and the meaning of Community Mental Health

Chapter 3
The Historical Roots of TCL or The Archeology of Coercion

Chapter 4
The Training In Community Living Model

Chapter 5
A Description and Critique of The Three TCL Studies

Chapter 6
The Replications: Programs of Assertive Community Treatment

Chapter 7
Concluding Comments

Appendix A
Acknowledgements

An individual intellectual effort entails the complicity of many.

First, I owe an immeasurable debt of gratitude to my parents, Ilona and Sandor, who had the foresight to emigrate and provide for their two children, George and Tomi, so that they both could thrive.

Second, I would like to thank my dissertation committee for their support and scholarly rigor.

Third, Dr. S. P. Segal has been a careful intellectual critic and a colleague, holding me to standards that have forced me to think and question my most closely held convictions and as a result immeasurably improve my work.

Fourth, to Dr. Eileen Gambrill, I want to say that I have received from her not only the most selfless support for my work, but also found in her a role model of what a truly ethical, deep thinking scholar and educator can be like, for that and her friendship I am eternally grateful.

Last, I came to realize fairly late in my early adulthood that I possessed some intellectual skill and critical acuity. This realization led to some individuals whose work has made my scholarly life absolutely vibrant ever since. They are; K. Popper, F. A. Hayek, E. Gellner, and A. Schopenhaur.
Introduction

“... the conjecture that Thales actively encouraged criticism in his pupils would explain the fact that the critical attitude towards the master’s doctrine became part of the Ionian school tradition. ... It was a momentous innovation. It meant a break with the dogmatic tradition which permits only one school doctrine, and the introduction in its place of a tradition that admits a plurality of doctrines which all try to approach the truth by means of critical discussion.

It thus leads, almost by necessity, to the realization that our attempts to see and to find the truth are not final, but open to improvement; that our knowledge, our doctrine is conjectural; that it consists of guesses, of hypotheses, rather than of final and certain truths; and that criticism and critical discussion are our only means of getting nearer to the truth. It thus leads to the tradition of bold conjectures and of free criticism, the tradition which created the rational or scientific attitude, and with it our Western civilization, the only civilization which is based upon science(though of course not upon science alone).”(Popper, 1962, p.151)

Karl Popper in his usual concise, and crisp language depicts the venerable trial and error method of critical reasoning by which science proceeds. This is the formulation of daring falsifiable conjectures tested by severe and ingenious efforts at their refutation(Popper, 1979, p.81) -It's history can be traced back at least 2400 years to Presocratic Greek philosophers such as; Anaximander, Pythagoras, Xenophanes, Heraclitus, and Parmenides(Popper, 1962, pp.136-165).

This well regarded if controversial approach to science plays little if any role in the development of social work ideas as reflected in the development of social welfare policy and the various ‘scientific’ intervention programs created by the ‘helping’ professions to meet the identified policy agendas. This in part, may be explained by the politically correct although false assumption, that as social workers, in an effort to maintain both our ‘empathic’ supportive professional
stance toward our clients, and the good will of our fellow social work professionals, we should not critique clients’, or colleagues’ behavior too closely, by, for example, asking them to be responsible for it, or, to not too directly point out empirical problems with professionally accepted intervention methods which rely on sacred guild myths like practice wisdom (Klein and Bloom, 1995) rather than scientific rigor. In short social agreement and professional authority appear to be prized above the truth.

There is further, in what used to be called the moral sciences a heated debate between two very different views about the role of science and the nature of knowledge. One view, best represented by the post-modern or hermeneutic school, claims that science ought to reach for understanding, to come to accept all ‘forms of life’ regardless of the nature of the beliefs of various communities or how much these beliefs may conflict with our own. This attitude argues for example, for accepting ritual mutilation of the female clitoris among certain groups in Africa and stand as uncriticizable by outsiders because of this practice’s functional fit and because we understand its meaning in that social environment. The other view, ostensibly representing the objective school of philosophy, contains two methodological approaches. The first approach, called justificationary (subsuming logical positivists, followers of Wittgenstein’s later philosophy-mind or language philosophy, and qualified realists) asserts that you can prove the truth of your claims essentially by cumulative positive evidence.

1 The current philosophic inclination among most social work professionals is to argue that there are no objective truths but many subjective ones each entitled to respect and non-interference from outsiders (see critique, Gomory, 1997; see Tyson, 1995 for contrary view).
inductively procured (Gomory, 1997), the second approach, sometimes referred to as Critical Rationalism or Fallibilism (Miller, 1994) a position with which I agree, strongly opposes the cultural relativism and the justificationary approach of the first two groups and argues that science ought to provide testable, that is potentially falsifiable, explanations (hypothesis) about how our world works (Gellner, 1974, Gellner, 1992).

The willingness to ‘endorse’ all cultures and their beliefs by the various justificationary approaches often translates into a philosophic relativism which holds that there is no real objective world outside of the particular culture or environment under scrutiny and every ‘fact’ is relative, including the findings of science, to these so called cultures or traditions. This relativistic position is regularly justified by the alleged necessity for cultural sensitivity. This notion claims in its extreme form that only the members of a particular group have the proper credentials to knowledgeably service, interpret, evaluate or discuss that group. Group membership is generally conferred by having such things as; skin color, sexual preference, gender, political status or drug habits in common. This stance bars any meaningful critical evaluation from outside of such groups or the programs targeting them because it assumes that the identified shared characteristics\(^2\) of researchers and group members provide privileged access to

\(^2\) There may in fact be many characteristics (sexual preferences, eating habits, ethical beliefs, hobbies, intellectual skills, etc.,) known or unknown that actually differentiate the individual members of these ‘homogeneous’ groups. But, out of either, current scientific impotence or ignorance as to what these characteristics are, or willful disregard of available research evidence identifying these characteristics, they are ignored in order to maintain category coherence. Of course, it is a necessary corollary of any effort at categorization that those qualities or aspects of an individual that conflict with the paradigmatic particulars of a category be ignored. This requires no specific political, or professional agenda (no conspiracy) but does point out the highly subjective process involved in categorizing per se (see Peckham, 1978 for an in-depth
and knowledge about that group that is not attainable by any other means. (Jarvie, 1969; Gellner, 1974; Gellner, 1979)

This stance avoids the essential critical tension that the confrontation of differences offers. This attitude is unfortunate because in the 'hard' sciences real progress has been made precisely to the degree that various conflicting positions or theories were placed in competition with one another and tested to determine which survived these critical tests (Popper, 1962, PP 33-65). Even Newtonian mechanics, the most well supported theory of all time, which for over two hundred years was thought to explain how the universe operated, was falsified and replaced by a more comprehensive new theory (Einstein’s theory of relativity) because Einstein’s theory when tested, provided some explanations of phenomena which Newton’s could not, while explaining all that Newton did explain. Such strong critical clashes are also the bread and butter of debates in Philosophy.

Examples of such critical efforts include; in *Abusing Science* by the philosopher of science Philip Kitcher who critiques the explanatory claims of ‘Creationism’ for humankind’s origins and development and compares the evidence for it to Darwinian evolution (Kitcher, 1982). Kitcher has also written about the problems of Sociobiology as developed by E. O. Wilson and others in *Vaulting Ambition* (Kitcher, 1985). Another important book length critique in biology is *Not In Our Genes* by three prominent scientists, R.C. Lewontin of Harvard, a population geneticist, Steven Rose, of London’s Open University a

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explanation of this phenomenon).
neurobiologist, and Leon Kamin a psychologist from Princeton (Lewontin et al., 1984). This book in detail refutes the claims of various genetic causal theories by examining the studies and data that purport to corroborate the genetic causal claims. Among these were the claims that genes determined IQ scores; that Schizophrenia is genetically determined or caused; and that genes determine social behavior.

The domain of Philosophy in large measure consists of critiques of various philosophic positions in order to demonstrate, through good argument, why one theory is superior to another. In our century some of the great philosophical debates still undergoing hard testing by conceptual and empirical analysis are Logical Positivism (truth can be found by sense observation of data adding up to verified theory) vs. Popperian Critical Rationalism (bold conjectures put to severe tests, with the currently surviving hypothesis tentatively held to be a better approximation to the truth) vs. Wittgensteinian ordinary language philosophy (words and their meaning are defined by a particular group’s life rules) vs. various forms of Relativism (such as Hermeneutics or post-modernism which claim that there is no objective truth, everything is interpreted or deconstructed) vs. out and out Skepticism (sure knowledge may be sought, but cannot be found, therefore no actions are justifiable, leading dogmatically to anarchy) (Munz, 1985).

Some contemporary critical tracts that are particularly outstanding are Legitimation of Belief, by the late Ernest Gellner (Gellner, 1974), Conjectures and Refutations, by Karl Popper (Popper, 1962), Criticism and the Growth of
I believe, as do Popper(1962; 1979), Jarvie(1969), Gellner(1973; 1974; 1992), Bartley(1984), Albert(1985), Agassi(1988), Munz(1985; 1993), Miller(1994) and a host of other scholars, that the application of criticism by falsificationary testing is essential to further knowledge, not only ‘hard’ scientific knowledge, but even the ‘softer’ kind pertaining to human beings and their behavior, the moral sciences (Miller, 1994). There are very few examples of such critical analyses in my area of interest, mental health.

One outstanding recent exception is The Selling of DSM (1992) by Stuart Kirk and Herb Kutchins, both graduates of Berkeley’s social welfare doctoral program. This book critically examines the claim of the mental health bureaucrats who have manufactured the various DSM diagnostic categories, that they have identified reliable diagnostic entities and finds this claim wanting.

The more important issue of the validity of the various mental disorders(are these disorders real?) is even less well researched. In fact no empirical data has ever been able to demonstrate for example, the existence of the 'sacred symbol' of institutional psychiatry, the syndrome of schizophrenia (Szasz, 1976, Boyle, 1990). The research community has simply assumed that the schizophrenia construct is valid. This they believe has been demonstrated long ago by its original inventors, Emil Kraepelin and Eugen Bleuler. The fact is that neither carried out any empirical research. They both simply described various symptoms and behaviors found among heterogeneous insane asylum
populations and claimed without any systematic data that they formed the core of the schizophrenia syndrome (Boyle, 1990, chapter 3 and my analysis in the first chapter to follow).

This lack of critical thinking is to be found in much of social science, but appears to be particularly endemic in mental health. The claims and results of primary research done in mental health are accepted for the most part uncritically as reliable and valid on their face even by comprehensive literature reviews published in scholarly journals as I will presently demonstrate. These articles compare and contrast the various reviewed studies and their outcome claims and on-occasion their differing research methodology (for example; Olfson, 1990; Attkisson et al., 1992), accepting the data and the conclusions as given by the researchers. Too rarely does any scholar dig further to question the reliability and validity of the results as reported in these primary research articles. What is needed, and the books I listed above can serve as models, is an approach which would analyze, how data were identified and gathered, what assumptions the researchers used in their research, the reliability and validity of the research variables and instruments used to capture this information, along with evaluating the research models employed to see if the claims made are validly asserted by models methodologically capable of testing the proposed hypothesis. Without this sort of detailed critical analysis of the data

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3 It may be argued that these articles are mostly, or all, to be found in peer reviewed journals and as such have been closely scrutinized for error by knowledgeable experts in that area and thus are not uncritically accepted. But as Flew (1994) points out self interest often plays a powerful role in how research is done and reviewed. Individuals who have a strong interest in supporting a position or a particular paradigm (often that in large part is what is meant by being an expert in a field) may not be the best people to review research in
that underlies the claims of research we may miss fundamental errors in methodology and accept false outcome claims based on them.

This problem also occurs more frequently then is supposed in much harder sciences, such as microbiology. Illustrative is the recent claim that a sheep was successfully cloned from another sheep's adult cells. This original research has not been reproduced in several attempts at replication. It appears that this cloning might have been the result of inadvertently using fetal cells (a very common way of producing genetic copies that is not the equivalent of adult cell cloning) because the sheep used was pregnant.

"Dr. Wilmut’s experiment, long accepted without demur, came under criticism last month from Dr Norton D. Zinder, a distinguished microbiologist at Rockefeller University. In a letter to the journal Science, Dr Zinder said Dr. Wilmut’s success rate, 1 in about 400 tries, was scientifically meaningless because any number of possible errors could yield false results. … Walter Gilbert, a Nobel laureate at Harvard[said that] [t]he paper should not have been published in the form in which it appeared … Nature, the journal that published Dr. Wilmot’s report, has not taken a neutral part in the controversy. It used Dolly[the cloned sheep] in its advertising … The dispute … raises the issue of how thoroughly a researcher should replicate an important result before publishing it, and the related question of what standards of proof a journal editor should demand before accepting a paper for publication.”(NY Times, 2/28/98, A6)

One of the purposes of my dissertation is to test for the existence of these previously noted research problems.

I propose to apply Critical Rationalist principles to the most well accepted model of community mental health treatment for the ‘severely and persistently mentally ill’, Programs of Assertive Community Treatment(PACT). These programs,
“... typically include aggressive clinical and social case management, training in community living skills, and crisis intervention” (Attkison et al, 1992 p 580).

More specifically I will examine the research available on one of these programs, Stein and Test's Training In Community Living (TCL) originally invented in Wisconsin in the early 1970's and now implemented in many sites throughout the United States\(^4\), Great Britain and Australia. An industry has developed around this model (Stein and Test, 1985). Many states are using the PACT model and using the results alleged to have been found in original TCL research as their justification for replication. I will evaluate all of the relevant replications as well as the original model.

With the development of ‘capitation’\(^5\) and its heralding by the mental health establishment as the new answer for ‘innovative’ mental health service financing and program provision, ‘assertive case management’ (PACT) is becoming more and more wide spread (Bloom et al., working paper p.28). There is an apparent assumption by the field that innovative means effective. However, it should be kept in mind that many current conventional, ineffective interventions and financing scenarios for which PACT and capitation respectively are the corrective solutions, were also considered innovative when they were first introduced.

The examination of the TCL model and its various replications is intended as falsifications.

\(^4\) There are at least 340 assertive community treatment programs in 34 states, as of 1992, that are funded or sponsored by the various states. There are many more that have been developed by the Department of Veteran Affairs, municipal mental health agencies and other private or voluntary agencies which, as yet, have not been counted (CSNN, 1997; Deci et al., 1995).

\(^5\) A method of paying a preset fee to mental health providers in exchange for delivery of all services deemed necessary for clients enrolled in capitation plans. These plans are often part of the more comprehensive ‘innovation’ of managed care currently in vogue in the health care arena.
a case study and is chosen because of its wholesale acceptance by the experts as a well tested effective community mental health program (Attkisson et al, 1992). A further advantage is that the original Training In Community Living model has been in existence for over twenty years and Stein and Test (two of the creators) both separately and in various collaborative efforts, have reported on the research extensively and have written voluminously about what this model is attempting to, and in their estimation is accomplishing. The public availability of research done over an extended period of time allows for a historical overview along with the empirical analysis of the program’s effectiveness. It is a rare opportunity to also examine, as described in their own words, the education, training and conceptual development over time of some of the individuals who currently play a major role in the treatment and identification of those who are considered to be severely mentally ill. The field of mental health is intimately involved with various forms of social control(Szasz, 1987, Szasz, 1994). By reviewing and critically evaluating the TCL groups’ written record we can perhaps come to a better understanding of the roles if any, of science, politics, coercion and power in how society defines, financially underwrites program research and development, and implements solutions for at least one major social problem, ‘severe and persistent mental illness’.

Methodological Confessions

To properly understand what I am attempting here let me describe my theoretical framework( point of view, professional bias, philosophical underpinning). This is done to simplify the evaluation of my work by others. This
should, in addition, promote clearer critical responses to my framework and the assertions I make, perhaps thereby eliminating or reducing ad hominem attacks. My critically - in contrast with uncritically or unconsciously - held beliefs that are applied in this dissertation are the following:

**Methodological Individualism**

“...the fact that it is concepts and views held by individuals which are directly known to us and which form the elements from which we must build up, as it were, the more complex phenomena” (Hayek, 1979, p.65).

Conceptual entities such as societies, cultures, professions, hospitals and mental health programs cannot be the final causal level of analysis in social science, but rather we must look to the human beings - the only ones who can actually formulate theories, plan, make decisions, apply 'interventions' and do good or do harm based on these theories - to constitute the proper level for explanations. This belief also accounts for my use of the first person. The third person point of view, standard in much of the research and theoretical writings of scholars, can distance the reader from the author with its assumed air of objective authority that really is not justified or necessary. These scholars are after all, fallible individuals who have done some research or some reflective thinking about some topic and now are offering their findings for review and critique. This of course may result in showing that the research is not only fallible but false.

In literature the third person point of view allows the author to present himself as the all powerful and knowing author of the narrative, the characters, and the alleged (in literature’s case imagined) facts, and in fiction he may be. But in
research we have no such authorities, just people claiming either through belief, empirical evidence or critical reasoning to have some interesting information to convey. By writing in the first person - and using as little jargon as possible - I hope to make the authorial wall less of a barrier and allow an intimacy in which the reader will be less inclined to be intimidated and more inclined to be engaged in a serious intellectual dialogue with the writer.

The use of the first person authorial voice is by no means new to scholars in the social sciences. Such distinguished individuals as sociologist, Edwin Lemert (1967), sociologist, Thomas Scheff (Scheff, 1975), psychologist, David L. Rosenhan (Scheff, 1975), sociologist, Irwing Goffman (Goffman, 1986), and Nobel laureate and economist, James Buchanan (1986) are among the many who have opted for this more familiar voice to discuss their research and ideas.

**Popperian Critical Rationalism** (Munz, 1993, Miller, 1994) -

Critical Rationalism is based on the following principles. Inductive reasoning is false. As a good Popperian, I know that no matter how often you find supporting evidence for a theory, an intervention, or for any concept or idea, that in no way assures you of having arrived at the truth or the proven in the sense of conclusive proof. The very next attempt may contradict and thereby falsify previous confirmations(evidence).

The classic illustration of this inductive problem - often referred to by

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6Sir Karl Popper (1902-1994), is the founder of Critical Rationalism, and one of the 20th century's greatest philosophers. He has falsified decisively the age-old doctrine of induction (Still inexplicably embraced by many social scientists) and has undermined all philosophies of science which seek to discover justifiable, ultimate scientific knowledge. In his numerous, lucid writings, he has powerfully argued for, a fallibilistic critical methodology as the core of the scientific method, and has identified the aim of science, as the
philosophers as Hume’s Problem (Popper, 1962 p. 42) - is the statement: all swans are white. As you observe more and more swans that are white, you appear to support the original hypothesis of the whiteness of all swans. This is all well and good as long as you don't get an opportunity to take a trip to Oceana. If you were to go, for example, to Lake Taupo in New Zealand, you would find black swans with red bills. This empirical observation, even of only one black swan, provided you are in fact looking at a black swan and not a raven or a blackbird instead, would disconfirm (logically falsify) your original hypothesis that swans are white and white only.

This thought experiment is part of an argument by Karl Popper, which demonstrates the logical impossibility of inductive reasoning as a tool for discovering what “is”. The simple fact is that you cannot make a sound inference from the particular (the known), no matter how often repeated, to the general (the unknown). One has no way of knowing whether the very next observation will not be the one to undo even the most well confirmed hypothesis. Witness the replacement of Newtonian mechanics with 200 plus years of empirical support behind it, by Einstein’s theory of relativity, as mentioned earlier. This analysis by Popper was a very important step, if not the decisive step in 20th century philosophy of science. By this demonstration he turned the very idea of scientific method on its head. The search for absolute, justified, perfect knowledge was over, it could not be done. Only the frailer version, hypothetical, human knowledge, tentatively held until falsified, was possible. A scientific theory must

unvarnished truth (Miller, 1994).
be open to the possibility of refutation entailing a critical test that can be empirically measured and independently evaluated. It should be a more comprehensive attempt at explanation that can explain the phenomena which the current theory(s) explains and something more. This new theory must ‘pass some new and severe tests’, proving itself against some new predicted outcome by agreement with the facts. The progress of science relies on bold and risky conjectures (hypotheses) severely tested and dropped when falsified(set aside by better, more comprehensive explanations)\(^7\).

**Unintended consequences flow from intended human action-**

This speaks to the limits of human(hypothetical) knowledge. Many of our greatest social problems occur as a result of planned (benevolent?) human actions which produce unexpected outcomes(welfare support resulting in dependency, college open enrollment resulting in decreased literacy, psychotropic drug use resulting in tardive dyskinesia etc.).

“To try to analyze these reactions and to foresee them as far as possible is, I believe, the main task of the social sciences.”(Popper, 1966, p.95).

**Situational logic-**

For explaining the actions of our actors, among which may be(in terms of our interests); the development of mental health programs, the creation of public policies and the production of theories (scientific or metaphysical), whether in the past or contemporaneous, I propose to use Popper’s method of *situational*

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\(^7\) Popper was not a naïve falsificationist(a single example of falsification will do), as he was derogatorily labeled by Lakatos, Kuhn, and many of their deciples, but rather, believed that in practice multiple, methodologically unproblematic, empirical falsifications should occur before a particular theory was considered to be tentatively false.
analysis, 

“The historian’s task is, therefore, so to reconstruct the problem situation as it appeared to the agent, that the actions of the agent become adequate to the situation.” (Popper, 1979, p.189).

This requires a thorough understanding of the problem(s) the agents were attempting to solve along with a comprehensive understanding of all the background knowledge (the philosophic frameworks available for explanations, the state of scientific knowledge about various interventions etc.) that may have been available to them as they attempted to solve their problem. This procedure is of paramount importance because,

“Our conjectural reconstruction of the situation may be a real historical discovery. It may explain an aspect of history so far unexplained; and it may be corroborated by new evidence, for example by the fact that it may improve our understanding...” (p.189).

In the alternative such evidence may prove decisive in falsifying the claims of these agents.

The preceding criteria comprise my method, which I believe also can be called the scientific method, if there is such a thing (see Popper, 1979, p.265). Method is not a panacea but a fallible guide for intelligent critical analysis and will allow those who wish to refute my arguments and conclusions to see the method in my method (Based on Jarvie’s superb analysis in Jarvie, 1969).

In the chapters to follow I apply the preceding set of methodological procedures to the critique and evaluation of the TCL model. I will begin by discussing in the first chapter the difficulties inherent in the commonsensical professional belief in the empirical reality of mental illness on which TCL and all
other mental health treatments by logical necessity are predicated. I do this by reviewing the widely based psychiatric claim that there is a well established and documented history of research done by the discoverers of schizophrenia (the paradigm model of all other mental diseases) that needs no further research and which unequivocally demonstrates the existence of schizophrenia and by analogy the existence of all other mental diseases and authenticates them as medical diseases, just as physical medicine has demonstrated the existence and authenticity of cancers, infectious diseases, etc.

In the second chapter I will offer a historical review of the TCL treatment population and a brief discussion of community mental health. In the section on the Treatment Population I review the various labels used for this group of people by the multi-billion dollar public mental health industry. I examine their significance both historically and for the type of treatment provided to the individuals ‘who’ are subsumed by these labels. They are most often called, ‘the severely and persistently mentally ill’ client, but are also known by the aliases of; the ‘chronic psychiatric patient’(Test & Stein, 1978), the ‘markedly impaired’(Test & Stein, 1976), or ‘persons with serious mental illnesses’ (Test, 1992).

These various labels are frequently found in the literature and used interchangeably. I will look at what the TCL group has said about these categories as well as what other researchers have written. I will explore these functionally descriptive labels and compare them to the more discrete DSM

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8 28.4 billion dollars were spent on mental health by government alone in 1990(Center for Mental Health Services).
categories that are used to clinically diagnose mental disorders. Of special interest will be to see how these various terms, including the DSM categories, are or are not used to direct the efforts for research and treatment. More specifically does having a diagnosis of schizophrenia, for example, indicate a different type of community mental health treatment then a diagnosis of bi-polar disorder? And what are the consequences of whether or not treatments relate to diagnoses?

The Community Mental Health Treatment section consists of a historical review of the various forms of treatment that were considered to be community mental health treatments in the United States. I will show that different types of treatments, often diametrically opposing one another, were considered to be beneficial community mental health interventions. Some of the issues to be evaluated are whether the changes in this treatment methodology were driven by empirical issues (is one treatment more effective than another?), or by other considerations, such as professional needs of the 'psychiatric experts,' or political agendas, or economic considerations. These issues will then be related to the status of client care to see if these various treatments considered, and to what degree, the desires and concerns of the clients.

Chapter three discusses The Historical Roots of TCL. Using situational analysis I describe how the inventors of TCL developed their theories about the clients their model claims to help, and demonstrate that they used coercion and force to effect 'client change' (or as I claim compliance), and argued their
therapeutic value. This chapter also suggests that the methodological problems I found in their 3 experimental studies (chapter 5) on the TCL model have had a long prior history.

My analysis is based on 10 original papers published by this group. These papers paint a powerful picture of how the TCL researchers came to conceptualize the severely mentally ill and I claim that these initial experiences at Mendota State were decisive in their development of TCL.

For example, in the first paper the researchers claim that these patients are;

“Obviously ... not ... a group of fragile, broken-spirited persons but rather ... tough, formidable adversaries who were ‘pros’ and who had successfully contended with many different staffs on various wards in defending their title of ‘chronic schizophrenic’(Ludwig et al., 1966, p.566).

And in a later paper they allege that there is a ‘code of chronicity’(perhaps genetically encoded?) this they suggest is so because of certain ‘clinical facts’

“Implicit in our discussion of the ‘code’ are five important clinical ‘facts’ which, we believe, underlie the behaviors of chronic schizophrenics. First, these patients can use their insanity to control people and situations. Second, they have an indomitable will of their own and are hell bent on getting their way. Third, one of the basic difficulties in rehabilitating these patients is not so much their ‘lack of motivation’ but their intense, negative motivation to remain hospitalized. Fourth, insanity and hospitalization effectively pay off for these patients in a variety of ways. Fifth, these patients are capable of demonstrating an animal cunning in provoking certain reactions on the part of staff, family, and society at large which guarantee their continued hospitalization and its consequent rewards.”(Ludwig et al., 1967, p.738).

Chapter four provides a conceptual analysis of The Training In Community Living Model. According to Test(Test, 1992) there are four essential characteristics that comprise the model.

“1) Core Services Team- “the primary provider of services in TCL is a core services team. This community-team consists of ... interdisciplinary staff who
serve as a fixed point of responsibility for ... young adults with schizophrenic disorders (elsewhere Test et al argue that all severely mentally ill persons should be treated by TCL, see Test, 1992, p. 166). The team's function is to see that all the patient's needs are addressed in a timely fashion. ... Having one team provide most of these services minimizes the ... fragmentation of ... care systems and allows for integrated clinical management.

2) **Assertive Outreach and In Vivo Treatment** - An essential ingredient in the way services are delivered ... is the use of assertive outreach. This means that staff ... reach out and take both biological and psychological services to the patient. Indeed, the majority of the treatment and rehabilitation ... take place in the community ... . The rationale for use of assertive outreach is to minimize patient dropout and to enable the provision of psychosocial services in vivo, right where the patients need to use them.

3) **Individualized Treatment** - Because persons with serious mental illnesses, even within the group diagnosed as schizophrenic, are greatly heterogeneous and both person and disorder are constantly changing over time, treatment and supports must be highly individualized. ... The content, amount, timing, and form of treatments/services provided vary enormously both between patients, and within the same patient across time.

4) **Ongoing Treatment and Support** - ... studies of community treatment using a variety of models have ... revealed a loss of gain following discharge(Test, 1981) It must be concluded that even very intensive community treatment models do not provide a cure for severe mental illness, but rather provide a support system within which persons with persistent vulnerabilities can live in the community and grow. It appears these supports must be ongoing rather than time limited."(Test, 1992 p.154-6)

These TCL treatment model characteristics were structured based on the "... broad biopsychosocial model of serious mental illness" presented by Strauss and Carpenter in their 1981 book called *Schizophrenia*.

"Such a model suggests that programs must provide interventions in all three areas (biological, psychological, and social), focusing not only on changing the person but also on changing the environment."(Test, 1992, p.156-7).

The preceding four characteristics as described by Test, plus the conceptual model of Schizophrenia originated by Strauss & Carpenter(1981), make up the justificationary framework of TCL.
In chapter four I will examine the TCL framework just described. This includes a critique of TCL empirical claims by evaluating their written record to see if the program in practice does what the theory claims that it does. I point out discrepancies, offer alternate explanations for some of their claims and in some instances refer to other empirical research which contradicts some of the TCL claims and also appears to offer better explanations.

The fifth chapter contains a methodological analysis of the original 3 TCL experimental studies. The three experimental studies conducted by the TCL researchers are each progressively longer, each claiming to build on the previous research findings and all claim success for their experimental intervention. I examine essential methodological questions in each of the studies. These include; the reliability and validity of the instruments used to collect data, the possible role of researcher and clinician bias, appropriateness of control groups, whether claims of significant outcomes for the experimental group result from the alleged effectiveness of the experimental intervention or such other matters as, administrative rules applicable to controls differentially than to experimentals, or the use of unreliable measures, or the renaming and redefining of variables during the research just as the study seems to be failing to provide expected results, or the action of unaccounted for psychotropic medications. The sixth chapter reviews and critiques all the randomized controlled experimental replications of TCL done to the present. The seventh and final chapter discusses my findings in summary and offers some brief thoughts about the implications of my analysis.
Chapter 1

**Conceptual Critique of Mental Illness**

“Indeed, if every inspiration that comes to one with such commanding urgency that it is heard as a voice is to be condemned out of hand by the learned qualification of a morbid symptom, a hallucination, who would not rather stand with Joan of Arc, and Socrates on the side of the mad than with the faculty of the Sorbonne on that of the sane?” (Huizinga, J. p.222, 1959)

**INTRODUCTION**

What follows is a rapid montage in quotes, from various authoritative sources, of the history of (mental) disease and its ‘sacred symbol’ (Szasz, 1976), schizophrenia:

“Of course, every conceivable human experience has been described, at one time or another, as the cause of psychoses which we now consider to be schizophrenia.” (Originally written in 1911 by Bleuler and translated and published in English only in, 1950, p. 347).

“We do not know what the schizophrenic process actually is”(p.466).

“It is not yet clear just what sort of entity the concept of dementia praecox actually represents. It probably includes one or very few diseases, in the narrower sense, which constitute the major portion of cases, in the same way as general paresis embraces the majority of all cases of the dementia paralytica of the previous century.”(p.279).

“Paracelsus(1493 - 1541) broke with scholastic tradition in medicine and recommended a complete re-evaluation of the fundamentals. In his teachings mystical, magical, and scientific elements were all blended into a single doctrine. Paracelsus was primarily a naturalist who trusted experience and mistrusted logic. Although he fought superstition and searched for natural causes, he did not hesitate to believe also in magic or unseen influences. Thus he firmly believed in the interaction of man and the stars. ... To him diseases were entities ... . Much of health and disease was attributable to chemical changes, and treatment was often carried out with chemicals.”(Engle and Davis, 1963, p. 122).

“One great Reason why these Patients are unwilling their Disease[ Madness or Insanity] should go by its right Name is, I imagine, this, that the Spleen and
Vapours are, by those that never felt their Symptoms, looked upon as an imaginary and fantastick Sickeness of the Brain, filled with odd and irregular Ideas."(Blackmore, 1724, pp. 98-9).

“The whole idea of dementia praecox originates with Kraepelin. Almost exclusively to his work we also owe the grouping and description of the separate symptoms.”(Bleuler, 1950, p.1).

“The development of the concept of dementia praecox constitutes a considerable part of the whole development of theoretical psychiatry. One can hardly be described without the other.”(p.4).

“I call dementia praecox 'schizophrenia' because (as I hope to demonstrate) the 'splitting' of the different psychic functions is one of its most important characteristics.”(p.8).

“However, the pathological anatomical findings exist, and even though the nature of their relation to the psychosis remains enigmatic, the above interpretations[of psychic origins] of primary manifestations appear forced in this light. ... Complete justice to all these factors can only be done by a concept of the disease which assumes the presence of (anatomic or chemical) disturbances of the brain; the course of the ... disorder is chronic ... the disturbance of the brain determines the primary symptoms.”(pp. 462-463).

“Hand in hand with the elaboration of the dementia praecox concept other disease - entities were defined, particularly the manic - depressive psychosis.”(p. 7).

“By 1896, Kraepelin was observing that some 10 to 15 percent of all admissions to mental hospitals and the vast majority of all chronic patients fit the criteria he was developing for dementia praecox. ... Kraepelin believed that the illness was somehow inherited.”(Gottesman, 1991, p.12).

“Schizophrenia is still diagnosed today[this book was published in 1991] as it was at the turn of the century: by its psychopathology; that is by its abnormal patterns of thought and perception as inferred from language and behavior. We wish we had a definite neuropathology(either chemical or anatomical) to pinpoint a valid diagnosis of schizophrenia, but that goal still eludes us.”( p. 17)

“Over a quarter of a century ago, in my presidential address to the American Psychological Association, I began by pointing out that the only non-symptom, ... you can know about a person that gives you better than an even chance in a wager that the person will develop schizophrenia is that his or her monozygotic twin is schizophrenic. ... Radical environmentalism was ubiquitous in those days, and the somatic phenomena of schizophrenia, known since the classic works of
Kraepelin and Bleuler, were either ignored or presumed to be psychosomatic in origin. ... I cannot say exactly when I began to adopt a strong genetic view as to the specific etiology of schizophrenia, but it must have been around the end of Word War II" (Meehl, 1989, p.935).

"Modern concepts of schizophrenia are based on the work of Emil Kraepelin and Eugen Bleuler, who made their main contributions just before and after the turn of the century, respectively. Kraepelin (1899), ... was the first to rearrange certain forms of insanity into one group, which he called ‘dementia praecox’. ... In keeping with this well-established tradition, the description of schizophrenia in this chapter[and the book] is based on the concepts of Kraepelin and Bleuler ..."(Straube and Oades, 1992).

"I take it to be obvious that the publication of this book[Schizophrenia and Genetics, by Gottesman and Shields] ‘settles’ a long - debated question, to wit Is there something - fairly ‘big’ something - genetic about schizophrenia? ... Nothing but American social - science prejudice ... can, I think, lead anyone who has read this book to persist in strong doubt as to the prime importance of genetic factors in schizophrenia. Hence I should now be inclined to adopt Otto Neurath’s maxim in the Vienna Circle, when ... he opined, ‘Feigl[Both leading members of this leading school of Logical Positivism], you know, one can seriously discuss only with those who are in the club’. For my part, I am no longer interested in a debate with anyone who continues t believe that we are all born with equal talents for developing schizophrenia, and who is prepared to exercise ... ingenuity ... in explaining away all genetic evidence by this or that ad hoc hypothesis."(quoting Paul Meehl in Gottesman and Shields, 1972, pp. 367 - 8).

“... I assume that mental disorder is best taken as a subspecies of medical disorder, ... I reject that mental disorder is an invalid concept."(Wakefield, 1993, p.161).

“Also as in DSM-III-R. it is assumed here that the concept of mental disorder is a straightforward extension to mental processes of the general concept of disorder used in physical medicine."(Wakefield, 1992, p.232).

“It has now been conclusively shown that individuals with schizophrenia and manic-depressive illness have brain diseases."(Torrey, 1995)

“Diagnostic criteria such as those provided in DSM - III or DSM - IV have a tantalizing simplicity that gives people the sense that we know what schizophrenia is when in fact we do not."(Shore, 1993, p3).

The fundamental ‘problem’ of this dissertation is human behavior, particularly those behaviors that come to the attention of the psychiatric helping professions.
These behaviors on the whole are not considered by these professionals as volitional acts of autonomous individuals, but rather as signs or symptoms of mental illness. Other descriptive categories used by the mental health experts in descending order of scientific support, most often as if no empirical differences existed among the various categories, are; mental diseases, syndromes, disorders, dysfunctions, and disabilities.

This view can fairly unproblematically be described as the medical model of mental illness. This model states that; mental illnesses are characterized by and manifested in various discrete groupings, most often called syndromes, “It is important to remember that schizophrenia remains a clinical syndrome comprising an unknown number of disease entities or pathological domains”(Shore, 1993, p.3).

Mental illnesses consist of public claims and/or exhibitions of certain types of unpleasant, unwanted or disturbing behavior, which are designated as signs or symptoms, of the underlying mental diseases which are hypothesized to be ultimately, in some yet to be specified way, physiological in nature and origin. They are claimed to be putative brain disorders; with the various possible etiologies running the gamut from genetic determinism, chemical imbalances, viral infections, hormonal irregularities, ‘abnormalities in the composition and structure of neural membranes’ and so on ad infinitum(pp. 1-22). The fact remains however, that after over 100 years of research, - with the research dollars reaching more than 100 million dollars by 1992 for just schizophrenia(p.2) - both the etiology and the nature of mental illness(es) remains as much a mystery as when Kraepelin invented one called dementia praecox, in 1896,

The psychiatric research community claims that at the current stage of its development, the science of Psychiatry is as yet unable to determine the causes, or even what, beyond being under medical purview, mental diseases are.

The representative quotes at the beginning of this chapter are meant to illustrate our intellectual as well as our scientific progress. We did not know what schizophrenia was in Kraepelin or Bleuler’s time, although we could name it both in Latin(dementia praecox) and Greek(schizophrenia), and according to the National Institute of Mental Health’s house journal Schizophrenia Bulletin in its 1993 Special Report on the latest research and treatments of schizophrenia we still do not know,

“The [DSM]criteria do not represent a comprehensive statement about either the defining features of the disorder or its essential nature. Diagnostic criteria such as those provided in DSM-III or DSM-IV have a tantalizing simplicity that gives people the sense that we know what schizophrenia is when in fact we do not.”(Shore, 1993, p.3).

Originally this chapter was going to review the validity of the concept of mental illness as argued for instance most recently and interestingly by Wakefield(1993) as a prelude to evaluating the experimental research on certain interventions that are claimed to help these type of diseases in the latter chapters. This approach seemed to be dictated by the simple inescapable fact that alleged effective medical treatments must have real medical diseases to treat. This appeared to require an inquiry into the nature of (medical)illness or disease in general and then perhaps to assess the validity of the claim that mental
disease can be a sub-category of the prior concept, and then finally to evaluate
the validity of the evidence for the claim that these distinct conceptual entities
(such as schizophrenia, manic depressive disorder or clinical depression) are
diseases under the criteria of the medical model. This seemed to follow from the
claims asserted by the National Institute of Mental Health experts that,

“Efforts to understand the nature of schizophrenia are ultimately linked to a
disease model. That is, schizophrenia is assumed to be a specific illness or
group of illnesses defined ultimately on the basis of its pathophysiological
mechanisms and its causes.” (Shore, 1993, p.2).

As I researched the topic it became clear that the definition of medical disease
has changed periodically, and has to, if it is to have any scientific status over
time. So part of my inquiry would have to consider the evolution of the concept
and the possible limiting parameters of it if known.

A quick and dirty review of the evolution of medical disease and its diagnosis

As we became more scientifically knowledgeable about the physical universe,
improving in our ability to both make bold conjectures (predictions) about the
world and to critically test these by severe and rigorous attempts at
falsification (Popper, 1962), the concept of disease and its diagnosis shifted
explanatory frameworks from a religious model of supranatural causality to a
model based on science and empirical evidence. The quality of the scientific
evidence also evolved as various conjectures about disease were tested and
corroborated or falsified as both practice and research in medicine became more
rigorous (Engle and Davis, 1963 I, II, III).

Medicine, disease and the procedures for identifying it, was seen as
indistinguishable from magic or religion in earliest human history and although in both ancient Egypt and Mesopotamia physicians observed ‘that certain symptoms occurred in combination of symptoms’ their causal explanations were still based on religion and magic (Engle and Davis, 1963 II, p.524). The science of medicine officially began with the Greeks. They were the first to attempt theories of physiology and medicine(p.524).

“The Hippocratic School at Cos directed its attention to disease in general as it was seen in individual patients. ... Diagnosis in the sense of naming the disease was not important. What was important was to study the natural history of disease and to derive from this some concept of pathologic physiology that would then be useful for the treatment of the patient. ... The rival school at Cnidos emphasized diagnosis and exact classification of disease. For example, they are said to have recognized seven diseases of the bile and twelve of the bladder. ... Both excluded magic as a cause for disease. The Hippocratic School ... developed the physiological concept of the four humors, which maintained its influence in medicine until the Renaissance”(p. 525).

The Stoic philosophers followed this period, their contribution was the stress on genuine and careful observations, but unfortunately they dogmatically stuck to prior generalizations refusing to alter them even when it was at the expense of individual patients(p.525). The Epicureans, in part as a reaction to this dogmatism, enlarged medical diagnosis by suggesting that true treatment was treatment that stood the test of prior experience based on anatomical research.

Galen(born 130 AD) influenced by Hippocrates, saw the importance of nature in healing of disease and wrote a book, *On the Natural Faculties*, in which he developed his theory of the four humors and discussed the importance of paying attention to how disease affected the particular individual who was ill(p. 526). The humoural theory of medicine was the predominant medical paradigm until
Paracelsus(1493 - 1541) in the 15th century speculated that diseases were discrete entities or species(p.525) and argued against the humoural theory of health and disease. He claimed that chemical changes determined health and illness and his treatment often consisted of the use of chemicals(p. 526). The humoural theory was falsified by the anatomical studies of “Vasalius(1514 - 1564), Michael Servetus(1509 - 1553), and William Harvey(1578 - 1657) and later by Morgagni (1682 - 1771) who laid the foundation of organ pathology”(p.526).

Thomas Sydenham during the 17th century went on to suggest that diseases were clinical entities and they should be carefully described and distinguished from each other, much like plants. He was able to classify the clinical picture of many of the infectious diseases for which the causal agents were found over 200 years later:

“Nature, in the production of disease, is uniform and consistent; so much so, that for the same disease in different persons the symptoms are for the most part the same; and the selfsame phenomena that you would observe in the sickness of a Socrates you would observe in the sickness of a simpleton Just so the universal characters of a plant are extending to every individual of the species; and whoever(I speak in the way of illustration) should accurately describe the colour, the taste, the smell, the figure etc., of one single violet, would find that his description held good, there or thereabouts, for all the violets of that particular species upon the face of the earth”(Sydenham, 1848, p. 15).

Sydenham postulated that a disease was a syndrome or a discrete constellation of symptoms and signs, “ ... with a characteristic time course and outcome.”(Kendell 1986, p.27).

As Engle and Davis point out, throughout the history of medicine a tension has existed and exists to the present between those medical researchers who believe in specific disease entities, objectively apart from the patient, and those
who believe in a more general concept of disease as represented within the
individual patient(p.527). By the 18th and 19th centuries pathology was
advanced enough to recognize the involvement of particular tissues not just
organs in disease processes,

“Corvisart, combining an interest in pathologic anatomy with clinical medicine,
associated clusters of symptoms with specific anatomic changes. ... Laennec ...
was able to ... correlate physical findings with the pathologic picture ... In
opposition were those ... who championed physiologic medicine and stressed
functional disorders.”(pp. 527 - 8).

In the mid 1800’s Rudolf Virchow developed and refined cellular pathology,
进一步帮助确定理论，即病理变化(病变)在正常
细胞是疾病原因。在同一年，Claude Bernard and Ivan Pavlov 强调了医学研究中
的实验性的重要性。

The Virchowian model of cellular pathology was modified in the early part of
the twentieth century when;

“ The concept of specific etiology of disease received a great stimulus after
Pasteur’s work and after the demonstration by Koch and others that a whole
series of diseases was produced by distinct microorganisms. Most of the
infectious diseases suddenly fell into categories that could be explained on these
bases. All diseases then came to be viewed from an etiologic point of view.
Classification of disease, which was once related to pathological findings,
suddenly switched to considerations of specific cause of the disease. The unity of
tuberculosis was affirmed through the finding of the tubercle bacillus by Koch.
Finding the cause of infectious diseases paved the way for specific
therapy.”(p.528).

Following the discovery of the role that microorganisms play in disease
formation came the era of functional diagnosis;
“... German medicine abandoned the old precepts and maintained that the chief method in the study of disease should be the collection of information at the bedside by every means available to modern science. Animal experimentation should be used only when it was impossible to gain similar information from direct observation of patients.”(p. 528).

The gradual elucidation of the roles played by heredity (the constitutional factor) and the environment in the concept of physical disease appear to be the most important achievements of 20th century medical research into disease(p.529).

A social worker’s attempt to stretch the definitional boundaries of medical disease

In an effort to clarify the impact of heredity and the environment and to relate them to ‘mental illness’, Jerome C. Wakefield, a professor of social work, recently proposed a new concept of disease as, ‘harmful dysfunction’(Wakefield, 1992).

“Wakefield proposed that the proper analysis of disorder incorporates both value and scientific criteria. Specifically, he argued that disorder is best conceptualized as a ‘harmful dysfunction,’ whereby ‘harm’ is a societal judgment regarding the undesirability of a condition (i.e., the value component) and ‘dysfunction,’ is a ‘failure of a [mental or physical]mechanism in the person to perform a natural function for which the [mental or physical]mechanism was designed by natural selection(i.e. the scientific component).”(Lilienfeld and Marino, 1995, p.411).

This proposal is a strategy to subsume, in what Wakefield believes is a logical and coherent research program(evolutionary psychology), mental ‘disorder’, a watered down and therefore less empirical version of disease(Kendell, 1986, p.34), under the rubric of medical ‘disorder’. He breaks little new ground but rather follows the party line of medical modelers, by identifying, without providing any evidence, mental ‘disorder’ as a sub-category of medical disease and therefore just like any other disease(Wakefield, 1992, p.374). His argument is not
a critical attempt at analysis (corroborating evidence is never provided), but
rather a justification by fiat of what appears to be a strong prejudice in support of
the medical model. He dogmatically asserts what he should critically argue for,

"My critique of Spitzer and Endicott's[leading DSM theoreticians] analysis should
not be confused with the common argument, which I reject, that mental disorder
is an invalid concept. Indeed it is only because there is a legitimate concept of
mental disorder, manifested in a large number of uncontroversial judgments
about which conditions do and do not fall under the concept, that it is possible to
judge whether a specific analysis .. is correct."(Wakefield, 1992, p.161).

Wakefield is mistaken. What precisely is at issue, is whether there are any
conditions that fall under the concept of mental 'disorder' in the natural world and
no amount of consensual agreement, contra Thomas Kuhn(Kuhn, 1962, 1970)
can legitimate (justify) this claim. It has to be scientifically evaluated and
corroborated (or falsified) by examining the empirical evidence.

One should note that consensual agreements(Wakefield’s so-called
‘uncontroversial judgments’), although appearing to be democratic and respectful
are deceptive. Initially arrived at perhaps through discussion and debate and
therefore not ‘uncontroversial’, once decided they must be imposed on those not
part of the original dialogue who disagree. These judgments declare that through
simple majoritarian vote some peoples’(the majority’s or the controlling epistemic
elite’s) opinions about the world are to be taken as true without the requirement
of corroboration by independent data('objective facts'). This is relativism(Jarvie,
1984), a philosophic perspective which appears to fulfill our need for being seen
as holding non-judgmental, politically correct and sensitive interpretations of
various cultures, of having in other words a non-imperialistic world view. The
difficulty with this approach is that it leaves us empirically impotent, unable to criticize any culture or society and its practices from any outside framework - all of us being thought of as equi-distant from the eyes of God, of having equal epistemic authority (Munz, 1985). No outside observer can claim moral, ethical or scientific priority over the observed culture or society. Conflicting consensual agreements about ‘the facts’, according to relativism, are of equal merit and are undecideable, leaving for example the belief in witches, a universally held belief in the 16th and 17th centuries (Thomas, 1971), and the Nazi’s notions about race and biology (not to mention psychiatry) empirically on par with Einstein’s theory of relativity.

Wakefield’s analysis has been reviewed by Lilienfeld and Marino (1995). “On the basis of the repeated failure of Wakefield and numerous others to provide a satisfactory formulation of disorder in terms of scientific criteria, however, we hypothesize that disorder is a nonscientific concept lacking clear-cut natural boundaries.” (p.417).

I believe their critique of Wakefield is interesting but a bit confused. It is not so much that the concept lacks clear cut natural boundaries but rather that the concept identifies nothing in the world. That is, much like other Wittgensteinian thinkers, Wakefield realizes that concepts are first, before anything else words, with a particular word having no immanent meaning. That, meaning is ascribed by people and any meaning potentially possible at all, is potentially ascribable (Peckham, 1979). The constraining factors on meaning ascription usually have to do with a particular language community’s need and ability to stabilize meaning. This is necessary in order to promote reasonable (normative)
behavioral responses to various words or 'concepts' which are in turn necessary for the maintenance of the stability of the language community itself. Or to put it differently, as Wittgenstein did in his later philosophy, a particular language community’s use of a word is the meaning of the word. No outside criticism of the closed community’s claim is needed or allowed (Gellner, 1979; Munz, 1985).

Wakefield appears to believe that if he can intellectually justify the use of the concept of mental disorder as medical disorder (the claim or ascription made by the language community of Psychiatry) by the use of conceptual analysis, which is really an imposition of meaning, in this case Wakefield’s use of his arbitrary notions of disorder, that by itself is sufficient. This is not so, since our organism is such that we are capable of hypothesizing (using conceptual analysis), or more simply, thinking about anything quite randomly, whether or not there are real world referents to our imaginings. Such ideas, if untested or untestable, are of little value in solving problems, especially when the particular concept we are playing with, the concept of medical disease, has been well tested and has proven its explanatory soundness in its older definitional form, physical disease. It seems reasonable therefore that more then just a simple assertion (the result of conceptual analysis is no more then that) must be provided as to why the category of medical disease should be allowed to incorporate negative behavioral happenings which have no physiological origins (cause). The verbal sign ‘mental disorder’ does not pass the test, by the use of non-verbal signs (intersubjectively examinable data) which would allow it to be subsumed
under the more regressive verbal sign, acting in this case as an explanatory regress, of ‘medical disease’, or more weakly ‘medical disorder’ (Peckham, 1979). In any case I believe Wakefield’s hypothesis is false and should be set aside (I argue this in detail in a paper currently in preparation).

I note with regret however that Lilienfeld and Marino in their critique of Wakefield instead of arguing for the removal of the concept, mental disorder (that by their own argument they appear to believe is false, p. 416), from the scientific dialogue about problematic behavior, recommend instead that we pay attention to certain;

“well validated conditions that are deemed to require intervention by mental health professionals. ... and ... encourage them to focus their efforts on questions of ... critical importance - and scientific relevance - to abnormal psychology, such as those regarding the etiology, assessment, treatment and prevention of psychopathological syndromes ... ”(p. 418).

The authors suggest that there are some unspecified entities called ‘well validated mental conditions’. I conjecture, since the authors neither state how they arrived at their claim for the existence of such ‘well validate conditions’, nor further how they know of the entities which they call ‘psychopathological syndromes’, that these entities are also metaconcepts just like ‘mental disorder’, and just like other Roschian concepts (p. 416) appear to be without any critical attributes grounded in nature (p. 418). I fail to see how these theoretical entities differ from Wakefield’s concept and therefore respectfully recommend that they join ‘mental disorder’ and be interred with other similar explanatory musings in the graveyard of false knowledge.

Unfortunately the claim that an entity is well validated suffers an even greater
burden, that of being impossible. Lilienfeld and Marino appear not to be familiar
with Karl Popper’s refutation of induction (Popper, 1962, Miller, 1995) which I
discuss in the introduction. Sir Karl demolishes the claim that you can use
discreet instances(data) to support or ‘justify’ empirical generalizations. Such
generalizations are notoriously under-determined. You may make a thousand
observations confirming your hypothesis (that the sun rises for instance,
‘supporting’ the anthropocentric position that the sun circles around the earth)
and they will not provide an iota of well validated support for even what may be,
or as in this case, was in times past, considered blatantly obvious. This follows
from the empirically discovered fact that the earth revolves elliptically around the
sun and spins on its own axis producing the optical illusion of sunrises and
sunsets. Science cannot prove anything, it is only capable of critically testing, by
very severe falsifying attempts, our theories or hypothesis, in order to distinguish
hypothetically true theories (those which pass these tests) from those which are
false, because they fail them (Popper, 1962; Miller, 1995). Of course all such
hypothetically true theories are always open to refutation by some later test.

The aim of medical research or what do we look for when we want explanations?

This brief review of the evolving and therefore changing historical definitions
of medical disease and the effort by psychiatric professionals of identifying and
naming certain human behaviors - only those which are deemed culturally
negative or harmful, and, unlike the sign and symptom constructs of physical
disorders which can be bi-directional (low and high blood pressure for instance),
ever those that are positive or helpful - as signs or symptoms of mental
disorders or diseases, indicates some of the problems science and society face in trying to categorize bizarre behavior in an experimental format that will be empirically testable.

Disagreement about the true nature and etiology of behaviors considered symptomatic of or asserted to be mental illnesses confound our ability to puzzle out truth from falsity. The apparent endless flexibility of the construct of disease (Boyle, 1990) offers opportunities for many misunderstandings,

"Many of those who apply the diagnostic terms of psychiatry and medicine seem to believe that their subject-matter is 'diseases', illnesses' or 'disorders'. ... These claims are suspect because they smuggle into existence the problematic terms disease and illness and give the false impression that there exist 'out there' readily recognizable phenomena called diseases which naturally form the subject-matter of psychiatry. In doing so, they obscure the fact that the terms disease and illness are popular or lay constructions whose referents change in idiosyncratic ways with time and place. This leads to reification and the erroneous belief that researchers and diagnosticians 'name diseases' which are somehow possessed by people. But it is no more possible accurately to define the subject-matter of medicine or psychiatry than that of, say chemistry ... . All that can be said is that people who call themselves psychiatrists or geographers or what-ever tend by convention or even by public demand, to study certain phenomena. ... The fact that some of these phenomena have, in our culture, been construed as 'illnesses' or 'diseases' and have had various popular assumptions made about them, is no more relevant to their scientific study than is the fact that some of the phenomena now studied by astronomers or physicists were once popularly construed as 'magical' or 'miraculous'. What psychiatrists and medical researchers therefore try to classify are putative [non-random]patterns of ... physical and behavioural phenomena" (Boyle, 1990, pp. 81-82).

Despite these difficulties, I believe based on this general method of science - the search for non-random patterns of phenomena which have explanatory power - we can test for the existence of the specific entities that have been asserted to be discrete mental disorders/diseases, such as schizophrenia, by seeing if such patterns can be found.
Since every historical and contemporary account of Schizophrenia research identifies Emil Kraepelin (1856-1926) and Eugen Bleuler (1857-1939) as either the individuals who isolated this putative disease or as the chief theoreticians of the concept, we can examine what Bleuler and Kraepelin actually did in order to receive this recognition. Modern researchers appear to take it for granted that these two German alienists did empirical research, and that this research identified a group of behaviors that cohered distinctly and non-randomly together in nature, forming a syndrome that was caused by the inferred, non-observable antecedent, schizophrenia (Strauss & Carpenter, 1981; Freedman et al., 1986; Meehl, 1989; Gottesman, 1991; Straube and Oades, 1992; Shore, 1993).

To evaluate the proposition that they, as claimed, did do such research and that this research demonstrated a discrete schizophrenic syndrome, requires us to: 1) identify what particular definition of disease or disorder was current during the time that Bleuler and Kraepelin were making their claims; 2) to examine the evidence regarding whether or not they did ‘research’ and of what sort, as they identified schizophrenia, and 3); to evaluate whether the evidence they provided passed any critical empirical tests attempting to falsify the ‘schizophrenic syndrome’ construct. The answers to these questions are important because if the answers turn out to be in the negative, and Kraepelin and Bleuler did no systematic empirical research which identified a coherent, regular pattern, but instead only made assertions based at best on some small biased sample of clinical observations which were never tested, then we have a potential empirical refutation of institutional psychiatry’s claim that the phenomenon of madness or
insanity is best considered a medical disease(s) - or more correctly a syndrome(s);

“The search for patterns and attempts to describe relationships between phenomena are central to scientific activity. They are central, too, to much non-scientific activity; of particular interest here is that they are central to lay or everyday attempts to understand behaviour and other events. What perhaps distinguishes the two is the scientist’s persistent demand for the provision of certain types of evidence that a pattern has been observed and the imposition of various publicly demonstrable criteria for evaluating it. This demand for evidence, and its public evaluation, is crucial in view of our apparent propensity to claim that certain events ‘go together’, in the absence of any direct evidence that this is the case (see, for example, Mischel, 1968; Shweder, 1977)” (Boyle, 1990, p.1).

My research hypothesis

I conjecture that Kraepelin and Bleuler did not observe any legitimate medical syndromes\(^{11}\). A syndrome, consisting of both signs and symptoms (Engle and Davis, 1963, p. 518), is the minimum non-random pattern that must be shown to exist to infer a medical disease. Rather, I think because like most alienists of their time they were committed adherents of the medical model of mental illness, a view with a long history\(^{12}\), used this paradigm to justify selecting certain ‘anti-social’ behaviors, calling them signs or symptoms and claiming that these behaviors occurred consistently, discretely and non-randomly together - forming

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\(^{11}\) According to Engle and Davis (1963), “In diagnosing different diseases we often use entirely different types of fundamental criteria. We may have a concept of disease and, therefore, diagnosis based upon gross anatomical defects, microscopic anatomical changes, so-called specific etiologic agents, specific deficiencies, genetic aberrations, physiological or biochemical abnormalities, constellations of clinical symptoms and signs, organ and system involvement and even just descriptions of abnormalities. Each diagnosis has its own degree of certainty.” (p. 517, italics added)

The least certain - diagnosis of the fifth order of certainty- are syndromes. A syndrome is, “... a group of signs, symptoms and laboratory findings which is not as definitive as a disease.” (p. 519).

\(^{12}\) This model had been claimed by the medical field for well over 200 years, since about the middle of the 17th. century, to at least theoretically account for madness (Scull, 1993, p.179). I say, theoretically, because medical knowledge was incapable of scientifically explaining even physical disease in the sixteen hundreds, since the false theory of disease, humoralism, was still the predominant medical paradigm (Footnote 16, p. 179). Nothing has been discovered in medicine since then to clear up the ‘mystery’ of madness. However at least one alternate conceptual model exists which better explains the behaviors currently subsumed under
a schizophrenic syndrome - without offering any further scientific evidence. All this was claimed to have been ‘discovered’ among the very heterogeneous populations of their asylums,

“Very early on in the history of the asylum, it became apparent that its primary value to the community was as a handy place to which to consign the disturbing, the vaguely menacing, the unwanted, and the useless - those potentially and actually troublesome people who posed threats to the social order and to the business of daily living which were not readily subject to control by the legal system.”(Scull, 1993, p.352).

This idiosyncratic identification of the ‘symptomatic’ behaviors with disease (which completely disregarded the strong likelihood that many of these behaviors may have been the iatrogenic results of the institutions themselves (Schull, 1993, chapters two & six) and not of mental illness) ignored arbitrarily, innumerable other ‘strange’ behaviors among this serendipitous group which could also have been asserted with equal (im)plausibility to have formed any number of alternate putative syndromes. My conjecture, if found to be hypothetically true, not falsified by the best available historical evidence, would substantially undermine the fragile theoretical edifice of modern psychiatry.

In order to play fair with the protagonists I will attempt to use their own words where ever possible. This use of primary sources is rare among modern mental health researchers. Most rely on secondary sources and the claims and interpretations found in them as evidence, a fatal mistake as I hope to demonstrate.

Definition of disease during Kraepelin’s and Bleuler’s time.

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insanity, Thomas Szasz’s Rhetorical Paradigm(Vatz & Weinberg, 1994).
“A psychosis is generally a complicated structure which may manifest itself in very different ways, not only from one patient to another, but in the same patient at different times. The manifestations were formerly taken for the diseases themselves, and even yet it is of practical value to emphasize them as *pictures of morbid states and syndromes*.” (Bleuler, p.161, 1924).

Although from the very beginning of scientific thinking about disease there had been an ongoing discussion among medical researchers as to whether disease entities were autonomous and could exist separate from and outside of the host body or whether they could only be considered as existing within the host (Engle, 1963, p 117), by the late 19th century the dominant paradigm was that of Rudolf Virchow (1821-1902) and cellular pathology,

“This science, which naturally includes a cellular theory of the living, proceeds from the fact that cells are the actual operative parts of the body, the true elements themselves, and that all vital action proceed from them. Just as life itself is only expressed by means of action, so is the knowledge of the various kinds of activity and its disturbances the actual task of pathology. ... Most diseases are not elementary processes but rather compound processes where alterations of several or many cell territories co-exist or range themselves alongside each other. As a consequence, further investigation of the cells or cell groups under consideration, indeed a repeated investigation of the place or places of the disturbances, is required not only for special pathology, but also for the theory of disease in general, i.e., of the ‘where’ of the disease, therefore of the anatomical parts involved thereby. This investigation is to be carried out not merely with the knife, but also according to the usual authentic anatomical methods. It is generally necessary to use experimental and clinical investigation.” (Virchow as translated by Engelhardt in Caplan et al., 1981, pp. 1990-1).

Virchow’s view of medical disease emphasizing cellular deviation was compatible and not in conflict with the view of the bacteriologists of his era, contrary to what has sometimes been claimed (see Boyle, 1990, p 9). Witness his explicit

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13 *Kahlbaum deserves the credit for having made a deliberate and sharp differentiation between ‘conditions’ and ‘diseases’ [in his book *Grouping of Psychic Diseases* (1863)], even though in his time the position of science had not yet made it possible to circumscribe natural morbid pictures more exactly.” (Bleuler, p.161, footnote 1, 1924).
acknowledgment, “That an actual parasite[bacteria], whether it is plant or animal, can become the cause of a disease ... “(Caplan et al., 1981, p. 193).

He characterizes ‘Scientific' medicine’s objective,

“... as ... the discovery of changed conditions, characterizing the sick body or the individual suffering organ. Its object is also the delineation of deviations experienced by the phenomena of life under certain conditions, and finally the determination of means by which these abnormal conditions can be counteracted. This therefore presupposes the knowledge of the normal course of the phenomena of life and of the conditions under which this course is possible. Its foundation is thus physiology. It is composed of two integrated parts: pathology, which transmits or should transmit the knowledge of changed conditions and of change phenomena of life; and therapy, which determines the means by which to counteract these conditions or to maintain the normal ones.” (p. 188)

The Virchowian physiological causal model of disease continues to serve as the basic model for disease in scientific medical discourse throughout the world. Fundamental abnormal physiological change in the organism is the **limiting parameter** for the definition of disease. Despite the not inconsiderable efforts by mental health experts to move away from this bottom line(Kendell, 1975a; 1975b; Kraupl-Taylor, 1979; Spitzer and Endicott, 1978; Wing, 1978), pathologists, the medical specialists who look for diseases, have not deviated. There simply are no pathological texts that recognize mental illnesses as medical diseases. For example, we are told by Stanley Robbins, a leading scholar and pathologist, whose *Pathologic Basis of Disease* is a standard text in medical schools that pathology is the discipline which, “deals with the study of deviations from normal structure, physiology, biochemistry, and cellular and molecular biology.”(Robbins, S.L. et al, 1984, p.1). Psychiatry or psychopathology is never mentioned.
Pathologists, the experts who study real diseases, only evaluate physiological abnormalities of the physical organism and only view these physical abnormalities as disease entities.

Kraepelin and Bleuler both believed in this model, and, as suggested earlier, conceptualized their theories about mental illness in line with it.

“GENTLEMEN,- The subject of the following course of lectures will be the Science of Psychiatry, which, as its name implies, is that of the treatment of mental disease. It is true that, in the strictest terms, we cannot speak of the mind as becoming diseased, whether we regard it as a separate entity or as the sum total of our subjective experience. And indeed from the medical point of view, it is disturbance in the physical foundations of mental life which should occupy most of our attention.” (Kraepelin, p. 1, 1905).

“The conviction that the key to the understanding of insanity was to be found in the condition of the brain had already focused the attention of alienists on this organ.” (Kraepelin, 1917, 1962, pp. 119-20).

Bleuler similarly applies the model to his definition of mental syndromes,

“Syndromes are complexes of symptoms that belong together genetically. A part of such pictures as manic depressive insanity, eventually with their corresponding delusions, represent at the same time such syndromes. One speaks, furthermore, of an ‘organic symptom complex’... and understands by this term the sum of the psychical fundamental symptoms of a diffuse atrophy of the cortex, or of a general lowering of the function of the cortex through shock or injury to the brain.” (Bleuler, 1943, p. 164).

He also identifies the usual medical notion of acute disease with those he calls mental diseases,

“The observation that an acute disease may result in permanent damage to the affected organ[the mind] has far greater significance in psychiatry than in any other field of medicine.” (Bleuler, 1950, p. 3).

The reasons why Kraepelin and Bleuler along with other alienists, adopted this medical framework have been well documented by a number of medical historians (see Scull, 1993; 1989; 1981; Porter, 1987; Skultans, 1979; Szasz
1994; 1987; 1977; 1976; Bynum, 1985; 1982). Much of it had to do with the need
to be seen as scientific, to gain professional recognition, prestige, power, and
financial success - this of course did not preclude other motives, such as the
desire to genuinely help an apparently troubled group of people. Perhaps more
importantly, there were no scientific rival frameworks about human behavior,
normal or deviant, to challenge the disease model. This lack of alternate
explanatory paradigms made it very convenient for alienists to use medical
explanations for strange overt behavior. In order to avoid or reduce anxiety about
the unknown human beings appear to require explanations for why things
happen. In fact the search for explanations may be the prime role of our cognitive
faculties (perhaps genetically determined, Peckham, 1978). This holds true
especially for difficult to comprehend events or unusual behavior. Where better or
true alternatives are not to be had, bad or false explanations will do, as long as
they appear to be plausible. Any explanation is apparently better then none for
reducing anxiety. For proof of this, witness the popular news media’s attempts to
explain the petty and major crimes and tragedies in our everyday lives
‘scientifically’ by the use of professional experts, often from the mental health
industry.

The use of existing frameworks(paradigms) without question by scientists is
identified by Thomas Kuhn as the practice of ‘normal science’(Kuhn, 1962,
1970).
“The skills that a scientist brings to bear on his subject are not natural, or innate. They are learned skills. And implicit in Kuhn’s description of normal science is the thesis that all learning is by example. A paradigm, then is a skillful achievement from which others learn by admiringly repeating the technique employed.” (Hattiangadi, 1987, p. 192).

Kuhn claims that this is the practice of most scientists most of the time and quite appropriate since he holds that scientific validity is decided by a central committee vote of the ruling scientific hierarchy of any particular time. Karl Popper disputes this position and believes the unquestioned acceptance of frameworks leads to poor science and often encourages ignorant and relativistic claims which go unchallenged because the currently favored paradigm is not allowed to be critiqued (Popper, 1994).

Be as that may, Kraepelin and Bleuler were both normal scientists in Kuhnian terms, faithfully adhering to the physiological causal model of illness in their writings. A crucial additional fact, the discovery and corroboration of the existence of a real physiologically determined disease which had behavioral consequences, namely Syphilis, seemingly lent strong empirical support to the approach heralded by these 19th century alienists. The title of alienist was by the end of the 19th century replaced with the more medical sounding and less socially 'alienating' honorific of psychiatrist in use since.

Boyle (1990) in a somewhat lengthy but important quote crisply lays out the essential arguments supporting the syphilitic model of mental illness,
“There can be little doubt that the growing body of evidence about the nature of infectious diseases, including syphilis, must have helped to convince psychiatrists that they were on the right track as far as methodology was concerned. For probably the first time in medical history, there were strong indications that adopting a particular theoretical framework would lead to a successful outcome. Moreover, medicine’s successes had begun with - indeed, would never have been attainable without - careful observation of phenomena as they presented themselves to the clinician, not by speculation about unknown causes. Dementia paralytica appears to have been particularly common in northern Germany and Kraepelin - who was greatly influenced by Kahlbaum - wrote extensively on the subject. The role of the discovery of the dementia paralytica - syphilis link may therefore have been to confirm and extend the belief in a particular way of working, rather than in a somatic theory of deviant behaviour, which was in any case already well-established. And the length of time it had taken to discover the link may have given psychiatrists hope in the face of their poor empirical results.

But it is notable that those who emphasized the importance of the clinical observation of symptoms, which included bizarre behaviour, did so in apparent ignorance of the considerable methodological problems of observing and recording behaviour; indeed, from their emphasis on brain pathology it was easy to lose sight of the fact that, in many cases, their subject-matter was behaviour. They also ignored the problem of knowing which behaviours should be thought of as symptoms, given that no pathology could be demonstrated on many inmates[of their asylums] and, in those in which it could, there were no clear links between it and behaviour. Instead, it was implicitly reasoned that people were confined to asylums because they suffered from mental disorders, Therefore, what the clinicians observed in the asylum, and considered abnormal, must be the symptoms of mental disorder. But the concepts of insanity and mental disorder were lay and not scientific concepts and their referents were idiosyncratic, many and varied.”(p. 41).

The evidence for the claim that both Kraepelin and Bleuler where relying on the syphilis model when looking for explanations of 'mental disorders' is to be found in their own writings.

First Kraepelin,

“The greatest step toward understanding the etiology of mental disease was the discovery that paresis resulted from syphilis. ... Recent research in syphilis, especially the priceless discovery of the Wassermann reaction, cast light on the importance of the dreadful affliction to psychiatry. We can expect this light to illuminate still other spheres of our science.”(Kraepelin, 1917, 1962, p. 130).
“The nature of most mental disorders is now obscured. But no one will deny that further research will uncover new facts in so young a science as ours; in this respect the diseases produced by syphilis are an object lesson. It is logical to assume that we shall succeed in uncovering the causes of many other types of insanity that can be prevented - perhaps even cured - though at the present we have not the slightest clue”(pp. 151-2).

In his *Lectures on Clinical Psychiatry* (1913), Kraepelin causally connects the problem of insanity to the use of chemical agents and syphilis, suggesting presciently that some insanity is the iatrogenic effect, like tardive dyskenisia is of psychotropic medications (Whitaker, 1993), of the drugs being prescribed by doctors for illnesses real and imagined,

“Even though the limits of his power against this mighty adversary [mental illness] are very narrow, opportunity enough is afforded to every practical physician to contribute his share to the prevention and alleviation of ... mental disease. Alcoholism and syphilis undoubtedly offer the most profitable points of attack, together with the abuse of morphia and cocaine, which so clearly owes its fatal significance to the action of medical men.” (Kraepelin, 1913, p. 3).

Then Bleuler,

“... we believe that an advance has been made [by the concept of schizophrenia] which is even greater than the progress made by the discovery of the etiology of general paresis. The latter syndrome too was for a long time obscured by many other symptom-pictures. We feel that the dementia praecox problem involves much more deeply the entire complex of the systematics of all the psychoses than the problem of general paresis ever did in its day.” (Bleuler, 1911/1950, p. 5).

“In recent years *anatomical investigations* have carried us forward very nicely, in that, for instance, a clearly circumscribed picture could be distinguished of dementia paralytica [a form of syphilis], of various forms of senility, and of several diseases that may be designated clinically as epileptic.” (Bleuler, 1924, p177).

“... all authorities tend to consider schizophrenias as poisoning or some similar novum of the body. In that case, the disease should be placed alongside the paresis group and it ought not to show any transitions to other diseases.” (Bleuler, 1911/1950, p 276).
By 1896, the time of Kraepelin’s earliest publications on dementia praecox, the great physician and syphilologist Alfred Fournier had for some twenty years been writing about the connections between syphilis and general paresis of the insane (GPI), and by 1894 had demonstrated the causal relationship between GPI and tabes (Quetel, 1990, p.163). The similarity between the late 19th century medical description of the symptoms of syphilis and Kraepelin’s description of the symptoms of dementia praecox (schizophrenia) is striking. Syphilis, “In 1884, the official dictionary of medical science definitively establishes the specific character of progressive general paralysis [meningoencephalitis of tertiary syphilis]. The onset is of variable length, with intellectual enfeeblement and disturbances of the personality and the moral sense, and it is marked by an apoplectiform ictus or a particularly violent fit of mania. Then follow the classic symptoms of impaired speech, pupillary inequality, disruption of movement (particularly in locomotion) and delusions of wealth and grandeur.”(pp.161-2).

Note how Kraepelin closely parallels the dictionary’s description of general paralysis when discussing the characteristics of dementia praecox,

Variability;
“The general course of dementia praecox is very variable.”(Kraepelin, 1919/1971, p. 181).

Enfeeblement;
“.. they lead to a more or less well marked mental enfeeblement.”(p.1).

Disturbance of the personality;
“.. the common characteristic of which is a peculiar destruction of the internal connections of the psychic personality.”(p. 3).

Disturbance in the moral sphere;
“.. sudden oscillations of emotional equilibrium of extraordinary violence may be
developed. In particular sudden outbursts of rage with or without external occasion are not infrequent and can lead to most serious deeds of violence."(35).

“Not infrequently ideas of sin also appear, and in connection ideas of persecution.”(p. 105).

Physiological disturbances;

“The spasmodic phenomena in the musculature of the face and of speech, which often appear are extremely peculiar disorders”(p. 83).

“Behaviour of the Pupils.-This is of great significance. ... Here and there one observes a distinct difference in the pupils.”(p. 77).

“Psycho-motor Domain.- Various and profound disorders are found here. Dufour has described disorder of equilibrium, staggering, adiadochokinesia, and tremor, which he regards as the expression of a ‘cerebellar form of dementia praecox.”(p.79).

Delusions of wealth and grandeur;

“Exalted Ideas.-In a large number of cases ideas of exaltation are added ... . He[patient] lives in Berlin gets a uniform must go to the Kaiser, can become minister and pope, will be a good match, will get a great inheritance, gets milliards from God; at the war-office there is gold deposited for him.”(pp. 29-30)

The description of the two ‘diseases’ is virtually identical. Perhaps the only difference being that Kraepelin and the rest of the medical world in the late 1890’s knew that syphilis was a real physical disease with organic antecedents, whereas the putative disease dementia praecox and its causes,

“.. are at the present time[even in the year 1996] still wrapped in impenetrable darkness.”(Kraepelin, 1919, p.224)

It should be noted that all of the quotes above support the claim that mental diseases are real based on their putative physiological origin and their apparent similarity to the symptom pictures of those diseases that have behavioral manifestations and have been found to have bio-chemical etiologies and effects
on the body. Kraepelin and Bleuler both believed that all mental diseases would turn out in the end to be caused by physio-biochemical agents just as these diseases.

"Kraepelin’s aim was to define disease entities based on definite organic pathology, and he always felt that dementia praecox was ultimately based on a faulty metabolism, although he could never prove it."(Arieti, 1959, p. 456).

This is still the expressed hope of modern mental health researchers and they also have failed to prove it.

Although Kraepelin and Bleuler are exceptionally ambiguous and hedge all of their claims about disease in general and schizophrenia in particular in their writings (as will be seen when we examine their ‘research’) making it very difficult to know exactly what they believe, the following quote from Bleuler, taken together with Kraepelin’s quote on p.43, about the ‘physical foundations of mental life’, is strongly suggestive,

“Complete justice to all these factors[the schizophrenic symptoms] can only be done by a concept of the disease which assumes the presence of (anatomic or chemical) disturbances of the brain; the course of the cerebral disorder is chronic, ... the disturbance of the brain determines the primary symptoms(disconnection of association, perhaps the disposition to hallucinations and stereotypies, a potion of the manic and the depressive syndromes and of the states of clouded consciousness).”(Bleuler, 1911/1950, p.463).

However to illustrate their confusing, self-contradictory statements about their beliefs compare the following statement about disease by Bleuler with the preceding,
“The whole difficulty lies in the fact that there is no definition of “disease” and there cannot be any. The fruitless controversies can only cease when the ambiguous and indefinite concept is entirely excluded. *It is easy enough* to examine how a person is and reacts, and draw conclusions *from the facts*, instead of from a concept, and then determine our actions accordingly” (Bleuler, 1924, p. 171).

The remarkable thing about this quote in a book claiming to be a textbook about mental diseases, is its similarity to the approach Thomas Szasz recommends in dealing with behavior of the deviant kind. Szasz believes that ‘insane’ behaviors, like all human acts, are volitional and meaningful, requiring contextual analysis and voluntary treatment if elected by client, and not entities constructed of symptoms(behaviors) which are by definition to be seen as unmotivated diseases or syndromes requiring treatment in all instances, coerced if patient consent is withheld (Szasz, 1961; 1965; 1970; 1976).

Kraepelin and Bleuler’s commitment to the organic etiology of mental illness has undergone a fundamental reinterpretation by modern psychiatric researchers. By emphasizing the historical plasticity of the definition of disease in its capacity to incorporate new medical discoveries, they wish to claim that psychiatrists’ observations of socially unacceptable behavior in and by themselves are sufficient to establish disease entities (Kendell, 1986, pp.23-45). They argue, that what they call clinical syndromes (consisting only of observed behavior) can be asserted to be real without requiring them to be grounded in physiology (see next section, ‘syndrome transformed’ for clarification).

This is contrary to the stated beliefs of Kraepelin and Bleuler (but not necessarily to their actual behavior) as shown previously, and is an assertion
without any credible evidence. Although it is quite true that various diseases have been found to have a variety of causes such as; cellular derangements, bacterial agents, abnormal chromosomes, genes, and molecules (all of which are physio-biochemical structures), medical researchers always assume a physio-biochemical etiology for their diseases and attempt to demonstrate such causal relationships through experimental research.

Kraepelin and Bleuler, having been brought up in the Virchowian paradigm supporting the idea of natural disease entities, derived further sustenance from the finding that, “single micro-organisms, with an independent existence, were responsible for certain clusters of phenomena with their own, apparently natural, course and outcome.” (Boyle, p.10).

This was the case in syphilis. As good normal scientists they assumed that the various peculiar behaviors of the inmates of their asylums were the result of the biological activity of some as yet undiscovered infection (of some micro-organism) just like the peculiar behaviors observed in some syphilitics. Based on their framework they accepted, more likely even expected, that inmate behavior would fall into natural behavioral clusters which deviated from some hypothesized normality and that such clusters would each have their own antecedent, a dementia praecox/schizophrenia ‘germ’ perhaps, with a unique capacity to produce a non-random cluster (syndrome) (Boyle, 1990).

In other words, having the syphilitic model as an apparently well corroborated guide, they imposed this model on their asylum charges and looked for similar behaviors, and, not surprisingly found them among this ‘selected’ population of
inmates. They then proceeded to claim that these behaviors formed a syphilis-like syndrome called the dementia praecox/schizophrenia syndrome. This is exactly the opposite of what is usually done in medical research. There, unselected populations are observed to see if new non-random groupings of signs and symptoms occur, or alternatively and more frequently, individuals come to the attention of doctors with certain clinical complaints, various signs and symptoms that seem to emerge, recur and group non-randomly over time. These groupings must and do contain independently testable objective signs like glucosuria for diabetes, not just symptoms (behaviors) which provide the empirical evidence necessary to corroborate research claims.

**Syndrome transformed**

Before I discuss what sort of research was done by Kraepelin and Bleuler, I want to identify a subtle maneuver, hinted at earlier, that was undertaken by psychiatric researchers as they attempted to establish the existence of mental disease as a sub-category of medical disease. As noted in footnote 11, both signs and symptoms must be present in a grouping before it can be called a medical syndrome, otherwise the postulate is only a putative clinical syndrome, a term which can only be suggestive and of almost no empirical value.

Psychiatrists altered this requirement slightly but with dramatic results. Without giving any reasons or even acknowledging the change, they shifted from requiring both symptoms (behavioral manifestations) and signs (objectively measurable observables, like temperature, blood pressure, glucose in the urine) to be present when looking for ‘psychiatric’ syndromes (the non-random
occurring clusters that infer unobservable antecedents like schizophrenia) to **only looking for symptoms**. This they had to do of course, since observed behavior is the only phenomena psychiatry has accessible to ‘research’. No objective psychiatric signs are available or possible, exposing the futility of the claim that mental illness is like any other medical illness and necessitating the fudging of accepted medical research requirements.

“By the presence of the symptom-complex so selected and defined, the great group of dementia praecox is characterized as a unit.”(Bleuler, 1950, p.1)

“These classifications[of the sub-groups of schizophrenia] are based on the prominent syndromes, that is, the characteristic symptoms”(Straube and Oades, 1992, p.15).

“... the diagnosis of schizophrenia can be regarded as dependent on the presence of certain syndromes, symptom clusters.”(Stromgren, 1991, p.34).

“The concept of a syndrome ... denotes a cluster of symptoms with a characteristic temporal evolution ... . In the case of schizophrenia and most other mental disorders those symptoms are a mixture of abnormal or distressing subjective experiences ... and observable abnormalities of behavior.”(Kendell, 1991, p.63).

This view of syndromes is unique to the mental health field. Physical medicine requires the presence of both signs and symptoms. Why is this shift a problem? Well, symptoms (e.g. pain, sweating, nausea, hallucinations or delusions) are often not directly observable by an onlooker but are the verbal reports or claims of an individual. Verifying the accuracy and veracity of such reports is problematic. The reliability of the accurate reporting of such observations therefore may be very low and the actual causes of the behaviors may be many, that is they are over determined. For example, headaches may be due to either a blow to the head or a brain tumor. If, on the other hand the symptoms are
'observable abnormalities of behavior' like 'blunting of affects', then we are left to rely on the accuracy of the subjective observations of those judging such affects, and again questions of reliability and validity present serious difficulties. Consequently these behavior clusters may be present by chance alone; or be non existent (false statements, willful or mistaken) and of little value in identifying the existence of an underlying process like schizophrenia reliably and validly.

For these reasons medical researchers demand that an apparent clustering of symptoms should be reliably (non-randomly) associated with some independently measurable occurrence(s) called signs. Signs are directly available to an observer eliminating the need to evaluate the reliability of the reports of the sufferer or of having to interpret the cause and meaning of the observable behavior (screaming, crying, bashing of body parts against walls, shivering) of the individual to be diagnosed.

"In order to clarify the distinction between signs and symptoms in medicine, Kraupl-Taylor(1979) use the example of glucose in the urine. This is designated as a sign because it can be reliably measured by an external observer. Its frequency of occurrence is less than is the individual frequency of events with which it may be associated (excess urine production, thirst, tiredness, [emaciation, sweet tasting urine,] .. ) and because there are plausible grounds for assuming that it is not a consequence of these. The term sign suggests that an event signifies, or is indicative of, another event which can be independently observed. The presence of glucose in the urine may signify hyperglycemia (itself an over determined event) or diminished glucose reabsorption in the renal tubes. Because hyperglycemia fulfills the criteria listed above, It too is designated a sign. Signs are often mistakenly said to be indicative of a syndrome name as, for example, when high blood glucose levels are said to be a sign of diabetes. Such statements are obviously tautological because the syndrome name does not refer to an observable event which can be measured independently of the sign"(Boyle, 1990, pp. 12-13).

Medical researchers by insisting on the presence of both signs and symptoms
for the legitimization of putative clusters of events as syndromes seek to
guarantee that they are using the best empirical data for their claims. People who
are identified with a medical syndrome will tend to share at least one sign in the
cluster, although they may have different symptoms. This may happen because
the same sign may have a multiplicity of symptoms. There may even be, among
various syndromes, shared signs as well as common symptoms. Nevertheless
keeping discrete groupings has proven to be of explanatory value because,

“Only some of those who show glucose in the urine, for example, will show
hyperglycemia and those who do will in turn, show a variety of pancreatic
abnormalities. It is therefore assumed that different processes led to the
appearance of the signs in the various groups. This assumption may be
supported by the observations that between-group variability in progress over
time is considerably greater than within-in group variance; that the groups,
without intervention, reach obviously different end-points, for example early death
versus average life-span, or that response to the same intervention is quite
different between groups.” (p. 13).

This shift, requiring only symptoms, which are further confounded in
psychiatric literature by sometimes being incorrectly called signs as well (see
such misuse in Straube and Oades, 1992, pp. 17-18), to be constitutive of
syndromes, has allowed psychiatry to appear to be identifying the same type of
constructs as are found in medicine when in fact they are identifying much more
unreliable phenomena. The important point to be made here is that neither
Kraepelin, Bleuler or any other psychiatric researcher has used the notion of
syndromes the way medical researchers do.

Putting this very important point aside temporarily - let’s accept this shift for
the sake of our investigation - but let’s keep in mind that the necessary condition
for inferring a hypothetical construct is a grouping of regularities reliably and
validly observed and that the claim for the existence of schizophrenia by these researchers implies that such regularities were observed in the behavior of their patients (Boyle, 1990, p.14). Therefore if Kraepelin and Bleuler can be shown to have found such a regular pattern by their research among their insane asylum populations then we have some evidence - remember their syndrome is a weaker construct then usually applied in medicine - to suggest the existence of the schizophrenic syndrome and by identity the existence of mental illness.

THE RESEARCH

Did Kraepelin and Bleuler observe the necessary set of regularities, even by their less ‘scientific’ syndrome definition, sufficient for inferring dementia praecox/schizophrenia?

It should be stated immediately that I am not interested in whether they observed strange behavior in their asylums. Clearly they must have, since the asylums were created to house people who behaved oddly. What I am interested in is whether Kraepelin and Bleuler observed syndrome-like regularities using good science, among this universe of bizarre behavior which would support their claim for the existence of schizophrenia. Good science then as today requires the gathering of systematic data, having reliable and clear descriptions of observations so others can replicate them, avoiding reliance on personal experience and belief in the absence of systematic evidence, and where possible using empirical tests to evaluate hypotheses. Rudolf Virchow(1821-1902) writing contemporaneously with our alienists, bases the validity of scientific medicine strictly on its ability to make careful observations and to test its hypotheses
through experimental research.

“The fact that, for a number of the most significant contagious and infectious
diseases, it is possible to verify not only the invariable existence of
microorganisms, but also the experimental proofs of their activity, is sufficient to
show the great progress made by natural scientific knowledge in this difficult
area, which only half a century ago was still endangered by the most arbitrary
assumptions concerning the causes and conditions of pestilences and
contagious diseases. ... the great and consoling difference in contrast to the
method of the earlier speculative and aprioristic pathologists lies in the fact that
every step on the way of the contemporary researcher can be controlled exactly,
and that even the greatest enthusiasts[those with dogmatic beliefs, must]start
from real things which are approachable by means of experimental

Since medicine of Kraepelin and Bleuler’s time used the above research
methodology for its pathological discoveries I will apply these criteria to them and
their ‘psychopathological’ discoveries and see how they fare in comparison.

Kraepelin’s writings dealing with dementia praecox can be separated into two
loose temporal categories of early - texts published in 1896-1899, the time from
which most experts claim the modern construct of schizophrenia originated - and
late - from the 1913/19 text. For the early material I will be relying on Boyle(1990)
for translations and quotes since these texts were not available in translation for
my review.

**Early writings:**

In 1896 Kraepelin wrote that to identify a hypothesized meaningful cluster of
bizarre behaviors he would use as the criterion similarities of onset, course and
outcome of such behaviors. Kraepelin, following Thomas Syndeham’s disease
definition(Kendell, 1986), was thereby asserting that if he could find a group of
people whose behavior was similar at a particular point in time(onset) and shared
similar development over time (course) and reached a similar end (outcome) then he would be justified in positing an as yet unobserved but real process or causal agent to account for this putative non-random clustering of behavior. The reason why Kraepelin used onset, course and outcome, especially outcome, as the relevant criterion for identifying dementia praecox was that syphilis had by this time been shown through experimental research to have very clear outcomes, and as I asserted earlier this model served as the prototype for Kraepelin’s view of mental diseases,

“Gentlemen,--- I think that our best guide to the clinical meaning of the various morbid conditions with which we meet is their termination. ... a purely empirical knowledge of the termination of diseases must be of the greatest value to the physician, as it gives him the power of predicting the further course of his cases. ... The first disease to be recognized as a unit, on account of the regular law of its development and in spite of the variety of its forms[just as he claimed dementia praecox manifested itself], was general paralysis of the insane[syphilis]. In this disease the delusions of grandeur and insignificance, the excitement and depression, the delirium and hallucinations, all terminate in a peculiar mental weakness of extreme intensity with physical enfeeblement. The end is everywhere the same” (Kraepelin, 1913, pp. 193-4).

Difficulties with the criterion of ‘onset, course, and outcome’

Boyle(1990) points out(p.45) that although these terms, onset, course, and outcome, appear to describe simple clear and distinct occurrences, when it comes to reliably and validly observing behavior they turn out to be quite complicated and continuous processes. The observations of these events by various researchers may be haphazard and subjective. Different observers at different times may see different events or behaviors as important, or as the appropriate cut off or transition points from which to infer onset, course, and outcome.
In large heterogeneous populations, as were in these asylums, a great variety of shared and distinguishing behaviors could be observed, making the identification of discrete groupings a very unsystematic procedure. For this reason in particular, the requirement of correlating an independently measurable, reliable event, a sign, with the observed ‘symptomatic’ behaviors is essential for the identification of a true cluster. Additionally, if ‘outcome’ is to be one of the criteria evaluated then every member of the cluster group must reach a point beyond which no more change can occur (where outcome can be said to have been realized). How one might actually establish this ‘final stage’, outside of the obvious and in the case of schizophrenia very rare occurrence of all members dying (Bleuler, 1911/1950, p.254), is highly problematic. How could one know that some further change might not occur sometime in the future? Finally the identifying of any new representatives who conform to this putative clustering must await their reaching the agreed to ending (what ever and when ever that might be); delaying the ability to diagnose beyond reason and severely limiting the progress of research.

Method of research

Although these are significant problems, explaining why most medical research uses other criteria, they don’t necessarily invalidate Kraepelin’s work. If similarities of onset, course and outcome were found and described adequately by him then we would have at least the beginnings of the search for potential signs to associate with a putative cluster. Ordinarily medical researchers when searching for regularities in heterogeneous populations present data
demonstrating that some members of this population share similarities which
are not found in other members of the population or if there are apparent
similarities their antecedents (causes) are different. The researchers also have to
demonstrate that the group showing the similarities is homogenous in some
essential way, that we are not witnessing a simple co-occurrence by chance
alone. After the presentation of the data the researchers then would end by
postulating a new construct. In 1896 Kraepelin did no such thing,

“Instead of concluding by inferring his construct, having presented evidence in
support, Kraepelin began with the construct and proceeded to describe what he
called cases of dementia praecox. He did not report the number of these who
conformed to the descriptions he presented. These data, however, are crucial to
claims that important shared features have been identified. Rather Kraepelin’s
descriptions are in the form, ‘one often notices’; ‘it is occasionally observed’; ‘in
some cases’; and so on. Thus Kraepelin wrote as if by some independent and
valid criteria, established by past research, dementia praecox had already been
inferred in this sample and he was merely engaged in recording his impressions
of the group. He wrote, that is, as if data supporting the introduction of his
concept had already been presented when in fact they had not.”(Boyle, 1990,
p.46).

This complete reversal of accepted medical research methods lends support to
my previous claim that he used the syphilis model as the basis for his own
putative construct, assuming that the asylum inmates behavior he ‘found’ and
declared a cluster, simply and directly demonstrated the obvious existence of a
non-observable causal agent which was much like a syphilis germ.

Kraepelin in his texts uses retrospective data for his claims. His Lectures on
Clinical Psychiatry (3rd English edition, 1913) is representative, being nothing
more then a retrospective narration and presentation of his opinions and beliefs
about the histories and diagnoses of various residents of his insane asylum,
whom he brings before students during his lectures as illustrative cases of various mental diseases. There is no empirical evidence presented anywhere in this book. He does not provide the sources, or how he gathered his information or if he made any attempts and of what sort to confirm the accuracy of the information collected. He simply provides global accounts from which the reader is left to separate the chaff, not from the wheat but unfortunately from other chaff. It may be objected that since this is a book for students, simple clear clinical illustrations are more pedagogically sound then technical 'scientific' material. This would be a reasonable explanation for the lack of empirical content if such was forthcoming elsewhere. What I want to emphasize is that Kraepelin never provides such data in any of his texts, period. Kraepelin furthermore, seems to have been completely ignorant of any of the methodological problems that may effect reliability and validity of research when using data gathered retrospectively, such as faulty or imagined recall of long past events, the conscious or unconscious shaping of such data to present circumstances, or to the leading questions of the interviewers. The potential for bias in retrospective data is extremely high and the onus is on the researcher to indicate what steps he has taken to avoid them. This Kraepelin fails to do(in fact he doesn't acknowledge them at all). When examining Kraepelin’s notion of onset Boyle after quoting Kraepelin about how onset can begin,

“... so imperceptibly and with such indefinite indications that those around imagine that they are confronted simply with the outcome of an unhappy development, perhaps even of some character fault.” (p.47 as found in Kraepelin, 1896, p 426).
and

"In more then half the cases, the upheaval occurs so imperceptibly and with such
indefinite indications that its actual beginning cannot be determined in
retrospect"(p.47, as found in Kraepelin, 1899, p.149)).

observes that,

"It is difficult to understand how Kraepelin could have made use of his stated
criterion of similarities in onset, course and outcome when he believed that onset
could not be observed in more than half of those to whom he applied the term of
dementia praecox.“(p. 47).

Boyle also provides quotes from Kraepelin which demonstrate that onset was not
always uniform in its behavioral manifestation contradicting Kraepelin's
requirement, but rather, that varied and opposite types of behavior could indicate
it,

"In the patient’s behavior, either a marked inertia and lassitude or [its
opposite]typically childish characteristics make themselves apparent.”(Kraepelin,
1896, 428)

or

“The psychosis begins as a rule with indications of a light or severe psychological
depression(1896, p442). In a second group of cases one sees the illness set in
with the sudden onset of a state of excitation with little prior warning.”(1896,
p.443).

The same problem - statements indicating large amounts of within group
variability - can be found when one looks for proof of similarity of behavior in the
early writings of Kraepelin about the course, and the outcome,

“The course of this process of illness can take the most varied forms”( Kraepelin,
1896, p. 426).

“The further course of the illness in these cases is a varied one insofar as the
imbecility sometimes develops more rapidly, sometimes more slowly and can in
fact stop progressing at very different stages.”( p.429)
"The common outcome of all severer forms of dementia praecox is idiocy."(p. 436).

"The end state[of one type of paranoid form of dementia praecox] is feeble minded confusion."(Kraepelin, 1899, p. 188).

"Most frequently, however, the illness seems to lead to an insane confusion."(p. 200).

Kraepelin fails to specify what behaviors constitute the concepts of insane confusion, feeble minded confusion, or idiocy. So it is impossible to know how similar were the behaviors of those inmates to whom dementia praecox was applied or how different were the behaviors of those without this label, leaving the reader without any clue as to how the differentiating of the outcome state of dementia praecox from other ongoing behaviors may have been accomplished.

This was further complicated by his admission that,

"Unfortunately, I have not yet been able to discover particular indicators for drawing conclusions about the likely outcome of the illness in individual cases."(p. 180).

Astrup and Noreik(1966) lent further support to this apparent falsification by Kraepelin of his own claims that he had identified a discreet grouping based on similarity of outcome, when they note that Kraepelin acknowledged in 1920 (long after his most 'critical theoretical work' was completed) that it was not possible to predict prognosis(outcome) even with careful observation.

In conclusion, I believe that I have shown that Kraepelin in his own early writings fails to provide any consistent evidence based on his own criterion (similarity of onset, course and outcome) for inferring a hypothetical construct. As a matter of fact he admits that he failed to find a way to identify dementia praecox
outcomes. This suggests that he must have looked for such a method, although he doesn’t provide any description of such a search or what data he may have gathered to reach his conclusion. The admission that outcome cannot be predicted is in itself sufficient to invalidate his dementia praecox hypothesis since the ability to spot the outcome is fundamental to it. You can recognize a new dementia praecox case only when you see its outcome’s resemblance to previous outcomes of cases labeled as dementia praecox. If little or no similarity is found we cannot predict any new cases since we cannot identify them.

The historical evidence further suggests that he had a ready made model in mind (syphilis) that he used to identify symptomatic behaviors. Using this model of disease he haphazardly and retrospectively collected data (behaviors) about which he offers no information (i.e. population size or other demographic data) and used this data as if he were simply confirming, by providing further data, the already verified construct of dementia praecox, instead of (as he should have been doing), offering evidence to determine if such a construct could be validly inferred in the first place from the empirical evidence.

Late writings:

In this material an interesting transformation takes place. Kraepelin changes the criterion by which he wants to identify dementia praecox. As we know, in 1896 the entity was to be found by looking for similarities of onset, course and outcome. Now,

“Dementia praecox consists of a series of states, the common characteristic of which is a peculiar destruction of the internal connections of the psychic personality.” (Kraepelin, 1919/1971, p. 3).
The new criterion is now to be some 'common characteristic' of some mental states. Why this change? Well because,

"It has since[1896] been found that the assumptions upon which the name chosen rested are at least doubtful, as will have to be explained more in detail later, the possibility cannot in the present state of our knowledge be disputed, that a certain number of cases of dementia praecox attain to complete and certain recovery, and also the relations to the period of youth do not appear to be without exception."(p. 4)

This shift was a direct response to criticisms leveled at his original criterion by other alienists including Bleuler(1911/1950), that some of those diagnosed with dementia praecox received this diagnosis as a result of a dramatic change in behavior for the worse at a later time of life (not in youth as postulated by Kraepelin) and many dementia praecox cases appeared at the outcome stage (really the point of last observation) to exhibit normal behaviors, not forms of dementia as hypothesized by the theory.

Ordinarily in scientific research when a hypothesis is falsified, as appears to have been done by the demonstration that uniformity of onset, course and outcome could not be found among those labeled with dementia praecox, the researchers are required to go back to the drawing board and search for some new and independent non-random grouping of uniformities. To be precise, there cannot be a discussion about ‘a certain number of cases of dementia praecox’ which do not conform to the original criterion, because the assertion of the existence of the construct of dementia praecox is exclusively dependent on identifying data which meets that criterion. If the criterion is not demonstrable, then no construct has been show to exist. It is therefore logically impossible to
talk about dementia praecox/schizophrenia as an entity if a valid criterion does not exist. The researchers must begin their search again for signs and symptoms, as discussed earlier, and demonstrate that some new and different set of regularities (beside onset, course and outcome) were found. This Kraepelin never did, nor did anyone else (Boyle, 1990, pp. 176-177; Kendell, 1991, pp. 66-67).

What appears to have happened is that after the invention of the name, the dementia praecox abstraction was reified and made concrete by constant discussion in the psychiatric literature as if it was proven to be real, with just some trivial difficulties remaining, mostly to do with what were the actual symptoms and the best name for the syndrome, instead of its existence never being demonstrated.

"Unfortunately we could not shirk the uncomfortable duty of coining a new name for this disease. But the present one seems too awkward. ... But there is a far more important and practical reason why it seems so unavoidable to me to propose a new designation [schizophrenia] beside the older one. The older form is a product of a time when not only the very concept of dementia, but also that of precocity, was applicable to all cases at hand, But it hardly fits our contemporary ideas of the scope of this disease-entity. Today we include patients whom we would neither call 'demented' nor exclusively victims of deterioration early in life." (Bleuler, 1911/1950, p. 7).

Why or how this expansion of the 'disease' took place neither Kraepelin nor Bleuler explain or give evidence for. And of course as I have been showing it would be logically impossible to expand the category of that disease without new research independently finding some new regularities allowing a new inference of a disease entity.

So having abandoned his original criterion and offering no evidence for his
new one, psychiatry was left to drift in a theoretical vacuum. This did not bother Kraepelin or the other alienists. Repetition of claims, like mantras, has a deceptively intoxicating effect. We human beings, needing exegeses for the tragic and problematic events (in fact for all experiences) in our lives, create abstract entities like God which appear to answer our prayers by ‘explaining’ why things happen and thereby alleviating our anxieties. But, we gain this calm at the price of often accepting unproved or false explanations as true. It’s obvious that the claim of various societies and religious groups that their particular god is the true one cannot be right. If one of them is the true god the others must be false, or what’s more likely, they all may be false. Similarly Kraepelin having posited his abstract concept of dementia praecox as an explanation for bizarre behavior, simply wrote volume after volume (several thousand pages in fact, Kraepelin, 1919/1971, pp. vii-xviii) as if it was real. Since he wrote ambiguously and unclearly about dementia praecox others were able, like the hermeneutic interpreters of the Bible (another book purporting to describe true events not fiction), to get whatever they wanted from it. The fact that Kraepelin in all these volumes used his personal authority instead of empirical evidence for support of the concept was not a problem for other psychiatric experts. Using his paradigm they were content to be Kuhnian normal scientists and to do research that supported not tested the construct of dementia praecox.
“Kraepelin assumed that his ‘dementia praecox’ or Bleuler’s ‘schizophrenia’ was a ‘disease entity’. He did so, not because he had empirical evidence on that score but because from the middle of the 19th century until the middle of the 20th century most diseases were assumed to be entities with necessary causes that were either present or absent. This was because most of the great diseases that dominated medical practice in the 19th century—tuberculosis, syphilis, typhoid fever, cholera, and malaria—had been shown, one after the other, to be caused by infective organisms that were quite distinct from one another; it was therefore natural to assume that this was the nature of disease in general.” (Kendell, 1991, pp.65-66).

Although I believe that by now it should be clear that Kraepelin failed completely in his attempt to demonstrate that dementia praecox was a natural entity, I would like to conclude this section by discussing how he used his authority instead of evidence for his theoretical claims and diagnoses because, as with much else in contemporary psychiatry, he serves as the exemplar of this profession’s scientistic pretensions.

Since as he points out,

“The diagnosis of individual cases of dementia praecox has to distinguish the manifold states from a whole series of diseases which outwardly are similar but which are totally different in their course and issue. Unfortunately there is in the domain of psychic disorders no single morbid symptom which is thoroughly characteristic of a definite malady.” (Kraepelin, 1919/1971, p257).

We might ask how he or any other alienist could select the appropriate symptoms which would accurately identify dementia praecox patients and differentiate them from other patients if the outward states (the patient behaviors) of the various diseases were similar. Well the answer is that you could not.
“... there exists to-day to a not inconsiderable extent the possibility of cases of amentia and of manic-depressive insanity being wrongly attributed to dementia praecox and visa versa ... In this uncertainty about the delimitation the statements of different observers can in the first place not be compared at all, not even the diagnoses of the same investigator at different periods of time separated by a number years. ... On this account also[concerning recovery] the figures of different investigators will of necessity diverge from one another.”(pp. 186-7).

He acknowledges the unreliability of the observers, in this case alienists including presumably himself, to diagnose accurately, but yet goes on to assert that dementia praecox never the less is a disease.

“Although I must doubt that all of the disease pictures of Kahlbaum actually belong together, I nevertheless feel that my extensive experience justifies the recognition of the great majority of these cases as examples of a single characteristic illness form[dementia praecox].”(Kraepelin, 1899, p. 160).

But without a regular method of diagnosis based on a valid criterion this claim is ridiculous. Since I have demonstrated that by his own account this was not accomplished empirically what method did he use? He used the age old adage of ‘when reason fails apply authority’. He simply asserts that his ‘extensive experience’ justifies his diagnostic decisions as to who is or is not to be considered to have dementia praecox. His monumental text on dementia praecox, *Dementia Praecox and Paraphrenia*(Kraepelin, 1919/1971) is filled throughout with language of self reference such as ‘I think’(p. 263); ‘As it appears to me’(p.263); ‘we meet with’(p.33); ‘As it seems to me’(p.75); ‘I would, however, lay no weight on this”(p. 299); ‘as far as I can judge from the few cases’(p.307).

Not to labor the point, the entire volume is a statement of his own beliefs about dementia praecox not a scientific treatise backed by evidence.

The exclamation point to my conclusions must be the totally irreconcilable
positions taken by him when he states that we must, “... even yet ... rely purely on the valuation of clinical experience .. “(p.255) for the identification of dementia praecox. And his admission, which completely undermines this claim of accurate clinical authority, that his clinical experience has lead to many mistakes and where used it only described idealized or artificial, not real entities.

“In several such cases, In which I believed with certainty that I had to do with undoubted dissimulation[people pretending to be mentally ill] I never the less saw dementia praecox develop later”(p.273).

“The presentation of clinical details in the large domain of dementia praecox meets with considerable difficulties, because delimitation of the different clinical pictures can only be accomplished artificially. ... We shall be obliged therefore, as in paralysis, to content ourselves at first for the sake of a more lucid presentation with describing the course of certain more frequent forms of the malady without attributing special clinical value to this grouping[because such group does not exist]”(p.89).

Confirming the highly problematic nature of clinical authority without evidence, Bleuler - who otherwise has a very high regard for Kraepelin(see below) - never the less writes this about Kraepelin’s clearly questionable clinical decisions,

“Kraepelin released many of his patients as cured since a clear concept of cure demands a restitutio ad integrum be demonstrated. None of these cases were considered as cured by Aschaffenburg[fellow alienist], who saw the same patients.”(Bleuler, 1911/1950, p.255).

Clinical authority in this context turns into a macho contest of whose authority is ‘bigger’.

Bleuler’s work

Bleuler’s magnum opus is his Dementia Praecox or The Group of Schizophrenias(1911/1950). This book acknowledges that,
“The whole idea of dementia praecox originates with Kraepelin. Almost exclusively to his work we also owe the grouping and description of the separate symptoms. It would be too tedious to acknowledge our debt to him in each and every instance ...” (p. 1).

“... Kraepelin finally succeeded in isolating a number of symptoms which were present in maladies with very poor prognoses [dementia praecox] while absent in other disease-groups.” (p. 3).

“The establishment of the dementia praecox concept has brought clarity and order ... The Kraepelinian dementia praecox is an actual disease concept. The concept includes symptoms which occur only and always in dementia praecox. Thereby the disease group is provided with concrete delimitations.” (p. 279).

“.. the concept of dementia praecox is a very well defined one. It has the advantage over symptomatological disease pictures that with it there is no ‘more or less’” (p. 283).

Bleuler, as can be seen, assumes that Kraepelin has found dementia praecox through research and does not question its real world existence. Bleuler does not describe any research of his own, gives no evidence to sustain any of his assertions and except for minor issues of interpretation writes as if all his claims had been previously established by Kraepelin. Since I have shown this was not the case, Bleuler simply compounds Kraepelin’s failure. Considering his complete acceptance and reliance on Kraepelin’s claims about dementia praecox my analysis of his work will be briefer as a consequence.

Like Kraepelin’s, his practice appears to be very different from what a good medical researcher would apply using the method outlined earlier in this chapter. His son Manfred Bleuler, who went on to become a well known psychiatrist himself, offers a vivid picture of his father’s modus operandi.
“Eugen Bleuler’s concept of schizophrenic psychoses was based first on his experience between 1886 and 1898 as a young man in the ‘Island Clinic of Rheinau’. There he devoted all his time and interest to the care of his patients, and lived among them. He was their Doctor, and he treated them for a broad range of conditions ... However, his main endeavor was to be close to his patients; working with them, playing and walking with them, even organizing dancing parties with them. He became their friend, and they called him father. All this was because E. Bleuler was not yet married, had no family life in Rheinau, and had no social life outside the hospital, which was very isolated at that time. It was at Rheinau that he realized that schizophrenics could not be ‘demented’ since they did not lose a lively and colorful inner life.” (Bleuler, M., 1991, pp. 2-3).

This quote illustrates the autocratic paternalistic notion of the doctor patient relationship that was associated with the views of alienists, and often with medical doctors as well, in the 19th century. The younger Bleuler’s emphasis that what most influenced his father’s conclusions about schizophrenia was his intimate personal involvement with the patients rather then information gathered from clinical or experimental research corroborates my findings that Bleuler did not do research according to the best scientific standards of his time, assuming that he did any at all(a very questionable hypothesis, since no written documentation of such research exists). His beliefs about the patients not being demented because ‘they did not lose their lively and colorful inner life’, was based on an obviously unscientific lay conceptualization of behavior which went untested and derived, according to his son’s report, from his over close personal association with his patients. Manfred Bleuler also tells us that by 1925 or so, his father had stopped trying to find the underlying process(in my usage, the disease entity) of schizophrenia because,
“He concluded that we should continue to study everything we can observe with schizophrenic patients, but we should not waste time in looking for mysterious backgrounds of the psychosis. This was Meyer’s(1948) common sense psychiatry. It helped Bleuler not to adhere too much to hypothetical ideas about specific processes underlying schizophrenic psychoses.”(p.40).

This remarkable admission by Manfred Bleuler that his father gave up hope of ever finding a disease entity only leaves us wondering why he did not then give up the notion of mental illness as well, since his assumption about mental illness, like Kraepelin’s, depended on the existence of such an underlying construct. One can only conclude that his vested interests as an alienist outweighed his commitment to good research methodology. The use of ‘common sense psychiatry’ as a scientifically valid method makes a mockery of the whole rationale of scientific research. The common sense assumption of ‘we are what we see’ is falsified by everything we know about science and knew back then. One of the main purposes of science is to find explanations for those phenomena that appear to be obvious and easily explained by ‘common sense’ but are not. Imbecility in the form of syphilis was thought to be inheritable for over two hundred years, till the late 19th century, but turned out to be caused by a syphilitic spirochete(Quettel, 1990, pp.165-170). Thousands upon thousands of women died in childbirth at the hands of doctors during the 18th. and 19th centuries. Ignaz Semmelweis(1818-1865) was crucified by his fellow doctors when he urged them to wash their hands before touching their patients, and as a consequence thousands of pregnant women continued to die of this iatrogenic effect of medical intervention until the germ theory of disease was finally corroborated early in the 20th. century. One could go on with many more
examples but what needs to be emphasized is the deeply unscientific thrust of such an acceptance of the empirical power of unsystematic common sense observation. This approach is as ubiquitous among modern psychiatrists as it was back in Eugen Bleuler’s time.

“There were professors of psychiatry who were eager to demonstrate the impressive catatonic signs to their students, and less interested in speaking with the patients or demonstrating their manners of thinking and feeling. It is good that this has changed, and it raises the interesting question of the degree to which the symptoms observed in schizophrenics have been dependent on the interests of their psychiatrists. Did it change the patient’s symptomatology for instance, if the psychiatrist was fascinated by demonstrating catatonic symptoms, and not interested in speaking empathetically with the patient.” (Bleuler, M. 1991, pp. 4-5).

Bleuler’s accomplishments, if they are to be thought as such, appear to be the following; he renamed Kraepelin’s entity dementia praecox as schizophrenia; he without any evidence declared this entity to be more comprehensive then Kraepelin thought; and finally he declared this disease not to be a single disease but a group of diseases. Reading his oeuvre simply leaves one baffled that modern psychiatric researchers would have claimed that he did meaningful scientific work to elucidate mental diseases. He practices precisely the very bad science he blames others for employing. He classifies hundreds of behaviors according to his own arbitrary disease designations(Bleuler, 1911/1950, pp.13-226), based on his own authority. This is his critique of previous researchers,
“These remarks lead us to a discussion of the actual psychiatric practice which shows up the old principles of disease classification with bitter irony. Our literature is replete with complaints about the chaotic state of the systemics of psychoses and every psychiatrist knows that it is impossible to come to any common understanding on the basis of the old diagnostic labels. No discussion among clinicians, even those with closely allied views, is possible unless each one outlines his special point of view as to classification. ... Thus, not even the masters of science can make themselves understood on the basis of the old concepts and with many patients the number of diagnoses made equals the number of institutions they have been to. It is obvious that every author of a textbook was obliged, above all, to construct his own system of classification since the systems established by his predecessors were useless to his way of thinking and to his method of observation. Even within the very same school ... Situations as the following are rather common: In a certain hospital there would be a big pot, labeled ‘dementia’. Now along comes a new physician who enlarges the pot standing along side the other one, and labels the second pot, ‘paranoia’. He then carefully proceeds to seize the old inmates of the institution by some vestige of a delusion and puts them, one by one, in his new pot - and doing this believes that he is correcting the errors of his predecessors.”(Bleuler, 1911/1950, p.277).

This ‘ironically’ is exactly what he does with Kraepelin’s dementia praecox,

“The concept[dementia praecox] also corresponds with reality since its criteria can actually be discovered very easily; and as far as I know, no one has as yet proven the presence of any contradictions in the facts of this concept.”(p.279).

After saying that Kraepelin had definitely identified an ‘actual disease concept’(p.278) and is uncontroverted by the facts(previous quote), he disassociates himself from Kraepelin’s latest version of this entity based on nothing but his own authority, no empirical evidence is cited for this disagreement although he alludes to Kraepelin’s authority based earlier texts for support.

“Since this was written[Bleuler’s support of Kraepelin’s disease entity], Kraepelin has somewhat narrowed down his concept. I cannot follow him in that direction and maintain my position on the basis of the sixth and seventh editions of this textbook of psychiatry.”(footnote 9, p. 278).

One would assume that Bleuler would have been able to follow Kraepelin’s new version if any evidence would have been presented to support this shift but it is
clear that there was no evidence presented or possible. It is equally clear that both Bleuler and Kraepelin were simply doing what Bleuler criticized others for doing in the quote cited above, arbitrarily making up and changing the content of various categories and claiming by this to be making scientific progress,

“Aside from this widening of the scope of the concept[adding catatonic psychosis] we are in complete agreement with Kraepelin”(p.287). This quote illustrates nicely how psychiatric science works. As long as the claim making authority is powerful enough, all that’s needed to advance the field, is a simple dogmatic inclusion by fiat of what others consider different and distinct concepts or entities. This is just how the numerous mental disorder classifications of the various DSM publications (for example homosexuality) are added or subtracted in current psychiatric practice(Kirk and Kutchins, 1992).

Bleuler of course like Kraepelin is unable to be consistent in his claims. While claiming that dementia praecox/schizophrenia has concrete delimitations(Bleuler, 1911/1950, p.279) he states on p.281 that,

“However, the concept is as yet not well defined in two directions: one is that of paranoia, the other that of alcoholic psychoses.”

and

“Schizophrenia, moreover, embraces the greater part of those cases whose designations emphasize hallucinatory excitements or confusions ... In this direction the limits are difficult to define because as yet we do not know the rather manifold though not very common diseases which must be considered in addition to schizophrenia.”(p.288).

and

“It is impossible to make a sharp distinction between schizophrenia and the degenerative psychoses described by certain writers”(p.326).
and most interestingly

“Schizophrenia cannot easily be distinguished from malingering” (p.327).

These quotes directly contradict his claim that clear boundaries exist between schizophrenia and other diseases (p. 279). It is even highly problematic, according to Bleuler, to distinguish it from plain lying, which he calls malingering. He also falsifies his previous assertion, that after Kraepelin’s discovery of dementia praecox the use of personal authority by different researchers is no longer necessary because now there is ‘clarity and order’ (p.278), when he calls on his own authority in asserting the following,

“Chronic alcohol-paranoia has frequently been diagnosed by others; I, however, have not yet seen such a patient, who gave me even the slightest reason to see in him anything other than an ordinary schizophrenic who also drank. ... For me, therefore, there can be no doubt that the greatest portion of the chronic alcohol-paranoias of other writers are only schizophrenias.” (p.282).

"It is obvious that we have to consider as secondary phenomena the well-know disturbances of memory and orientation”(p.354).

"It should ... need no proof that the disturbances of the complex functions of intelligence ... are comprehensible only in connection with the already named secondary symptoms ... Also negativism is certainly a complex secondary phenomenon."(p.354).

For evidence of how arbitrary and belief based his diagnoses can be, see his statement following, regarding why a certain condition pertains to some cases rather than to others,

“I believe this particularly because, from a certain point on, the patients led such a senseless life that it is better explained by dementia praecox than by simple alcoholisms”(p.283).

These statements are not accompanied by any critical argument much less any
Bleuler goes on to reveal his hopeless lack of understanding about proper medical practice while providing succor for those future psychohistorians (Barzun, 1974) who without ever personally treating sundry famous artists and writers proceed to diagnose mental illnesses by interpreting their ‘artistic productions’, by reading secondary sources and by interviewing their relatives and friends when he says,

“In many instances, the patient’s history provides such a good basis for a diagnosis that it is possible to recognize with certainty the existence of schizophrenia from the reports of relatives. In fact the behavior of many of our patients is so characteristic that it can be adequately described for us also by laymen.” (Bleuler, 1911/1950, p.297)

How fortuitous for science that the lay notions of ‘he acts and looks crazy’, or has been reported to be by others, or perhaps paints or writes like he is crazy is sufficient to demonstrate insanity. This of course harks back to our earlier discussion of common sense psychiatry. As with prior statements by our alienists, this claim is also contradicted by other accounts of Bleuler’s asserting the complete incompetence of the lay public to accurately diagnose mental diseases.

“I cannot understand how Strohmayer ever arrived at the erroneous conclusion that affective deterioration is usually detected by ordinary lay people. Some time ago, I failed to make the diagnosis of schizophrenia ... because I believed the relatives ... ”(p.257).

These very confusing and incompatible statements should come as no surprise once we understand that psychiatrists cannot make coherent objective decisions about diagnosis because they have no way of determining when a
behavior(sympptom) is ‘prominent’ enough to be symptomatic of a disease.

“Just how prominent the various symptoms have to be in order to permit a diagnosis of schizophrenia can hardly be described. Indeed we have no objective standards by which to measure the gradations of complicated psychic processes.”(p.298).

Making the whole psychiatric diagnostic exercise resemble the magical incantations of medieval wizards as they attempt cures of sick spirits, as Keith Thomas in his Religion and The Decline of Magic, makes clear,

“All three constituents of primitive healing were thus present at one time or another: the spell, the medicine and the special condition of the performer. But no coherent theory underlay the visits of the clients to the cunning men[wizards]. Indeed the patient was often kept in ignorance of the formula employed, which like the details of much modern [psychiatric]medicine, might be deemed too secret to be entrusted to laymen.”(Thomas, 1971, p. 182).

If only Bleuler would have admitted Psychiatry’s cognitive inability to identify objectively the level of behavior intensity necessary for a behavior to be classified as symptomatic of a mental disease at the beginning of his book and not kept us mystified till page 298, then perhaps we could have proceeded for the next 100 years in the field of psychiatry to pursue potentially more fruitful venues of research.

CONCLUSION

Claims for the existence of mental diseases have been based on asserting the belief that they are precisely the same entities as other medical - biological - diseases. I offered as documentary evidence for this claim the quotes of the psychiatric authorities who are responsible for our contemporary ‘scientific’ understandings about mental illness, declaring this belief as if it is scientific fact. I reviewed the history and definition of physical diseases and went on to
demonstrate that the psychiatric claim that mental diseases have been
determined to exist under the same criteria as physical diseases is false. I did
this by analyzing the ‘research’ of the two 19th century psychiatrists, Emil
Kraepelin and Eugen Bleuler, who have been recognized by all modern mental
health experts as the individuals who ‘established’ the existence of the
paradigmatic psychiatric disease, schizophrenia, and others such as manic
depression(Kraepelin, 1905; 1913). I found after careful review of their writings,
that they did no research that met the methodological requirements of medical
researchers investigating physical diseases during the same historical period.
What I found instead were narrative reports of haphazard and conflicting
personal observations of asylum inmate behaviors and putative explanations for
them, which the two alienists tried unsuccessfully to put into a coherent theory of
mental disease without using any empirical evidence. They failed in their effort
not because strange behaviors cannot have a relationship to real disease (the
bizarre behavior of syphilitics puts that to rest) but because their claims were
tautologically constructed to fulfill their a priori expectations and remained
theories which were never empirically tested or corroborated. By using their own
words as evidence to test and as a result falsify their hypotheses I believe I have
avoided the inevitable heuristic turn taken by modern researchers who, with little
historical consciousness(Lukacs, 1968/1994), have built pseudo-scientific
theories on the house of cards of secondary sources.

Kraepelin and Bleuler, like other thoughtful people were puzzled and perhaps
troubled by strange behavior. As educated men of their time they were surely
aware of the traditional pre-scientific explanations and theorized causal
collections between madness and illness. Lacking obvious alternate paradigms
and being medical doctors predisposed to look for explanations under the
medical model of their time, they naturally assumed that the strange behaviors of
their inmates must be caused by some infectious agent as had been
demonstrated in syphilis. They applied that model and used it to define some of
their inmates’ peculiar behaviors as symptoms of an underlying process,
declaring the existence of the mental disease of dementia praecox by edict,
rather than through independent empirical research.

Gerald Grob, the renowned historian of American mental health not
unsympathetic to the use of the medical model in psychiatry, nevertheless has
this to say about the 19th century psychiatric efforts at applying this model to
behaviors classified as mental illnesses,
In general medicine, the demonstration of a relationship between the presence of certain symptoms and a specific bacterial organism had led to the development of a new classification based on etiology rather than on symptomatology, at least for most infectious diseases. The inability to pursue a parallel course left psychiatry with a classification system based on external behavioral signs that tended to vary in the extreme. Conclusive evidence that paresis (general paralysis of the insane) was actually the tertiary stage of a disease that began with a prior syphilitic infection offered an attractive model for psychiatric diseases. Nevertheless neither psychiatrists nor pathologists were able to identify other comparable specific psychiatric disease entities. … Like physicians generally, psychiatrists advanced explanations that appeared to embody the latest available knowledge. The reasons are not difficult to grasp. Unlike those outside the specialty, psychiatrists were responsible for thousands of patients in mental hospitals. Their existential involvement with such a large and varied group precluded consideration of the claim that mental disease could neither be understood nor treated. … Moreover, the training and education of psychiatrists – which did not differ from that of other physicians – rendered untenable any abandonment or modification of the traditional therapeutic role. Such role, of course, required some sort of theoretical justification. Had psychiatrists rejected an effort to explain mental disease, they might have impaired their professional legitimacy and prepared the way for other groups willing to fill an existing void and meet perceived social needs." (Grob, 1985, pp. 20-21)

This is all quite obvious when one examines their work. What is puzzling, is why the vast majority of contemporary psychiatric thinkers\(^{14}\) refuse to see the ‘obvious’. One possible answer might be that changing paradigms is difficult (Kuhn, 1962/1970), and is resisted by people for various reasons. The paradigm has worked for a long time (Newtonian mechanics for example); we have come to depend on it to explain the coherence of our world; it guarantees continuity of our expectations; it reduces our anxieties about the unknown, it insures, as Grob in the previous quote suggests, employment to many working under its boundaries; as well as the further entrenchment of some established

\(^{14}\) A hard-core minority relying on empirical evidence stoutly maintains a dis-loyal opposition (Breggin, Szasz, Gomory, etc.)
power elite; and if found to be false it may expose to ridicule the experts whose careers’ are built on its foundations.

Regardless, the dichotomy between the horde of mental health experts claiming the reality of mental disease and the paucity of the empirical evidence in support is striking. Since neither Kraepelin nor his acolyte Bleuler ever demonstrated the existence of dementia praecox/schizophrenia, although they and their psychiatric offspring who themselves made no serious further headway toward empirical validation, have acted as if they had, modern psychiatric assumptions about mental disease have no well tested scientific legs to stand on.

Today, the field of mental health finds itself in the same position vis-a-vis psychiatric research as Kraepelin found himself 100 yrs. ago, in 1896. We are still waiting to find, by the application of science, a coherent non-random construct of (objective) signs and symptoms that is connected to some underlying process that we could call a mental disease. Or if this is not, (as I believe), based on the available evidence a very fruitful research program then as good methodologists (Miller, 1994) we should turn to other promising and testable hypotheses for explanations.

Knowing that the widely held notion that ‘truth will out in the end’ is false, and mendacity and irrationality must be resisted forcefully at every turn, leads me to conclude, that the more often and the more vigorously we investigate and test the original sources of truth claims and refuse to accept uncritically the secondary source based interpretations of contemporary psychiatric authorities, the more likely it is that we will have a meaningful critical forum for the evaluation
of such claims and, as a result, scientific progress.

This chapter attempted an excavation of a foundational psychiatric ‘truth’, that mental illness is just like any other medical illness. This turned out to be, after careful review, the intellectual equivalent of quicksand. Once this claim’s assumptions and evidence were examined no empirical evidence or logical coherence was found to support the weight of the mental disease explanatory edifice. Even though this chapter provides telling evidence falsifying the medical model of deviant human behavior that ideally should serve to bolster the search for other better, alternate models of explanation for these behaviors and the strategies necessary for coping with them, the rest of this dissertation will be devoted to evaluating a particular treatment approach highly recommended by the proponents of the medical model. If I am correct in my claim in this chapter then this approach and all other psychiatric interventions treat intellectual constructs not medical diseases and as such ‘operate’ metaphorically not medically (Szasz). I nonetheless undertake this evaluation without irony, recognizing that I could be wrong and the medical modelers could be right, and as a result their research should be assessed on its merits.
Chapter 2

TCL Treatment Population and the Meaning of Community Mental Health

“If we examine the definition and practice of psychiatry,....we find that in many ways it is a covert redefinition of the nature and scope of ethics. According to Webster’s, psychiatry is 'a branch of medicine that deals with the science and practice of treating mental, emotional, or behavioral disorders esp. as originating in endogenous causes or resulting from faulty interpersonal relationships'; further, it is ‘a treatise or text on or theory of the etiology, recognition, treatment, or prevention of mental, emotional, or behavioral disorder or the application of psychiatric principles to any area of human activity(social psychiatry).’

The nominal aim of psychiatry is the study and treatment of mental disorders. But what are mental disorders? To accept the existence of a class of phenomena called ‘mental diseases’ rather than to inquire into the conditions under which some persons may designate others as ‘mentally ill’, is the decisive step in the embracing of the mental health ethic. If we take the dictionary definition of this discipline seriously, the study of a large part of human behavior is subtly transferred from ethics to psychiatry. For while the ethicist is supposedly concerned only with normal(moral) behavior, and the psychiatrist only with abnormal(emotionally disordered) behavior, the very distinction between the two rests on ethical grounds. In other words, the assertion that a person is mentally ill involves rendering a moral judgment about him. Moreover, because of the social consequences of such a judgment, both the 'mental patient' and those who treat him as one become actors in a morality play, albeit one written in a medical-psychiatric jargon.”(Szasz, 1991, p.26-7).

Treatment population

I will now focus on the group the multi-billion dollar public mental health industry claims to serve. They are functionally diagnosed as ‘the severely and persistently mentally ill’, a conceptual construct in search of substance. They are also known by the noms de plume of; ‘chronic psychiatric patient' (Test & Stein, 1978), ‘markedly impaired’ (Test & Stein, 1976), or ‘persons with serious mental illnesses’ (Test, 1992).These various descriptive labels, although no more than commonsensical observational statements, are provided by the TCL experts
as if they each distinctly had scientific significance in distinguishing a particular
group of people by characteristics that at a minimum makes them behaviorally
(psychiatrically) unique. Let’s look at each definition separately and see what
each describes. The ‘chronic psychiatric patient’ is simply a descriptive label for
an individual indicating a lengthy involvement with the mental health system. This
usually means that he or she has been hospitalized and medicated over many
years. The key adjective is ‘chronic’, implying that if you have a record of being in
a psychiatric hospital repeatedly, that in itself is sufficient to prove disability. The
physical experience of hospitalization is thus a life time sentence, a finding of
‘psychiatric guilt by hospitalization’. Among the homeless for example we
routinely ask whether they have ever been in a psychiatric hospital. If the answer
is yes, then we cite this as supportive evidence for a finding of mental illness,
often the only evidence.

The second label, that of being ‘markedly impaired’, appears to describe some
deficit, either psychological or physical, suggestive of someone who is distinctly
less intact mentally in some ways then the normal population. This impression is
false. Test & Stein tell us that,

“By ‘markedly impaired’ is simply meant those patients who traditionally have
been treated by public mental hospitals and aftercare programs.”(Test & Stein,
1976, p.72).

Again, no identified illness is necessary, it is the treatment, the mere involvement

15 Unlike Pirandello’s literary creations who are in search of an author, these psychiatric constructions have
plenty of authors (the dozens of DSM bureaucrats in charge of mental disorder claiming and naming). What
they lack is the Platonic essence.
with institutional psychiatry (guilt by association) that diagnoses the disability. Of course, there is an underlying assumption (a bias) by the authors that these people have something called Mental Illness. My point is, that what characterizes the assumption that people have such illnesses has nothing to do with scientific fact, but is instead akin to characterizing people who spend a lot of time in water, competitive swimmers for example, fish like. If you swim well you swim like a fish. Likewise, if you can get admitted to or are judged to require a psychiatric hospital stay you must be mentally ill. Only ‘the insane’ or perhaps people with no other source of social support, food, shelter or income would want to do that.

Vogel (1991), offers a good description of who populated and still populates psychiatric hospitals, many patients were and are simply old, or homeless, or socially without support.

The third label of ‘persons with serious mental illnesses’ appears to address a disease state of some sort. This perhaps is the situation where a medical expert has the scientific tools to, and does identify the disorder(s) afflicting a person, which may (usually) be amenable to treatment by some disease specific intervention. Test, et al., (Test & Stein, 1978 p.351, or Marx, Test, & Stein 1973, p. 506), claim that ‘persons with serious mental illnesses’ fall into a variety of different clinical diagnostic categories, most being schizophrenic,

“Such patients represent a wide spectrum of diagnostic categories; the majority, however, carry the diagnosis of schizophrenia” (Test & Stein, 1978, p.351).

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16 See Bachrach, 1988, for an interesting, although inadequate, critique of this concept and its limited utility for diagnosing mental illness. The critique is of minimal value because this author is clearly a supporter of institutional psychiatry and does not examine any of the most fundamental problems inherent in psychiatric nosology, as described here.
Based on these findings one might expect that these individuals would have
diagnosis specific problems or difficulties to resolve. Nothing could be further
from the truth. In fact members of this disparate group (consisting of various DSM
categories) all share ambiguously defined characteristics of, what Test and Stein
describe as ‘severely and persistently mentally ill’ individuals. The characteristics
are the following; 1) High vulnerability to stress, 2) Deficiencies in coping skills,
3) Extreme dependency, 4) Difficulty with working in the competitive job market,
and 5) Difficulty with interpersonal relationships (Test & Stein, 1978, p.351).
These attributes are remarkable for their lack of resemblance to any of the
disease characteristics one might find in medical text books describing
scientifically well corroborated disease processes such as, cancer, heart disease,
venereal disease, or diabetes. They describe instead immature, socially
repressed, educationally backward, emotionally dependent, and economically
vulnerable individuals who are, for a variety of reasons, none of them, I claim,
mental illness related, unwilling or feel unable to take charge of their lives (Szasz,
1994).

I conjecture that Test & Stein are fully aware of this difficulty and would very
much like to avoid discussing this problem. They never once consider alternate
explanations to the disease model for such behaviors, although a large and
diverse sociological and anthropological literature is extant (I cite most in the
present bibliography). They prefer instead, to go on ‘helpfully’ meddling (Flew,
1995) with the various existential problems of the mentally ill despite the lack of
empirical evidence for these hypothesized disease entities or their solutions. My evidence for this charge is in their research. They pointedly ignore the identified individual clinical disease diagnoses of the various group members when ‘treating’ these people, and attempt instead to procure very similar functional changes throughout the treatment population based on the previously enumerated behavioral characteristics which the researchers correctly note cut across all diagnostic categories. For example;

“Deinstitutionalized patients, whatever their diagnoses, tend to share a number of deficits that are independent of diagnosis and that usually are found among severely ill individuals who need psychiatric services over an indefinite period of time.”

Furthermore,

“These patients, under moderate stress, tend to develop severe symptoms. Symptoms cut across diagnoses and may include the indicators of unremitting thought disorders ... . Severe behavioral disorders, including disruptive, dangerous, suicidal, unpredictable and offensive behaviors, are often common parts of the clinical picture. Abuse of a variety of substances ... may further complicate symptom presentation ”(Cotton, 1983, p. 56).

The discerning reader will notice that in the preceding two quotes, certain behaviors, volitional activity, such as drug taking and becoming angry, are called symptoms which are alleged to ‘indicate’ or constitute, singularly or in various groupings, disorders, which are tautologically also called symptoms. This of course is nonsense(literally), but typical of the obscurantism which passes for argument among mental health experts. Note also that volitional acts(Flew, 1995) are described as if they were happenings (non-acts called symptoms, like a runny nose). Behaviors, some of which may be labeled ‘dangerous’ or ‘offensive’ are renamed and called symptoms and by psychiatric prestidigitation transformed
into disorders. To whom these behaviors are ‘dangerous’ or ‘offensive’ and who defines them so are important questions left unexamined by institutional mental health experts.

The cavalier distortion of allegedly scientific entities (disorders, symptoms) and what constitutes them is unfortunately the rule rather than the exception in psychiatric methodology. Test & Stein, in lock step agreement with Cotton’s list of the defining behavioral characteristics of the severely mentally ill go on to say;

“In summary, the chronic psychiatric patient is one whose emotional disabilities are so serious and persistent that without special support he or she is unable to maintain a stable adjustment to community life. Such patients represent a wide spectrum of diagnostic categories…” (Test & Stein, 1978, p.351)

The question which then arises is: If these patients all have these apparent difficulties-in-living in-common, regardless of their diagnosis (many non-psychiatric dependents share these difficulties see Szasz, 1994), why are such discrete diagnoses necessary? My conjecture is that without such clinical diagnoses (really medical and therefore socially authoritative ‘stamps of approval’) mental health workers would not have the scientific authority required to treat these people as mentally ill patients, losing both their professional status and their field’s justification for existence.

I would challenge Test & Stein to offer empirical evidence along with an explanation as to the relevance a specific diagnostic category has to the way they provide treatment. That is, test the hypothesis that diagnostic categories distinguish treatments among severely disturbed mental health clients. If they are unable to specify in detail how the use of such categories targets their treatment,
or if this hypothesis is experimentally falsified, as ethical researchers they
should admit that this hypothesis is false and stop using it. I conjecture that this
would be an unlikely occurrence, not because they would be able to confirm the
scientific value of these psychiatric diagnostic categories for their work, but
because the falsification of this hypothesis would undermine their whole
theoretical edifice eliminating the justification for their program.

Community Mental Health Treatment

“The dawning of all great truths on the consciousness of humanity has usually to
pass--says Tolstoy--through three characteristic stages. The first is: ‘This is so
foolish that it is not worth thinking about.’ The second: ‘This is immoral and
contrary to religion.’ The third: ‘Oh! This is so well known that it is not worth
talking about.’ “(Polanyi, 1948 p. v)

The belief that community mental health treatment in the least restrictive
environment is the most beneficial approach to help the severely mentally ill is
held dogmatically. I assert this because this ‘psychiatric truth’ is defended
uncritically in the form of belief. Much like religion, the empirical evidence is at
best tenuous. Furthermore the concept of community mental health treatment
has been defined differently in the past and with as much logic as this
contemporary definition. Two psychiatrists, Dr. Thomas Szasz and Dr. E. Fuller
Torrey, who are in diametrically opposed camps when attempting to explain the
concept of Mental Illness have surprisingly similar views about much of the
community mental health movement and its history. Both recognize that, the duty
to care for the severely disturbed of our society has a long-term historical claim
on the community at large. Torrey;
“The care of the mentally ill in America had been the responsibility of the states since even before the Constitution had been written—since 1766, in fact, when Governor Francis Fauquier had gone before the Virginia House of Burgesses and asked funds to open a public psychiatric hospital because of the accumulating numbers of mentally ill individuals who could be seen on the streets of Williamsburg.” (Torrey, 1989, p.54), Szasz;

“During the early decades of the century, virtually everyone considered to be seriously mentally ill was confined in a state hospital. Psychiatry was synonymous with hospital psychiatry .... The permanent confinement of the mental patient in the insane asylum was accepted as society’s proper response to its double duty, to the deranged patient endangered by his disease, and to society endangered by him.” (Szasz, 1994, p. 169-170).

The concept of community treatment can mean any number of things, as determined by current philosophic frameworks, politics, acceptable methods of social control, and tolerance for deviant behaviors. Some versions may be in marked opposition to each other. In the ‘bad’ old days the proper role of community treatment was defined as periodic, often long term and involuntary stays in state mental hospitals (or asylums in earlier times) for those alienated from society; thus, the original name of alienists for psychiatrists who were essentially administrators of massive institutions. The grounds for this type of treatment were; that tranquility, spiritual healing, and the acquisition of a sense of responsibility, the then presumed keys to curing the insane, could best be had through the provision of structured, physically rigorous but uncomplicated work provided in the physical isolation of an institution where the patient could best be free of the stress and pressure of the everyday world (the community-at-large) (Scull, 1979). In the current ‘good’ enlightened days, community treatment is described as treatment in the least restrictive environment (ironically the
community-at-large), preferably done ‘in vivo’. The trumpeting of the so called Community Mental Health Movement of the early 1960’s as a new, freshly discovered approach to help those most severely disturbed is puffery of the worst sort. It is merely a repackaging of a very old concept, government responsibility for institutional psychiatry (pre 1963 resting mostly with local and state government, post 1963 much of it borne by the federal government).

The fundamental difference between the ‘old’ and the ‘new’ treatment is the location of the treatment. One, the old, is provided in a confined space, called a hospital, and the other, the new, is provided in neighborhoods called the community.

One is truly at a loss to understand why the current rationale for neighborhood community treatment is more compelling then the model of community care in psychiatric hospitals that was offered earlier in our history (Grob, 1983). There is nothing about living in a neighborhood that logically compels a better outcome (whatever that may be) for so called mentally ill individuals. The plight of the homeless in various communities attests to that. This is an empirical question that requires further research and as far as I can tell has not been decided.

Today’s leading proponent of the biological school of psychiatry, E. F. Torrey, illustrates the lack of critical thinking around these issues. He enthusiastically supports, what he and we euphemistically now call outpatient commitment, in a linguistically more honest world we used to call it involuntary treatment. This

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17 This term generally is defined as - ‘in its natural environment’. The use by TCL of this term is strange because the natural environment of human beings is usually not defined by such things as their residency.
approach is really the old view of community treatment, forcing clients against their will to take psychotropic medication and undergo any other treatment mandated by psychiatrists, but misleadingly now portrayed as outpatient or in-community treatment and as such innovative or at least vastly underrated in therapeutic value. In order to carry out such treatment the client has to be restrained physically if he objects, and compelled by force to accept treatment. This differs little from being involuntarily hospitalized (physically confined) except for the place of confinement, some community treatment facility (in the new sense) and generally for a shorter period, as long as it takes to complete treatment. Of course if compliance cannot be secured in the outpatient setting, the client is involuntarily hospitalized in the old sense (in a mental hospital) thereby completing the circle of coercion (Torrey & Kaplan, 1995).

Much of this psychiatric rhetorical activity (stretching and altering meanings of concept words in-order to suggest change in professional treatment behavior which de facto remains the same) rather then research toward more effective treatment alternatives may be explained by the differing problem situations (historical contextual differences) which currently require presenting coercive treatments more subtly then in the past, along with the lack of any meaningful alternatives to the ‘tried and true’ under the governing paradigm.

The claim I will be advancing in the next chapter is that all so called PACT programs are to a large degree coercive and that this activity results

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Sometimes, but not always, provided in neighborhoods where clients grew-up, these locales are asserted to be therapeutic and more conducive to autonomous client behavior then are psychiatric hospitals.
tautologically in the misattribution, wittingly or not, of patient and worker behavior. Forced or imposed client change is passed off as client internalized or learned change. These approaches are ineffective, or no more effective than other interventions, with the addition of possible unwanted side effects. Coercive measures may result in involuntary compliance but may not win the hearts and minds of those so treated. I will also discuss how the development of TCL was strongly influenced by the early experiences of its inventors doing research and providing community mental health treatment in a Wisconsin state mental hospital. Their training in this setting included the use of electric shock as punishment and control of uncooperative patients, along with other coercive measures. These experiences defined their view of mental illness and have shaped the nature of the theories and methods they applied in the development of the TCL model.
Chapter 3

The Historical Roots of TCL or The Archeology of Coercion

"Between World Wars I and II the commitment to institutional care of the mentally ill remained unchanged. ... Indeed, dismal economic conditions did not appreciably diminish the spirit of therapeutic innovation that had begun with the introduction of fever therapy in the 1920's and continued in the succeeding decade with insulin and metrazol shock therapy and prefrontal lobotomy. Many of these new approaches had been developed by European psychiatrists. Their incorporation into American medical practice was related to several variables: the migration to America of European refugee physicians after Hitler came to power in 1933; the seeming effectiveness of such novel therapies; and perhaps most important of all, the fact that these new approaches were compatible with a scientific psychiatry and were beyond the competence of the newer nonmedical occupational groups within the mental health field. Having been trained and socialized as physicians, institutional psychiatrists were receptive to somatic therapies that went beyond custodial care of patients. The autonomy and independence enjoyed by physicians also precluded any legal or informal barriers that might have been imposed against the introduction of novel therapies whose effectiveness was questionable." (Grob, 1983, p.288-9). "The receptivity toward therapeutic innovation was understandable. In one sense it grew out of psychiatry’s attempt to emulate the alleged successes of scientific medicine. Just as surgery symbolized the success of scientific medicine, so too novel psychiatric interventions would demonstrate the specialties ability to influence the outcome of mental diseases." (p.291)

The book edited by Stein and Test reporting on the first ten years of the TCL effort states that,

"Ideas rarely arise de novo; they are generally formed from the building blocks of prior knowledge and experience. To become lasting, they must be nourished in an environment that is willing to set aside the accepted attitudes and practices that resist new concepts." (Stein and Test, 1985, p.7).

Stein and Test highlight in the above quote and the one following, what they believe was the value of their experience at Mendota State Hospital to their own development as well as to the development of TCL.

"In the mid 1960’s, ... Several psychiatrists who had just finished their residency joined the hospital staff. These psychiatrists were imbued with the therapeutic zeal frequently found in young, uninitiated physicians. In addition,
Arnold Ludwig joined the staff as director of research and education. His first two projects involved many members of the hospital staff. One of Ludwig’s projects involved the formation of a Special Treatment Unit (STU), a research unit whose primary goal was the development of new treatment techniques for chronic schizophrenic patients that could reduce or otherwise modify the chronicity that these patients had established. Through the programs of the STU, Ludwig, Marx, and Test demonstrated that a variety of novel psychosocial treatment techniques could make an impact on previously unresponsive patients and significantly enhance their in-hospital functioning. When Ludwig left in 1970, Stein took over his position as director of research and education, and Marx and Test assumed leadership of the STU. These changes in leadership made possible a shift in the direction that STU programs would take. The new project, Prevention of Institutionalization Project (PIP) was an extension and outgrowth of findings from the STU’s research treatment programs for chronic schizophrenic patients. It extended what had been learned about chronicity and its treatment to prevention of chronicity.”

This descriptive quote is especially revealing. It establishes the intimate involvement of all the creators of TCL in the research done at Mendota State after the appointment of Ludwig as director of research and education, and suggests that what they learned from these projects formed the core of their understanding and treatment of clients (inpatient and TCL). The authors provide a list of ten papers published during the heyday of the STU experiments, I will highlight the research done and the ‘lessons’ learned by this group by going directly to a selection from these sources.

Here again, I will allow the authors to speak for themselves,
Client descriptions-

“It is becoming fashionable to view mental patients, especially chronic schizophrenics, as poor, helpless, unfortunate creatures made sick by family and society and kept sick by prolonged hospitalization. These patients are depicted as hapless victims impotent against the powerful influences which determine their lives and shape their psychopathology. Such a view dictates a treatment philosophy aimed at reducing all the social and institutional iniquities responsible for the patients’ plight. However, in the process of leveling the finger of etiologic blame for the production and maintenance of chronic schizophrenia, theoreticians and clinicians have neglected another culprit- the patient himself. Professionals have overlooked the rather naive possibility that schizophrenic patients become ‘chronic’ simply because they choose to do so.” And further, “In our own experience, the problem is not so much modifying factors outside the patient, but rather in changing certain patient attitudes and consequent behaviors.” (Ludwig et al., 1967, p.737).

They identified what they describe as the ‘Code of Chronicity’ through the research conducted in the special training unit (Ludwig et al., 1966).

“Implicit in our discussion of the ‘code’ are five important clinical ‘facts’ which, we believe, underlie the behaviors of chronic schizophrenics. First, these patients can use their insanity to control people and situations. Second, they have an indomitable will of their own and are hell bent on getting their way. Third, one of the basic difficulties in rehabilitating these patients is not so much their ‘lack of motivation’ but their intense, negative motivation to remain hospitalized. Fourth, insanity and hospitalization effectively pay off for these patients in a variety of ways. Fifth, these patients are capable of demonstrating an animal cunning in provoking certain reactions on the part of staff, family, and society at large which guarantee their continued hospitalization and its consequent rewards.” (Ludwig et al., 1967, p.738).

The researchers found these patients to be;

“Obviously ... not ... a group of fragile, broken-spirited persons but rather ... tough, formidable adversaries who were ‘pros’ and who had successfully contended with many different staffs on various wards in defending their title of ‘chronic schizophrenic’ (Ludwig et al., 1966, p.566).

These descriptions of clients as active, volitional agents\(^\text{19}\) contrasts

\(^{19}\) Admittedly not in ways that this group of researchers would like, but still ‘pros’ who are ‘successfully’ outwitting professionals. One can’t help but reflect that such success would perhaps require some planning and organized effort, perhaps worthy of the term rational.
remarkably with both Test's more contemporary definitions of SMI's quoted earlier; as well as with Stein's current(1989) stated view of these clients as having a malady which causes a ‘whole host of problems’ like;

“stigma, poverty and loneliness, which are ongoing problems- and, intermittently, the terror of psychosis, with its frightening delusions and hallucinations. ... . Over the years, I have grown to respect and admire a great many persons who struggle day to day with the terrible burden of having a malady that causes them enormous problems and is so little understood by the professionals who are trying to help them. They are some of the most courageous people I know.”(Stein, 1989, p.133, emphasis added).

Stein in 1973, rather then displaying the professional humility and ignorance this 1989 quote captures, apparently ‘knew’ a great deal more about these patients and certainly back then did not view them as courageous or demonstrate much respect for them; as I report in the next section.

**Treatment Approaches used by this group**

In 1973 Stein reported a study, *The Use of Punishment As A Treatment Modality: A Case Report*(Brandisma & Stein, 1973), on the use of electric shock, without client consent, as punishment to reduce allegedly unprovoked assaultive behavior of a ‘retarded, adult, organically damaged’ twenty-four year old woman. This single case study was a follow up to Ludwig, Marx et al’s 1969, study of the use of electric shock on a paranoid schizophrenic patient, *The Control Of Violent Behavior Through Faradic Shock*, (Ludwig et al., 1969). Ludwig justified his study by its uniqueness. He listed four general areas of uniqueness of which the third was, “the fact that this procedure was administered against the express will of the patient”(Ludwig et al., 1969, p.624).
Prior to Stein’s ‘experiment’, his subject, an organically brain damaged woman, while at another institution had been;

“... secluded ... permanently and [the institution] tried various drug therapies including Mellaril, Proloxon, Stelazine, Compazine, Phenobarbital, and Dilantin. These efforts failed to significantly affect her behavior and ... Carol was transferred to ... the state hospital[Mendota State Hospital] with the recommendation that she receive electroconvulsive therapy.”

At Mendota State Hospital the following ‘therapies’ were tried;

“1) High doses of Phenothiazines and combinations of phenothiazines: Results no apparent effect on her behavior. 2) Primodone in the vain hope that her attacks represented adherent psychomotor seizures. ... [if you know that a treatment won’t work why subject a person to it?] 3) Dexamethasone in the hope that the paradoxical inhibition often found in hyperactive children would result[same question about the efficacy of this as of prior treatment]. 4) Daily electroconvulsive therapy(twenty sessions). Results: she became progressively more aggressive ... ”(Brandsma & Stein, 1973, p.31.)

It should be obvious that these institutions were not providing thoughtful, high quality treatment based on empirical research, but arbitrarily throwing every available coercive chemical and physical agent (most of them highly toxic or dangerous) at this organically damaged individual to make her stop behaviors which due to her real physical problems (difficulties in moving about, being hard of hearing, affected by epilepsy and possible tardive dyskinesia), may have been out of her control(Brandsma & Stein, 1973, p.31). The experiment itself is shocking in its crass inhumane treatment of the client.

To get a ‘baseline’ measure of this brain damaged client’s assaultive

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20Surely some behavior was effected, since these psychotropic drugs along with the other drugs Stein’s team administered at Mendota State Hospital were even then, known to cause brain damage(tardive dyskinesia) in a very high percentage of patients treated with them. As early as 1972 estimates ranged up to 41% of long term users being affected by Tardive Dyskinesia(Kazamatsuri et al., 1972; Marrocco, 1972) and by 1973 estimates ranged to over 50% of people suffering from this disorder among those patients using such drugs for 3 years or longer(Crane, 1973).
behavior, the client was baited and ridiculed in an analogue situation in order
to get her to hit, curse kick etc.;

“During the first session heavy canvas mittens were placed on the patient, ... . The staff(five or more) people would sit very close to patient with a young female within striking distance. The patient was required to sit in an armchair throughout. ... .During the base rate week the staff quickly developed a consistent provocative approach in order to ensure a high frequency of behavior from the patient and be generalizable to the frustrations she would encounter outside of treatment. This consistently involved: 1) ignoring the patient in conversation; 2) refusing to give the patient candy or snacks when others were eating them; 3) denying all requests, for example, during the session if she asked if she would be able to go for a walk that afternoon, she was immediately told, 'No you can’t.'; 4) refusing to accept her apologies or believe her promises of good behavior; 5) The above mentioned female sitting next to her often leading the provocation; 6) using provocative labels for her behavior, i.e., 'animalistic, low grade'; 7) discussing family related frustrations, i.e., her mother’s refusal to write or visit, how her dead grandmother would be displeased with her present behavior if she were alive.” (Brandsma & Stein, 1973, p.32-33, emphasis added).

This methodology is crass scientism. Their misuse of professional authority is demonstrated in the previous quote, where aggressive, artificial incitement by the staff is used to provoke an angry response from the patient; this elicited behavior then is used as a representation of the allegedly ‘unprovoked’ assaultive behavior of the client. This speaks more about the uncritical attitude of these clinicians and their abuse of power over an involuntary subject, then about the alleged assaultive behavior of this client. A true baseline for assaultive behavior would have to have been collected when it occurred, in situ, without artificial provocation by staff.

The involuntary use of electric shock is a clear human rights violation, even in the case of war, and was back in 1973. The notion that a person who is

\[21\] Not electric convulsive therapy (ECT) which is a highly problematic but widely accepted psychiatric
organically diminished due to repeated grand mal seizures and suspected brain damage (Brandsma & Stein 1973, p.31), and had spent almost all her life confined in various institutions, is appropriate to be the involuntary subject of punishment, a method which already had been shown to be ineffective in increasing desired behaviors (Azrin and Holz, 1966, p.438-443), is reminiscent of the pseudo-scientific justifications for the inhumane experimentation on the frail, the deviant, and the racially impure during the Nazi era in Germany. Stein cites the Azrin and Holz (1966) source to support his use of punishment (Brandsma & Stein, 1973 p.36). In fact this classic review argues the opposite, that punishment is ineffective in many situations, especially those involving human subjects, as a method of behavioral change; and, in the bargain, has many disadvantages,

“The principal disadvantages of using punishment seem to be that when the punishment is administered by an individual, 1) the punished individual is driven away from the punishing agent, thereby destroying the social relationship; 2) the punished individual may engage in operant aggression directed toward the punishing agent; and 3) even when the punishment is delivered by physical means rather than by another organism elicited aggression can be expected against nearby individuals who where not responsible for the punishment. These three disadvantages seem especially critical for human behavior since survival of the human organism appears to be so completely dependent upon the maintenance of harmonious social relations.” (Azrin & Holz, 1966, p.441).

The callous disregard of the then available scientific evidence falsifying their approach; as well as the deep disrespect for the personal autonomy and human rights of their patients as manifested in such research, is in dramatic contrast to their self serving contemporary declarations about mental patient suffering and courage, which never make mention of these never repudiated experiments.
The potpourri of experiments carried out by the Special Treatment Unit staff, and their bewildering lack of ethics, logic, empirical coherence, scientific reliability or validity makes for fascinating, if chilling, reading but is difficult to briefly summarize. As I read these papers I got the sense that these researchers followed their personal whims in deciding what they should subject the captive Special Treatment Unit patients to. To get the full flavor of the attitude of the researchers toward the clients requires extensive quotation from their papers. In one of these papers (Ludwig, 1967) the researchers described an artificial social system to propel, 16 male and 14 female patients who were residing in the Special Treatment Unit, into sanity—that is, the researchers sanity.

“Rather then settle for the unhealthy and unstructured social system of patients, we decided to create a new artificial system based on certain rational principles of responsibility and sanity. Within the framework of this artificial patient society, we wanted to minimize reinforcement for crazy and maladaptive behavior and to maximize the rewards for responsible, healthy behavior. Since we felt it would be helpful for patients to gain a clear conception of where they stood in relation to other patients in terms of sanity, we constructed a social caste and class hierarchy consisting of seven separate levels. This artificial social system was designed to encourage vertical mobility, whereby patients could move up or down the levels depending on scores they received on their weekly behavior rating. The privileges and responsibilities of patients are strictly contingent upon their weekly social level.” (Ludwig, 1967, p.391, emphasis added).

This early pre TCL research sets the methodological tone for all the future research. No validity measures were reported for any of the instruments used in this paper. Ludwig on page 396 did report a very high (.95) inter-rater reliability for one instrument, the STU Behavior Report, and claimed a study was done to ascertain it. He provided no citation for such a study or the methodology used.

forcing behavior change.
The behavior report (Chart D, Ludwig, 1967, p.392) which these researchers used utilized a points system ranging from 0 to 4, for various sets of behaviors (see next page)
<table>
<thead>
<tr>
<th>Chart D: STU Behavior Report</th>
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<tr>
<td><strong>Personal</strong></td>
</tr>
<tr>
<td>1. Dirty</td>
</tr>
<tr>
<td>2. Sloppy</td>
</tr>
<tr>
<td>3. Bad taste (clothes)</td>
</tr>
<tr>
<td>4. Lousy Posture</td>
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<tr>
<td><strong>Work</strong></td>
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<td>5. Dirty</td>
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<tr>
<td>6. Sloppy</td>
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<tr>
<td><strong>General</strong></td>
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<tr>
<td>7. Goof-Off</td>
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<tr>
<td>8. Snotty</td>
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<tr>
<td>9. Inefficient</td>
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<tr>
<td>10. Crazy</td>
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<tr>
<td>11. Obnoxious</td>
</tr>
<tr>
<td>12. Big mouth</td>
</tr>
<tr>
<td>13. Hatred</td>
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<tr>
<td>14. Belligerent</td>
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<tr>
<td>15. Greedy</td>
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<tr>
<td>16. Irresponsible</td>
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<tr>
<td>17. Stubborn</td>
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<tr>
<td>18. Close-Mouthed</td>
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<tr>
<td>19. Globo</td>
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<tr>
<td>20. Lazy</td>
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<tr>
<td>21. Passive</td>
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<tr>
<td>22. Blah</td>
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<tr>
<td>23. Vulgar</td>
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<tr>
<td>24. Tramp</td>
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<tr>
<td>25. a. Queer</td>
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<tr>
<td>b. Lesbian</td>
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<tr>
<td><strong>Total Behavior</strong></td>
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<tr>
<td><strong>Less: 1/2 # wrong on</strong></td>
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<td><strong>Total Score</strong></td>
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Chart D reveals the subjective and highly prejudicial nature of the various ‘behavioral’ categories. For example, the awarding of 0 points, a low score (bad), to men for ‘queer’ behavior (what ever that might be), and a high score (good) of 4 for ‘masculine’ behavior (what ever that might be) or similarly for women, 0 points for ‘lesbian’ behavior (bad) and 4 points for ‘feminine’ behavior (good) discloses the biases held by the clinicians about sexual behavior. (Ludwig, 1967, p.392).

These behavioral ratings were assessed by the clinicians. Depending on the weekly totals, which could range from 0 to 100 points, the clients were put in one of the seven ‘social castes’ each week. No criteria were provided as to how these assessments were made. Ludwig did say:

“Where disagreement concerning particular[behavior] ratings occurred among the staff, a vote was taken in the presence of the patient - the patient receiving the majority staff rating.”(Ludwig, 1967, p.391-92)

It appeared to be no problem for these researchers to replace science with the methods of popularity contests. Another example of the uninformed, prejudice riddled approach employed by the researchers is the name they gave to one of the social levels (charts E & F in appendix A ). Taboo behavior, those of-course the researchers defined as taboo, such as ‘fornicating’, ‘performing perverted sexual activities’, or ‘elopement from the hospital’, fell under the ‘well accepted’ empirical category of ‘mortal sin’.

The kind of coercive and intolerant approach, masquerading as science, exemplified by this piece of research I believe, speaks for itself as to its social and therapeutic usefulness. In my opinion it has none. The assessment
instruments used were subjective and biased, without any credible evidence offered by the authors for their use or their reliability and validity. They declared the scientific validity of their experiments while using professional authority to impose them on the patients, who were all, confined in the institution involuntarily. The authors theorized about the nature of the problems of their charges and contrived experiments to alter the patients’ ‘problematic’ behavior as if the patients’ imprisoned status had no impact on their behavior or on the outcomes. I believe that this research created the framework from which TCL grew. The view that these clients were cool calculating customers ‘hell bent’ on making trouble and therefore in need of severe punishment in order to force them to be ‘sane’ permeates this early research.

Assertive community treatment is a euphemistic label for coercion, which has a long history in institutional psychiatry and is exemplified by the research done in the STU unit of Mendota State Hospital during the Ludwig, Stein, Marx, Test era. Let me conclude this section with Ludwig's views on punishment. I believe that these represent the past and current views of the researchers, since none of them has ever renounced them and references to these articles have appeared in recent papers on the Training in Community Living Model, by these researchers (Stein & Test, 1985, p.15);
“One of the immediate ethical issues involves the use of punishment for patients. Without delving into all the aspects of this problem, ... , we will simply say that this issue is largely artificial or moot, for there are no psychosocial techniques for instituting human behavioral change which do not employ the very potent tools of both reward and punishment. Even those programs which espouse only benevolent approaches make liberal use of such negative reinforcements as withholding privileges, withdrawing love or approval, restraints, and seclusion, ECT[electric convulsive therapy], and drugs for the avowed purpose of ‘controlling’ patient behavior, but the rationales offered are often only euphemistic or socially condoned excuses for subtle or blatant punishments. The issue is not whether punishments should be used; they are and will be- this is simply a fact of all clinical and social life. The real issue is whether punishments will be administered openly, non-apologetically, and in a consistent, systematic, goal-oriented manner rather then on a disguised, apologetic, whimsical and haphazard basis.” (Ludwig, 1967, p.746-7)
Chapter 4

The Training In Community Living Model

"A very popular error: having the courage of one's convictions; rather it is a matter of having the courage for an attack on one's convictions!" (Nietzsche as quoted in, Kaufmann, 1958, p.34)

Description of the TCL model

According to Test (Test, 1992) there are four essential characteristics that comprise the model.

1) Core Services Team- “the primary provider of services in TCL is a core services team. This community-team consists of ... interdisciplinary staff who serve as a fixed point of responsibility for ... young adults with schizophrenic disorders (elsewhere Test et al argue that all severely mentally ill persons should be treated by TCL, see Test, 1992, p. 166). The team’s function is to see that all the patient's needs are addressed in a timely fashion. ... Having one team provide most of these services minimizes the ... fragmentation of ... care systems and allows for integrated clinical management.

2) Assertive Outreach and In Vivo Treatment- An essential ingredient in the way services are delivered ... is the use of assertive outreach. This means that staff ... reach out and take both biological and psychological services to the patient. Indeed, the majority of the treatment and rehabilitation ... take place in the community ... .The rationale for use of assertive outreach is to minimize patient dropout and to enable the provision of psychosocial services in vivo, right where the patients need to use them.

3) Individualized Treatment- Because persons with serious mental illnesses, even within the group diagnosed as schizophrenic, are greatly heterogeneous and both person and disorder are constantly changing over time, treatment and supports must be highly individualized. ... The content, amount, timing, and form of treatments/services provided vary enormously both between patients, and within the same patient across time.

4) Ongoing Treatment and Support-... studies of community treatment using a variety of models have ... revealed a loss of gain following discharge (Test, 1981) It must be concluded that even very intensive community treatment models do not provide a cure for severe mental illness, but rather provide a support system within which persons with persistent vulnerabilities can live in the community and grow. It appears these supports must be ongoing rather than time limited." (Test, 1992 p.154-6)
These TCL treatment model characteristics were structured based on the, “
... broad biopsychosocial model of serious mental illness” presented by Strauss
and Carpenter in their 1981 book called *Schizophrenia*.

“Such a model suggests that programs must provide interventions in all three
areas(biological, psychological, and social), focusing not only on changing the
person but also on changing the environment.”(Test, 1992, p.156-7)

The preceding four characteristics, plus the conceptual model of Schizophrenia
originated by Strauss & Carpenter, make up the justificationary framework of
TCL.

Taking this framework seriously raises some preliminary concerns. First, the
researchers, beyond stating that they(TCL) will adopt the Strauss & Carpenter
mental illness model(strictly speaking it is only a model for schizophrenia) offer
no explanation why this makes sense. Is this model well tested? If so, where's
the evidence? Even a cursory reading of Strauss and Carpenter's book makes
clear that these authors are only guessing and perhaps hoping that their notions
are genuinely explanatory. They provide no well-tested corroborating empirical
evidence in support of their model, although like other contemporary psychiatric
theorists they pay homage to Kraepelin and Bleuler (p. 7).

“The introduction of this model [Kraepelin’s] has had a profound impact;
Kraepelin’s discrimination of dementia praecox from manic depressive illness is a
cornerstone of scientific psychiatry.” (p. 3).

Of course it is precisely Kraepelin's failure to do just that, discriminate his
concept of dementia praecox from any other putative mental disease
scientifically, that I demonstrated in my analysis of his research in chapter one
among other things.
Strauss and Carpenter without much effort to hide the nature of their conceptual effort freely admit,

“Since the synthesis of information into a working concept of schizophrenia cannot be accomplished without a point of view [more correctly without testable, falsifiable, hypotheses], ... it is useful to clarify our bias from the beginning.” (Strauss & Carpenter, 1981, p.4, emphasis added).

What follows the quoted text in the chapter, is a list of assertions and as admitted by the authors their biases as to what they believe characterizes their hypothetical construct of schizophrenia, but, as stated before, no well-tested evidence is offered anywhere in the book for these claims.

The book is a rehashing of most previous theories (biological as well as psycho-social) about the possible etiology and nature of the claimed disease and some treatment suggestions. These treatments similarly are a rehashing of various current interventions. Note their attempt at mystification;

“Since Kraepelin and Bleuler originated the concept of schizophrenia, steady progress has been made in the acquisition of knowledge necessary for understanding this disorder. Despite this progress, the essence of the puzzle remains unsolved.” (Strauss & Carpenter, 1981, p.7).

If indeed we have made progress, it would be nice if the authors would share the available corroborating evidence, or at least cite it. If such progress does exist at the periphery, since the essence according to these authors, is still a mystery, what possible information does this claim convey?

They also state in one of their four principles, “The boundaries of schizophrenia are not so well defined as the core” (Strauss & Carpenter, 1981, p.5). This appears to contradict their previous claim of progress in general knowledge, or perhaps it doesn't, it's most difficult to tell. What is clear however,
is that if you assert that in schizophrenia, “... social, psychological, and biological processes are involved” (Strauss & Carpenter, 1981, p.5), you are asserting a triviality and not a profundity. All human activity entails these processes and therefore this claim is not informative. The TCL researchers by using the Strauss & Carpenter biopsychosocial model to explain mental illness, are using, not a scientific, but a pseudo-scientific, possibly a metaphysical, model of explanation, which is not testable even in principle. It is too broad. Anything and everything may be subsumed by it. Any and all behavior may comprise one or more components of this model. The vague and imprecise nature of this model provides no independent way to test its claims outside the framework. For example, principles three and four read as follows:

“Because social, psychological, and biological processes are involved in schizophrenia, it is incumbent upon the investigator and the clinician to grapple with genetic, biochemical, and neurophysiological mechanisms on the one hand and psychological, social and cultural mechanisms on the other.”

“Patients with schizophrenia vary in their prognostic status, and a full range of outcome functioning is possible. Course of illness is not irrevocably established early in illness, but many factors (often socioenvironmental) interact with the ill individual to facilitate or impede recovery.” (Strauss & Carpenter, 1981, p.5).

Beside the considerable technical problem of identifying, describing, locating and proving the causality of the various putative mechanisms, Strauss and Carpenter allege, are involved in Schizophrenia, this all too comprehensive model provides a fertile environment for the growth of endless numbers of alleged etiological explanations. In their book you find; the genetic explanation, the various bio-chemical explanations (the dopamine hypothesis, the transmethylation hypothesis, the neuromuscular dysfunction hypothesis, the
various viral theories, the enzyme hypothesis), the psychophysiological explanations, and the psychological and social explanations (developmental explanations, life stress explanations, social environmental explanations, family impact explanations) (Strauss & Carpenter, 1981, chapters 7 and 8).

It is obvious that almost any rationale for etiology is allowable in this framework, nothing is excludable (refutable), and ad hoc statements may be used to explain away any potential falsifications. The rule in science is, the fewer (one, is the best), unfuted explanations, the better (the more likely that our theory approximates reality). In pseudo-science relativism rules, the more theories, the merrier. Power and subjective authority replaces evidence when searching for truth. Note for example, the many religious and mythical explanations for the origin of our species vs. the single scientific, Darwinian, evolutionary explanation. This problem, along with the confusing and reifying of autonomous behavior as psychiatric symptomatology, subverts the effort to use a critical scientific methodology in institutional psychiatric research.

Additionally, as I argued in chapter 1, the inability of psychiatry to empirically demonstrate the existence of a discrete set of non-randomly recurring signs and symptoms, that could possibly corroborate the existence of a syndrome of schizophrenia fatally undermines any current attempt at a scientifically meaningful explanation of schizophrenia as a disease. This lack of evidence for schizophrenia’s empirical substance logically forecloses the possibility of finding legitimate (scientific) treatments targeted at such a putative entity as well. But of course, I will proceed to evaluate just such treatments in later chapters despite
what I believe to be the overwhelming falsifying evidence, because such mental health research is constantly done, human nature being what it is.

So much for the scientific bases of the TCL model. Of course the researchers could argue that the Strauss & Carpenter model is only heuristic. That would be fine, but would change the character of their claims about mental illness. It would then be just a conceptual guide, a hypothesis to be tested (and rejected if falsified) and not the allegedly well proven entity claimed and used to justify the expenditure of public tax dollars.

**Conceptual Difficulties**

The TCL model as detailed by Test et al., and described previously has some fundamental conceptual difficulties. The idea that a Core Service Team can provide *all the necessary* services that a person needs is philosophically naïve and according to some important social thinkers, utopian\(^{22}\) (Hayek, 1979; Popper, 1961). Such utopian efforts are logically untenable (see footnote 22); and

\(^{22}\) Popper, criticizes Utopian (Holistic) social intervention on the grounds of logical impossibility (Popper, 1961, p.70-93). It is not possible to identify, comprehensively and conclusively, all needs a person may have, even, at a fairly specific point in time. Different people may identify different needs and different numbers of needs depending on their particular perspective and situation. Many putative needs are social constructions while others are concrete and objectively definable necessities such as food and clothing. Examples of some socially constructed are; a need for social skills, or a need for skills of daily living. Most of these types of skills are contextual. The nature of the client’s social and material environment will dictate the kind of ‘skills’ required. Consequently, there is no way to train a team of experts to provide the virtually innumerable interventions potentially needed for the satisfaction of all the possible needs covered by this claim. This doesn't take into consideration the services necessary for such unexpected and potentially unforeseeable needs as services for the iatrogenic effects of the interventions themselves. These are the services needed to cope with such problems as, tardive dyskinesia and sexual dysfunction caused by psychotropic medication, for example (see generally Breggin, 1983).
so would be the TCL claim, if it were intended to be taken literally, rather than, as the use of poetic license to enhance the model’s market value.

It is undeniable that TCL(PACT) proponents such as the National Alliance for the Mentally Ill(NAMI) are heavily engaged in promoting this model throughout the country,

“... the NAMI/PACT Initiative Steering Committee concluded that a comprehensive strategy is necessary to influence the dissemination of assertive community treatment programs and closer adherence to the PACT model specifically. The goals of the initiative are to:

Design and implement a means of rapid and effective replication of the PACT model of ACT;

Promote a consensus among public mental health authorities, advocates, and service providers for adoption of national standards to set minimum criteria for ACT programs; and

Influence state and local mental health authorities that have not already done so to adopt ACT as a core program within their service delivery system.

To carry out the work of the NAMI/PACT Initiative, a new organization will be established, Programs of Assertive Community Treatment Incorporated (PACT, Inc.). PACT Inc. will be a private, nonprofit corporation with national focus and representation of consumers, family members, clinicians, administrators and researchers dedicated to the dissemination of the PACT model.” (CSNN, 1997, p. 10).

I will assume here that although there is a self-serving marketing component to this claim, the program inventors also sincerely believe in their approach.

Due to its all-encompassing service agenda we could perhaps rename this effort the ‘valet service’ concept of mental health treatment. This claim of

Finally, the reader should note the naive optimism expressed in an approach that assumes that all human problems have solutions (that all needs are satisfiable). This is just another aspect of the Utopian outlook, which can be contrasted with a more realistic view of the world where much of existence is not under human control, and not, as a matter of course, malleable to our interventions. This Critical Realist outlook, further sees life as problematic, full of inherent difficulties. These problems may or may not be solvable. The sense of the Tragic (Unamuno, 1972) is recognized as the defining characteristic of life as supported by human history and does not view existence from the narrow modern perspective offered by the very unusual circumstances of late 20th Century America. This narrow perspective can be characterized by the ‘can do’ attitude that finds solutions everywhere. This naive world view is mediated and supported by the relatively little material deprivation present in contemporary American society.
exhaustive client need fulfillment is also a problem because the researchers are proposing to use the Core Service Team interventions on a conceptual construct,\textsuperscript{23} the Strauss & Carpenter model of schizophrenia, for which we have no experimentally well tested evidence indicating its empirical reality as explicated earlier. Cures without a disease are like sex without an object, conceivable but not very effective or interesting.

Moreover, the nature of the services actually provided have more to do with what the providers define as needs then what the clients desire. The idea of clients having specific ‘needs’ must assume some ‘gaps’, as defined by the experts, in the social, environmental, or personal domains impacting the client, which according to the TCL model, \textit{must} be, ‘compensated’ for by one or more of the following; employment training, skills training, rehabilitation, education, environmental and behavior modification\cite{Test, 1994, p.154-8},

\begin{quote}
“Such a model\cite{the broad biopsychosocial} suggests that programs must provide interventions ... focusing not only on changing the person but also on changing the environment.”\cite{Test, 1994, p.156-7}.
\end{quote}

This agenda for changing the person and environment must further assume as a rationale some notion of \textit{necessary} change; from some behavior or situation defined as negative, unhealthy, inappropriate, abnormal, to some other behavior

\textsuperscript{23} A whole school of philosophy has focused on conceptual analysis leading nowhere. Analytic philosophy although still very prominent is barren of any analytic fruit about what is true (professor Wakefield mentioned earlier is one of its foremost proponents in mental health and social work and his work illustrates well this approaches failings). It endlessly explicates words and their meaning, as if unaware of the infinite regress innate in all such efforts\cite{since all concepts and words have to be defined in terms of other concepts and words, which themselves have to be further defined in terms of other words and concepts, which in turn have to be further defined in turn etc.}, ignoring the original reason for analysis as argued for by Bertrand Russell, the inventor of analytic philosophy; which was, to be as clear and explicit in stating explanatory hypotheses as was possible in-order to allow the most critical tests of their truth value in the real world\cite{Magee, 1997}
or situation presumably felt to be better, which, *must be* provided to the client. Professionally defined expectations of client change for the better can be coercive and patronizing, not to mention just plain wrong, if not identified as also needed by the person who is to undergo the change. The authors, nowhere in their writings explicitly state or even indirectly suggest that this decision is the autonomous right of the TCL clients. Furthermore, appropriate change of environment or behavior, is somebody’s version of such change. The values and ethics that frame this determination are of primary importance. Who should be authorized to define appropriate change of client environment and behavior? Should it be; Test, Stein, Marx, Ludwig the various inventors of TCL or the clients?

The idea, that whatever a client’s needs are, is what will be addressed by the TCL program is ingenious as a marketing concept, but what does that mean in practice? Stein explains;

"The ACCT( the team) serves as a fixed point of responsibility ... and is concerned with all aspects of their(lives) lives that influence their functioning, including psychological health, physical health, living situation, finances, socialization[could this include defining proper sexual behavior, gay or straight? see section on treatment approaches], vocational activities, and recreational activities. The team sets no time limits for their involvement with patients, is assertive in keeping patients involved ... In addition to the day to day work ... the team is available 24 hours a day, seven days a week ... "(Stein, 1990 p.650, emphasis added).

This methodology²⁴ if implemented as described is highly intrusive. Typically,

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²⁴ The description of the technology of intervention of TCL, is very vague in all the papers published by the researchers. It is remarkable that the voluminous writings of the TCL inventors does not include even one(as far as I've been able to determine) detailed case example of the methodology at work. They provide what they call a case example in Stein and Test, 1978 on pp. 50-52, which is a vague and unhelpful example. They assert in this ‘case example’ that in the admissions office, at the first meeting with John(the future client) and
TCL activity may include such coercive moves as becoming the representative ‘financial payee’ of the client. This ‘helpful’ service provides opportunities for blackmail of the clients by the service providers. For example, forcing medication compliance on a client, by threatening to withhold monies belonging to the client (Stein & Test, 1985, p.88-9). Another typical coercive activity appears to be, forcing treatment on clients who do not want it (p.91-92). On-going (eternal?) assertive community treatment, desired or unwanted is justified by the claim that when such treatment stops the intervention effect evaporates (Test et al, 1991).

This they say is so, because although,

his family, John was “in the midst, of a schizophrenic episode, but was not immediately suicidal”(p.50). How this was assessed is not described nor are any of the therapeutic interventions that helped in John’s dramatic clinical improvement ‘within a week’ explained. The most helpful information is in a paper by Test (Test, 1992). This paper is still very imprecise, as for example on p. 157, under the heading of ‘Direct Assistance with Symptom Management’, the interventions for this problem are described thus; “The minimizing of psychotic symptoms and the prevention of relapse is a continuing priority of our program ... . Specific interventions employed ... include medication ... , 24- hour crisis availability, and occasional brief hospitalization. Additionally, we provide each patient with a long-term one-to-one relationship aimed at problem solving, at assisting them to learn about their illness, and at enhancing their own coping strategies for dealing with serious symptoms.”.

Beside dispensing medication and the brief hospitalization, which are concrete interventions, all the others are vague and open to many interpretations and do not tell an interested professional anything about what the TCL model services actually (specifically) do.(To see the further vagueness of program description, read Test, 1992, p.156-58).

Final point. This, allegedly, comprehensive and intensive program(TCL) spends 21.4% of its contacts with patients(at least during the first six months, no published data is available for contacts over longer periods) medicating them. This service takes up the second greatest number(21.4%) of worker contacts with clients. First in number of worker contacts is, one to one support(24.9%), which, from what Test writes(Test, 1992, p.157), might for the most part be spent convincing the clients that they are mentally ill and therefore in need of psychotropic medication. With the program already spending 21.4% of its client contacts dispensing meds, could TCL be spending 46.3% of total client contacts dispensing psychotropic meds and providing related management? Compare this to 10.9% of client contacts spent on vocational issues, 2.5% on their living situation, 2% on physical health, 12.1% on social recreation, 11.3% on psychotherapy(whatever that is)/case monitoring, and 9.2% on activities of daily living(for service contact breakdown see, Brekke and Test, 1992, p.240).

Surely, for enhancing client change, as opposed to enforcing control over the client, those array of interventions which are not ‘medication and related management interventions’(i.e. clients learning about their alleged illness) should be the ones deserving the larger portions of such contacts.
“It is not clear why, across a range of empirically supported biological and psychosocial interventions, the positive effects of treatment end when the intervention ends, but a likely hypothesis is that the underlying psychobiological vulnerabilities and/or deficits of schizophrenia persist for many patients. This suggests that these patients may need ongoing rather than time-limited special supports in order to survive in the community.” (Test et al., 1991, p. 240).

I believe an alternate explanatory hypothesis might be worth considering, especially since the construct of schizophrenia, is highly suspect. My conjecture is that, instead of the intervention being effective while applied and effect vanishing when treatment’s stopped, what actually happens is that the ‘hyperactive’ program while being provided, using aggressive coercive methods, counts(substitutes, mistakes, or confuses) the very active effort of workers as the effort or outcome of clients. If, for example, you invade a reluctant client’s home and force the client to go to work, by threats and by use of your authority(Stein and Test, 1974, p. 268), even when the client, both by her actions and words, insists that she doesn’t want to go to work(Test and Stein, 1976, p. 77), then you have not accomplished your claimed goal of increased employment of such a client. The most that has been demonstrated is that such pressure by a worker on a client can get the client to the job site very reluctantly, but certainly no claim can be made of having improved the actual work effort of the client(nothing has been internalized independently by the client as to the value of doing the coerced activity absent duress).

The fact that clients don’t show up for work on their own after these assertive community treatment interventions are discontinued tends to corroborate my conjecture. The ‘effect loss’, is an artifact of workers no longer coercing the
activity that is being measured. This program failure or ‘effect loss’ is found in all of the TCL research although never investigated from the point of view I am presenting. I believe my argument more comprehensively explains the so called ‘loss of gain’ (Test, 1994, p. 156) of these programs then the explanation provided by the TCL group; the problem is explained away by the researchers as the result of the alleged incurable nature of mental illness which can be treated but not cured. A perhaps incidental but professionally convenient outcome of the researchers’ claim is, that if ongoing treatment is ‘found’ to be necessary, a long term stream of funds would have to be forthcoming to support the program, providing a steady source of income for the experts involved.

To validate the use of assertive outreach the TCL researchers rely on two studies, one of which is their own (this study is evaluated in the next chapter). They claim that the, “Studies that have used such assertive approaches have demonstrated their feasibility and effectiveness (Beard et al., 1978; Stein & Test, 1980)” (Test, 1981, p. 80).

The definitions of outreach used by Beard et al., and Test et al., do not coincide and Beard et al., doesn’t support the type of assertive approach Test et al., use. This is how Beard et al., describe their program’s ‘reaching out services’;

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25 The concept ‘assertive’ as used by Test and company differs from how the research literature usually defines this concept. The field of behavior therapy has for a long time been interested in assertion training. Assertive behavior is defined in that literature for example as, “... effective social influence skills that are acquired through learning,” (Gambrill, 1995). And as opposed to the TCL approach (as demonstrated in the present paper), “Fundamental to the concept of assertion is a concern with basic human rights,” (Ibid., p. 82). Assertive and aggressive behaviors are carefully distinguished both by their form and their effects (Ibid., p. 85). Test et al., do not differentiate ‘assertive’ from ‘aggressive’ behavior. They appear to be interchangeable in TCL. In this instance as in many others the TCL researchers appear ignorant of the research or care not a fig for it.
“Reaching-out services consisted of phone calls, letters, and home and hospital visits made by both staff and members. Through such contacts, subjects who dropped out were provided with further information ... . It was hypothesized that such information would lead to participation in the (Fountain House) program. ... In those ... instances when an individual requested that no further contacts be made, his wishes, of course, where respected.” (Beard et al., 1978, p. 624, emphasis added).

The emphasis, on respect for the wishes of people who choose not to be involved in the Fountain House program, by Beard et al., is a striking contrast to the coercive methods used by Test et al.,

“For instance, a staff person attempting to assist an ambivalent patient to a sheltered workshop in the morning is likely to receive a verbal and behavioral ‘no’, if he approaches the patient with a firm, ‘It’s time for you to go to work; I’ll wait here while you get dressed,’ the likelihood of compliance increases. The latter method allows less room for the patient to ‘choose’ passivity ... .” (Test & Stein, 1976, p.77).

Two questions immediately come to mind. First, who decides that the patient in the above quote is ambivalent as claimed, as opposed to let’s say, as a close reading might more correctly indicate, resolutely opposed to going to work? Second, is there not in this quote a professionally opportune, but never-the-less mendacious redefinition of the active refusal of the client (who uses ‘a verbal and behavioral no’), into ‘passivity’ or non-action? The disregard of clients expressed wishes, and the reinterpretation of client behavior to justify programmatic interventions, appear to be the outstanding characteristics of programs using TCL style ‘assertive’ methodology. The claim of effectiveness for their assertive approach, is not substantiated by the single other program they have used to justify this assertive methodology. The Fountain House model (Beard et al., 1978, p.622), in its outreach effort establishes contact with program dropouts and gives
them information in the hope that this additional contact will reconnect them to
the program. But, if they (the program) are asked to stop their outreach effort by
the dropouts, all outreach is discontinued. TCL on the other hand assumes that
clearly expressed volitional behavior, such as declared unwillingness to go to
work, which contravenes TCL goals, is in fact, non volitional behavior caused by
the mental illness. The Fountain House model of outreach does not resemble the
TCL assertive outreach model, except to the extent that both are called outreach
efforts. Beard et al., however never call their outreach, assertive, leaving Test et
al., with only their own research as supporting the effectiveness of the type of
assertive model they advocate. Coercion is not a part of the Fountain House
model of ‘Reaching-Out Services’, it is a vital part of the TCL model.
Chapter 5

A Description and Critique of The Three TCL Studies

“We have now sunk to a depth where the restatement of the obvious is the first duty of intelligent men.” (George Orwell)

The originators carried out three studies on this model. Each is reviewed and critiqued in the sections to follow.

A general note on methodology:

One study (Marx et al., 1973) claimed to have used blind and independent researchers (raters) in gathering data, the other two studies (Stein and Test, 1980; Test et al., 1991) only claimed the use of independent researchers. Stein and Test do not describe how research staff was trained in any of their studies nor do they explain what is meant in these studies by blind or independent. The three studies used an array of instruments to collect the data and to evaluate various outcomes. No information is provided about the reliability and validity of these instruments in any of the published papers on these three studies, although citations referencing articles on various instruments is provided. This vagueness and lack of information makes it impossible for interested scholars to evaluate the various claims for the TCL model made by the investigators, by simply reading their articles. It requires following up citations that they provide for the various instruments, evaluating these articles to see what if anything they provide regarding the validity and reliability of the measuring tools. This can be time consuming, although essential as I demonstrate, if meaningful evaluation of claims is to be attempted. Few people may be willing to undertake this task and
consequently questionable research claims can go unchallenged and programs are implemented based on them.

**The first study: Extrohospital Management of Severe Mental Illness (Marx et al, 1973)**

This study involved 61 inpatient clients at Mendota State Hospital who had a variety of diagnoses with most (79%), labeled schizophrenic. At the time of the study they had been hospitalized from three to eighteen months. They had been given a ‘prognosis’, by the hospital staff of “not currently capable of sustained community living” (for complete criteria see, p.506). The subjects were randomly assigned to either the Training in Community Living experimental group (Community Treatment Group, CTG, n = 21), or to one of two control groups. The Research Unit Controls Group (RUC, n = 20), received inpatient treatment from the same clinicians who provided the experimental group’s Community Treatment, while the other control group (Other Unit Controls, OUC, n = 20), remained in their referring wards and received the routine treatment provided by the clinicians assigned to those wards.

The results after five months of treatment were claimed to:

“... indicate(d) that ... the experimental group in the community, ... following treatment ... had attained more autonomous living and employment situations than controls.” (ibid., p. 505).

To be more precise, as a sympathetic reviewer of the Assertive Community Treatment literature wrote in his 1990 review;
“Marx and colleagues found that after five months of treatment, the TCL patients had spent significantly less time in psychiatric institutions and more time in semi-sheltered settings than either control group. The experimental patients also achieved higher occupational functioning than the controls, but were not significantly improved on various measures of psychiatric symptomatology.” (Olfson, 1990. p. C-70).

Critique:

The first important point to be made is that only the Research Unit Controls (RUC) can be considered an adequate comparison control group because only this and the experimental group (CTG), were evaluated *blindly* and found not to differ significantly at base line for psychiatric symptomatology. The Other Unit Control (OUC) group did not receive this evaluation nor was administered any of five other measures provided to the experimental (CTG) and research unit control (RUC) groups at varying intervals during the study (Marx et al., 1973 p. 507). The researchers claimed that for various administrative reasons they could not test this group (Marx et al., 1973 p. 508). This situation makes the assumption of group equivalency problematic for the Other Unit Control group. As Cook and Campbell pointed out,

“Obviously, with a small initial sample of persons who (may)differ widely on a dependent variable of interest, random assignment may result in dissimilar groups.” (Cook & Campbell, 1979, p. 341)

Since the largest group n was only 21 it cannot simply be assumed (as the researchers would like), that this group is comparable just by the initial randomizing of subjects to the three groups. This hypothesis was corroborated by the fact that 9 patients from this (OUC) group vs. 4 for the RUC group and 1 patient for the CTG group were found to be institutionalized at the end of the
study (presumably the OUC group differed in the severity of symptomatology, as can be seen in table 3, appendix B). A similar claim can be made by looking at table 4 (appendix B) which lists the employment status of the various groups' members at the end of the study. Fifteen patients of the OUC group were unemployed as compared to 6 for the RUC control and 8 patients for the experimental (CTG) group, suggesting again that some initial group differences might have been at work, making the OUC group inappropriate for inclusion in the analysis.

The Second methodological concern was that the medicating of the patients with psychotropic drugs appeared to be very high in this study (about 90%, p.508). Possible medication effects may blur any causal outcome claims directly attributable to TCL treatment versus medication compliance. The authors note, “We can not partial out how much of the gains made by the E subjects was secondary to the success of the TCL program in gaining medication compliance as compared with its psychosocial interventions”(Stein and Test, 1980, p. 396). This appears to be the case for all three studies done by the TCL group. The first study (Marx et al., 1973), was the only study of the three that provided the number of clients receiving psychotropic drugs. Neither of the other studies discussed this issue. The well known problems with these drugs, should have necessitated serious discussion both about the harm they might have caused to the patients' health as well as to the effect they might have had in distorting the so called outcomes of the TCL interventions. Did these drugs by their general tranquilizing effects ‘eliminate’ or reduce unwanted, or disliked behavior.

The most often promoted claim of all assertive community treatment programs,
including TCL, is that there is a significant reduction of inpatient hospital stays (measured in days) in the treated as compared to the control group. I contend that this statistically significant greater reduction in hospital stays is not the result of the experimental program’s services, as suggested by the researchers, but is the result instead of the initial policy decision not to admit (hospitalize) any patient in the experimental group and to carry out all treatment of the experimental group in the community, regardless of the symptomatology or difficult behavior of the patients (see under Procedure p.506). The outcome of reduced hospital stays is therefore tautological to the program design.

If you place the experimental group in the community and keep them there regardless of their behavior, which in controls would have led to hospitalization, than it follows tautologically that they will spend (much)less time than controls, who are not kept out of the hospital by a rule not to hospitalize, in the hospital. The arbitrary decision not to hospitalize the experimental group fatally confounds any meaningful analysis of the living situations of the control and experimental groups in this study as well as of the various intervention effects, since we have no way of knowing what kinds of living situations people in the control group(RUC) would have been in if they also would have, by administrative fiat, not been allowed to stay in the hospital although treated there. It should also be noted that the experimental group members spent time in a penal institution 41 times as often on the average as the control group(RUC), 6.48 days for the experimental group as compared to .16 days for the controls (see table 2 appendixx B).
The authors’ claim that the experimental group had more autonomous employment situations, table 4 (appendix B) then controls has serious flaws. We cannot use the OUC control group as a valid comparison group. As explained earlier, this group was potentially significantly different from the other two groups at the start of study. So the claim of more unemployed among the controls (15 patients among the now disqualified control group, OUC) was reversed, because only 6 patients from the RUC control group were unemployed as compared to 8 patients from the CTG experimental group.

Here by the way, was a typical example of selectively choosing supporting data without rhyme or reason, just as long as it appeared to support the TCL claims. For how else to justify the use of one control (OUC) group’s outcome (in this case unemployment status) showing apparently a favorable outcome for TCL, verses the other control (RUC) group’s outcome, showing an unfavorable outcome? Setting aside the fact that the OUC control group was disqualified from consideration (due to issues previously discussed), shouldn’t the TCL experimental group have had significantly fewer people unemployed as compared to both control groups for the TCL researchers’ claim to more autonomous employment situations by the experimental group to have been valid?

The claim that the experimental group achieved higher occupational functioning (competitive employment is considered higher occupational functioning than sheltered employment) was also only partially supported by the experimental results. According to Table 4 (appendix B) identical number of
clients were employed (either in sheltered or competitive employment) from each group (13). Again I am excluding the invalid OUC group from the analysis. The CTG (experimental) group had 4 in sheltered employment as compared to 10 for the RUC (control) group and 9 in competitive employment as compared to 3 for the RUC group.

It is known that serious experimenter bias issues arise when dispositional judgments are made by non blind researchers evaluating and treating both control and treatment groups (Rosenthal, 1976). Even the most fair minded researchers may inadvertently influence interpretation of results as well as the treatment outcome. The placement of the control clients more often in sheltered employment, as compared to experimental clients, may have been confounded by experimenter/clinician bias. It is also important to note that in the competitive employment category the same number (2) of patients from both groups worked full time. If the criteria of employment success was measured by which group had the most number of patient members achieving the most autonomous employment situation (full time competitive employment) than control and experimental interventions worked (or did not work) equally well.

In Summary

The claim that experimental patients were living in more autonomous situations (not in the hospital) due to the TCL treatment was clearly false as demonstrated by my analysis. No experimental intervention was needed to create this situation. All that was necessary was the simple refusal to admit anyone in the experimental group into the hospital regardless of the severity of their problems.
Marx et al's data even seems to suggest that the experimental program led to more time spent in penal institutions.

The claim of higher levels of occupational functioning by the experimental group was not well supported. First, the data indicated that more people were left unemployed (8) by the experimental program than were unemployed (6) by the control (RUC) program (see table 4, appendix B). Second, a good argument can be made for bias by the clinicians against the control group due to both clinician and experimenter bias. Third the identical number of patients achieved full time employment in the competitive employment category from both groups. These three issues make the claim by the researchers to more autonomous employment by the experimental group at least highly problematic if not incorrect. As a result, this study is stripped of the most interesting of its claims, leaving us with the rather unremarkable and by me uncontested finding that it was, "feasible to treat the experimental group in the community, ... "(ibid., p.505).

The second study: *Alternative to Mental Hospital Treatment* (Stein and Test, 1980)

This study was a follow up to the previous pilot study by Marx et, al. This study consisted of 130 subjects who;
... where randomly assigned by the admission office staff (of Mendota State Hospital). Control subjects (n = 65) were treated in the hospital for as long as necessary and then were linked with appropriate community agencies. Experimental subjects (n = 65) did not enter the hospital ... but instead received the TCL approach for 14 months before integration into existing community programs. Assessment data on all patients were gathered at the baseline (time of admission) and every four months for 28 months through face to face interviews by a research staff that operated independently of both clinical teams." (ibid. p. 393)

The authors claimed that the results of this study supported the findings of the first study that experimental subjects spent significantly less time hospitalized than controls, along with having spent significantly less time unemployed and having earned significantly more in full time competitive employment than controls (although no significantly more time was spent in full time competitive employment by experimental group members). The most important claim that they made was that TCL treatment was able to reduce psychiatric symptomatology significantly among the experimental group as compared to the controls. This claim, if correct, is a very important piece of evidence supporting the effectiveness of TCL treatment and would help buttress the arguments which support the current widespread use of this model. I will discuss 5 major dependent variables:

**Living situation:**

Here, as in the first study reported, the claim of reduced hospitalization as an outcome of intervention was actually the result of choosing, by way of a simple a priori rule not to hospitalize clients in the experimental group regardless of
behavior, not the result of any program interventions. Nothing new was added to the structure of the experimental program to alter this conclusion from the first study. Table 1 (appendix C) indicated that at 8 months there was a statistically significant difference (p=.01) in the independent living situation favoring the experimental group and at 12 months a smaller statistically significant difference of p=.05 also favoring the experimental group. There was no difference in the independent living situation between the groups at 4 months. Both groups spent much more time living in independent than in supervised situations when not in institutions, with the most statistically significant measure (p=.01 at 8 months) accounting for only a 16.45 mean percentage of difference between the groups. Although this was statistically significant it had no practical significance. The authors used the well worn and erroneous practice of talking about statistical significance as if it was the equivalent of practical significance. Studies often leave the reader mystified as to how the word ‘significance’ is being used. We are primarily looking for tangible differences in programs costing tax dollars and effecting people.

26 This study by using only an ‘independent’ research staff is notably different from the first study where there was blind and independent evaluation as to patient symptomatology (where no differences in symptomatology were found). Obviously this undermines and strongly prejudices any claims at gathering unbiased data for evaluation by the current projects research staff.

27 Since this Table (1)(ibid., p.394) is labeled Mean Percentage of Data-Collection Periods Spent in Various Living Situations we don’t really have a very clear idea of how big the variation is among the individuals staying in independent living situations in these two groups. The standard deviations clearly suggest a greater range among the control group whose length of stays may be influenced by the re-hospitalizations which may have occurred to them, but which by administrative fiat were extremely unlikely for the experimental group. For additional provocative, and highly relevant criticism of the use of significance testing in the social sciences see, Oakes, 1986, especially chapters 1 and 2.
Employment status:

The program claim, for the experimental group spending less time unemployed and more time in sheltered employment (p.394) was explained, I assert, by the greater amount of time spent hospitalized by the control group. Re-hospitalization made it much harder, if not impossible, to maintain a job. While re-hospitalized and not working, the time-spent unemployed also must have increased. To state the tautology explicitly; not working constituted being unemployed. Re-hospitalization and less sheltered employment were the results of the rule permitting frequent hospitalization of the controls and, in the alternative, not permitting the hospitalization of the experimental group. This conjecture was also supported by the fact that there was no difference between the groups in the amount of time spent in competitive employment (p. 394). This confirms that the experimental program was not superior to the control group’s competitive employment training program, if there was one. The section on Control Treatment (p. 394) is unclear as to whether such control interventions even existed for enhancing competitive employment skills.

The claim that the experimental group earned significantly more income through competitive employment was again a result of the allowable re-hospitalization of controls. Since the controls were routinely re-hospitalized and experimentals were not, the controls had to have shorter lengths, although no less total time, of competitive employment. This required that they work more part time, therefore
lower salaried jobs, consequently earning on the average less money.28

Satisfaction with Life:

The researchers reported simply that, “E subjects were significantly more satisfied with their life situations than were C subjects at 12 months.” (p.395). No data were presented for life satisfaction for any other period. Elsewhere we found that the 12 month life satisfaction measure of statistical significance favoring the experimental group was (p=.05)(Test & Stein, 1978, p. 69). This result was unremarkable because the existing research literature indicates that most people who are treated and live in the community are happier than people who are treated in institutional settings, whether for medical or psychiatric reasons(Timko et al., 1993; Rosenfeld, 1992; Lehman, 1986; Polak, 1978; Wilder et al., 1966). This higher self reported satisfaction with life, I conjecture, is largely the result of the amount of freedom and autonomy the individual feels in his own home verses the restrictions experienced in institutions and is only indirectly, if at all, connected to a particular community treatment29. It appears that, almost any community treatment when compared to restrictive inpatient treatment seems to have higher self-reported client life satisfaction measures. This theme will reoccur throughout the balance of the research analysis.

28 My conjecture is substantively supported by a less-well-known and slightly different version of this report, describing the first 12 months of this program. The researchers state, “...that during two of the three data collection periods, E subjects earned significantly more income through competitive employment than did C subjects. Other data [not provided in any published reports] indicate this was probably the result of the fact that E subjects spent significantly more time in full time competitive employment situations than did C subjects, who spent more time in part time competitive employment.”(Test & Stein, 1978, p.65, emphasis added).

29Findings of two recent studies suggest that the less community treatment, especially case-management, the better(Huxley & Warren, 1992; Dietzen & Bond, 1993). One of the studies also found that only the monitoring component of community treatment correlated to increased quality of life measures and satisfaction with services for all clients.(Huxley & Warren, 1992)
Self-esteem:

This measure was not effected by the experimental treatment. There were no statistically significant differences after the initial baseline significant difference found in favor of the experimental group, (Stein & Test, 1980, p. 395). This finding of an apparently higher measured amount of self-esteem in the experimental group at baseline suggests an interesting hypothesis. Since there was this initial baseline difference and at all later data collection points there were none, it is possible that the experimental program had a negative effect, by reducing the self-esteem of the experimental group over time (possibly by its paternalistic and coercive interventions). There is suggestive data in Test & Stein, 1978, p.65, where they reported 6 experimental patients (10% of experimental group) leaving the program, against the researchers advice, between 8 and 12 months and seeking and gaining admission to psychiatric institutions not involved with the study. This may indicate that the coercive elements in the treatment program served to undermine the self esteem of these troubled clients leading them to request re-hospitalization elsewhere. Of course we cannot investigate this intriguing question because the data necessary (self-esteem measures of both groups over the length of the study) are not provided in any of the published articles.

Symptomatology:

The finding by the researchers in this area was unambiguous. The data, it was claimed, indicated a clear reduction of symptomatology favoring the experimental group at all data collection periods following baseline, culminating
with improvement in seven of the 13 symptom categories by the 12 month data collection period (Table 4, appendix C). My analysis of this finding will focus first on the table (table 4, appendix C) then on the instrument used by TCL to assess the psychiatric symptomatology and finally on statistical issues. First, a review of table 4 (appendix C) shows a remarkably fortuitous conjunction of seven symptom measures of statistical significance favoring the experimental group at twelve months. This served to convince the researchers of the effectiveness of the experimental model. But, if this happy occurrence was a demonstration of effectiveness it was of a curious type. Only three of the seven statistically significant symptom measures at 12 months (Anxiety or Fear, Thought Disorder, and Global Illness), were found to be significant in the measurement period at 8 months, and, none of these seven significant measures at twelve months, was found to be significant the measurement period at 16 months. Note also that three other symptoms of the seven symptoms showing statistical significance at 12 months (Depressed Mood, Suicidal Trends, Paranoid Behavior), showed no statistical significance at any other data collection point and only two symptoms (Anxiety or Fear, and Global Illness) had significant differences from the control group at both the 4 and 8 month measurement points.

This almost random variability (now it's significant and now it's not) of the symptom categories is not a pattern that supports the claim that a program
provides interventions that creates change in symptom intensity over time. Evidence for this claim would require a consistent finding of significance favoring the experimental group in the symptom categories over the entire intervention period. The data do not support this contention. An analysis of the development, construction, and validation of the instrument used to measure psychiatric symptomatology and its duration, called the Short Clinical Rating Scale (SCRC), suggests that it is of questionable utility. The instrument was developed by Heninger et al., 1970, for nursing staff in acute clinical settings. It includes 13 items, each regarding a defined pathological behavior such as depressed mood, measured on an 8-point scale. Heninger et al., published a table showing the ‘intraclass' correlations of rating pairs using the SCRC (table 1, Heninger et al., 1970, p.243). This table provided data collected in four different situations by different groups of raters. I have calculated the mean correlations found across the various rating settings for each symptom category and for the entire instrument. This allows for the broadest test of reliability claims. Although some higher reliability for certain categories was found in some settings none was consistently higher in all categories. The average reliability ratings across four situations for each of the 13 items was as follows: .32 for Hallucinations; .47 for Depressed Mood; 47 for Motor Retardation; .478 for Verbal Anxiety; .53 for Social Withdrawal; .546 for Paranoid Behavior; .57 for Global Illness; .62 for Motor Agitation; .63 for Physical Complaints; .655 for Expressed Anger; .686 for

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This may be the place to once more remind the reader that the symptom assessment was done by evaluators without being blind as to group membership. This suggests to me, among other things, the recommended use of metaphorical sunglasses by the raters to prevent an over-exposure to Negative Halo
Suicidal Trends; .72 for Hyperactivity/Elation; .76 for Thought disorder. The over all average for the entire instrument is .59. Succinctly stated, the reliability of this instrument in confirming the presence of symptomatology and its level of intensity was slightly better than a layman’s guess.

To further clarify the highly problematic use of this scale I want to analyze in a bit more detail the only category that appeared as statistically significant in table 4 at all treatment phases (although it was non-significant at all follow-up periods), the category of ‘Global Illness’. What does ‘global illness’ evaluate? Is it similar to GAS, a comprehensive global assessment scale for symptomatology? No. In fact it is nothing more than, “ … the rater’s assessment and is not based only on scores of the preceding items [the other 12 specific symptomatology categories]”(French & Heninger, 1970, p. 240). The category is not further described and no effort is made to specify what criteria is to be used for the evaluation of global illness by the assessors and is clearly a subjective guess of the individual assessor. The raters are asked to score the patients with 0 points=no evidence of illness, 2 points= mildly ill, 6 points markedly ill and with 8=severely ill. If this was not troubling enough, the limited reliability of this category further undermines confidence in the utility of this scale.

Table 1, on p. 243 which provides, for this and the other 12 categories, the findings of intraclass reliability in four differing settings (sometimes only in three as in this case where the usual rating by a psychiatrist was not done), shows the following; with the smallest group or rating pairs (7 nurses) and therefore the

Effects(see for example, Lance et al., 1990)
easiest to achieve better reliability (fewer people to train to see things similarly) the correlation is .72, when you raise the group membership or rating pairs however to 15 and 18 nurses you get a dramatic drop in agreement to .45 and .55 respectively (p. 243). Averaging the three different rating situations gives a clearly unacceptable .57 result (the same as getting results by chance alone). The category with the next most numerous statistically significant results during the treatment period, Anxiety or Fear, averages .478 (worse then chance alone), and so it goes.

In addition a fundamental statistical problem was not mentioned by the researchers. This is the well-known problem of statistically significant values appearing by chance when many variables or measures are included in a statistical analysis. There are 104 measurement points in Table 4. Thus depending on the P-values employed, from one to five of the significant values in the table could have occurred by chance alone.

By the use of either or both methods of analysis above, it is clear that the researcher’s claim to have demonstrated reduction of symptomatology in the experimental as compared to the control group is false.

In summary

The post treatment results can be summarized by saying that whatever effectiveness was claimed for the TCL model on the experimental group during
the treatment phase disappeared almost immediately\textsuperscript{31} after treatment. This more extended version of the first experimental program effort did not yield greater success. Alleged differences, such as higher earnings or less institutional stays, were due to rules related to hospitalization not to program interventions or their possible effectiveness. There was even a possibility (supported by data mentioned by the researchers indirectly in another context, Test & Stein, 1978, p. 62-5), that the experimental interventions reduced client self-esteem and drove clients into leaving the program and to seek institutionalization on their own.

Furthermore, the most important potential finding, of reduced symptomatology, was much like Ahab’s Great White Whale, fiction. Methodological difficulties, as explained previously falsify this claim.

The third study: *Long - Term Community Care Through an Assertive Continuous Treatment Team* (Test et al., 1991)

**Limit of Analysis**

Before I begin my analysis I must point out a troubling issue. Even though this study was publicly presented at the Annual Meeting of the American Psychiatric Association, May/1994, the lead investigator, Dr Test, refused me permission to cite the findings of the study in this dissertation, complicating the study’s critique. In responding to my written request, she wrote that she wanted to be the first to publish such results, six years have passed since the completion of the study in 1992, yet no published data have been made available for critical review. At the

\textsuperscript{31}See for example, the mysterious extinction of any and all significant differences, alleged to have been found for psychiatric symptomatology at 12 months favoring the experimental program, by the very next measurement period at 16 months, following only by 2 months the conclusion of the experimental
same time, Dr. Test and her colleagues have made at least three public presentations using the data she reported to the APA, to promote the TCL model as a very effective mode of intervention confirming its early success by the results they obtained in this long term study. She writes,

“Results supported the hypothesis that the early positive effects of comprehensive community care delivered through the PACT model are sustained when that care is ongoing and long-term”(CSNN, 1997, p. 6).

Now it may be a possibility that the absence of published data is no fault of the principal investigator, but due rather to the fact that she, having attempted publication has not been successful up to the present. This is clearly a possible alternate hypothesis as to why data have not been provided through the official channels, to my own hypothesis that these results (the ones she presented at the APA and elsewhere) have not been published because, subjected to critical analysis, they are highly problematic for TCL effectiveness claims and would interfere, if widely disseminated, with its promotion as a valuable model of mental health treatment worthy of continued government funding. The alternate hypothesis (‘Test is not deliberately withholding study findings hypothesis’) appears to conflict however with Dr. Test’s own rationale for not publishing. In a private communication to me, she said in summary, that difficult personal circumstances have prevented her from making any significant progress in publishing the long-term results although these circumstances have not prevented her from traveling as far as Australia to promote the ACT model. This claim tends to falsify the previous alternate hypothesis but certainly not

program(Stein and Test, p. 395-6, especially Table 4 (in index C, this paper)).
conclusively. In any case the chief impact on my evaluation below is that it
limits what I can say about this study to material from the APA presentation that
is not a ‘result’, and to material Test and associates have published about this
long term study elsewhere. Obviously as a consequence, my analysis is
somewhat circumscribed and will appear undernourished and perhaps strained,
but hopefully will offer some new information furthering our understanding about
PACT as well. Before this refusal by Dr. Test I did an analysis of her results as
she reported them in the APA presentation. My findings, which I hope I will be
able to present sometime in the near future unexpurgated, I believe, fatally
undermine her public claims.

The Study

This study was a long term(1978-1992) study of so called ‘Young
Schizophrenics’.

“In this study we randomly assigned 122 young(schizophrenic or
schizoaffective) adults ... to either the PACT model(n=75), or to a control
condition(the existing treatment in Dade County, during the study period, for
community mental health care, n=47). In this study, instead of being discharged,
clients remain in their respective treatment conditions for a period of at least
seven years”(Test et al, 1994, p.7).

“Dr. Test stated that results across seven years revealed PACT clients to have
sustained community tenure that was significantly more favorable than those
individuals in the control condition. Results supported the hypothesis that the
early positive effects of comprehensive community care delivered through the
PACT mode are sustained when that care is ongoing and long term, thus
facilitating consumers to remain in the community and experience a decent

This long term study was the crowning effort(and hoped to be the crowning
achievement) of over twenty years of TCL research. Based on their previous
efforts the researchers attempted to support, what I labeled earlier as, the
‘valet service’ model of community treatment, which they claimed, must be
maintained continuously with severely disturbed clients. There is no cure, only
continuous treatment. If treatment is discontinued effects disappear. This study,
due to its length, should have been the test to corroborate TCL’s effectiveness
claims or perhaps, as I conjecture, refute them once and for all if improvement
was not shown or was found not to be the direct result of the TCL program. As
with their two previous studies the researchers provided no information on the
validity and reliability of their research instruments. Also the same potential bias
issues occurred here as in the second study since the raters were not blind to
client group membership.

The researchers this time selected a ‘narrow’ category of clients,
schizophrenics, “...since these disorders characterize the largest portion of
patients who require intensive, long-term care.”(Test et al, 1985, p.18). The
empirical research however, strongly questions the existence of any discreet
concept of schizophrenia and suggests that so called ‘symptom overlap’, the high
occurrence of behaviors, which are hypothesized to uniquely distinguish
schizophrenia, but are found in other distinct mental disorders (such as
Delusional Disorder, Brief Reactive Psychosis, Manic Depressive Disorder) is
high (Bentall et al., 1988; Bentall, 1990; Miller & Flack, 1991). Bentall et al.,
conclude that the schizophrenia construct,

“... seems to have no particular symptoms, which follows no particular course,
and which responds to no(or perhaps every) particular treatment”(Bentall et al.,
1988, p 314).
The disregard of well-researched data seems to be the hallmark of the TCL group.

The authors assert that the clients in the experimental group did significantly better in sustained community tenure than the control group over the whole experimental period (CSNN, 1997 p. 6). There is an interesting definitional problem that appears over time, as to what situations or settings would be representative of valid measures of reduced sustained community tenure. In their 1991 (Test et al., p. 243) paper reporting on the first two years results, the authors relied on the findings of statistically significant less total time and lower percentage of patients who spent any time in inpatient psychiatric settings and skilled nursing homes (IP+SN) favoring the experimentals. According to the researchers, “We combined time in inpatient psychiatric and skilled nursing homes (IP+SN) in our analysis because in Wisconsin these facilities have much in common” (p. 243). Thus psychiatric and skilled nursing homes (IP+SN) served in this paper as proxy for poor settings, which if significantly more often utilized by one group over the other, indicated less sustained community tenure for that group.

The finding in this paper, of significantly more total time spent by and higher percentage of control group members in these settings any time is, contrary to the claims of the researchers, of no great moment. My prior analysis of their earlier research demonstrated that this outcome is entirely dependent on the differing administrative rules applicable to controls and experimentals, not the
particular treatment. The sooner you return individuals to the community for
treatment and refuse to readmit them to inpatient types of settings the sooner
you can use them as data for community tenure, and the more ‘sustained’ their
community tenure will be. This paper just as importantly noted,

“A community program is not successful if patients are avoiding hospitalization
but are spending much time in situations clearly indicative of poor quality of life.
Therefore, we also studied time spent in jail or other penal settings and in
homelessness or homeless shelters. Throughout the first two years the time
spent in these settings was small and did not differ significantly between the
groups.”(p. 244).

This clearly establishes that at least for the first two years of the study, only
tautologically determined procedures and not the experimental model (just
exactly as in the earlier studies), provided justification for claims of effectiveness.
There was no differential impact of the experimental treatment on either time
spent in penal institutions or in homelessness.

However in the later unpublished papers (all publicly presented and handed
out) a new category called ‘all poor’ settings is introduced. It consists of, “… the
mean number of days spent each year in in-patient psychiatric settings plus
skilled nursing homes plus penal/jail settings plus homelessness or homeless
shelters.”(Test et al., 1994, p. 10). These are, “… usually indicative of either poor
functioning or poor quality of life”(Test et al., 1994, p. 9). Although I am prohibited
from directly citing the researchers’ ‘findings’ in their unpublished papers, based
on this newly introduced combined variable (it appears in the 1994 paper for the
first time), their outcome claim as reported and allowed by Test for quotation in
Community Support Network News, states that,
“… results across seven years revealed PACT clients to have sustained community tenure that was significantly more favorable than those [sustained by] individuals in the control condition.”(p. 6).

This claim and their introduction of this new combined variable is puzzling since it is clear from what I indicated earlier, that in the first two years of the study the treatment had no statistically significant effect on homelessness and penal stays. This makes the claim of experimental effect over the entire seven-year period false if it is to apply to homelessness and penal stays as well as nursing homes and inpatient psychiatric settings. One explanation for the use of this expanded version of ‘all poor’ settings (which I believe is supported by the data, although not available for citation), is that since reduced hospitalization (whether in psychiatric hospitals or nursing homes) is a rule based, not treatment based outcome as recognized by the researchers themselves twenty years ago, in-order to legitimate the actual effectiveness of treatment, they had to try to find non-tautological outcomes,

“Community treatment results in less time spent in the hospital. This finding is certainly not surprising since experimental patients were usually not admitted to hospitals initially and there were subsequent concentrated efforts to keep them out”(Test and Stein, 1978, p. 353-354).

Since homelessness and penal stays are not directly effected by TCL administrative rules not to hospitalize, and do represent the types of settings we would want fewer people to be in, reduced patient participation in those settings as a result of TCL would validate the utility of TCL. Unfortunately for the researchers, at least for the first two years of data (this is all that is available for analysis), by combining these various settings into ‘all poor’ settings they merely
succeeded in demonstrating that the significance found for the tautologically
driven components of hospitalization and skilled nursing homes, could
statistically carry(outweigh) those other non-significant components. Penal stays
and homelessness, when measured on their own were found to have no
statistically significant differential impact on the control and experimental groups.
It is difficult to see why this combined variable was created, other then to suggest
that there was a program effect, in reducing homelessness and jail time, when
none existed. Since I am prohibited from using actual data from the unpublished
results I can only suggest that this hypothesis is not falsified by the results of the
five other years of data. This analysis confirms that this study’s very long-term
results are as of little value as either the very first study’s brief intervention, or the
second study’s longer intervention. Hospital stays can be reduced by fiat (no
program needed) but the program is no more likely to reduce homelessness or
jail time in seven years as it is in two years, or fourteen or five months. Perhaps
the researchers will now claim that seven years, for some early entry patients it’s
14 years, is not long enough for such ill patients. The final intriguing piece, in
what seems to me a puzzling history, is that this program was extended two
years beyond its original time line of 1978 to 1990, to 1992. As late as 1992 (after
the presumed end of the research) Test stated that, “... the duration of the study,
... ran from 1978 to 1990”(Test, 1992, p. 160). In a personal communication(letter
dated July 8, 1994) to me, Test wrote that the study was extended through 1992.
What could have happened between the writing of the article just cited, where
Test states that the study ended in 1990 and the letter she wrote to me, that
would have resulted in a two year extension?

Without a direct report we can only conjecture. Perhaps, it became clear to the researchers somewhere toward the original planned end of the study, that results would not corroborate previous TCL claims and the entire TCL approach was going to be revealed as ineffective bringing to a disappointing end twenty year of hope that this model had a unique claim to effectively treat the severely mentally ill. This, understandably hard to swallow, potential threat might have led to a decision by the researchers to extend the project, with the aim of ‘finding’ some reportable positive results. That this conjecture is not without evidence must await publication of the yet unquotable data.

What is absent from these reports on the long-term study is probably more telling then what is included. The affect of TCL on psychiatric symptomatology is not discussed at all. The published results of the long term study only discusses sustained community tenure and make no mention of symptomatology change. Does this mean that TCL over the time of the present study was unable to demonstrate any reduction in symptoms among the experimental group as compared to controls? Was TCL treatment effective, ineffective, or harmful in dealing with psychiatric symptomatology? Based on the previous history of TCL research reports one might surmise that this outcome data was left out because it did not support TCL effectiveness. Improvement in the symptomatology of clients is a claimed benchmark of effective TCL. These researchers, ever since their second study (twenty years ago), have searched for and declared how
important a finding of such improvement would be. Hence their silence on the subject of psychiatric symptomatology is deafening.

In their initial report of the long term study (Test et al., 1985) after discussing the importance of focusing on symptoms and describing how in this study special interventions will be used to manage symptoms (p. 21), the authors state,

“\text{We are well aware of the problems when hospitalization is used as a measure of outcome and we look forward to the examination of our short-term (two-year, Test’s parenthetic comment) findings on other measures of psychosocial functioning.”}(p. 25).

Although they further reassure us in their 1991 article that, “Analyses of data related to additional outcome variables, including symptomatology, ..., during the first two years are currently in progress and will be reported in future papers.”(Test et al., 1991, p. 244). They have never published (including the 1997, CSNN report) any such short or long term data analysis. We can only conclude that it would not have supported TCL claims. They have published so much and so often, certainly data showing TCL program effectiveness on symptomatology would be the last thing to be neglected. What we do know from what is available right now is that this study does not provide any new data that shows TCL to have any effect beyond what the earlier more time limited studies described. These showed only a reduction in hospital stays in TCL programs which, were not however due to TCL treatment. What is important about this study when compared to these researchers’ previous research efforts is its apparent cynicism. Their attempts in this long term study to find non-tautological results to validate the research model by the creation of the misleading combined
variable of ‘all poor’ settings, or by extending the study for two years, reflect a
determination regardless of contradictory empirical evidence to manufacture
positive outcomes where none can be found.

Conclusion

I believe my analysis undermines all the claims for the treatment effectiveness of
the TCL model on ‘the seriously and persistently mentally ill’ as argued for by its
inventors in these three papers describing their research, and thereby seriously
also damages the outcome claims of PACT models in general. By reviewing the
PACT replication efforts in the next chapter I put this hypothesis to the test.
Chapter 6

The replications: Programs of Assertive Community Treatment

INTRODUCTION

One possible indication of the success of an idea, theory or approach in any domain may be measured by the number of its convinced believers, or users if you will. If this criterion of success is applied to Programs of Assertive Community Treatment (PACT or ACT) this approach ought to be considered a resounding success. The current number of replications of the TCL program originated 25 years ago (Marks ET al, 1973; Stein & Test, 1980), is estimated to be around 397 in the U.S. (CSNN, 1997). The rapid rate of implementation of these programs is indicated by their growth from 223 in 1992 to 397 programs by 1996 (ibid. p. 3). Total expenditures, almost all from tax dollars, currently (1997) exceed 157 million dollars per year. These numbers are based on information from only 14 states and are a conservative estimate of all existing PACT's in the United States (ibid.).

The enthusiastic acceptance by the mental health field of this approach may indicate as Laurie Flynn, Executive director of the National Alliance for the Mentally Ill claims that,

“Because the PACT model has had the benefit of being continuously refined in research based settings, we are able to bring forward compelling data regarding the superior effectiveness of its particular set of interventions. The effectiveness of the model has been proven, not only in terms of clinical care, but also in terms of the quality of life and satisfaction of clients.”(CSNN, 1997, P. 19, italics added).
This explosive growth has been accompanied by numerous studies evaluating and testing the various versions of Test and Stein's original invention. The question this chapter will address is whether there is ‘compelling data regarding the superior effectiveness’ of this model in these studies as asserted above.

There have been a number of reviews from time to time of this research (Olfson, 1990; Solomon, 1992; Burns & Santos, 1995; Scott & Dixon, 1995 Draine, 1997 for example). Their conclusions when compared to Ms. Flynn's unrestrained zeal are sobering if not unexpected considering my findings on the earlier research by Test and Stein. Olfson in his review, the first focusing on PACT, cautiously states,

“The most consistent finding- that assertive community treatment reduces hospital utilization-has been replicated in several evaluations ... The superior efficacy of assertive community treatment in controlling clinical symptoms, improving social functioning, and promoting occupational functioning has been restricted to the original research in Madison and the TCL replication in Australia. Other researchers have tended to find assertive community treatment to be no more or less effective than standard care in these critical outcome areas.”(Olfson, 1990, p. 640).

Solomon (1992) in a somewhat more expanded and inclusive review of various case management approaches, also finds that,

“... the evidence seems to indicate that case management is most effective in reducing the number of hospitalizations .... Other areas.... produced limited positive outcomes.”(p.176).

Correspondingly Burns and Santos (1995) report that,

"Recent studies of assertive community treatment continue to show a strong experimental effect of the intervention on psychiatric hospitalization. ... The
absence of an experimental effect on functional outcomes in most studies is also consistent with the findings of previous research” (p. 673).

As I demonstrated in ch. 5, reduced hospitalization cannot be considered an ‘experimental effect’ of ACT. Reduced hospital stays can be implemented by any type of community program by simply imposing a rule to not hospitalize and return all SMI’s regardless of the severity of symptomatology to the community for services. This obvious maneuver has been noticed by several reviewers (Olfson, 1990a; Solomon, 1992), although they semantically obscure the simple mechanism by which reduced hospitalization is obtained, by describing it as an independent or process variable,

“The clinical significance of the observed decrease in inpatient service utilization is difficult to assess. Restricting the clinical criteria for hospitalization is an explicit tenet of assertive community treatment. Under such conditions, reducing hospital utilization becomes more of an independent or process variable then an outcome variable” (Olfson, 1990a, p. c-75).

Contrary to Olfson’s claim, the clinical significance of this ‘observed decrease in inpatient utilization’ is not difficult to assess, there is none. The decrease is not intervention dependent; it is an administrative action. By calling the prohibition of inpatient treatment of the experimental group (in some instances just its severe limitation) an independent variable the reviewers appear to suggest that its result, less hospitalization, is related to some ‘deeper’ scientific activity then the arbitrary rule that actually produces it.

Despite these reviewers finding only reduced hospitalization and patient satisfaction as consistent measures (not really outcomes of treatment as argued above), the latest review of the PACT research, as part of Psychiatric Services’
comprehensive promotion of PACT in its July, 1995 volume, surprisingly concludes that,

“Research on assertive community treatment has advanced since the reviews of Olfson and Test largely by providing further evidence of the positive effects of assertive community treatment in reducing hospital use and increasing patient and family satisfaction; by testing assertive community treatment in a wider range of populations, including patients with recent-onset schizophrenia, veterans, dually diagnosed clients and homeless persons and by studying the integration of other empirically validated methods ... into assertive community treatment.”(Burns and Santos 1995, p. 674).

This claim of advances in ACT research based on its use on ‘a wider range of populations’ is highly problematic. A moment’s reflection should make it clear that these populations are not distinct from one another, but very much alike. They are a part of that vast subsuming category institutional psychiatry has labeled the severely mentally ill (SMI’s). Members of this category, regardless of diagnosis, all share the functional characteristics(chapter 3) as well as the treatments (ACT, medication, social welfare benefits, imprisonment) of social dependents (Test & Stein, 1978, p.351; Szasz 1994).Their alleged population differentiation is primarily dependent on the particular perspectives from which various researchers choose to look at these individuals and their behavior, not on discrete distinctions in personal or social characteristics.

Being a veteran for example does not identify a unique population, since many different sorts of persons may and do become soldiers. ‘Population’ is a word whose most concrete definition according to The Random House Dictionary is, “ ... the number or body of inhabitants of a particular race or class in a place: the native population”(p.1119)
The term veteran, is merely an administrative convenience helpful for broadly categorizing a highly heterogeneous group for whom the federal government has a responsibility and as a consequence a need to track. By describing veterans as a population, the researchers attached to the Veterans Administration’s mental health bureaucracy are using it in very loose lay terms, meaning some collective or other, but are hoping that it will be (mis)understood by those in the research community as if it was being used in its more formal sense defined above. This approach provides justification for their psychiatric research (research being a necessary activity for professional validation) of the only group of individuals over whom they have hegemony, by looking at them as if they were a homogeneous group deserving of special study.

Similarly, depending on the claimed expertise of particular lead researchers, other individuals labeled as SMI’s are variously identified as research populations of ‘homeless’ (i.e. ‘homeless’ veterans, ‘homeless’ dual diagnosed veterans, ‘homeless’ dually diagnosed, ‘homeless’ schizophrenics) or ‘schizophrenic’ (i.e. ‘schizophrenic’ homeless drug abusers, ‘schizophrenic’ drug abusing veterans), or ‘dually diagnosed’ (i.e. ‘dually diagnosed’ schizophrenics, ‘dually diagnosed’ veterans, ‘dually diagnosed’ homeless veterans) etc. The point is, that these people do not necessarily differ in any meaningful way, and research on these varying groups is not research of wider populations receiving PACT (they are all SMI’s), but, serve rather as convenient statistical data, in the form of highly flexible and interchangeable research categories, for experts who need to justify
their particular research programs as advancing the knowledge base of the field.

When you examine the social and demographic characteristics of these distinct ‘populations’ as described in the various studies there are few if any functional differences among them regardless of psychiatric diagnosis or population category. They share such characteristics of social dependents as being mostly male, relatively young, unemployed, on government benefits, single, very poor, generally without a high school diploma, often homeless, housed often with parents, frequently (involuntarily) hospitalized, highly involved with the criminal justice system, frequent users of various legal and illegal drugs, mostly white, over-representedly African American, and poor managers of money (Bond et al, 1991; Curtis et al, 1992; Hornstra et al, 1993, Lehman, 1993; Rosenheck et al, 1995; Solomon & Draine, 1995; McFarlane et al, 1996; Meisler et al, 1997; Morse et al, 1997).

Based on these facts, the mere duplication of the findings of prior research on the outcomes of (re)hospitalization and service satisfaction by this more recent research cannot be called an advance in the field. Further, knowing reduced or less frequent hospitalization is not dependent on a particular treatment but on an arbitrary rule, and knowing, that the report of superior service satisfaction favoring community treatment cannot be based on the patient’s clinical or functional improvement as a result of the specified treatment because, the research indicates no correlation between them; the finding of increased patient satisfaction may better be explained by viewing hospitalization for what it
functionally is, incarceration, and any treatment in the community like what it most closely resembles, probation. From this perspective it immediately becomes clear why most patients favor community treatment (probation) in any form regardless of its efficacy, over hospitalization (imprisonment) periodic or long term. It is not because of alleged treatment differences favoring PACT but for the obvious reason that most people prefer freedom, even if somewhat circumscribed, to involuntary detention. Be that as it may (and of course the truth of these claims by me will depend on the analysis to follow), the most telling difference between my approach and the previous reviews is that I use fallibilist criteria for evaluating the research.

These earlier reviews accept the claims of statistical significance made in the various studies at face value, a serious problem if these claims are false. They rarely evaluate the methodology employed (an exception is Draine, 1997) and often explain away the findings of non-significant results (the most frequent finding among most of the studies on the variables of symptomatology and functioning), as if the reviewers wanted to find value (justification) for PACT methodology even where the research suggests none.

“Difficulty achieving an experimental effect [clinical or functional] may be due to the limited gains that can be achieved by patients with severe mental illness particularly within the short study periods, which typically have ranged from three months to two years.” (Burns & Santos, 1995, p. 673).

or

“Case management is a system management service more so than a clinical service . . . . Therefore, its is not surprising that case management appears to affect system outcomes, e.g. rehospitalization, more then clinical outcomes.” (Solomon, 1992, p. 177).
The first of the above claims is unsustainable by the analysis of the evidence of clinical outcomes in the long term studies (see analysis to follow). The second contradicts the fundamental philosophy of ACT providing all necessary biopsychosocial services (these typically are clinical interventions) needed by the client through “a core services team” (Test et al, 1997, p169-72). Both are representative of the justificationary rhetoric common in psychiatric research employed by all of the reviewers I examined, seeking always to find support or proof for, rather than test by attempted falsifications, professionally approved approaches. Viewed from the perspective of rational self-interest this is not surprising. All of these reviewers are also committed researchers in this area and applying fallibilist criteria would force them to reject all research, even if tentatively, which shows non-significant results and would prohibit the use of ad hoc explanations as excuses for failure (Popper, 1979). Such falsifications could undermine the usefulness of future ACT research and short circuit careers based on this approach, some of which are of 25 yrs. duration, including their own.

My own emphasis in the present review on the patient outcomes of symptomatology and social functioning, rather than on what are often labeled system or process outcomes (although program cost will be evaluated) is for the following two reasons. First, there is I believe little empirical controversy presently over the question of the feasibility of systemic implementation of community treatment approaches. In fact, as early as 1954 Bockoven and Solomon published data on patients receiving such treatment in which,
“Boston Psychopathic had demonstrated in the 1940's and 1950's: [that] patients with major mental illness can be given better care outside of the confinement of a hospital” (Bockoven and Solomon 1975, p. 798).

In their 1975 paper (before Test and Stein have published), they document the use of early discharge as a therapeutic intervention criterion much like the TCL model, and as a way of reducing hospitalization,

“When the Solomon Center patients were admitted in the late 1960's the clinical administrative practices of mental hospitals had changed. ... Indeed, discharge itself became part of the patient's treatment program. This hospital showed a steady yearly reduction in census starting in 1951.” (p. 799).  

Second, systems or process research has been the central focus and primary agenda of the field for at least the last two decades, currently focused on managed care, with little to show for this effort on client outcomes. Perhaps this is so because what amounts to program success in these two outcome categories may be incompatible with one-another. That is, the ability to successfully implement a program (the methodology required to evaluate gate keeping functions; manage care; control, pacify, socialize, educate or otherwise conventionalize often troublesome people evaluated by the so-called process or system outcomes), may be in conflict with the methods needed for improving individual functioning or the autonomy of clients from the clients perspective.

What counts as socially acceptable program implementation is always based on normative judgments rooted in the ethics and beliefs of the authorities in charge, while the effort to improve personal functioning and autonomy in-terms of the client may often require acceptance of unconventional and possibly anti-social behavior. One of the more serious difficulties with mental health research
into outcome effectiveness is the determination for whom these interventions should work; the individual patient or society at large. Psychiatric authorities contend that these programs are unproblematically scientific and are applicable to both the identified patients and others, for instance, their families and society. I believe this to be untenable. We need to evaluate these components separately and decide which groups’ agenda to affirm programmatically. Individual behavior and functioning, the imparting of freedom and autonomy from the perspective of the patient, and how that may be improved or aided, will inevitably come into conflict with the moral principles and values of the psychiatric experts in charge. This is so because their agenda as the representatives of the system, the interest of which is social order, must include the imposition of heteronomy and conformity, in line with current societal norms, on all individuals. This dilemma of the acknowledged imbalance of power favoring society and the state was very clear to our founding fathers, who provided the strong safeguards for individual rights found in our laws and constitution as protection against the potential abuse of this power.

The social workers, clinical psychologists, psychiatrists, the public welfare system and its employees, who must implement these programs cannot be considered to be equally the agents of clients, families and society. The various agendas among these groups rarely coincide. In fact these professionals’ actions are proscribed by their particular organization’s rules and regulations as to what extent they can truly represent the perspective of clients (as for example criminal defense attorneys do). This is best illustrated by recalling the well-worn truism,
‘he who pays the piper calls the tune’. The salaries of these various experts are paid from the public treasury or by private corporate entities such as third party health care providers (HMOs) or various non-profit agencies, not from the pockets of clients. So, the commitment of these bureaucratic employees to clients is limited by an important economic constraint, the threat of job loss if agency rules are violated, and ends when the clients needs are in opposition to an agency’s regulatory mandate.

Hence, what follows is a critical review of the research claiming to show statistically significant improved functioning and symptomatology among the ‘severely mentally ill’ clients treated by PACT. Ultimately such improvement is the principal justification for using ACT argued for by its proponents, as Don Gilbert Commissioner of the Texas Department of Mental Health and Mental Retardation makes clear,

“We first piloted Assertive Community Treatment a few years ago in Alvin, Texas. Through this pilot, we realized that Assertive Community Treatment works. As we focused our attention on Assertive Community Treatment, we kept in the forefront, that when it is all said and done it’s really about people. It is about the people we serve and their many stages of recovery from mental illness”(NetworkNews, 1997, p.1 & 16).

Studies

I chose to review all the studies found in the University of California’s Melvyl data base which self identified as studies about ACT type programs and excluded those that did not. What qualifies as an ACT program at a minimum, is not clear in the literature, and of course is a serious problem when comparing various studies. I will however, not be examining that important issue but instead,
will stick to reviewing the claims of treatment effectiveness of all those programs that see themselves as using assertive community treatment no matter how broadly defined (On program fidelity see McGrew et al, 1994; McGrew et al., 1996).

I believe this is an appropriate approach because the studies that claim to be testing this model evaluate implementations ranging from low to high levels of adherence to the original model. According to the CSNN (1997) survey most of the current programs in the United States “moderately adhere to the PACT model” (p. 4) with only 2 states reporting high adherence and 1 reporting low adherence out of the 14 states reporting data. Since most of these studies are of moderate replications and as a consequence not identical to the original model (TCL), which in any case I believe has not demonstrated any statistically significant advantage in improving client symptomatology or functioning, it would be of some interest to see if any of these hybrid versions have any superior client outcomes. If they do, and their particular methods are detailed in the studies, then that information might offer us some clues as to what interventions may be useful. If they don’t, and perhaps even are found to contain harmful elements, then the whole broad category of PACT might, at least tentatively, be considered to be, in my terms, falsified. This result would argue for the setting aside of this approach as a valid method of treatment, and the redirecting of research toward identifying some better tested yet unfalsified alternatives for dealing with these problems.

Table 1. lists 27 studies of which 17 discuss randomized control trials (RCT).
All the RCT’s which were available for my review (either because they are published or if unpublished available from authors for my review) are included. There is only one RCT on PACT to my knowledge in the literature (as of December, 1997) which I could not access, (Mulder, 1985). It is an unpublished manuscript, which I was not able to review, although I attempted by phone and mail to reach the author. It has been commented on in the existing literature reviews I cited. I don’t list this particular study in the table because I could not review the actual research.

**Non-randomized pre-post studies**

The 10 studies which are not RCT’s are non randomized pre-post studies, 5 with and 5 without controls (see table 1). The limitations of such studies are well known and consequently I will not analyze them in this review, although I provide the findings in table 1. Never-the-less a brief review of their limitations may be of value. Non-randomized pre-post studies without controls allow no valid conclusions to be drawn about either the representativeness of the sample (Is it identical with the population one wishes to test?) or the effectiveness of experimental intervention. One may comment about population change if found, but not as to its cause (Campbell & Stanley, 1963; Cook & Campbell, 1979).

Non-randomized studies using controls are open to the following sources of invalidity; the experimental group may not be representative of the study population, the controls and experimental may not be similar, there may be differential selection-maturation interaction, difficulties with instrumentation, possible differential statistical regression or an interaction of selection and local
history. These potential alternate causal explanations make it virtually impossible to attribute change to experimental intervention in these models (Cook & Campbell, 1979, p. 103-07).

Both these non-randomized research approaches are of such little value in causal research (Cook & Campbell label them “generally uninterpretable” (p. 96)) that the question arises, why do them? Often, it is asserted that these designs are helpful for “suggesting new ideas” (Cook & Campbell, 1979, p. 95) which if found to be of sufficient interest could then be tested by RCT’s. Or, that even though treatment circumstances prevent the employment of RCT’s, using some form of ‘quasi’ experimental research is better than no research at all. The word quasi is illuminatingly defined by the Oxford American Dictionary as, “seeming to be something but not really so, as in a quasiscientific explanation” 1980, p. 548, Avon Books, N.Y.), nicely pointing out the logical incoherence inherent in such approaches. Finally, it is argued that in those circumstances where the effectiveness of the intervention has already been proven by previous RCT’s these non-random studies provide further proofs of treatment value.

“... despite their shortcomings, such studies flesh out the findings of earlier [experimental] controlled trials which used dedicated research teams and special funding.” (Hambridge & Rosen, 1994, p 443).

The proper response to such tortured reasoning is, that if, the methodology of a study makes it implausible to assert anything about the causal effectiveness of the experimental treatment then these studies cannot be used to explore preliminary hypothesis in any reliable and valid fashion about causality. In addition, since formal inductive reasoning is an impossibility, as explained in the
introduction, the accumulation of discrete research to support previous
research findings is of no value. The accumulation of positive findings by
induction is not accumulation of scientific knowledge; the theory ‘supported’
thereby may still be falsified by the very next hard test. Science can only
eliminate false knowledge not provide support for true knowledge(Popper, 1979).
These models are technically incapable of connecting an experimental
intervention and any change found causally, period.

In fact there are no empirical differences among imagining, intuiting,
daydreaming, guessing, or non-randomized quasi-experimental research as
possible techniques for arriving at useful new ideas to test. They all may offer
new notions worthy of further evaluation. The one noticeable difference between
non-randomized quasi-experimental research and the others, is that imagination,
intuition, dreaming or guessing are substantially less expensive activities but can
prove to be just as, perhaps even more suggestive i.e. Einstein’s mind
experiments. What is of primary importance is not how we get an interesting new
idea, but how we test that idea. It seems to me that our research emphasis ought
to be focused on taking an interesting idea discovered by any means and seeing
if a critical test can be arranged for it, perhaps a small scale randomized control
trial. It would be of greater scientific value, as well as more cost effective, to
consider only those ideas that are critically testable by potential falsification. By
choosing this approach we would avoid spending public money on ‘research’
which cannot deliver any more information, in fact a great deal less judging from
Einstein’s impact on science, then his mind experiments, which cost nothing.
All this said, such designs continue to be utilized and evaluated in the literature because of the public demand for ‘scientific’ answers to serious social problems (whether such answers are possible or are forthcoming is another issue altogether), and scholarly egos which must respond to such demand and publish, being what they are (often suggesting the possibility of solutions were none may be had due to our cognitive closure\textsuperscript{32}, McGinn, 1993, pp. 5-7), they will continue to be published despite their invalidity in causal research.

Randomized controlled trials

Turning to the randomized control trials (RCT’s), 17 were reviewed. Only two had true no treatment control groups (#’s 22 & 24 in table 1). The others employed various versions of routine treatment groups as the controls. Without a ‘no treatment’ control group the only thing that can be asserted about significant outcomes favoring the experimental treatment, is that such a treatment leads to a statistically significant better outcome than the alternate intervention, but we cannot say if the experimental treatment is better than no treatment at all. This is important because if a ‘no treatment’ or a referral or an-information-only control alternative can be shown to be as effective as the experimental intervention then significant savings may be realized by using minimal intervention with no apparent detriment to the patient. Only one study used blind and independent evaluators (#15). This study compared two different versions of assertive

\textsuperscript{32}The natural world can transcend our knowledge of it precisely because our knowledge is a natural fact about us, in relation to that world. It is a general property of evolved organisms, such as ourselves, to exhibit areas of cognitive weakness or incapacity, resulting from our biological constitution; so it is entirely reasonable to expect naturally based limits to human understanding. We are not gods, cognitively speaking. A creature’s mental powers are things in the natural world, with a natural origin, function, and structure, and
community treatment and cannot be used to evaluate such treatment for effectiveness since it did not involve either a routine (the treatment regularly provided to SMI’s in a particular community) or no treatment control group alternative for comparison. Some of the studies used independent raters (#’s 1, 5, 10, 11, 12, 14, 20, 24) some used program staff (#’s 2, 6, 19). The others did not record how they collected the data. Only eight studies (#’s 1, 6, 10, 11, 12, 14, 23, 25) showed some significant findings other than reduced hospitalization or service satisfaction both of which, if unaccompanied by other clinical or functional improvement, are not true treatment outcomes.

I will focus my attention on these eight reports since I believe they provide the most important data for evaluating whether this type of program is effective in terms of client outcomes. Most particularly I will concentrate on three efforts, the first two of which claim to be close replications of the original program, one done in Australia (#’s 10 & 11), the second done in England (#’s 1, 14, 21), and the third (#23) in the U.S. The U.S. study is very important because it asserts the gold standard of ACT, that is, long term clinical effectiveness, and it is the largest RCT study, with the most people, over multiple sites, ever done on this type of treatment.

These three studies are among only five RCT studies in the literature to claim clinical improvement for ACT interventions and two of these studies, which appear in Burns & Santos’ review, I believe are not valid. One article cited, McFarlane et al., 1992 is not about a completed study but is a chapter in a book

there is no necessity that this part of the world should be capable of taking in the rest.” (McGinn, 1993, p. 5).
containing a summary of outcome claims without statistical analysis, “We have not included statistical analyses because this study is still underway.” (p. 45)

Clearly a study still underway with unanalyzed data should not be used for making outcome claims, although Burns & Santos appear not to see the problem and list these incomplete and tentative data in their table 1 (p. 670). The second study, Merson et al (1992), which Burns & Santos also identify as an appropriate study (see table 1 p. 670), calling the intervention ‘ACT-like’ (Burns & Santos, 1995, p. 671), is also not acceptable. Merson et al clearly state that their treatment program differs from PACT type programs (they don’t self identify as an ACT program, my criteria for inclusion in the analysis), because,

“... the community services in these studies [done by Stein & Test, 1980; Hoult, 1986; Mujien, 1992; Burns, 1990] were experimental, and generalizations from them may not be valid because some of the gains may have stemmed from novelty and from the enthusiastic commitment of research orientated teams.” (p. 1311).

They further differentiate themselves when they declare that their service “... is not a crisis intervention service and does not have 24-hour cover” (p. 1311), both of which are critical elements of PACT interventions according to Test (1992) and McGrew et al., (1994).

Analysis of the Randomized Controlled Trials:

**Bush et al., 1990 #6** - This was a program claiming to use TCL methodology although they call their effort ‘intensive outreach’ (p. 648). How similar this intervention was to the original and how one would determine that is not stated and is unclear. The paper also doesn’t disclose who collected the data. The only significant finding besides reduced hospitalization is the finding that, “the
experimental clients more frequently achieved appropriate living arrangements and relationships as defined by the case managers” (p.648). The obvious problem with this finding is that it appears to be dependent on a subjective evaluation of client behavior by the case managers based on their own individual definitions for each of the variables. This outcome measure is nothing but an opinion apparently lacking even intersubjectively agreed upon criteria for the variables of interest, since each case manager apparently defines appropriate living arrangements and relationships individually. It is also unclear from the text whether one group of case managers or two separate teams of case managers provided the control and experimental interventions. Obviously if it is the same set of case managers providing both interventions serious bias issues arise not unlike the issues I discussed about the Test & Stein research in chapter 5. Finally, with the small number of individuals in each group (n=14) statistical power is severely curtailed undermining the scientific authority of any significant findings (Cook & Campbell, 1979, p. 354) even if the other issues mentioned were not of serious consequence.

**Solomon & Draine, 1995 #25** – This study arose as part of a larger RCT trial of 200 homeless SMI's leaving an urban jail system (#24, p. 260 in table 1). It aimed at testing the effectiveness of ACT by comparing the treatment provided by an ACT team, to that of an individual case manager, using a no intervention referral of clients to a community mental health center as control. The results of the larger trial showed no significantly different outcomes in any domain among the three treatment groups even though the researchers were looking to find support
for their hypothesis that ACT would be significantly superior in "a variety of psychosocial and clinical outcomes" (Solomon & Draine, 1995a, p. 256). This result argues strongly for the use of no treatment control groups in every RCT that tests PACT in order to determine whether any specific treatment is more effective than any minimum or no treatment alternative.

This study was significantly biased by the high attrition rate of the study participants, 53% by 12 months, making any outcome findings unlikely to be representative of the original set of homeless persons leaving jail (Cook & Campbell, 1979, p.53) as Solomon & Draine acknowledge (Solomon & Draine, 1995a p.261). The actual group we have for analysis in this their second study is one which contains, “ ... subjects entering the service phase [who] were not representative of the original jail sample, but were comparable across conditions.”(p. 261).

During the large scale study (#24) the researchers noticed a very high recidivism (56%) among those individuals in the ACT group when compared to the other two groups, 22% among individually case managed individuals and 36% among those receiving a referral(Solomon & Draine, 1995b p. 168). They decided to explore further what might explain this highly unexpected phenomenon, in the present study (#25). They compared 22 clients in the ACT to 29 clients in individual case management. All clients were included who were being served for at least 3 months by spring 1992 (p. 169). The only significant findings of this study were; a.) that clients of case managers who sought stipulations (agreements that clients would be returned to jail if they didn't follow
through on recommended treatments or a particular housing situation) were more likely to return to jail (p. 170), b.) case managers were more likely to initiate a violation of probation process as an intervention strategy with clients for whom they sought stipulations and c.) clients for whom stipulations were actively sought returned to jail faster (p.170). These activities all occurred in the ACT intervention and as Solomon & Draine state,

“These findings raise provocative questions regarding the possibility of deleterious consequences of intensive case management services for seriously mentally ill people, homeless people and others who are vulnerable to jail detention.”(p.171).

This conclusion by the researchers is noteworthy because it is the first public (published) acknowledgment that ACT interventions are likely to be harmful, countering previous statements that ACT is useful even in those situations where it is no more effective then an alternate, because it does no harm. Solomon, one of the principal investigators of the present study, used just this explanation in the past to argue for the continued use Of ACT (Solomon, 1990, p.176).

Solomon & Draine as result of this new disturbing finding identify coercion as the most important ethical problem of ACT treatment and question the prior unquestioned assumption of its moral and scientific validity as a mental health intervention,

“... coercive case management may defeat the goal of increased independence and is antithetical to the general principle of client self-determination”(Solomon & Draine, 1995b, p. 171).

This finding to say the least does not help the ACT cause.
Australian replication

The two articles (Hoult et al., 1983 & Hoult, 1986) listed as #10 report on one research project claiming to closely replicate the original TCL model and,

“... to be significantly more satisfactory and helpful [to] patients ... achieved a clinically superior outcome, and cost less than standard care and after-care.”(Hoult et al., 1983, p.160).

This study utilized two treatment groups of 60 patients each. One ACT, the other routine brief hospitalization with after-care. The clinical claim is based on the findings resulting from the use of three clinical instruments, the Present State Examination (PSE), a structured mental status interview schedule; the Brief Psychiatric Rating Scale (BPRS), which provides global ratings of psychiatric symptomatology and the Health Sickness Rating Scale (HSRC) which estimates patient total functioning (Hoult et al., 1983, p. 162).

The BPRS showed no significant differences in the degree of severity of symptoms between groups before and after treatment. According to the HSRC there was a 4-point difference (p<.05), 50 for experimental group, 46 for control group, out of a possible score of 100 favoring the ACT group. Both groups at baseline had scores of 26, demonstrating a very large improvement among both groups. The PSE showed statistical significance (p<.001) favoring the experimental over the control group in having fewer symptoms on the total score, as well as having fewer symptoms in the four sub-scale scores. Unfortunately there was also a significant difference (p<.01) favoring the experimental group at baseline in 2 of the four sub-scales.

The authors list this finding but do not discuss the serious problems, which
result from it. If 2 of 4 sub-scales of a mental status exam are significantly
different between groups at baseline then the potential failure of the initial group
randomization process has to be addressed. It is quite likely that these groups
were unequal at the start of the project. Table 6 (p.164) which lists the PSE
scores, clearly shows lower baseline scores favoring the experimental group on
all five scales of the PSE; 30.9 vs. 30.5(mean total PSE score), 8.4 vs. 8.2(mean
DAH sub-scale), 7.3 vs. 7.0(mean BSO sub-scale), 5.0 vs. 4.5(mean SNR) and
11.6 vs. 10.5(mean NSN) with the last two scores statistically significant. As can
be seen by the numbers, what is considered significant, a .5 difference in the
SNR sub-scale and what is not, a .3 difference in the BSO sub-scale differs very
little, suggesting that the other two baseline non-significant sub-scales were
marginal in their non-significant status favoring the experimental group, further
supporting the finding that the groups were not equivalent from the beginning.
Some statistical testing should have been done to evaluate this problem and to
control for it. Since this was not done, in fact no mention is made of this difficulty
in the paper, the results of the PSE cannot be considered valid simply on its face.
This leaves only one (the HSRC) of the three clinical instruments as showing a
statistically significant (p<.05), although practically, a non-significant difference in
functional improvement. The authors seem to admit this when they state,

"On the Health Sickness Rating Scale, scores for both groups changed from a
level at baseline that was indicative of clear-cut and overt psychosis (mean
scores of 26 for both groups), to a level at 12 months, that was considerably
higher (mean score of 50 for project patients and 46 for control patients), but still
indicative of unsatisfactory or unstable adjustment and far from an ideal state of
functioning (i.e. a score of 100)."(p. 164)."
These results, one instrument (BPRS) showing no significant differences between groups; one instrument (PSE) showing significant difference favoring the experimental group at follow-up but also showing significant differences at baseline favoring this group (strongly suggesting difficulties with the original randomization of the sample, undermining the equality of the control and experimental groups and the validity of significant differences found at follow-up); and one instrument (HSRC) showing an unproblematic statistical significance favoring the experimental group (although even this finding according to the authors is of limited practical significance for treatment), are hardly a clear cut finding of clinical efficacy of ACT treatment. One might argue that they speak equally well of findings of no significant differences between treatments.

The authors also state that the cost of the experimental intervention was less than the control treatment. This appears to be so, if the numbers they provide are accurate (control-$5669, experimental-$4489), by $1170 per patient, but this is not a statistically significant finding and is not claimed to be one. Furthermore, this difference in cost is due to the much higher hospitalization rates (hospitalization costs more then community treatment) imposed on the controls then on the experimentals (p. 165). As I argued before, the reduced hospitalization, and, as a consequence the reduced expense of ACT, is due to the rule of not permitting experimentals, even very disturbed ones, to be hospitalized and not to the treatment.

Finally, the claim of client satisfaction favoring the specific methods of assertive community treatment made by the authors of this study is contradicted
by the data. I would argue instead that their data provide strong support for my thesis that it is the greater freedom and autonomy provided by any community treatment, not the particular interventions of ACT (which are never consistent across studies anyhow), that is the cause of this increased satisfaction. They asked about patient preferences at a 12 month follow-up (there was no baseline taken). To quote the authors,

“The majority (80%) of experimental group patients who were not admitted to hospital were pleased and grateful about it; only 30% of control group patients were pleased and grateful about being admitted to hospital, whereas 39% were upset and angry.” (Hoult, 1986, p. 142).

Or to put it positively,

“Treatment preference was explored by asking all patients whether they prefer admission to Macquarie Hospital or treatment at home by a community team. The majority of the project (87%) and control (61%) patients preferred community treatment.” (Hoult et al., 1984, p. 163)

What is telling here for my argument is that the majority of the control group (61%), the group that didn’t experience the community treatment (so would not know the specific interventions), still preferred it. This must mean, since the survey question only asked the patients where they preferred to be treated, in a hospital or at home, that the majority even of the group unfamiliar with the specifics of the community treatment, preferred to be at liberty, regardless of what the accompanying treatment actually consisted of, then to be incarcerated in an institution. In fact the experimental group members felt that the most important elements of treatment were the availability of staff for frequent caring personal contact, the hallmark of any sensitive therapeutic relationship, and freedom.
"Most thought that the Community Team was helpful, caring and supportive and many indicated that they appreciated the 24-hour availability and the freedom that community treatment enabled them to have." (Hoult et al., 1983, p. 163).

In #11 (Hoult & Reynolds, 1984) a subset of schizophrenic clients were evaluated using data from the larger study discussed previously (#10). This smaller study was done, because no prior research had been focused on the effects of ACT specifically on this group while using strict diagnostic criteria without allowing client exclusions due to symptom severity (p. 360). In this subset there were 65 patients diagnosed as schizophrenics who were divided into two groups, 33 experimentals and 32 controls. The problematic nature (coercion) of ACT is made manifest in this paper when they discuss how patients were selected for the study,

"The sample consisted of those patients who presented voluntarily[n=23] or were taken involuntarily[n=42] to Macquarie Hospital ... . As each patient presented, the Admission Office nurse opened a sealed envelope in which there was a card stating ‘experimental’ or ‘control’. ... No patients who met the eligibility criteria were excluded from the study, so that even the very ill (e.g. acutely psychotic or suicidal patients) were included." (p. 360-1).

What about those who didn't wish to be treated by ACT or otherwise? No difficulty there,

"Some acutely psychotic patients did need admission. These were patients who either fell asleep when given tranquilizing medication at the Admission Office and so stayed in hospital overnight or who refused initially to co-operate with treatment and so had to be admitted in order that they could be compulsorily treated. After 2 or 3 days they agreed to co-operate and so were discharged." (p. 363).

It’s notable in the above quotes that prior to the National Institute of Mental Health’s Decade of the Brain (the 1990’s) mental health professionals
unselfconsciously admitted the purpose for which psychotropic medications were actually used, to tranquilize, and demonstrated how hospitalization was employed to blackmail patients to follow psychiatric orders. It also appears that none of the patients involved in this or the larger study had a choice about being in these studies. As the authors state all patients were placed in them, even those who, as described in the above quote, refused to co-operate. No data are presented, regarding refusal rates as is common if clients are permitted to refuse participation in any of the 3 associated papers.

Clinical outcomes are even less positive in this sub-sample than the larger study. Here neither the BPRS nor HSRS show any statistical significance favoring the experimental group. The only clinical instrument showing significance is PSE (p. 367-8). The researchers here however do not compare PSE baseline measures to follow-up measures as they did in the larger study, but just the follow-up measures between control and experimental groups. The problem here is that we can’t be sure whether (as in the larger sample) there were PSE sub-scale differences at baseline among this sub-sample or not. The authors do not mention the issue. However they report that two of the three clinical measures at follow-up are statistically non significant and only one is significant. This single claim of significance is only valid if PSE sub scale differences were not present at baseline in this sub-group as they were in the overall sample, information that is not provided. My judgment that this study does not corroborate functional improvement resulting from ACT is echoed by Olfson’s review (1990a) of this study, “In this study, however, there were not significant
differs in the functioning of the two schizophrenic sub-samples”(p. C-75).

My earlier analysis of the larger more inclusive sample which, argued that the asserted significant difference in client satisfaction favoring the experimental group based on ACT effectiveness is invalid, holds as well for this sub-group of schizophrenics. The questions used for both analyses where asked of all participants at the same time and were the same and consequently the same critical arguments apply to both study results.

**English replication**

Five published articles discuss this particular effort (Muijien et al., 1992a; Muijien et al., 1992b; Marks et al., 1994; Knapp et al., 1994; Audini et al., 1994). The first four articles refer to phase one and the last article to phase two of the controlled study. This study attempted to replicate the Test and Stein Training In Community Living (TCL) model using information gathered from that group as well as information obtained from the Australian replication of that model.

“The DLP was modeled on the experimental intensive community care services developed in Madison and Sydney, providing continued care with ‘assertive outreach’”. (Knapp et al., 1994, p. 195).

“To allow replication of the Madison approach, members of the Maudsley and Madison CC teams, and some of the Maudsley and Sydney teams, made 10 exchange visits.”(Marks et al., 1994, p. 180).

The 189 patients (Control group=97, DLP=92) of the study were selected during the first 26 months (from October 1987 to December 1989) and were evaluated for at least 18 months or longer (p. 180). It is important to note that the criteria of selection for the sample required the functional diagnosis of SMI (severely mentally ill), not a particular clinical diagnosis of major mental illness,
“Patients: (a) had SMI requiring urgent hospital admission (including patients who were violent, suicidal, or detained under a Section of the Mental Health Act 1983).” (Marks et al., 1994, p. 180).

This subtle strategic shift from clinical to functional evaluation by Psychiatry, reflective of the times, is an indirect but important admission of failure by the psychiatric experts. The attempt at blurring and collapsing diagnostic categories into the broad and consequently vague construct of ‘the seriously and persistently mentally ill’ both limits the value of the results of the studies based on it (most replications) and indicates that even the experts find diagnostic categories seriously flawed scientifically for classifying or targeting treatment (no evidence of reliability and validity). However, rather then admit that people with these problematic behaviors and situations are perhaps not ill, an ad hoc argument is advanced to switch the ‘treatment’ effort to so called behavioral outcomes from symptom improvement. These behavioral outcomes are such things as employability, stability in different housing situations, improved socialization etc., but these problems are what all impoverished, educationally and socially deprived individuals might need help with, they are not mental health problems per se.

All patients in this trial were asked before randomization if they had any objection to either community or inpatient-hospital care (Muijien et al., 1992b, p. 750). This was done in order to insure that patients who might not want community care could be excluded. Apparently, the thinking among the researchers was that some patients might not feel that community treatment was
as effective as the standard inpatient treatment and as a result would not want to be subjected to it. The researchers note that, "If community care was rejected patients were to be excluded from the study, but this never occurred." (Muijen et al., 1992b, p. 750). The researchers don’t appear to care however, whether the patients ever rejected inpatient hospitalization even though they collected this data, and never appear to think that this is important information. The obvious explanation is that all these patients were admitted through the emergency clinic and were ‘facing emergency admission’ (Marks et al., 1994, p. 180). That is, they would be admitted involuntarily if deemed to be in need of care by a psychiatrist regardless of their desire not to be hospitalized, so there would be no point in noting any patient objections to hospitalization.

The finding by the researchers in this study that patients didn’t resist community treatment is, as noted before about patients in earlier studies, a clear indication of a preference by patients for freedom over imprisonment. Here again, according to the self-reported responses of patients they were willing to participate in any community treatment provided without knowing the actual treatment content. The nature of DPL (the experimental treatment) was not explained to them at the time they were asked for their responses.

“All patients and their relatives were asked by the clinic staff before randomization whether they objected to either hospital or community care .” (Muijen et al, 1992b, p.750).

It would have been helpful for a more accurate analysis if the researchers would have provided the data on client refusal of in-patient care. This would have provided data to test my hypothesis, that many patients would have objected to
such treatment. But this clearly was of little concern to the researchers who as professional psychiatric workers were expected, as part of their regular administrative requirements, to involuntarily hospitalize, and so no such data was included.

The claimed results of the trial were the following. For Phase I

“Outcome was superior with home based care. Until month 20, DLP care improved symptoms and social adjustment slightly more, and enhanced patients’ and relatives’ satisfaction. From 3 to 18 months DLP care greatly reduced the number of inpatient bed days as long as the DLP team was responsible for any inpatient phase its patients had. Cost was less. DLP care did not reduce the number admissions, nor of deaths from self-harm (3 DLP, 2 control). One DLP killed a child. … (Beyond 20 months most gains were lost apart from satisfaction)”(Marks et al 1994, p. 179).

For Phase II

“The slim clinical and social gains from home-based v. out/in-patient care during Phase I were largely lost in Phase II. Duration of crisis admissions increased from Phase I to Phase II in both DLP II and DLP control patients. During Phase II, patients’ and relatives satisfaction remained greater for home-based than out/in-patient care patients. … . Such satisfaction was independent of clinical/social gains.”(Audini et al., 1994, p. 204).

**Analysis: Phase I**

The first point to be noted is the high numbers of patients who did not complete the rating instruments after baseline. Baseline completion percentage of all patients-control group=98%, DLP=99%; 4 month completion percentage of all patients-control group=63%, DLP=74%; 11 month completion percentage of all patients-control group=67%, DLP=72%; 20 month completion percentage of all patients-control group=73%, DLP=78%(Marks et al., 1994, p.182) with statistically significant differences found for fewer missing ratings for DLP men (n=48, 52% of sample) v. control men (n=46, 47% of sample)(p<.04) and for DLP
'old' patients v. control 'old' patients (p < .05). Resistance by the patients was cited as the main reason for the incomplete follow-up data (Marks et al., 1994, p. 182). The finding that patients were 'resistant', suggests the lack of voluntary involvement of many patients in both the treatments and the study itself. Further, because at the follow-up points from 22% to as much as 37% of the patient data was missing, serious questions about the reliability and validity of the data to reflect accurately the effect of the experimental treatment arise.

The second point of note is that on the PSE sub-scores at baseline, “DLP 'old' patients had less severe PSE neurotic subscores than control 'old' cases (p < .009 SNR, < .003 for NSN) and also than 'new' DLP patients (p < .009 for SNR and for NSN) ... Across diagnostic subgroups, fewer DLP than control patients were 'old' BM cases (17 v. 34, 18% v. 35% p < .01).” (p. 182)

These results, since no evidence was given for controlling for the significant differences found in analysis at baseline between the control and DLP groups favoring DLP, suggest lack of baseline group equivalence and problems with the randomization process, strongly undermining any research findings favoring the experimental group.

As to the findings themselves: on clinical measures GAS scores were non-significant at all measurement points, BPRS was significant (p = .03) only at the final measurement point, PSE total was non-significant at all measurement points with the delusional and hallucinatory syndromes sub-scale (DAH) significant (p = .03) at the 4 month measurement point only, the behaviour, speech and other syndromes sub-scale (BSO) significant (p = .05) at 4 months only, the specific neurotic syndromes sub-scale (SNR) significant (p = .04) at the final
measurement point only, while the non-specific neurotic syndromes sub-scale (NSN) was non-significant at all measurement points (p. 184.). The patient satisfaction scores were non-significant at 4 months, favored DLP at 11 and 20 months (p = .001) but are only marginally or not at all related to clinical outcomes (program effectiveness), since of the 14 possible measures of clinical outcomes at 11 and 20 months only two are found to be significant (20 mo. BPRS p = .03, and 20 mo. SNR sub-scale p = .04). As both are final measurement point results at 20 mo. my argument for exit bias as in the VA study (see analysis of VA study to follow) is applicable here as well in explaining the 20 mo. patient satisfaction and clinical results (p. 184).

The results of this study corroborate what I found in the earlier studies and they offer little empirical support for PACT claims. The authors of this study echo my findings in their rather pallid claims for the outcome effectiveness of the experimental treatment.

"Over 20 months, home-base care improved symptoms and social adjustment slightly more than did standard in/out patient care, and was definitely preferred by patients and relatives. Home-based care reduced the duration (but not the number) of crisis admissions by at least 80% but only if the community team was responsible for discharge. Cost was less. Beyond the 20-month study, consumer satisfaction with CC continued despite loss of most other gains."(p. 191, emphasis added).

This study also adds further support for my claim that it is the administrative procedures rather then treatment that reduces hospitalization duration. Only when the experimental team made the discharge decision as noted in the highlighted portion of the previous quote, was there a significant reduction in the duration of crisis admissions.
But this study also suggests some serious problems. Most troubling is the report by the researchers that,

“In the cohort of 189 patients, five died of self-harm in the 20 month study (three DLP, two control). As with SMI suicides in Madison[Test and Stein’s] study such deaths were unexpected and occurred despite recent contact with staff.”(p.187).

This is of concern because neither the experimental nor the control treatment at its best was able to do anything about the suicides; the authors spend considerable article space attempting to demonstrate that especially the DLP was carefully and comprehensively provided to these patients, although with no success. Several DLP patients were judged to be improved by the DLP experts right before they committed suicide. DLP in-fact had a higher suicide rate then the control treatment (see p.187-8) This points to the problematic nature of psychiatric claims to evaluate so called SMI clients. Their tools appear to be highly unreliable both in preventing suicides and in identifying suicidal individuals (Gomory, 1997). One might go further, although more careful research is needed in exploring the possible harmful elements in assertive treatment that may contribute to both suicidal behavior and completed suicides, and question the scientific validity and professional ethics of using any coercive methods in working with such patients (Gomory, 1997). I say this because the authors assure us that these individuals had very close attention (unwanted?) paid to them by the assertive treatment team. “The … three DLP suicidal patients had had unusually persistent care from the DPL”(p. 187). The questionable utility of in-patient and coercive out-patient care in general, is further supported by the findings reported in this study that,
“Outside the DLP study, among in-patients and newly discharged cases in the 480-bed Bethlem-Maudsley Hospital suicides were found to occur surprisingly, about once in 7 weeks.” (pp.187-8).

This is highly suggestive but without comparative suicide rates of the surrounding community or London perhaps; we are left to our conjectures. It would have been helpful if the researchers would have provided such rates for comparison to improve the rigor of outside evaluations of their effort. Alas the absence of such data permeates PACT research.

Phase II

The second phase of the study evaluates the usefulness of ACT for long term treatment (covering 45 months of treatment). In this second phase the patients in the first phase experimental group were randomly redistributed into a standard care control group and an experimental group which, continued to receive the DPL treatment.

Findings:

“Method: Patients, aged 18-64, had entered the trial at month 0 when facing emergency admission for SMI. After at least 20 months home-based care (Phase I), patients were randomized at month 30 into Phase II (months 30-45) to have either further home-based care (DLP II, n=33) or be transferred to out/in-patient care (DLP-control, n=33). They were assessed at 30, 34, and 45 months. Phase I control patients (n=70) were assessed again at 45 months. Measures used were number and duration of in-patient admissions, independent ratings of clinical and social function, and patients’ and relatives’ satisfaction.

Results: The slim clinical and social gains from home-based v. out/in-patient care during Phase I were largely lost in Phase II. Duration of crisis admissions increased from Phase I to Phase II in both DLP and DLP-control patients. During Phase II patients’ and relatives’ satisfaction remained greater for home-based than out/in-patient care patients. At 45 months, compared with the Phase I control, DLPII and relatives were more satisfied with care. Such satisfaction was independent of clinical/social gains [compared to both Phase I and Phase II controls].” (Audini, 1994, p. 204).
In depth analysis of Phase II is not required since the researchers admit candidly that the experimental treatment had little if any superiority as determined by their own data.

“Our controlled results are rather chastening. The anticipated superiority of DLPII over DLP-control patients did not materialize on most measures. Many of the gains from community care which had been present at month 20 were lost during months 30-45(Phase II) in both groups. There was no DLPII v. DLP-control patients’ superiority on duration of admissions or on most clinical or any social measures. Patients … nevertheless continued to be more satisfied with DLP than control care”(p. 209).

These findings corroborate my previous findings. I might point out once again that this research strongly suggest, as I believe do all the other previous studies analyzed in this chapter, that freedom, in the form of the least restrictive environment, regardless of the actual methodology employed, is what is favored by patients over all alternatives that are more restrictive. In this study, as in most of the other previous studies, no statistically significant positive relationship exists between targeted experimental treatment and increased client satisfaction favoring ACT.


This final study to be analyzed in this chapter, is perhaps the most important one because it claims to demonstrate long term clinical improvement, cost effectiveness, and improved client satisfaction favoring ACT treatment using a large sample (n=873). The sample is the largest by far of any of the PACT RCT’s. Patients are evaluated across ten separate sites in two different types of hospitals (Neuro-Psychiatric -NP or General Medical and Surgical Hospitals-
GMS) all in a large public health care system (the VA system) under routine circumstances.

“Intensive community treatment of high hospital users has different outcome profiles at different types of facility. At acute care hospitals[GMS] it is associated with greater improvement in long-term (2-yr.) clinical outcomes and, when fully implemented, is cost neutral. At long-stay hospitals[NP] treating older patients with lower levels of functioning at the time of program entry, it is not associated with clinical or functional improvements at two years but generates substantial cost savings.”(Rosenheck & Neale 1997, p. 3).

More specifically,

“On measures of clinical status and social adjustment there was only one significant difference between groups across the [4]follow-up periods[every 6 mo.]: IPCC[ACT] patients at GM&S[GMS] sites had higher community living skills. However at the final interview IPCC at GM&S sites showed significantly lower symptoms (BPRS); and higher functioning (GAS, Community Living Skills) and satisfaction with services. IPCC treatment resulted in reduced hospital use at both GM&S and NP sites but these differences were significant only at the longer stay NP sites. Comparison of IPCC and standard treatment patients on total societal costs, including the cost of IPCC, showed significantly lower costs for IPCC at NP sites ($82,454 vs. $116,651, p<.001). but significantly higher costs at GM&S sites ($51,537 vs. $46,491, p<.01). However when two sites that incompletely implemented the model were dropped from analysis, costs at GM&S sites were only $38 greater IPCC (p=ns, p. 2).”

These findings appear to strongly corroborate the long-term effectiveness of ACT across all important outcome variables, including the clinical, even if differentially distributed across patient groups.

**Analysis by hospital type:**

**NP hospitals** resemble traditional state mental hospitals both in their history and current use (Rosenheck et al, 1995, p. 130). There were four NP sites with total n=345. This group of patients was found to be older, more likely to be white, with poorer social-vocational adjustment, with greater hospital utilization and were also more likely to carry a clinical diagnosis of schizophrenia or other psychoses
then the GMS patients (Rosenheck et al., 1995, p.133). On clinical and functional outcomes on NP hospital patients, the ACT treatment was no more effective then the standard treatment. Cost was the only outcome which was statistically significant.

The 1995 paper only examined VA health care costs, while the 1997 paper examined total societal costs. Both were found to be significantly lower in NP IPCC treatment when compared to the standard VA treatment. The average total VA health care costs per patient for IPCC patients were found to be $33,295(29%) less than for STD-VA patients at the NP sites (p=.0016), while the total societal costs were $34,197(29%) less (p<.001) per patient for IPCC, when other(societal) costs totaling $902 per client were added to the VA health care costs found in the 1995 study. Although the authors declare, “Overall, average societal costs per patient were $34,000 lower for IPCC treated patients at NP sites, a statistically significant difference.”(R & N, 1997, p. 13). The societal cost significance is driven only by the VA healthcare costs because, “[d]ifferences in non-health care costs were small and not statistically significant”(p. 13). So the claim of significance for 'societal' costs is hyperbole, because other then the original statistically significant cost differential found by the researchers in 1995 involving VA health care costs, no additional statistically significant costs, societal or any other, have been discovered. This inflated rhetoric is similar to the claims made by Test et al for their ‘all poor settings’ variable favoring ACT critiqued earlier, where all of the significance was apparently the result of just one component variable, the reduced psychiatric hospitalization/skilled nursing home
stays, and none the result of the other two, reduced jail time or homelessness, both of which were non-significant (see ch. 5) but were included nonetheless by the researchers. The use of ‘umbrella’ variables such as ‘societal costs’ and ‘all poor settings’, falsely suggest effectiveness in areas beyond the legitimate purview of the specific data that produces the statistical significance of these collapsed variables.

Regardless, the purported monetary savings favoring IPCC is clearly something to consider when choosing between interventions, despite the lack of any clinical outcome differences. Things however are not always what they seem. The apparent substantial dollar difference between the cost of treatments is based on some rather insubstantial data. In describing the measurement of VA health care costs in their 1995 study the researchers note that,

“Total VA health care costs for each patient were determined by multiplying the units of service consumed by the site-specific average cost per unit of service ... using the VA’s standardized national Cost Distribution Report. ... Using accounting procedures standardized across the entire VA system the CDR identifies and distributes to each major type of health care service both direct healthcare costs and indirect costs.”(p. 132).

So far, it sounds like a well-organized bureaucracy providing accurate cost analysis. Unfortunately the authors in the very next sentence inform us that,

“However, data on the duration of visits, the number of clinicians involved in each outpatient visit, and the intensity of inpatient services are not available from VA data bases, so the precision of these cost estimates is limited.”(p. 132).

I would think that it would be extremely limited, close to non-existent, entailing ‘guesstimates’ since the researchers describe no accurate alternate method for getting this data. If you don't know the duration of visits or the number, and of
course as a result, the type of clinician (MD, psychologist, psychiatrist, M.S.W., nurses etc.) in attendance, you cannot know with any degree of precision any of these important and costly expenses. This use of ‘guesstimates’ strongly undermines the validity of the significant cost differentials favoring ACT claimed by the authors.

This is especially important because according to table 5 (p. 137) there was among the four NP’s only one (NP-4, p<.001) which had a statistically significant difference in cost favoring ACT. Of the others all with non-significant differences, one (NP-1) actually had a non-significant difference favoring the standard treatment by 6%($7,210). The over all significant cost finding in NP settings (p<.001) was the result of one hospital (NP-4), which had a 56% difference of $93,852(p<.001) favoring ACT. The text only provides the aggregated findings of all four hospitals (p. 135) and does not discuss the potential problems associated with using aggregated data, which when desegregated (as in table 5, p. 137), unmask the significance claim’s misleading effect (see discussion of umbrella variables above). Such maneuvers obscure the primary role of the one hospital (NP-4) which produced the statistical significance. The fact that the other three hospitals’ results were non-significant strongly suggests that the institutional practices (decision rules for hospitalization for example) at NP-4 were different from the other three and are responsible for the statistical significance found and not due to the advantage of the experimental treatment per se.

**GMS hospitals** according to the authors, “provide shorter term, crisis-oriented inpatient care akin to that offered in academic and community general hospitals
(Rosenheck et al., 1995, p. 130). There were 6 GMS sites with a total n=528. In these hospitals the authors found that ACT, “is associated with greater improvement in long-term (2-year) clinical outcomes and when fully implemented is cost neutral”(R &N, 1997, p. 3).

The clinical improvement found in these settings favoring the experimental treatment unlike the results of no difference found between control and experimental treatments in the NP sites is explained by the fact that the GMS sites had, “younger, higher functioning patients, with greater subjective distress, -- patients who may be more likely than those at NP facilities to have the capacity and motivation for change.”(p. 17).

The claim of clinical effectiveness is based on the result of significantly higher community living skills found across all measurement periods favoring IPCC patients, and with the findings, at the exit (final) interview and at no other follow-up period, of significantly lower symptoms (BPRS), higher functioning (GAS, Community Living Skills) and the proverbial increase in satisfaction with services (p. 2). In these two papers as noted in my previous comments on other studies, instruments requiring observer ratings (BPRS, GAS) are open to observer bias (observers in this research were independent but not blind) and results gathered by instruments using self-report measures (BSI and Self-report measure of community functioning) may be biased by the effect of the environment on the patients’ responses. That for example, the statistically significant finding of better community living skills reported by the GMS ACT patients may be strongly influenced by the sense of competence and autonomy engendered in these
patients by being free (while receiving IPCC treatment), compared to the sense of institutional dependence promoting a sense of incompetence produced in the control patients by being frequently imprisoned (while receiving standard care). Leading he patients to attribute the impact of the environment falsely to ACT(IPCC).

The finding by the researchers of significantly higher reported community living skills across all treatment periods favoring the IPCC group is also belied by the data provided in table 2 and fig.3 of their 1997 paper. At 6 mo. (IPCC-14.11 vs. STD-VA-14.40) and at 12 mo. (IPCC-14.00 vs. STD-VA-14.23) the actual measurements of community living skills competence favors the control treatment. The graph provided by the authors in fig. 3, after indicating an initially slightly higher score of 14.66 for IPCC Vs 14.58 for STD-VA (at baseline), shows that the control group out performed the experimental group (reported higher competence) for well over 12 mo., with the most impressive difference favoring the experimental treatment occurring at 24 months, the exit interview (is this what drives the significance?). The material in the table and figure 3 puts in puzzling light the claim of the researchers that, “Comparison of outcomes at GM&S sites across all time points using random regression analysis showed significant differences on ... the measure of community living skills.”(p. 12).

Is it possible that after fighting hard for over a year to attain a sense of competence, even in the ‘higher functioning’ population found in the GMS sites, the sense of futility engendered by repeated confinement finally took its toll on the control group? Or perhaps in the alternative or in addition, exit response of
clients in experimental programs is influenced by that fact (the relief at being at the end of a new and ‘innovative’ program or a desire to meet the program’s and researchers’ expectations). This of course is speculation. what follows is not.

In their 1995 paper the researchers identify post facto, two GMS hospitals (GMS-2 and GMS-5) as sites that did not fully implement the experimental treatment. GMS-5,

“... provided virtually no community-based contact or rehabilitation services. This site, in effect, developed a low-intensity patient tracking program rather than IPCC services.”(Rosenheck et al., 1995, p.134).

The other site (GMS-2) provided substantially fewer community based services and under performed in most ACT categories when evaluated for ACT program implementation fidelity compared to the other 4 GMS sites(R. & N. 1997 p. 11). The researchers in their attempt to reduce the significant difference of ‘total per patient VA health care costs’ found favoring the control treatment in these settings (p. 2), a problematic finding for their research agenda, decided to reanalyze the data with these two sites excluded. The aim of this reanalysis was to ‘prove’ that the increased costs of IPCC were the result of these 2 now decertified sites’ higher costs due to their ineffective ACT implementation. The reanalysis eliminated 34%(n=181) of the original sample. This proved fruitful from their perspective, because the statistically significant difference found during the original analysis of cost was reduced to a non-significant difference, although still favoring control treatment (p. 2). From my point of view, it was interesting that, by eliminating these two hospital sites in their re-analysis the researchers created an unintended experimental situation to test other outcomes besides cost,
including clinical outcomes.

The clinical outcome measures of IPCC treatments at the remaining GMS sites in this reanalysis should have increased in their statistically significant impact. This hypothesis is based on the assumption that the dropped programs (those decertified) were either not significantly different in clinical effect from their control comparison programs (STD-VA) or better yet, were less effective. This is the only reasonable interpretation of their ‘decertification for cause’ (lack of resemblance both in structure and in effectiveness to IPCC) of these two sites, that would allow the authors to drop them from the secondary analysis. Low and behold, a paradox,

“Exclusion of these two sites from analysis did not change results of the clinical outcome data” (p. 12).

With over a third of the original sample removed, about half of whom were treated in one of these sites (GMS-5) essentially by periodic contact (almost a no treatment group), there was no change in ‘the results of the clinical outcome data’. If this in fact was the case, then the decertified experimental treatments had to have had the same superior clinical results, and statistical significance (even the one program at GMS-5, that was just a minimal contact treatment) as the other more devoted ACT’s. Being or not, an ACT made no difference to clinical outcome.

If true, for the first time in the history of ACT research, we have an empirical falsification of the claim that ACT is more effective clinically because of its unique qualities (based on assertive methods), by relying, entirely on the research
findings of the ACT experts themselves. That is, even if we assume that there were no methodological difficulties with their study, and we accept all of their claims regarding the experimental treatment, the results wind up falsifying all of the conventional ACT claims, and support my argument, reiterated throughout this paper, that any significance found, is just the obvious desire of people for freedom, which any treatment in the community offers more of, although, older patients who have been conditioned by long institutionalization, like those in the NP sites, may have even lost this basic human desire (p.17).

It seems obvious, that if there would have been statistically significant differences found favoring the clinical effectiveness of the fully implemented IPCC’s over those which were decertified, the authors would have provided those results in their paper. This data would have contributed the final coffin nail needed to entomb the critics of ACT (if there are any beside the present author) by demonstrating how head to head a community treatment with little (GMS-2) or no (GMS-5) ACT is less effective then ACT in toto. This they do not do, and consequently corroborate my hypothesis stated previously.

I called one of the principle authors, Dr. Rosenheck, who was kind enough to share with me his yet unpublished 1997 paper, which I used for this analysis. He confirmed that he believed that the clinical results of these two sites were in the same direction and with similar significance as the results found at the other sites, although he had not done this particular statistical test. He seemed a little surprised by my questions, and said that they raised interesting issues and he should have thought of some of them himself.
Be as that may, it is clear, that even if the alleged statistical differences found for clinical outcomes were real as claimed by the researchers, and not due to bias and measurement difficulties as suggested earlier, they could not be attributed to the ACT program in particular. Both the decertified as well as the fully implemented ACT sites seem to have had identical clinical results, so the clinical effect must be ascribed to some as yet unidentified planned or random activities and/or occurrences at all of these GMS sites.

In speculating as to what might explain this totally unexpected phenomenon an interesting alternate explanatory hypothesis, suggesting a potentially fruitful research program in psychiatric treatment, occurred to me. I call it the ‘Placebo Effect of Psychiatric Rhetoric’. In psycho-pharmacological research ‘sugar’ pills, pills having no chemical impact on symptoms, have been found to help people ‘diagnosed’ as suffering from various mental health ailments any where from 10 to 40% of the time, often comparing very favorably with the chemicals specifically targeted for effective treatment. There are also comparable research findings investigating psychotherapy which indicate strong placebo effects when compared to talking cures (Proleau, Brody & Nathan, 1983, pp. 275-310). I’m hypothesizing, that since great efforts were made in this study to identify, select, and train staff in specific sites for participation in the study, part of each site’s effort to qualify for selection required the provision of evidence by each site, in the form of appropriately conforming language and behavior, that they looked and acted like(appeared to be) good ACT programs,
“Conceptualization of the IPCC program was based on a comprehensive review of the literature and on consultation with an expert in the Wisconsin ACT model [Test & Stein’s]. Ten IPCC programs were selected for study after competitive review of proposals from 22 VA hospitals in the Northeast. Proposals were first reviewed by a panel of VA professionals. The final selections were then reviewed, modified, and approved by a panel of three national experts on community-based mental health treatment. Consultation, including several site visits and attendance at programwide staff meetings, was obtained from a second expert in the PACT approach. ... To maximize consistency and quality in implementing IPCC treatment at all sites, monthly telephone conference calls involving all program clinicians; two programwide training conferences; 30 site visits by the central management/evaluation team; and frequent telephone contact with program managers was used” (Rosenheck et al., pp. 130-31).

This meant that all the professionals working in these programs had to live and breathe the rhetoric and ideology of ACT. They had to come to ‘believe’ that this type of program was a program that worked. Very few people can be effective at their jobs if they don’t believe that the work they do is valuable and meaningful, in other words effective, this is especially true for new and innovative programs. It is then a very short step from the above assumption to the notion that the enthusiasm of a staff can have a therapeutic (Hawthorn/placebo) effect on the clients being treated, separate from any potential causal effect of the overt intervention. This hypothesis would then help explain why 2 programs which turned out retrospectively to have been non ACT in their actual implementations were found to have similar clinical outcomes to the more faithful replications.

Workers who in fact did little or nothing technically resembling ACT (according to Rosenheck et al.), made believers out of their clients by acting and speaking as if they were doing ACT. Clients who because they were allowed freedom (treatment in the community) felt better about that existential situation, and accordingly experienced feeling and reported being ‘clinically improved’, and
likewise were found by clinicians, who were looking to find them thus, also ‘clinically improved’. This is only a tentative hypothesis, which needs to be tested, but it certainly offers one potential explanation for the paradox of these researchers’ data.

These findings all add up to exactly the opposite of what Rosenheck and Neale wish to claim when they state,

“that although the clinical benefits of intensive community care may not be apparent for as long as 18 months of treatment, they may yet appear after further treatment.”(p. 16)

These benefits did not appear and cannot appear (there are no empirically demonstrable unique ACT treatment effects) as corroborated by this, the largest and best-implemented randomized controlled trial of ACT ever done.

CONCLUSION

This chapter reviewed all currently available controlled experimental trials bearing on ACT and found little support for the claims of its superiority over ‘standard' treatment and where available ‘no’ treatment as well, either as to its clinical effectiveness or its claim to be related to increased client satisfaction. Importantly some evidence of this treatment’s harmful effects was found.
I have argued the following:

a) Institutional psychiatry is addicted to justification. This methodology, used to support its claim to professional validity based on declaring that some bad behavior is medical disease, is philosophically bankrupt because of its reliance on induction, a practice falsified by Karl Popper.

b) The disease hypothesis was not corroborated when the research used to argue the reality of schizophrenia was examined.

c) Even thought the problem situation is thus, I examined the historical background and the most current research on assertive community treatment (PACT or ACT). According to institutional psychiatry this treatment is the best tested and most effective treatment for the severely mentally ill, most of whom are identified as schizophrenics. A review of the data on ACT’s provided through controlled trials, found no evidence to support this treatment’s efficacy over alternate or minimal treatment and in certain instances indicated harmful effects. At best the evidence was ambiguous, if one can suspend disbelief and imagine empirical treatments for nonexistent illnesses.

d) The examination of the professional history of the creators of PACT indicated an education in psychiatric treatment acquired in state mental hospitals that was inconsiderate of the person-hood of their patients and documented the
use of pseudo-behavioral methods more akin to torture than treatment to force behavior change.

e) This learning history explains the coercive methods used in ACT. Where effectiveness or client change as the result of ACT were touted, I argued that, it was better explained by the coercion and the meddling activities of the ACT workers. The results of the coercive actions of the workers, intentionally or not, were mistakenly taken for voluntary behavior change undergone by the treated patients. The alleged inability to maintain treatment effect after treatment is stopped (the claim of most mental health treatments) or the inability to generalize treatment effect at least in PACT’s, is therefore a phantom, since no treatment effect was ever demonstrated.

f) Despite the lack of any meaningful evidence corroborating any treatment specific effect, the experts of assertive community treatment continue to argue its value. The reasons can be but two, either these experts have no more fruitful alternate ways to deal with their perceived problems and as ‘helping professionals’ they feel compelled to do something rather than nothing, and/or, they have self-serving agenda’s that require activity that can be asserted to be helpful in order to maintain professional status, funding, careers, or their own positive sense of self as helpers.

Discussion

I have argued that Psychiatry uses a justificationary philosophic perspective in its attempt to explain unwanted or unpleasant behavior by the medical model. I further argued that Karl Popper has falsified this inductive approach to doing
science on logical grounds, and thereby nullified it as a valid procedure for scientific theory development. Despite this current problem situation, induction is still widely used both in the physical as well as the social sciences\textsuperscript{33} (see Chapter 1, & Gomory, 1997b).

In the field of mental health it has led to countless numbers of controlled experimental and other types of research\textsuperscript{34} aiming at validating mental diseases and their cures or treatments. This is typically done by having psychiatric experts write review articles containing long lists of research results which demonstrate nothing individually, but when summed together are purported to indicate, or clearly suggest, or show an emerging picture of, or can be understood as showing evidence for, such diseases. One of the chief proponents of biological psychiatry, Nancy Andreasen, in a recent article provides an example of how ambiguous language and data can be used to aver the reality of mental illnesses. As she explains,

“The challenge in developing a scientific psychopathology in the 1990’s is to use the power of multiple disciplines. … Contemporary psychiatry studies mental illnesses as diseases that manifest as mind and arise from the brain. It is the discipline within cognitive neuroscience that integrates information from all these related disciplines [cognitive psychology, neuropsychology, neuroanatomy and neurobiology] in order to develop models that explain the cognitive disfunctions of psychiatric patients based on knowledge of normal brain/function.” (Andreasen, 1997, p. 1586).

Beside the sheer arrogance of her unsubstantiated claim that psychiatry is the central science to be utilized for understanding human behavior, it is also telling

\textsuperscript{33} For a denial that anything other then inductive methodology is possible when doing science see Tyson, 1995.

\textsuperscript{34} PsychINFO Data Base for example as of 3/98 provides 27,842 citations for schizophrenia and 4335 citations for schizophrenia research, by far the most on any of the various DSM type diseases.
that one of the leading advocates of the disease model of mental illness is forced to admit that psychiatry, despite its widely publicized claims for having established this reality is in actuality still looking in the 1990’s to ‘develop a scientific psychopathology’.

In the same article she also makes the following statement, “There are at present [1997] no known biological diagnostic markers for any mental illnesses except dementias such as Alzheimer’s disease.”(p. 1586). This illustrates both the scientific failure and the long-term strategy of mental health experts. Rather then discovering mental illnesses scientifically, they simply concoct arbitrary groupings of displeasing behaviors, perhaps those that were earlier seen as sins(Szasz 1970), or re-label well established neurological diseases such as dementias as mental diseases, and use the reality of these physical diseases to assert that all the other touted mental diseases, like the ones reconstituted from sin to sickness based on psychiatric mythology(see chapter 2), are just as real as the organic dementias. They argue that these are currently unidentifiable only because of our limited technology. They never once entertain or test the obvious alternate hypothesis, even after 110 years of empirical failure, that the misbehaviors asserted to constitute, schizophrenia, clinical depression or bi-polar disorder do not form medical disease entities and may be amenable to explanations of an other sort,
“The to-be-discovered lesions that define the remainder of mental illnesses[other than dementias] are likely to be occurring at complex or small-scale levels that are difficult to visualize and measure, such as the connectivity of neural circuits, neuronal signaling and signal transduction, and abnormalities in genes or gene expression. Despite their lack of defining objective index such as glucosuria is for diabetes, however, these illnesses are very real.” (p. 1586).

This is transparent mendacity. At best, based on her own statements, the most that could be asserted is that she is hypothesizing their putative existence, but with no objective tests identifying these disease entities forthcoming, her claim is without empirical force but not professional authority. It is of course easier to rely on authority then critical tests when claim making, especially if the authority can profess a medical pedigree. The problem here is not that Andreasen is looking to use the medical model to explain certain behaviors, but rather that, she dogmatically asserts what she needs to demonstrate by using hard data. I contend that in science we should be free to hypothesize potential solutions(explanatory theories) as we will, with no prior restraint as to what is or is not permissible(or under what conceptual framework we must work), as long as the resultant hypotheses are testable. If we want correct explanations for and consequently be able to offer appropriate help(treatments) to people with troubling experiences, thoughts, or behaviors, (be they called normal or abnormal), we should test our ideas among all populations - especially unselected ones35 - to see if they occur in and identify discreet or heterogeneous

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35 The restriction of psychiatric research to selected(psychiatric) populations and the nonutilization of unselected(normal) populations when seeking to test notions about behavior that is alleged to be symptomatology of psychiatric illness, prevents the testing of at least one important alternate hypothesis: that such behaviors( hallucinations, delusions, thought disorder etc.) occur in normal populations as well. The corroboration of this hypothesis(see Boyle, 1990) can falsify psychiatric claims that these behaviors are unique to and descriptive of specific mental diseases.
groups and/or circumstances. The search for potential causes and ‘cures’ should be carried out without a priori expectations and beliefs that distort the focus of independent research, letting the experimental chips fall were they may.

It may turn out that discreet differences are found among the behaviors of various groupings as a result of open explorations, which may indeed lead to the identification of various diseases similar to Alzheimer’s, neurosyphilis, pellagra psychosis or encephalitis lethargica, all of which were, at one time thought of as mental diseases. Andreasen discusses some of the current efforts along these lines testing out various alternate cognitive models to explain ‘mental illness’ in psychiatric populations (p. 1587-1592).

She describes for example *The lesion method and neuroimaging* (p. 1591) and the resultant findings which are being used to construct various cognitive models of depression that she believes may eventually help explain why similar symptomatology is found among putatively discrete syndromes like clinical depression and schizophrenia.

“Because these frontal regions have also been implicated in ‘psychomotor poverty dimension’ that is related to the alogia and avolition of schizophrenia, and because psychomotor retardation is an important symptom of depression, these findings support the possibility that similar symptoms observed in different disease conditions may share a common neural substrate” (p. 1591).

This tentative explanation, and the equivicating language used – ‘these findings support the possibility that’, or ‘may share a common neural substrate’ etc., - makes clear it is no more then that, should be tested against other possible alternate hypotheses for best fit.

Since the studies Andreasen mentions, note consistent overlaps in important
symptoms(behaviors) present in at least two of what are thought by psychiatric experts usually as distinct mental diseases, these findings might need to be tested under an alternate hypothesis such as this: “The results indicate the vast overdetermination of most symptoms(behaviors), and their appearance in all sorts of settings and populations without the presence of objective signs as part of the syndrome picture blur and thereby undermine meaningful mental disease categories(perhaps requiring a reformulation of their nature or construct, perhaps even falsifying them) and be better explained by psycho-social rather than biological theory” (Bentall, 1990; Boyle, 1990; Breggin, 1991). Whether either or neither is a correct explanation is of course an empirical question yet to be determined.

Andreasen suggests that as a result of this and some other research there are some findings that appear to support her contention about the biological origins of schizophrenia and analogously depression.

“The common thread in all these observations, spun from four different starting points is that schizophrenia reflects a disruption in a fundamental cognitive process that affects specific circuitry in the brain and that may be improved through medications that affect that circuitry. The various teams use different terminology and somewhat different concepts ... but they convey a common theme.”(p.1590).

She mentions very little about theoretical modeling and the search for explanations of troubling behavior among normals, but the apparently regular occurrence of behavior, hallucinations for example, which she describes as ‘common symptoms of severe mental illnesses’(p. 1587), in normal populations tends to undermine rather then corroborate her claims(Bentall, 1990).
Although I bring only a social scientist’s perspective to what is often biological and medical claim making, it is clear after a close reading of Andreasen’s article that despite her claim, no conclusive evidence is offered to corroborate the biological disease explanation of schizophrenia or 'mental illness'. This is for good reason. All of the research Andreasen reviews is still ongoing and as she states, “... increasingly sophisticated strategies and conceptualizations are [still] emerging” (Andreasen, 1997, p. 1592).

I would just briefly reiterate once more that her assumption, illustrated in the previous two quotes, that proving the biological causation of mental illness can be based on inductive accumulation of evidence or on the summation of findings from various domains or from different perspectives has been falsified long ago by Karl Popper (Miller, 1995). Science is incapable of proving anything, it can only point out what is false. Her apparent unfamiliarity with what philosophy has labeled Hume’s Problem suggests that there is a fundamental error in her methodology and understanding as to how Science progresses - by the critical testing of theories, eliminating false theories and providing as a consequence more comprehensive ones which explain all that the old theories did and add something more. This difficulty may be fatal to her arguments.

By way of concluding this part of my discussion I would like to briefly identify a number of difficulties that biological psychiatrists would have to overcome in order to realise in fact what they currently simply assume to be the facts. First researcher’s like Andreasen would have to find a solution for the rather old but critical problem of reliably and validly identifying discrete groups of
schizophrenics whose controlled experimental research results can meaningfully be said to lead to conclusions like the one she makes that, “The applicability of this model to schizophrenia has been shown through neuropathology studies that demonstrate increased cell packing density in the prefrontal cortex in schizophrenic patients which is consistent with a loss of neuropil.” (p. 1589).

Even those who believe in the explanatory value of this model without reservation, such as Jamison and Goodwin, who wrote the leading text on manic depression, admit to the problematic nature of these diagnostic categories (questionable reliability and validity) which have led to highly heterogeneous membership, certainly making the claim that such ‘cell packing density’ is attributable to some organic brain deviation present in a discrete grouping of schizophrenics who are reliably and validly separable from various other groups of psychiatric or normal populations problematic.

“Maneros and colleagues and Maj noted a high proportion who undergo syndrome shifts - that is, at one point in their history they can be diagnosed schizophrenic, at another as having an affective disorder.” (Goodwin & Jamison, 1990, pp. 88-89)

This leads David Cohen, a social worker and well respected mental health researcher to conclude after citing the research that, “A careful and skeptical reading of the evidence would reveal that not a single biological abnormality of any sort has been identified consistently with schizophrenics and only schizophrenics, no matter how broadly or narrowly diagnosed. This I suggests, so far constitutes the ‘biological basis of schizophrenia’” (Cohen, 1989).

Another objection that would need to be overcome is the charge of using very simplistic reductionistic reasoning when arguing the linear causality in the above quote maintaining that brain circuitry disruption is responsible for (mis)behavior
(Andreasen, 1997, p.1590). Many researchers (Rose, 1997 especially cogently) have pointed out the difficulties with assuming that micro elements (cells, genes, neurons) control or determine – as opposed to contribute in an unquantifiable way to – macro processes (cognitive development, physical structure, and volitional behavior) of the human organism,

“The phenotype of an individual cannot be broken down into the separate contributions of genotype and of environment, because the two interact to produce the organism” (Lewontin et al 1984, p.97).

Adding to this, our absolute inability to explain how and why we make choices or behave as we do by any non-trivial scientific theory, makes such efforts perhaps futile. Noam Chomsky, the noted cognitive scientist and one of our clearest thinkers says the following,

“… when we turn to such matters as causation of behavior, It seems to me that no progress has been made, that we are as much in the dark as to how to proceed as in the past, and that some fundamental insights are lacking. Roughly, where we deal with cognitive structures, either in a mature state of knowledge and belief or in the initial state, we face problems, but not mysteries. When we ask how humans make use of these cognitive structures, how and why they make choices and behave as they do, although there is much that we can say as human beings with intuition and insight, there is little, I believe, that we can say as scientists.” (Chomsky, 1976, p. 138).

Andreasen would also need to face the related reductionistic difficulties inherent in a posteriori or ex juvantibus arguments - explanations of etiology and pathogenesis of a putative disease state based on the assumed mechanism of action of the treatment employed, such as are claimed for certain pharmacological agents in Andreasen’s article (pp. 1589-91) and for all psychoactive drugs used on the mentally ill. The criticism goes something like this,
“... the disease models inferred on the basis of drug mechanisms of action are grossly inadequate from at least two viewpoints. First they ignore the fact that complex behavioral problems require consideration of historical, socioeconomic and cultural factors, and cannot be adequately described in pathophysiological-neurochemical terms which do not provide any account, e.g. of important cognitive aspects. ... Secondly, the models tend to confound hypothetical ‘downstream’ mechanisms contributing to the production of a particular type of symptom with a complex array of both proximate and remote causes and mechanisms placed ‘upstream’, both in the organism showing the disturbance and outside.” (Bigami, 1982, p 97).

"When you have a cold, your nose runs. Yet despite the invariable correlation of the two, it would be a mistake to believe that the cold was caused by the nasal mucus; the chain of cause and effect runs in the reverse direction. ... After all, when one has a toothache one can alleviate the pain by taking aspirin, but it does not follow that the cause of the toothache is too little aspirin in the brain." (Rose, 1997, p. 292).

Finally, the confounding effects of long-term psychotropic drug use or other invasive treatment, like ECT(such as organic brain damage like tardive dyskinesia or brain trauma), on the brain research outcomes in all psychiatric studies should be indicated. Andreasen, like most of her colleagues, doesn't provide the drug use history of the experimental groups in any of the studies she describes. Since long term use of these drugs along with ECT causes permanent brain damage in a substantial number of psychiatric patients it would be important to know what was done to control for this potential confounding effect, because otherwise the alleged brain changes noted in schizophrenics may be explained by the damage caused by these treatments, not by the ‘schizophrenic disease process’ (Breggin, 1991, 68-114).
“The effects of neuroleptic drugs constitute crucial variables in the interpretation of signs of neurological and cognitive dysfunction in chronic patients. Neuroleptic drugs affect practically every brain cell, organ, and regulating system in the human body and, like all neurotoxic substances, produce brain damage after prolonged exposure at high doses.” (Cohen, 1989, p. 256).

This brief list is not meant to be exhaustive but is provided to illustrate some of the roadblocks, several of which appear to be formidable, that would have to be eliminated before Andreasen and her fellow medical modelers can scientifically assert the biological causal explanation of misbehavior as disease. As I illustrated in my review of Kraepelin and Bleuler's work in chapter 1 and here in my analysis of Andreasen’s approach, biological psychiatrists, theoreticians, or researchers, never make clear claims of having established the validity of the disease model based on their data alone (the data plainly doesn't provide such evidence), but invariably have to go beyond the research, that is, exaggerate what can be asserted, and rely on their authority to convince, knowing that few if any will look beyond the claim. This Wittgenstinian strategy seems to have been successful, since only a small handful such as, Richard Bentall, Mary Boyle, Peter Breggin or Thomas Szasz have publicly identified it.

Research efforts looking to justify hypotheses are doomed to fail not only because induction is logically false, but perhaps as importantly, because justification leads to tunnel vision which when faced with any negative research outcome uses ad hoc explanations to save favored theories, refusing to entertain failure (for examples see chapter 6). This commitment to preferred theories makes the search for alternate, perhaps better explanations far less likely.
“Some genuinely testable theories, when found to be false, are still upheld by their admirers - for example by introducing ad hoc some auxiliary assumption, or by re-interpreting the theory ad hoc in such a way that it escapes refutation. Such a procedure is always possible, but it rescues the theory from refutation only at the price of destroying, or at least lowering, its scientific status. One can sum up all this by saying that the criterion of the scientific status of a theory is its falsifiability, or refutability, or its testability.” (Popper, 1962, p. 37).

These tactics and the consistent need to justify as correct to retain professional authority, also contributes to the difficulties inherent in the scientific community’s peer review system. The only way to survive both as a scholar (tenure committees look for peer reviewed publications) and as a researcher is to be approved by this system in both the hard and soft sciences. As Peter Duesberg, a well respect if controversial professor of molecular and cell biology at the University of California at Berkeley makes clear in his 1996 book Inventing The Aids Virus,

“The scientist who is very productive, most able to sell research, and well liked for not offending his peers with new hypotheses and ideas is selected by his peers for funding. The eccentric, ‘absent minded professor’ with ‘crazy’ ideas has been replaced by a new breed of scientist, more like a ‘yuppie’ executive than the quirky genius of old academia. These peers cannot afford a nonconformist, or unpredictable, thinker because every new, alternate hypothesis is a potential threat to their own line of research. Albert Einstein would not get funded for his work by the peer review system, and Linus Pauling did not (for his work on vitamin C and cancer even though he received two Nobel Prizes). The only benefit of the numerous cascades of competitive tests and reviews set up by peer review is the elimination of unsophisticated charlatans and real incompetence. In sum, the review of too many by too many achieves but one result with certainty: regression to the mean. It guarantees first-rate mediocrity. As these armies of new scientists flood the peer review system, they even act to suppress any remaining dissension by the few remaining thoughtful researchers. Peer review after all, can never check the accuracy of experimental data; it can only censor unacceptable interpretations. A scientist’s grants, publications, positions, awards, and even invitations to conferences are entirely controlled by his competitors. As in any other profession, no scientist welcomes being out-competed or having his pet idea disproved by a colleague.” (p. 65-66).
In chapter 1, taking the claims of biological psychiatry seriously, despite the evidence, I analyzed the data for the existence of the prototype of mental disease, schizophrenia. If the biological psychiatrists are right then surely schizophrenia, the disease alleged to explain almost every senseless human act imaginable, must have been demonstrated by now to exist (Szasz, 1987). Some of the human acts which have been attributed to this disease and not to the person supposedly suffering from it, who under other circumstances would be called a perpetrator, but here considered a victim of schizophrenia, are presidential assassinations, uni-bombings, abortion clinic bombings and child murders. By using a fallibilistic approach to science (Popper, 1962), I have argued that no schizophrenia entity exists and based on the reasons presented in chapter 2 could not be found to exist in the future. This does not preclude, through the use of independent well tested theories, explanations in the future of some strange human behaviors by the use of the disease model, but such diseases would have to be, following well established medical convention, considered to be neurological not mental diseases (see chapter 2).

Next I went on to review the gold standard treatment36 of individuals diagnosed as the severely (sometimes called seriously or persistently) mentally ill. The treatment, Assertive Community Treatment, was evaluated by reviewing both the historical information available on the individuals who invented it and the research done by them and those who replicated their approach. My analysis of

36 I call it the gold standard based on L. Stein’s 1974 article Gold Award: A Community Treatment Program appearing in Hospital and Community Psychiatry, 25, 669-672.
the research concentrated on the controlled experimental trials of this
treatment because as I argued in chapter 6 this is the only research model that
may claim to validly test for causal connections between treatment and outcome.
The analysis of the experimental research strongly suggested that this treatment
was at its best, contrary to the claims of its proponents, no better than other
routine or minimal community or alternative interventions and sometimes more
harmful (the increased incidence of suicide as a result of psychiatric treatment as
noted in chapter 6 for instance). Although people often improved behaviorally,
sometimes dramatically in both the control and experimental treatments, no
evidence was found to causally link this improvement to the specific techniques
employed by ACT. This kind of behavioral change is corroborated by the
improvement found among depressed individuals by the simple passing of time
for instance. In fact, what my analysis suggests is that the troubled or trouble
causing individuals who are involved with the state or public mental health
system, when reporting greater personal satisfaction for ACT where not doing
that, but were, when given a choice, favoring less coercion and more freedom (in
psychiatric jargon, community as opposed to in-patient treatment) regardless of
such treatment’s effectiveness on symptoms (behavior), employment, or social
functioning(chapter 6). To be more specific, where ACT offered more
opportunities for freedom then the alternate treatment the patients tended to say
they were more satisfied regardless of actual treatment effects, and where there
were no differences in the amount of freedom between treatments, no
differences in satisfaction were noted. Rosenheck and Neale’s (1997) research
result, where similar amounts of freedom were experienced by both the GM&S ACT patients and the two formerly GM&S ACT but now decertified program’s patients (chapter 6) exemplifies such outcomes.

I also demonstrated that reduced hospitalization favoring ACT was simply the result of an administrative ploy available to any type of ‘community’ program. A community treatment program merely has to put in place a rule severely limiting or prohibiting completely the hospitalization of those individuals under its control regardless of their behavior, while insisting on providing treatment to them in the community only \(^{37}\) (chapters 5 & 6).

Further, I believe I showed that claims of any statistically significant functional or symptomatological patient improvement based on ACT are equivocal at best and without corroborating data at worst. The researchers attempting to justify ACT, claim to show such effects by using the results only of the instruments providing positive measures and ignoring the results of other instruments measuring these outcomes which don’t (see chapter 5 & 6). The ethical approach would have been to state the results as found in all the measurement instruments and based on the evidence, admit that no conclusions one way or another may be drawn from the ambiguous results. The researchers’ justificationary approach and the self-serving necessity of showing the value of the intervention (for reasons of funding, career building or institutionalizing the treatment program as noted in chapter 4) prevent this kind of straightforward reporting of results. There

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\(^{37}\) This may vary somewhat depending on the sense of commitment a program has to this administrative rule and also in how confident the program is in controlling the problem behaviors of its patients. ACT programs less confident in their coercive capacities may hospitalize their patients, some even more frequently then
are exceptions to this biased reporting, see especially the research reported by those doing the English replication in chapter 6.

One of the most problematic if unacknowledged confounding variables in ACT research is the use of brain damaging psychotropic medications. These drugs, at one time thought to be the long searched for genie in the bottle offering the often wished for ideal of ‘normality’ by providing miraculous chemical succor to all who are smitten in the magic kingdom of Psychiatry, are even by their most fervent believers acknowledged now to be of limited value due to their ineffectiveness (however defined) in at least 40% of mental health patients (Breggin, 1983; 1991). Although Test mentions in her earlier studies that almost all of the patients in the studies are routinely given such drugs, neither her research group nor any of the others, who also generally acknowledge the very high percentage of medicated clients, discusses the implications of long term medication. The use of medications complicates the assessment of outcomes attributed to ACT because of their well established tranquilizing affects which may confound causal claims, and also because of their impact on the strange physical behavior observed in these patients that often is the ‘psychiatric symptomatology’ used to diagnose as severe mental illness what is actually drug-induced organic disease (Cohen, 1989, p. 256).
“The first phenothiazine derivative, chlorpromazine, was synthesized in France in 1950 and introduced into the United States in 1952. Phenothiazines, commonly used at that time as an insecticide and as a treatment for parasitic intestinal worms, came to be employed as a treatment for Parkinson’s disease, surgical shock, motion sickness, nausea, vomiting, itching, and as a general sedative and potentiator for analgesics and anesthesia. ... Within five years after the introduction of phenothiazines, it was noted that they produced neurological side effects, including dystonias (involuntary muscle spasms), akinesia (immobility), akathisia (severe restlessness), and Parkinsonism (tremor, shuffling gate). ... The bucco-lingual-masticatory (BLM) triad comprises the most frequent and notable of TD symptoms, including sucking and smacking motion of the lips, lateral jaw movements, and often rhythmical, thrusting, or ‘fly-catching’ movements of the tongue. Other involuntary movements include movements of the trunk and abnormal gait and may involve nearly all muscles of the body.” (Brown & Funk, 1986, pp. 117-18).

It appears to be the case that psychiatrists have created, where perhaps there was none, a real mental health problem as a result of the iatrogenic affects of these medications. According to one estimate, as of 1985 there were 38.5 million people suffering from just one of these drug induced organic disorders, tardive dyskinesia (Breggin, 1991, p. 90). By now we must have well over 45 million of these organically damaged people, not an insignificant number exhibiting admittedly strange behavior. Paradoxically psychiatry can use these patients’ bizarre behavior to ‘prove’ the validity of its hegemony, while actively resisting over decades, admitting its complicity in making healthy people sick by coercively treating patients with these drugs (Brown & Funk, 1986).

My analysis in chapter 3 of the professional learning history and earlier research of the principal inventors of ACT provided evidence helping to explain the nature of ACT. Their early work with the mentally ill at Mendota State Hospital contained very questionable coercive ‘experiments’, based on particularly odious views of such patients (see quotes under client descriptions in
chapter 3), as a way of changing their behavior through the use of electric
prods. This procedure conflicted with the best scientific research available at the
time indicating that punishment of any sort, not to mention the type employed in
this groups research, was generally unacceptable when it involved human
beings(Azrin & Holz, 1966). I hypothesized that this history was foundational to
and is an explanation of the view more fully developed in their community
treatment that such patients need to be very closely controlled, the researchers
call it assertively treated, by the treatment team in-order to get results. The
various methods of control in ACT include, medicating for the purpose of
tranquilizing the overwhelming majority of patients (see Australian replication
analysis ch. 6), blackmailing them to comply with treatment by threatening the
loss of welfare benefit payments, as well as emotionally and physically harassing
them to do such things as get up, go to work, or attend various treatment
programs against their declared wishes (ch. 4). I believe the evidence reviewed
strongly corroborates, that all ACT’s are coercive, being, as they all are, founded
on assertive treatment developed and philosophically grounded in the early
Mendota State Hospital experience as just explained. These programs expect
compliance by and childlike acceptance of psychiatric authority enforced by the
techniques described above for any failure, rather than autonomy and adult
responsible behavior. Any disagreement by the client with the treatment team is
explained as illness related and therefore irrational and not to be regarded as
volitional activity and rational choice. Treatment compliance is in fact assumed to
be a positive treatment outcome of this model (as in complying with treatment
equates to better mental health), making the use of coercion appear to be
‘therapeutically’ justified.

The over arching result of my analysis is to highlight the intellectual cul-de-sac
institutional psychiatry has gotten itself into. In its effort to identify itself as a
medical specialty it has had to assert a series of claims about human behavior as
a medical disease and in order to keep up appearances also to do ‘research’ on
these claims. As I pointed out in chapter 1, psychiatric research had to resemble
the other medical research done contemporaneously, but only as much as it took
to fool the lay public, since in the past other, non-psychiatric medical researchers
paid scant attention to this research or it’s claims. The reason, as even Woodruff,
Goodwin, and Guze, widely recognized experts of psychiatric diagnosis and
leading organic researchers, candidly admit in their classic text, *Psychiatric
Diagnosis* (1974) is that,

“There are few explanations in this book. This is because for most psychiatric
conditions there are no explanations. ‘Etiology unknown’ is the hallmark of
psychiatry as well as its bane. Historically once etiology is known, a disease
stops being ‘psychiatric’ ”(p. xi).

But, having long ago allied itself with social control and as a consequence the
state psychiatry has served society well by providing an explanation, albeit false,
for certain socially unacceptable behavior(Szasz, 1984, Gomory, 1997; Connery,
1968 provides an especially clear description of this incestuous relationship in
America). Based on this explanation it offered various ‘treatments’ which didn’t
cure or treat disease in the medical sense, since no mental disease entity has
ever been corroborated by the available research, but instead, during
psychiatry’s early history (19th and early 20th century) using various chemical agents, tranquilized into insensibility, and isolated by involuntary hospitalization, its victims, effectively making them invisible and non problematic for the vast majority of society. In our current social situation the psychiatric population is still tranquilized (currently we claim to be correcting chemical imbalances in the brain by the selfsame psychotropic medications, see Breggin, 1983 for critique) and isolated by being ghettoized in run down inner city communities or in special subsidized housing developments for the mentally ill. But now, by using the refractive lens of language, the spin doctors of psychiatry have recast this coercion as ‘treatments in vivo’ and ‘community treatment’. This appears to satisfy our social conscience without solving the behavioral problem which has continued to escalate unabated (Gomory, 1997, Szasz, 1994).

Having said all this, one may suggest that my effort in this dissertation presents an unusually mean spirited picture of psychiatry and psychiatric researchers, who such a critic may claim, are just trying to be helpful in dealing with an intractable social problem. I would respond to such criticism by pointing out that my pessimistic view of psychiatry is based on the historical record and the data provided by the psychiatric research establishment, which on close inspection falsifies its own claims.

In closing I would like to offer quotes from two prominent psychiatrists one representing institutional psychiatry, and the other its critics who both interestingly seem to agree with much that I argued here. The quotes which follow are extensive. They are I believe necessary because as I argued
throughout, the written opinions and ideas of the various protagonists and the historical record of the research done by the supporters of particular positions as well as their critics provide the material required for the objective assessment of claims. This material may be offered initially by each as self-serving evidence of justification to sway popular opinion. But because of their public (published) nature, often contrary to the expectations and wishes of their authors, they can be subjected to full and vigorous critical assessments based on the facts. These in turn may themselves be vigorously dissected, offering at each iteration better and better opportunities for reasoned decisions both lay and professional.

First, C. Macfie Campbell (1876-1943), who taught at John Hopkins in the Department of Psychiatry from 1913-1920 and then directed Boston Psychopathic Hospital and taught psychiatry at Harvard Medical School. In this letter dated December 14, 1934, to C. M. Hincks, the general director of the National Committee for Mental Hygiene, he explains in plain language - much too plain for most of us not used to using language exhibiting racial prejudice so blatantly - the self-serving necessity of using false claims about the nature of schizophrenia and by analogy all mental diseases, as a strategy to dupe both the lay public and the funders of psychiatric research. Allowing for his often misplaced metaphors, using illness where he obviously means troubling behavior for instance, and where he talks about philanthropists remembering to also include government funding for up to date relevance, his analysis is as applicable

38 The very first precursor organization to NIMH and the organization which offered the first concrete rationale for federal intervention in mental health, the importance of having the citizens of America mentally fit through the practice of good mental hygiene,-perhaps on the model of dental-hygiene.(Connery, 1968).
today as when it was written,

“I started more then two weeks ago to submit a brief memorandum to you on schizophrenia . . . . I am somewhat hampered in presenting the topic because in one way I do not believe that there is such a thing as schizophrenia, and on the other hand I think it is the most important topic for investigation in our field. To put it another way, I do not think that there is a disease schizophrenia, but on the other hand, it is useful at this period to have some group term for an extremely large number of cases of mental disorder of the more serious type.

From the point of view of one who is angling for money to support his work, it is a great advantage to believe in a specific disease. He can then give figures with regard to morbidity, he can specify the general group of forces within which the real cause is to be found like the nigger in the woodpile, he can budget the funds necessary for the investigation and even suggest certain time limits within which the nigger will be discovered, the woodpile will be safe, schizophrenia will be eliminated, the taxpayers’ burden will be alleviated.

The philanthropist who may have little knowledge of detailed biochemical or bacteriological problems will thus feel that recondite studies are going to bring about a practical social result. He will have the comfort of seeing a specific piece of work going on, well delimited, with clean-cut formulations and with steady output of scientific facts which at least will be useful by-products even if the nigger will not be found. He may be willing to accept the comparison of the needle in the haystack, and realize that he must think in generous terms of time, money, and personnel. The main thing is that he is working with a definite disease, that the general concepts are those already familiar to medical science and to philanthropy, and that schizophrenia may be put in the same group as yellow fever and those other scourges of humanity which have been brought within control.

To one who looks at schizophrenia in a somewhat different way and who sees it just as a useful term embracing a very heterogeneous group of mentally sick people, angling for funds is a somewhat more disheartening process. He can of course, use the same bait as the person who believes in the nigger theory of schizophrenia, but that may go against his conscience. He may have to tell the philanthropist that he does not believe there is such a thing, that the schizophrenic patients whom he has studied most thoroughly seem to represent people of very varied physique and personality, brought up in the most varied circumstances, exposed to a great variety of life situations and who break down in many complicated ways which are perhaps a combination of their physical status, their personal difficulties and the social and cultural situation in which they are enmeshed. He may have to tell the philanthropist that the topic of schizophrenia is almost as broad as the topic of human nature, in fact the chief value of its investigation may be that it is the clearest and most striking
demonstration of the real facts of human nature, facts of human nature which are concealed by the ordinary conventional repressions of the so-called normal man. The normal man is the person who so adapts himself to social regimentation that he manages to conceal the underlying crudities of human nature so that practically nothing about them can be learned from a study of him.

A research into human nature would naturally send a chill down the spine of a practical philanthropist who wishes to budget for definite projects and who would like to see, if possible some return for his money. He may suspect the person who rejects the nigger-in-the-woodpile theory as being a somewhat loose thinker, fond of generalization, unable to give precision to his problems, with no definite lead as to the further course of his investigations, and with no promise that his investigations will have any practical value." (In Grob, 1985, pp. 48-49).

And Thomas Szasz, the much maligned critic of mental illness as disease, who has been witheringly denounced by institutional psychiatrists, but rarely critiqued accurately on the facts and arguments he actually presents (Szasz, 1993, pp. 797-800 for a partial but strong rebuttal to his critics) summarizes his take on the history of Institutional Psychiatry in Cruel Compassion (1994) thusly,

"The history of psychiatry, unlike the history of medicine, exhibits a distinctive pattern of cycles of patient abuse and institutional reform. Each cycle is characterized by the psychiatrist's staunch claim that he is a genuine medical healer, that his involuntary subjects are sick patients that the buildings in which the subjects are imprisoned are hospitals, and that the inmates' detention and subjection constitute medical treatments.

The cycles begin with the confinement of the insane in private madhouses. Soon, their proprietors are accused of incarcerating sane persons. The abuse is attributed to the profit motive. The solution is the public madhouse system, managed by physicians on the public payroll, supervised by authorities accountable to the public.

Once established, the public mental hospital system turns out to be a method of warehousing society's undesirables. Its managers and staff are even more corrupt and sadistic than the keepers of private madhouses had been. The problem is attributed to insufficient funding and inadequate doctors. The solution is spending more money on psychiatry and more time on training psychiatrists.

Meanwhile, mental hospitals multiply and flourish. Psychiatrists claim therapeutic success for one new intervention after another. Mental patients are subjected to
bleeding, cupping, tranquilizing chairs, ice-cold showers, threats of drowning, and other sadistic measures. After a few decades, the treatments are rejected as useless or harmful.

Toward the end of the nineteenth century, genetic explanations of diseases become fashionable. Psychiatrists declare that earlier therapeutic enthusiasms were naïve and misplaced. Insanity is an incurable, hereditary disease. Once a person is insane, he is destined to remain so for the rest of his life. The prominent psychiatrists of this era, exemplified by Emil Kraepelin, do not pretend to cure their patients. Instead, they model themselves after the pioneering pathologists who studied cadavers and classified diseases. In short the great state hospital psychiatrists were nosologists. They studied living corpses, called chronic mental patients, and classified their alleged diseases, creating mythological entities such as dementia praecox, manic-depressive illness, paranoia, and schizophrenia. Almost a century ago, psychiatry’s most celebrated madman, Judge Paul Schreber, faulted his psychiatrist, the famous German psychiatrist Paul Flechsig, for focusing on diseases rather than persons. In his Memoirs, Schreber wrote: “[Flechsig] did not understand the living human being and had no need to understand him, because … he dealt only with corpses.”

During the nineteenth century, society opened a second front in its war against mental illness. Psychiatrists and jurists join force and expanded the hitherto limited scope of civil commitment and the insanity defense. Coerced psychiatric examinations and psychological testing were introduced into every nook and cranny of the social fabric, from schools to divorce courts. The closer the alliance of psychiatry with the law and with education grew, the more indispensable coerced psychiatric interventions seemed to become.

After World War I, medical scientists made rapid advance in controlling infectious and metabolic diseases, notably the contagious diseases of childhood and diabetes. Psychiatrists imitated these discoveries by introducing into the practice of psychiatry so-called somatic treatments, such as insulin coma, convulsions caused by metrazol and electricity, and lobotomy.

And so we arrive at the present scene, drugs and deinstitutionalization. Once again, politicians and psychiatrists clamor for mental health reform. Now they claim that the patients are sicker than we thought they were; that mental illness makes them refuse to take the medications that make their maladies manageable; that the drugs previously hailed as having revolutionized the treatment of chronic mental illness are “ineffective or inadequate for as many as 60 to 80 percent of the patients”[According to Alan Breier, research psychiatrist at the National Institute of Mental Health; quoted in Acker, C. ‘New drugs quite world for those with schizophrenia. Detroit Free Press, June 29, 1993], that it was a mistake to give mental patients freedom, which they only abuse by not taking the drugs that keep them sane and law abiding. The reforms proposed are
predictable: More money for mental health programs and for research on new psychiatric drugs; more legal and medical control of mental patients; more mental health education to teach the truth about mental illness."(pp.195-6).

AMEN.

The sound byte summary of two-hundred some odd pages:

“IF the evidence doesn’t fit, then the disease model of mental illness is not legit!”
Appendix A
<table>
<thead>
<tr>
<th>ACT As of Dec. 1997 Studies and Timeline</th>
<th>Study population</th>
<th>Design and method of assessment</th>
<th>N</th>
<th>Attrition</th>
<th>Clinical status ACT exp. group</th>
<th>Social function ACT exp. group</th>
<th>Work function ACT exp. group</th>
<th>Independent living ACT exp. group</th>
<th>Service satisfaction ACT exp. group</th>
<th>Red. hospital use ACT exp. group</th>
<th>Lower costs ACT exp. group</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Audini et al, 1994, (45mo.)</td>
<td>SMI</td>
<td>RCT/indp. but not blind</td>
<td>66</td>
<td>26 of 92 from 1st phase(28%), 9% of exp. &amp; 15% of control at 45 mo.</td>
<td>GAS, PSE, &amp; BPRS ns. All measures incl. SAS showed deterioration</td>
<td>both groups worsened slightly. ns</td>
<td>ns</td>
<td>nr</td>
<td>sig. but did not relate to clinical or social outcomes</td>
<td>ns</td>
<td>nr</td>
</tr>
<tr>
<td>2. Bond et al, 1998 (6 mo.)</td>
<td>high risk of hospitalization</td>
<td>RCT/ACM clinical staff</td>
<td>16 7</td>
<td>45 of 212(21%)</td>
<td>ns</td>
<td>ns</td>
<td>nr</td>
<td>nr</td>
<td></td>
<td>centers A &amp;C</td>
<td>ns</td>
</tr>
<tr>
<td>3. Bond et al, 1991 (18mo.)</td>
<td>S MI substance users</td>
<td>Quasi-exp. w. control / clinical staff</td>
<td>97</td>
<td>6mo.-17(18%) 12mo.-28(29%) 18mo.-22(25%)</td>
<td>ns</td>
<td>ns</td>
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<tr>
<td>4. Borland et al, 1989 (5yrs.)</td>
<td>diagnosed Schizophrenic or bipolar</td>
<td>pre-post no control / client staff</td>
<td>72</td>
<td>81 of 168 ‘selected’ 9 lost(11%)</td>
<td>ns</td>
<td>ns &amp; may have worsened</td>
<td>ns</td>
<td>nr</td>
<td>nr</td>
<td>sig. change</td>
<td>ns</td>
</tr>
<tr>
<td>5. Burns et al, 1993 (12mo.)</td>
<td>S MI</td>
<td>RCT/ind.</td>
<td>172</td>
<td>337 randomized, 172 entered study(40% attr) 28% control &amp; 21% of exp. group refused to</td>
<td>ns</td>
<td>ns</td>
<td>nr</td>
<td>nr</td>
<td>ns</td>
<td>sig.</td>
<td>ns</td>
</tr>
<tr>
<td>Study</td>
<td>Population</td>
<td>Design</td>
<td>Setting</td>
<td>N</td>
<td>Follow-up</td>
<td>Results</td>
<td>Control</td>
<td>Costs</td>
<td>Staff</td>
<td>Client</td>
<td>Sig.</td>
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<tr>
<td>6. Bush et al, 1990 (12mo.)</td>
<td>v. high hospitalization rates</td>
<td>RCT/ nr</td>
<td>28</td>
<td>nr</td>
<td>nr</td>
<td>sig. by cm judgement</td>
<td>nr</td>
<td>nr</td>
<td>nr</td>
<td>sig.</td>
<td>nr.</td>
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<tr>
<td>7. Curtis et al, 1992 (up to 52 mo.)</td>
<td>SMI</td>
<td>quasi case-control / parandom.</td>
<td>43</td>
<td>nr</td>
<td>nr</td>
<td>nr</td>
<td>nr</td>
<td>nr</td>
<td>nr</td>
<td>sig.</td>
<td>higher rates then control</td>
</tr>
<tr>
<td>8. Hambridge &amp; Rosen, 1994 (12mo.)</td>
<td>SMI in cm for &gt;12mo</td>
<td>pre-post no control / client staff</td>
<td>64</td>
<td>nr</td>
<td>ns</td>
<td>sig. for 1st 6mo., ns. at 12mo.</td>
<td>nr</td>
<td>nr</td>
<td>nr</td>
<td>sig.</td>
<td>nr</td>
</tr>
<tr>
<td>9. Hornstra Schiz</td>
<td>none rand/quas-</td>
<td>22</td>
<td>nr</td>
<td>ns</td>
<td>nr</td>
<td>nr</td>
<td>nr</td>
<td>nr</td>
<td>ns</td>
<td>nr</td>
<td></td>
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<tr>
<td>Study</td>
<td>Year (Duration)</td>
<td>Condition</td>
<td>Design</td>
<td>Sample Size</td>
<td>Outcome Measures</td>
<td>Effect Size</td>
<td></td>
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<tr>
<td>et al, 1993 (24mo.)</td>
<td>o-phrenic</td>
<td>expl. with matched control / staff</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>10. Hoult et al 1983; Hoult 1986 (12mo.)</td>
<td>SMI</td>
<td>RCT/ind. but not blind</td>
<td>120</td>
<td>12% of exp. group, 16% of control group</td>
<td>PSE-sig. HSRS-sig. BPRS-ns Exp. group sig. lower at baseline in 2 subscores of PSE</td>
<td>nr ns nr sig. sig. ns?</td>
<td></td>
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<tr>
<td>11. Hoult &amp; Reynolds 1984; (12mo.) part of larger study above</td>
<td>Schizophrenic</td>
<td>RCT/ind. but not blind</td>
<td>65</td>
<td>6% of exp. group, 12% of control group</td>
<td>PSE-sig. HSRS-ns BPRS-ns</td>
<td>nr ns nr sig. sig. ns</td>
<td></td>
<td></td>
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<tr>
<td>12. Lafave et al, 1996 (12mo.)</td>
<td>SMI</td>
<td>RCT/ind.</td>
<td>65</td>
<td>45(40%) referred out of 110 didn’t receive service,</td>
<td>ns ns ns sig. ns sig nr</td>
<td></td>
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<tr>
<td>Study Reference</td>
<td>Design/Methodology</td>
<td>Sample Size</td>
<td>Effect Size</td>
<td>Statistical Results</td>
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<tr>
<td>13. Lehman et al, 1993 (12mo.)</td>
<td>RCT/ nr</td>
<td>54</td>
<td>None</td>
<td>ns</td>
<td></td>
<td></td>
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<tr>
<td>14. Marks et al, 1994 (Knapp et al, 1994) (min. of 20 mo.)</td>
<td>RCT/ind. but not blind</td>
<td>189</td>
<td>4 mo.-60 (31%)</td>
<td>GAS-ns BPRS-sig. at 20mo. only</td>
<td></td>
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<tr>
<td>15. McFarlane et al, 1996 (24mo.)</td>
<td>Schizophrenic</td>
<td>68</td>
<td>6 (9%)</td>
<td>NS</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>16. McGrew</td>
<td>Post no</td>
<td>212</td>
<td>sig.</td>
<td>sig./legal problems per</td>
<td></td>
<td></td>
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</tbody>
</table>

Note: GAS = Global Assessment of Functioning, BPRS = Brief Psychiatric Rating Scale, SAS = Social and Impulsive Behavior Scale, PSE = Present State Exam, ACT = Assertive Community Treatment, sig = significant, ns = not significant, nr = not reported.
<table>
<thead>
<tr>
<th>Study</th>
<th>Homelessness</th>
<th>Control / Case Managers</th>
<th>Client sig. Increase</th>
<th>Other Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>17. McGurrin &amp; Worley, 1993 (3yrs.)</td>
<td>SMI</td>
<td>Pre-post w. control no rand./case managers</td>
<td>18 3</td>
<td>approx. 25%</td>
</tr>
<tr>
<td>18. Meisler et al., 1997 (4yrs.)</td>
<td>SMI</td>
<td>Pre-post retrospective/clinical staff</td>
<td>11 4</td>
<td>none</td>
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<tr>
<td>19. Morse et al., 1997 (18mo.)</td>
<td>SMI homeless</td>
<td>RCT to 3 treatments/clinical staff</td>
<td>16 5</td>
<td>30(19%)</td>
</tr>
<tr>
<td>Study</td>
<td>Year</td>
<td>Intervention</td>
<td>Method</td>
<td>Sample Size</td>
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<tr>
<td>Morse et al, 1992 (12mo.)</td>
<td>1992</td>
<td>SMI home</td>
<td>RCT to 3-treatments/ind. staff</td>
<td>17</td>
</tr>
<tr>
<td>Muijen et al, 1992 (18mo.)</td>
<td>1992</td>
<td>SMI</td>
<td>RCT/nr</td>
<td>12</td>
</tr>
<tr>
<td>Qunlivan et al, 1995 (2yrs)</td>
<td>1995</td>
<td>SMI</td>
<td>RCT/nr</td>
<td>90</td>
</tr>
<tr>
<td>Rosenheck et al, 1995 Rosenheck &amp; Neale, 1997 (2yrs)</td>
<td>1995</td>
<td>SMI veterans</td>
<td>RCT/nr</td>
<td>87</td>
</tr>
<tr>
<td>Solomon &amp; Draine, 1995 (1y)</td>
<td>1995</td>
<td>SMI home</td>
<td>RCT/ind. staff</td>
<td>20</td>
</tr>
<tr>
<td>Study</td>
<td>Design</td>
<td>N</td>
<td>Exp. Group</td>
<td>Control/Staff</td>
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<tr>
<td>25. Solomon &amp; Draine, 1995 (5yrs.)</td>
<td>As in # 24</td>
<td>RCT/n</td>
<td>51</td>
<td>ns</td>
</tr>
<tr>
<td>26. Wood &amp; Anderson, 1994 (2yrs.)</td>
<td>Chronic psychiatric patients</td>
<td>non exp. matched control / staff</td>
<td>12 8</td>
<td>10(8%)</td>
</tr>
</tbody>
</table>

nr-not reported, ns-not significant, sig.- statistically significant at p<.05, cm-case management, RCT-randomized controlled trial,
ARTICLES:


NY Times (vol. CXLVII no. 51,082) 2/28/98 A6, Cloner of a sheep moves to persuade the skeptics.


BOOKS:


