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IN REVIEW

Treatment Outcome of Personality Disorders

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Objective: To review the treatment outcome of personality disorders.

Method: A literature search of studies pertaining to personality disorder and outcome was conducted, and studies that focused primarily on Axis II were retained. Of these, naturalistic outcome studies were distinguished from those that addressed treatment outcome specifically. The treatment outcome studies were examined in terms of type of treatment intervention, dependent variables, and outcome.

Results: Contrary to contemporary assumptions about Axis II, a substantial number of treatment outcome studies were identified. Trends in the assumptions underlying psychosocial and pharmacologic approaches were identified on the basis of dependent variables.

Conclusion: There is evidence that effective treatments exist to alleviate symptoms and reduce symptomatic behaviours that accompany personality disorders. What these results hold for the idea of remission from personality disorder is considered.

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Key Words: personality disorders, treatment outcome, outcome, Axis II, symptoms, symptomatic behaviours, functioning, stability, remission

The notion of personality disorders as stable and enduring traits was crystalized with the distinction of Axis II in the Diagnostic and Statistical Manual (DSM-III) (1). As such, the treatment outcome of personality disorders became an oxymoron that begged the question, is treatment outcome possible in personality disorder? The Axis II assumptions underlying the current definition of personality disorders imply that treatment outcome is not a relevant concept. This has been reinforced by an abundance of recent treatment outcome studies in which the implications of comorbid Axis II psychopathology are studied in relation to Axis I. Not surprisingly, the uniform finding is that Axis II psychopathology negatively effects the treatment outcome of Axis I disorders (that is, the adding of Axis II to Axis I renders Axis I less treatable). As we attempt to examine treatment outcome in personality disorders, we are faced with 3 basic questions. First, what, if any, evidence exists that personality disorders are changeable? Second, if such evidence exists, what is the nature of that changeability, or how can it be described? Finally, is the changeability of personality something that can be influenced by treatment? In other words, is there such a thing as the *treatment* outcome of personality disorders?

A literature search using the broad classifications of treatment outcome and personality disorders reveals 3 classes of studies. The first and most prevalent class of studies focuses on comorbid Axis II psychopathology in relation to Axis I outcome. The tack encompassed by the majority of these studies examines the impact of personality disorders on Axis I treatment outcome, an idea that is very different from the notion of treatment outcome of personality disorders. Because these studies are not

concerned with the treatment outcome of personality disorders specifically, they will, for the most part, be excluded here. The second class of studies directly addresses the stability hypothesis by examining the natural course of personality disorders. The methodologies of these studies consist of naturalistic, longitudinal (prospective or retrospective) assessments. The third class of studies directly addresses treatment outcome. These are the studies designed to examine the effects of a particular intervention which is employed with the intention of ameliorating *at least some specific aspect* of the psychopathology of Axis II. Of this latter class, the subdivision between psychosocial and pharmacologic interventions reflects characteristic differences in underlying assumptions and approaches to targeting change.

That there are enough treatment outcome studies of personality to warrant this review seems at odds with the currently held assumptions about Axis II. From a broader historical perspective, notions about whether or not personality can change have been widely divergent (2). The data are integral to the assumptions that underlie our nosology. Some authors have suggested that once something on Axis II shows treatment responsiveness, there enters a risk of it being “moved” to Axis I (3,4). We will return to problems of taxonomy regarding the stability and changeability of personality traits and personality disorders raised by this review. First, the evidence of the changeability of personality disorders is reviewed by examining the naturalistic studies followed by those studies that are aimed at capturing the effects of treatment designed to intervene with specific psychopathologies of personality.

Table 1a. Studies of natural course and outcome of personality disorders

Study	Personality disorder (sample size)	Length of time for follow-up	Method; sample	Outcome variable(s)	Findings
Robins (7)	Psychopathic (n = 94)	30 years	Retrospective; outpatient	Symptomatic behaviours; multidimensional functioning	Good premorbid functioning associated with a more positive outcome; more than half did not remit
Grinker and others (8)	Borderline syndrome (N = 41)	1.5 to 3 years	Prospective; inpatient	Multidimensional functioning	No improvement; low functioning; one-third rehospitalized
Werble (9)	Borderline syndrome (N = 51)	3 to 5 years	Prospective; inpatient	Multidimensional functioning	Stable work functioning; impaired social functioning; one-half rehospitalized
Maddocks (10)	Psychopathic (N = 59)	5 years	Retrospective; outpatient and forensic	Syndrome; functional behaviour	Impulsivity appeared stable; recidivism declined; 5% suicided
Carpenter, Gunderson (11) Carpenter and others (12) Gunderson and others (13)	Borderline syndrome (N = 24)	2 years	Prospective; outpatient	Syndrome; multidimensional functioning	Relative stability; negligible improvement; modest levels of symptoms and functioning
Skodol and others (14)	DSM-II BPD (N = 30)	2 years	Prospective; outpatient	Symptoms; Symptomatic behaviours	Anxiety, affective instability persisted; treatment compliance poor in BPD
Akiskal (15)	DSM-II BPD (N = 100)	0.5 to 3 years	Prospective; outpatient	Symptoms; syndrome	Moderate comorbidity with affective disorder; BPD diagnosis relatively stable
Pope and others (16)	DSM-III/DIB BPD (N = 33)	4 to 7 years	Prospective; inpatient	Symptoms; multidimensional functioning	BDP more stable than depressive disorder; high comorbidity of other PDs
Copas and others (17)	Psychopathic (N = 194)	3 and 5 years	Retrospective; inpatient	Syndrome; multidimensional functioning	More than 6 months of treatment associated with better outcome; multiple offences and self-damaging behaviours impeded improvement
Barasch and others (18)	DSM-III BPD (PDs) (N = 76)	3 years	Prospective; outpatient	Syndrome; multidimensional functioning	Stability demonstrated; BPD distinct from affective disorder
Perry (19) Perry (20) Perry, Cooper (21)	DSM-III BPD/ASPD (N = 82)	1 to 3 years	Prospective; outpatient and forensic	Symptoms; symptomatic behaviours	Symptoms (mood related for BPD; impulsivity for ASPD) persist for both disorders; slight improvement in GAS for both disorders
Plakun and others (22) Plakun (23,24)	DSM-III BPD/STPD SCZD/NPD (N = 131)	13.6 mean years (SD = 6.6)	Prospective; inpatient	Symptoms; functioning	Self/other report in improvement in GAS and social functioning

McGlashan (25,26) Bardenstein, McGlashan (27)	DSM-III/DIB BPD (N = 89)	15 mean years (range 2 to 32)	Retrospective; inpatient	Syndrome; multidimensional functioning	Good outcomes increase with time; good work function; 3% suicided; interpersonal stability by avoiding intimacy
McGlashan (28)	DSM-III STPD (mixed) (N = 109)	15 mean years (range 2 to 32)	Retrospective; inpatient	Syndrome; multidimensional functioning	Mixed social and work functioning; validated distinction from BPD
Modestin, Villiger (29)	DSM-III BPD (N = 18)	4.6 mean years	Prospective; inpatient	Symptoms; multidimensional functioning	Some improvements in social functioning; symptoms remained stable; two-thirds rehospitalized
Paris and others (30,31)	DIB BPD (N = 322)	15 years	Prospective; inpatient and outpatient	Symptomatic behaviours; multidimensional functioning	Less impulsivity with time; 23% rehospitalized; unstable social functioning; limited pleasurable activities

AVPD = Avoidant Personality Disorder; ASPD = Antisocial Personality Disorder; BPD = Borderline Personality Disorder; STPD = Schizotypal Personality Disorder;
SCZD = Schizoid Personality Disorder; NPD = Narcissistic Personality Disorder; PD = Various unspecified personality disorders; DIB = Diagnostic Interview for Borderlines;
DIN = Diagnostic Interview for Narcissism; PCL = Psychopathology Checklist.

The Stability and Natural Course of Personality Disorders

As this paper primarily focuses on *treatment* outcome, we will present only a cursory summary of longitudinal studies that examine the natural course of personality disorders. These studies are many, and several reviews are already available (5,6). For the present purposes, we will focus our examination of the naturalistic studies with a primary question in mind: Can personality disorders change?

Table 1 lists those studies that we have classified as longitudinal or naturalistic (7–51). To be classified under this rubric, studies primarily had to address personality psychopathology over time, either prospectively or retrospectively (in a few cases, cross-sectional designs were employed) to plot the “natural course” of personality disorders. Although most of these studies employed subjects who were undergoing some form of psychiatric treatment, these treatments largely were neither uniform nor controlled interventions. As such, we do not view them as treatment outcome studies specifically. It should be noted that for a study to be truly naturalistic, nontreatment-seeking subjects should be studied. Otherwise, uncontrolled treatment variables may introduce noise into what might otherwise be an unadulterated course of the disorder.

Inspection of Table 1 reveals that the vast majority of longitudinal studies have focused on borderline personality disorder (BPD), or some variant thereof, and comprise most of the studies in our review. A substantial number of studies have also examined antisocial personality disturbance. Two studies examined schizotypal personality disorder, and 1 study looked at personality disorders from clusters A and B from the DSM-III-R (52). The prominence of BPD and antisocial personality disorder is something that we found across all types of outcome studies which we examined, and this raises its own questions. Is it the case that other Axis II entities are not really regarded as disorders and have been neglected by investigators (and clinicians)? We, of course, cannot reasonably attempt to answer this question here, but it is certainly an important trend that is worthy of more consideration.

Table 1b. Studies of natural course and outcome of personality disorders

Study	Personality disorder (sample size)	Length of time for follow-up	Method; sample	Outcome variable(s)	Findings
Stone (32) Stone and others (33)	BPD (n = 205) PDs (N = 550)	10 to 23 years	Prospective; inpatient	Multidimensional functioning	GAS improvement in functioning after 5 to 10 years
Tucker and others (34)	DSM-III BPD (N = 40)	1 to 2 years	Prospective; inpatient	Symptomatic behaviours; multidimensional functioning	Improvements in symptomatic behaviour; improvements in social and work functioning
Costa, McCrae (35)	NEO five factors (N = 983)	6 years	Prospective; community	Five-factor dimensions	Stability on five factors demonstrated
Hart and others (36)	PCL (N = 231)	2.3 to 1.3 years	Retrospective/ cross-sectional; forensic	Functional behaviour	Low PCL associated with higher probability of remaining out of prison

Serin and others (37)	PCL (N = 93)	1 year	Prospective; forensic	Functional behaviour	PCL predicted recidivism
Silk and others (38)	DSM-III/DIB BPD (N = 9)	1 to 3.5 years	Prospective; inpatient	Syndrome	Seven of 9 retained DSM-III diagnosis; 4 of 9 retained DIB diagnosis; social isolation accompanied interpersonal stability
Arboleda-Florez, Holley (39)	DSM-III-R ASPD (N = 39)	25 to 51 years	Retrospective; forensic	Functional behaviour	Decreases in criminality at the same time
Harris and others (40)	PCL (N = 169)	10 mean years	Prospective; forensic	Symptomatic behaviours	PCL scores predicted recidivism
Mehlum and others (41)	DSM-III-R BPD (N = 29)	2.8 mean years	Prospective; outpatient	Symptoms; multidimensional functioning	Moderate improvements in symptoms and functioning except for social functioning (social functioning average to begin with)
Paris and others (42)	DSM-III-R/DIB-R BPD (N = 39)	2 years	Retrospective; outpatient	Syndrome	Sexual abuse possible risk factor for reduced recovery in DIB lifetime BPD
Vaglum and others (43)	DSM-III cluster A and B (N = 73)	2 to 5 years	Prospective; outpatient	Syndrome	High diagnostic stability
Garnet and others (44)	DSM-III BPD (N = 21)	2 years	Prospective; adolescent inpatient	Syndrome	PDs not stable in adolescents
Mattanah and others (45)	PDs (N = 70)				
Harper, Hare (46)	PCL (N = 889)	na	Cross-sectional; forensic	PCL factors	Affective-interpersonal stability; deviance declined with age
Black and others (47)	DSM-III ASPD (N = 71)	29 mean years	Retrospective; inpatient	Syndrome; functional behaviour	Stability demonstrated; high comorbidity with other disorders; frequent legal difficulties
Najavits, Gunderson and others (48)	DIB BPD (N = 37)	3 years	Prospective; outpatient	Symptoms; symptomatic behaviour; functioning	Symptom reduction; improvement in GAS (shift from "poor" to "fair" level of GAS); reduction in suicidal behaviour
Sabo and others (50)					
Ronningstam and others (51)	DIN; DSM-III-R; DSM-IV NPD (N = 20)	3 years	Prospective; inpatients and outpatients	Syndrome	Diagnostic instability in NPD demonstrated

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SCZD = Schizoid Personality Disorder; NPD = Narcissistic Personality Disorder; PD = Various unspecified personality disorders; DIB = Diagnostic Interview for Borderlines;

DIN = Diagnostic Interview for Narcissism; PCL = Psychopathology Checklist.

The early longitudinal studies of personality psychopathology painted a less than optimistic picture for prognosis and outcome. Borderline Personality Disorder (or syndrome or organization) was a label that, once earned, was difficult to relinquish. Studies that addressed borderline disturbance examined such pathology in patients for time periods of 5 years or less, which suggests that these findings really applied to the shorter haul. A study by Grinker and colleagues produced findings that suggested no improvement in symptoms, continued low levels of social functioning, and a substantial rehospitalization rate (8,9). There was a glimmer of hope in the follow-up report in which findings suggested that work functioning stabilized to a limited degree, but, overall, prospect for improvement was bleak (9). A series of reports from the International Pilot Study of Schizophrenia found essentially similar findings as the early studies that examined patients diagnosed with BPD and schizophrenia (11–13). Those designated with BPD showed little improvement at best and only a modest reduction in symptomatology. Findings were similar for antisocial personality disorder, where Maddocks demonstrated persistence of impulsive behaviour (10). That study, however, interestingly showed a decrease in recidivism with an increase in age, which is a somewhat prescient finding which suggests that people with personality pathology settle down as they grow older. Regardless, the findings from the early group of studies left the field with the notion of personality disorders as stable, persistent, and largely not amenable to treatment.

Table 2a. Treatment outcome of psychopharmacology interventions

Study	Personality disorder	Study design	Treatment intervention	Dependent variable(s)	Outcome(s)
Vilkin (55)	Borderline (N = 45)	Single-blind crossover	¹ Phenothiazine ² Diazepam	Symptoms	– ¹ + ²
Klein (56,57)	Pseudoneurotic schizophrenia (n = 32)	Randomized double-blind	¹ Imipramine/placebo ² Chlorpromazine/placebo	Symptoms	
Klein (56,57)	EUCD (n = 43) HCD/PACD (n = 37)	Randomized double-blind	¹ Imipramine/placebo ² Chlorpromazine/placebo	Symptoms	
Hedberg and others (58)	Pseudoneurotic schizophrenia (n = 28)	Randomized double-blind	¹ Trifluoperazine ² Tranlycypromine ³ Combination	Symptoms	– ¹ ± ² – ³
Reyntjens (59)	DSM-III personality disorders (N = 120)	Open trial	Pimozide	Symptoms; goal functioning	+ ±
Rifkin and others (60)	EUCD (N = 21)	Random assignment/ double-blind crossover	Lithium/placebo	Symptoms	
Tupin and others (61)	DSM-II ASPD (N = 27)	Open trial	Lithium	Symptomatic behaviours	
Sheard and others (62)	DSM-II ASPD (N = 66)	Random assignment/ double-blind	Lithium	Symptomatic behaviours	
Brinkley and others (63)	Feighner criteria (84) borderline syndrome (N = 5)	Open trial	Low dose ¹ Perphenazine ² Thiothixene ³ Thioridazine	Symptoms	
Leone (64)	Gunderson, Kolb (85) BPD criteria (N = 80)	Random assignment	¹ Chlorpromazine ² Loxapine	Symptoms	± ¹ + ²
Serban, Siegel (65)	DSM-III BPD/STPD (N = 52)	Random assignment	¹ Thiothixene ² Haloperidol	Symptoms	
Goldberg and others (66)	DSM-III BPD/STPD (N = 50)	Random assignment	Thiothixene/placebo	Symptoms	±
Hymowitz and others (67)	DSM-III STPD (N = 17)	Single-blind	Haloperidol	Symptoms; global functioning	± –
Soloff and others (68)	DIB/DSM-III BPD/STPD (N = 61) ^a	Random assignment	¹ Amitriptyline/placebo ² Haloperidol/placebo	Symptoms; symptomatic behaviours	– ¹ + ² – ¹ + ²
Soloff and others (69)	DIB/DSM-III BPD/STPD (N = 90) ^a	Random assignment	¹ Amitriptyline/placebo ² Haloperidol/placebo	Symptoms; symptomatic behaviours	– ¹ + ² – ¹ + ²

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^aNote that the change in sample size at the 2-year follow-up study for Soloff and others (52,53) was due to “extending the analysis to 90 inpatients and deleting outpatient trials to enhance homogeneity” (55, p 239).

Several caveats to the early view of personality pathology as intractable must be accounted for. First, these findings may have been an artifact of diagnostic criteria that, at the time, were not clearly laid out in reliable terms. Additionally, the prevailing psychoanalytic zeitgeist might be considered in which the common lore suggested a somewhat tautological assessment procedure of “the borderline” as a patient who could not remain on the couch, that is, who was *defined* by treatment failure. Finally, in perusing the early literature, one cannot help but notice that patients or subjects were routinely referred to somewhat pejoratively as “borderlines,” as opposed to a perhaps less ignoble manner such as individuals suffering from borderline pathology. Nevertheless, it was clear that those people exhibiting personality pathology were more difficult to treat

and among those patients with less than favourable outcomes.

Two substantial changes occurred in the next generation of studies that examined the natural course and outcome of personality disorders in general and BPD in particular. First, these studies employed more systematic attempts than had previously been made in refining both research and clinical diagnostic procedures. Such refinements included the development of systematic research criteria for studying personality pathology, the most notable of these being Gunderson's Diagnostic Interview for Borderlines (DIB) (53) and DSM-III. Second, this next generation of studies was conducted over much longer time periods than those in the first generation. In several instances, these studies spanned 15 years or more.

The more notable among these studies included the Chestnut Lodge Follow-up Studies, which were conducted by McGlashan; a general hospital sample, which was followed by Paris and colleagues; and the New York State Psychiatric Institute Studies (also known as the PI-500), which were conducted by Stone (25–28,30–33). In summary, these findings were important because they were the first to demonstrate tangible improvement in functioning or to suggest the possibility of at least some degree of remission. The overarching finding was that age was the best predictor of prognosis, which suggested a longitudinal process in the direction of remission. Simply, it seemed that those people suffering from personality disturbance burned out as they grew older and that the whirlwind of impulsive and unstable relationships and erratic functioning began to ease. The picture was not entirely optimistic, however, because there was some indication that patients relied on some degree of social isolation to calm their interpersonal world, which was, perhaps, a worthwhile trade-off in the interest of stability and improved functioning. Importantly, the length of time over which these studies spanned allowed the identification of this process and provided some hope for the possibility of a better outcome. At the very least, if patients who suffered from severe personality disorders could be kept alive long enough (no small matter—estimates of the suicide rate approach 10% [5]), there was some promise that things would get better. The fact that these changes could occur at all may have given rise to the idea that treatment interventions, if designed and carried out correctly, might actually speed the process along.

Table 2b. Treatment outcome of psychopharmacology interventions

Study	Personality disorder	Study design	Treatment intervention	Dependent variable(s)	Outcome(s)
Cowdry, Gardner (70)	DSM-III DIB BPD (N = 16)	Double-blind crossover	¹ Alprazolam ² Carbamazepine/placebo ³ Trifluoperazine/placebo ⁴ Tranylcypromine placebo	Symptoms; symptomatic behaviours; global functioning	$-1 \pm 2 -3 +4$ $-1 +2 +3 -4$ $-1 +2 \pm 3$ $+4$
Norden (71)	DSM-III-R BPD (N = 12)	Open trial	Fluoxetine	Symptoms	
Parsons and others (72)	DSM-III/PAF BPD (N = 330)	Double-blind preceded by single-blind period (10 days)	¹ Phenelzine/placebo ² Imipramine/placebo	Symptoms	
Teicher and others (73)	DSM-III-R BPD (N = 11)	Open trial	Thioridazine	Symptoms; symptomatic behaviours	\pm $+$
Coccaro and others (74)	DSM-III-R ASPD/BPD (N = 3)	Open trial	Fluoxetine	Symptomatic behaviours	\pm
Cornelius and others (75)	DSM-III/DIB BPD (N = 5)	Open trial	Fluoxetine	Symptoms; symptomatic behaviours	\pm \pm
Links and others (76)	DIB BPD (N = 19)	Randomized/double-blind crossover	¹ Lithium ² Desipramine	Symptoms; symptomatic behaviours	$--$ $\pm -$
Markovitz and others (77)	DSM-III-R BPD/STPD (N = 22)	Open trial	Fluoxetine	Symptoms; symptomatic behaviours	$+$ $+$
Soloff and others (78)	DSM-III-R/DIB BPD (N = 108)	Randomized/double-blind	¹ Haloperidol ² Phenelzine sulfate/placebo	Symptoms; symptomatic behaviours	$-1 +2$ $\pm 1 \pm 2$
Frankenburg, Zanarini (79)	DSM-III-R/ DIB-R BPD (N = 15)	Open trial/ratings blind to baseline	Clozapine	Symptoms	
Salzman and others (80)	DSM-III-R (SCID-II)/ DIB-R BPD (N = 22)	Randomized double-blind	Fluoxetine/placebo	Symptoms; symptomatic behaviours	\pm \pm

Kavoussi and others (81)	DSM-III PDs (N = 11)	Open trial	Sertraline	Symptoms; symptomatic behaviours	+
Markovitz, Wagner (82)	DSM-III-R/DIB BPD (N = 39)	Open trial	Venlafaxine	Symptoms; symptomatic behaviours	+

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^aNote that the change in sample size at the 2-year follow-up study for Soloff and others (52,53) was due to “extending the analysis to 90 inpatients and deleting outpatient trials to enhance homogeneity” (55, p 239).

Table 3. Treatment outcome of psychosocial interventions

Study	Personality disorder	Study design	Treatment intervention	Dependent variable(s)	Outcome(s)
Avoidant personality disorder					
Argyle and others (87)	Socially compromised (N = 16)	Random assignment	¹ Psychotherapy ² Social skills training	Symptomatic behaviours; social functioning	-1 ± 2 $\pm 1 \pm 2$
Marzillier and others (88)	Socially inadequate (N = 21)	Random assignment	¹ Social skills training ² Systematic desensitization wait list control	Symptoms; symptomatic behaviours	$\pm 1 \pm 2$ $+1 \pm 2$
Stravynski and others (89)	Socially avoidant (N = 22)	Random assignment	¹ Social skills training ² Social skills training with cognitive modification	Social functioning	
Stravynski and others (90)	Socially avoidant (N = 22)	Process analyses	Behavioural treatments combined	Symptoms; symptomatic behaviours	\pm $+$
Cappe, Alden (91)	SAD APD (N = 52)	Random assignment	¹ Graduated exposure plus skills training ² Graduated exposure alone wait list control	Symptoms; symptomatic behaviours	$\pm 1 \pm 2$ $+1 \pm 2$
Alden (92)	DSM-III APD (N = 76)	Random assignment	¹ Graduated exposure ² Skills training ³ Intimacy focus wait list control	Symptomatic behaviours; social functioning	$+1 \pm 2 \pm 3$ $+1 \pm 2 \pm 3$
Stravynski and others (93)	SAD APD (N = 28)	Random assignment	¹ Behaviour therapy ² Behaviour therapy plus in vivo augmentation	Symptoms; social functioning	$\pm 1 \pm 2$ $+1 \pm 2$
Borderline personality disorder (and schizotypal and antisocial)					
Linehan and others (94)	DSM-III BPD (N = 44)	Random assignment	¹ DBT ² Treatment as usual	Symptoms; symptomatic behaviours; global functioning	
Linehan and others (95)	DSM-III BPD (N = 39)	Random assignment	¹ DBT ² Treatment as usual	Symptoms; symptomatic behaviours; global functioning	
Shearin, Linehan (96)	DSM-III BPD (N = 4)	Open intervention/ process study	DBT	Symptomatic behaviours	
Karterud and others (97)	DSM-III BPD/STPD (N = 97)	Open intervention	Day hospital	Global functioning	\pm
Stevenson, Meares (98)	DSM-III-R BPD (N = 30)	Open intervention	Structured psychotherapy	Symptoms; symptomatic behaviours; social functioning; global functioning	\pm $+$ \pm \pm
Munroe-Blum, Marziali (99)	DSM-III-R BPD (N = 79)	Random assignment	CBT Group therapy augmentation	Symptoms; symptomatic behaviours; social functioning	$\pm \pm$ $++$ $\pm \pm$

Davidson, Tyrer (100)	DSM-III-R BPD (N = 3) ASPD (N = 3)	Open intervention (behavioural analyses)	CBT with DBT principles	Symptomatic behaviours	
Personality disorders					
Lieberman, Eckman (101)	DSM-II depressive neurosis (N = 24)	Random assignment	¹ Insight-oriented therapy ² Behaviour therapy	Symptomatic behaviours	± ¹ + ²
Pollack and others (102)	DSM-III-R PDs (N = 31)	Random assignment	Brief adaptional psychotherapy/ wait list control	Symptoms; symptomatic behaviours;	+ +
Winston and others (103)	DSM-III-R PDs (N = 32)	Random assignment	¹ Psychodynamic ² Behavioural therapy/wait list control	Symptoms; symptomatic behaviours;	
Winston and others (104)	DSM-III-R PDs (N = 81)	Random assignment	¹ Brief adaptive therapy ² Short-term dynamic wait list control	Symptoms; social functioning	
Monsen and others (105)	DSM-III-R PDs (N = 21)	Open intervention	Object-relations psychotherapy	Symptoms; global functioning	+ +
Piper and others (106)	DSM-III-R PDs (N = 120)	Matched control group	Day-hospital/ wait list control	Symptoms; symptomatic behaviours; social functioning	± + ±
Springer and others (107)	MCMI BPD/PDs (N = 31)	Random assignment	¹ CBT group (based on DBT) ² Control, discussion group	Symptoms; symptomatic behaviours	

APD = Avoidant Personality Disorder; ASPD = Antisocial Personality Disorder; AVPD = Avoidant Personality Disorder; BPD = Borderline Personality Disorder; CBT = Cognitive Behaviour Therapy; DPT = Dialectical Behaviour Therapy; PDs = Various unspecified personality disorders; SAD = Social Avoidance Distress Scale; STPD = Schizotypal Personality Disorder.

In addition to the previous studies, a study conducted by Pope produced important findings, although it was somewhat limited in the amount of time that patients were followed (16). In Pope's study, it was found that the presence of a major affective disorder was a prognostic indicator for positive outcome, despite the relative stability of the BPD diagnosis. Those findings echo results of an Axis I treatment study conducted by Woody and colleagues, which found that substance-abusing antisocial patients who suffered from an affective disorder were more responsive to treatment than those without an affective disorder, evidencing a better outcome (54). The final study among the more prominent studies of this generation was done at Austen Riggs by Plakun and colleagues (22–24). They found an improvement in overall functioning in patients as measured by global assessment scale (GAS) scores with both schizotypal disorder and BPD. These findings, however, although consistent with other studies from this generation, came from a sample with extensive attrition in which outcome was determined from mailed follow-up self-reports without independent validation. The third generation of follow-along studies took advantage of the best diagnostic technology available using structured interviews such as the DIB and those designed for Axis II of the DSM-III and DSM-III-R. These studies have been conducted over a shorter time span than the major longitudinal studies but, interestingly, have demonstrated change in time periods of less than 5 years. Modestin and Villiger showed improvements in social functioning despite the fact that the symptom picture remained stable (29). Gunderson and colleagues reported on a 3-year prospective-outcome study that suggested significant reductions in symptomatology and increased levels of functioning (48–50). As the authors duly noted, however, these findings were limited by a small sample size and selection procedures that were influenced by a patient's ability to complete an ambitious research diagnostic protocol which might have potentially favoured more highly responsive individuals (48). Nonetheless, the hypothesis of shorter-term change was now actively being researched. This shifting emphasis was also marked by research that identified risk factors which impeded outcome, such as a history of sexual abuse, which allowed for the development of more specific treatment interventions (42).

In summary, it appears that personality disorders, or more correctly, BPD and antisocial personality disorders, show some degree of remission over the long haul. It is less clear if this constitutes real change in the sense of a substantial reorganization of personality or a more "surface-level" change. In other words, has "personality" actually changed, or is it simply being expressed differently? For instance, an individual diagnosed with BPD may simply learn to avoid relationships in the interest of a more stable, albeit subdued, interpersonal livelihood. While some might argue that such a distinction is not entirely relevant, it does seem clear, however we think about it, that some sort of "mellowing out" occurs. This has also been found in

antisocial personality disorder where, although there is stability in the diagnosis, there appears to be a reduction in aggressive and impulsive behaviours in individuals over time (39,47).

By looking at the big picture, we see a very interesting progression. The earliest generation of studies painted the bleakest picture. With refined diagnostic criteria and studies conducted over substantial time periods, the possibility of change was introduced. The most recent generation of longitudinal studies, although shorter in duration, have led to a renewed interest and optimism in the changeability of personality disorders. Not surprisingly, we see that increasing attention has also been paid to the examination of treatments which are specifically designed to change personality disorders.

Treatment Outcome

We now turn to those pharmacologic and psychosocial studies that directly address the treatment outcome of personality disorders. Consistent with the contradiction that you cannot expect treatment outcome with something that is by definition stable, enduring, and not expected to change, the dependent variables in these studies are interesting to note. They range from symptoms and symptomatic behaviours to global levels of functioning and, typically, do not include syndromal classifications (that is, whether the patient continues to meet criteria for the diagnosis). In contrast, Axis I studies routinely include syndromal variation (for example, remission and relapse) and symptom change in their definition of outcome. For personality disorders, however, relapse and remission are presently dubious concepts at best. For example, there are no research diagnostic interview measures designed to look at the remission of personality disorders at the *syndrome* level. This makes for a scattering of dependent variables in treatment outcome studies. While the variation makes it difficult to generalize findings, there are some interesting trends, such as the differences between the psychopharmacologic and psychosocial treatment studies. We begin with psychopharmacologic studies.

Psychopharmacology Studies

In all, we located 28 outcome studies for the psychopharmacologic treatment of personality disorders (Table 2, 55–82). Of those reviewed, 17 of the more recently conducted studies employed the DSM-III or DSM-III-R frameworks. While some of the earlier studies adhered to the DSM-II criteria for personality disorders, we additionally included studies that examined phenomena which would have likely been classified as personality disorder in modern parlance in order to provide the most comprehensible picture possible (83). These included the historical diagnostic concepts of emotionally unstable character disorder (EUCD), hysterical character disorder (HCD), passive-aggressive character disorder (PACD), pseudoneurotic schizophrenia, and the admittedly occasionally rather loosely applied “borderline” designation.

In studies past and present, there has been much variation in how diagnoses were made. Some studies simply do not explain their procedures and provide only a brief statement that a certain disorder was studied (55). Two of the studies refer to specific research diagnostic protocols. For instance, Brinkley, Beitman, and Friedel used research criteria put forth by Feigner and his colleagues (63,84). Leone used the diagnostic characteristics described by Gunderson and Kolb to identify patients with BPD (64,85). In addition to the different taxonomic systems used to identify subjects with various personality disorders, there were also variations in the methods. Some studies employed structured clinical interviews, while other studies relied on chart diagnoses. The limitations secondary to variations in diagnostic procedures are important to bear in mind as we attempt to surmise results from these diverse protocols.

The range of personality disorders studied was rather limited. As with the naturalistic studies, we see that BPD (or related variations) gets the primary attention and accounts for 19 out of 28 (68%) of the studies. Next is schizotypal personality disorder (SPD) (including pseudoneurotic schizophrenia) with 8 of the 28 studies (29%). In 5 out of 8 cases, SPD is comorbid with BPD. Finally, antisocial personality disorder, or some related variant thereof, was explicitly addressed in 3 of the studies (11%). Examining this profile, it is logical to consider what was targeted for change in these studies. Generally, for BPD, it was depressed mood, which was followed by affective lability and transient psychotic symptoms or anxiety. In almost all cases for SPD, it was low-level psychotic symptoms, such as paranoia, ideas of reference, or odd communication. In the studies that examined antisocial personality disorder, impulsive and aggressive behaviours were targeted. The 5 classes of medication tested were neuroleptics, antidepressants, mood stabilizers, anticonvulsants, and anxiolytics.

Some differences in the trends of medications employed are noteworthy in pragmatic terms—that is, market availability. For

instance, closely following its introduction, Valium was tested for treating “borderline” pathology (55). More recently, for depressive symptoms, selective serotonin reuptake inhibitors (SSRI) medications have been the choice (71,74,75,77,80). Similarly, for the neuroleptics, we see the progression from the traditional neuroleptics (such as chlorpromazine and haloperidol) to more recent iterations of drugs (such as clozapine) that target psychotic-spectrum symptoms (56,65,79).

The variations in the methodological design of studies are important to note as well. The approaches range from single-group “open-label” trials to randomized double-blind crossover. Obviously, there is much heterogeneity of methodological rigor, and this must be factored in when weighing the evidence. Although an open-label trial is a reasonable procedure to secure exploratory findings, we would reserve the double-blind randomized clinical trial as a standard for declaring evidence of efficacy, and these studies are deserving of more attention as we attempt to sort out the evidence. Even these have potential limitations, however, because medication side effects can sometimes “break” the double-blind aspect. With these caveats plainly stated, we will briefly consider each of the 3 personality disorders studied.

Antisocial Personality Disorder. The evidence for efficacy of psychopharmacological approaches to the treatment of antisocial personality disorder is limited, not only by the number of studies, but also by diagnostic approaches. Two studies examined the effect of lithium on “aggressive prisoners” (61,62). In both of these studies, subjects were selected purely on the basis of their incarceration and histories of aggressive and assaultive behaviours. While these selection criteria certainly imply at least a degree of psychopathy, heterogeneous makeup in these samples is likely. Perhaps more accurately, this work examines the impact of behaviour that often accompanies the antisocial personality disorder diagnosis. In the Tupin study, subjects were not screened for a history of psychotic-spectrum illness, including the schizophrenia diagnosis, which was historically carried by at least 8 and possibly up to 12 of the 27 subjects (61). Furthermore, past clinical records indicated that only 12 of the 27 subjects had been designated “sociopathic.” Despite these limitations, including the open-trial design, a significant reduction in aggressive and assaultive behaviours, as operationalized by incident reports of such behaviour, was evidenced in the medicated subjects.

The study by Sheard and his colleagues was more rigorous from a diagnostic standpoint (subjects with a history of psychotic and organic illnesses were excluded) and by design (double-blind, randomized) (62). A significant reduction in aggressive behaviour was demonstrated in that study as well. The authors of that study, however, cautiously noted that 80% of the medicated subjects correctly guessed the active medication group status, which suggests that the double-blind may have been unblinded, possibly by side effects accompanying the medication. The final study, which examined the effectiveness of fluoxetine, produced equivocal results, and with only one subject with the antisocial personality disorder diagnosis, the findings are indecipherable (74). Overall, lithium is impressive in treating antisocial aggressive and assaultive behaviour and deserves more investigative attention than it is receiving.

Schizotypal Personality Disorder. The primary approach to the psychopharmacological treatment of SPD has been to target the low-level psychotic symptoms, such as odd and unusual thinking and ideas of reference. Accordingly, the major pharmacologic agents studied for this purpose have been neuroleptics. Several well-controlled studies demonstrated a moderate degree of efficacy that was primarily limited to the symptom realm (66,68,69). The study by Goldberg and colleagues further demonstrated that it was the more impaired subjects who tended to respond better to the medication (66). Consistent with these findings, less disturbed patients were found to be more reactive with side effects (67). These results generally support the careful and cautious employment of neuroleptics for patients suffering from SPD.

As with most of the pharmacologic studies, symptoms or symptom clusters are the dependent variables. The exception is a study of fluoxetine, which showed a reduction in symptomatic behaviours (such as self-injurious behaviours) in SPD and BPD subjects alike (74). Because this study was an open-label trial, results must be tempered and followed-up with more vigorous designs. Tricyclic antidepressants, however, do not appear warranted. A comprehensive study by Soloff and colleagues with a 4-year follow-up found that amitriptyline was less effective with patients exhibiting a schizotypal patterning of symptoms (68,69,86). This study further demonstrated that it was patients who presented with more severe schizotypal symptoms who responded best to neuroleptic medication, in this case, haloperidol. These results were impressive both for the methodological and diagnostic rigor of the project and particularly for the length of time for which it was carried out.

Borderline Personality Disorder. There are 4 pharmacologic treatment strategies for BPD. The first and second address the depression of mood and regulation of affect, respectively. Medications include antidepressants and mood stabilizers. The third

strategy is aimed at transient, psychotic-spectrum symptomatology with neuroleptics. Fourth, some attempts to medicate have included targeting impulsive and self-injurious behaviour with anticonvulsant medications. Interestingly, several of the studies in Table 2 have examined both BPD and SPD, and in these studies, the targets of treatment are the shared symptom domains enumerated above rather than the disorder.

As in the case of SPD, the findings for the use of neuroleptic medications with BPD suggest that this approach should be reserved for the most disturbed patients; that is, those who exhibit transient, stress-induced disorganization or dissociation. In fact, most of the studies from which the evidence of efficacy has been obtained included patients with a stronger component of the psychotic spectrum, whether stress-induced or more persistent (65,66,68,86). Specifically, these patients typically express some combination of the more severe symptoms of borderline and schizotypal pathology, but they do not belong clearly to one category or the other. This subgroup of patients expressing symptoms such as ideas of reference, paranoid ideation, or dissociative reactions to stress apparently respond to neuroleptic medication (66). Those patients who do not exhibit more severe (such as psychotic spectrum) symptoms do not appear to benefit and may suffer a negative impact stemming from neuroleptic side effects (67). Positive findings from an open label trial of an atypical neuroleptic (clozapine), however, deserve further exploration (79). We suspect that more conclusive findings as regards the diagnosis, and beyond the level of medicating symptoms, will require further study with the current diagnostic framework.

One of the more ambitious studies was carried out by Cowdry and Gardner (70). In a double-blind cross-over design, they examined the effects of alprazolam, carbamazepine, trifluoperazine, and tranylcypromine on outcome in BPD. Trifluoperazine seems to have a beneficial impact on behaviour and functioning; however, the results must be tempered because not all patients in this group completed the trial. Tranylcypromine had a positive impact on symptoms but not necessarily on symptomatic (impulsive) behaviour. Alprazolam appeared to initiate an increase in the severity of behavioural dyscontrol, a disinhibiting effect which is usual for the benzodiazepine medication class, as the authors note. When receiving carbamazepine, patients demonstrated a marked decrease in the severity of behavioural dyscontrol and evidenced some improvement in mood. It was described that, when receiving this medication, patients seemed to possess a greater ability to reflect on and, therefore, sometimes interrupt impulses before they translated into action. Interestingly, it was the physicians, not the patients, who noted improvement in mood, and this result was attributed to a "halo" effect on the part of the physicians. It might also be pointed out that patients were better able to tolerate negative affect without resorting to acting out, which suggests that this medication might be a useful adjunct to psychotherapy for those patients who exhibit difficulties controlling impulsive behaviours. Overall, the study usefully highlights the possible advantage of anticonvulsants for symptoms and symptomatic behaviours in BPD.

Finally, there are many studies that address the depressive symptomatology which accompanies BPD. Summing up, evidence for the efficacy of tricyclics is equivocal or negative. Evidence is stronger for the monoamine oxidase inhibitor (MAOI) antidepressants, although serious side effects limit their use to patients without significant self-destructive potential. The more recent studies that focus on the newly developed class of SSRIs are promising but not sophisticated. One randomized, double-blind study of fluoxetine is more equivocal, which suggests the need for many more trials (80).

Overview of Psychopharmacological Evidence. The pharmacologic studies are organized around symptom domains that are the intended target areas of the agent studied, and, to a great extent, these override diagnostic considerations. The evidence modestly suggests that neuroleptic medications can be used to target psychotic-spectrum symptoms and to reduce anxiety in comorbid SPD and BPD. The new atypical neuroleptic medications are clearly promising and require further testing. Selective serotonin reuptake inhibitor antidepressants show some effectiveness in treating depression in BPD, while lithium and anticonvulsants remain viable but generally unsung agents for the treatment of mood instability and behavioural dyscontrol.

Medication interventions target the symptom-based indicators of a personality disorder and not those indicators that are generally considered to be "personality traits." It might be speculated that impacting the accompanying symptoms of personality disorders over time will alter the course or expression of enduring "personality traits." This is a hypothesis in need of empirical testing, however. The majority of the pharmacologic studies are limited, not only by the dependent variables studied (primarily "symptoms" and not the syndrome level), but also by medication trials that, in most studies, last a matter of weeks. This is a very narrow scope of time given the enduring nature of these disorders and the snail's pace of changeability, which is suggested by the longitudinal and naturalistic studies and studies of comorbid Axis I and Axis II disorders

suggesting that the presence of an Axis II disorder, which suggest decreases treatment responsiveness.

Psychosocial Studies

An overview of the studies that have examined the psychosocial treatment outcome of personality disorders is shown in Table 3 (87–107). In general, 2 personality disorders, avoidant and borderline, have garnered the most empirical attention (Table 3). A final assemblage of studies looked at heterogeneous groups of individuals who displayed various personality disorders. We will consider each in turn.

Two strategies prevail for handling control groups in the experimental psychosocial studies. The first strategy is to compare 2 specific treatments or one treatment augmented by an additional intervention (99,101). This makes it difficult to interpret a lack of differences, however, which is a likely problem because this design by nature does not seek to maximize between group differences. The second, more optimal approach has been to compare the treatment of interest to treatment as usual. This approach is more likely to generate meaningful conclusions in the test of a specific treatment, as in the case of dialectical behavioural therapy (DBT), because the period of time can be extended to examine the longevity of change (94,95). This is in contrast to wait-list controls, where the idea of keeping an individual on a waiting list for a long enough time to examine change in personality disorders is often not ethical. Limitations of the treatment-as-usual approach include much within group variation (of the treatment as usual group), and that may introduce some problems in interpreting findings. In the long run, however, this represents a conservative control to the “active” treatment group, which suggests that demonstrated effects are much more compelling.

Avoidant Personality Disorder. The studies that examined avoidant personality disorder have included treatments which are mainly behavioural and targeted specifically at the social deficits which characterize that disorder. Most interventions have included some form of social-skills training (87–90). Some studies have employed behavioural modification interventions, which target specific avoidant behaviours, such as shyness (93). Methodologically, subjects were randomly assigned to treatment in the majority of studies. In many cases, there were multiple treatment groups. Some of the studies additionally included a wait-list control group (87,88,92), which appears to be the most viable analog of a placebo group in psychosocial clinical trials.

As is characteristic of most empirically based behavioural intervention studies, the dependent variables in the avoidant studies were closely linked to the interventions themselves. Anxiety was the focus for avoidant personality disorder at the symptom level. Slightly more specific to personality disorder, symptomatic *behaviour* was also examined in many of the studies. Usually, this took the form of shyness or social reticence. Finally, the outcome of social functioning included measures that tapped the quality of social-interpersonal relationships, which is the quintessential variable one might hope to impact in treating an individual who suffers from avoidant personality disorder.

Borderline Personality Disorder. The studies that examined BPD specifically comprised 7 of the 21 studies we reviewed (33%). Three of these studies had an “open” design and looked at treatments that were more broadly based (psychotherapy, day hospital, and inpatient treatment). The remaining 4 studies randomly assigned patients to varied treatment conditions. The least equivocal results came from the most well-controlled studies.

The most rigorous of psychosocial treatment outcome studies for BPD are those by Linehan (94,95). Using randomized trials in which patients received either DBT or “treatment as usual,” her results demonstrated improvements across the areas of symptoms, symptomatic behaviours, and global functioning for the patients who were in the DBT condition. The premise of DBT is to address symptomatic behaviours directly, such as parasuicidity, and to use systematic behavioural interventions to extinguish these behaviours. Principles derived from empirical studies in social psychology are sometimes employed to carry out these interventions and to maintain a therapeutic relationship. Elaborate contingency plans in the delivery of these interventions are part of the treatment manual, which is well developed. The extensive manual facilitates the possibility of conducting clinical trials, which is another advantage to this approach.

Results from Linehan’s seminal studies suggest that DBT is an efficacious psychosocial intervention. It was demonstrated that patients who received DBT engaged in fewer medically severe parasuicide attempts than did treatment-as-usual controls. Additionally, DBT subjects more consistently stayed in treatment and required fewer hospitalizations. Improvements in

symptomatic behaviour and global functioning persisted at the 2-year follow-up. The promising nature of these results, especially given the lasting quality, is suggestive of substantive change, and the DBT approach may prove to be a route to understand better the possibilities of long-term personality change, although more studies are needed. An investigation conducted by Springer and colleagues failed to demonstrate the superiority of DBT over treatment-as-usual in an inpatient treatment setting (107). Limitations in this study, however, included subject groups of varying personality disorders, in addition to borderline, and other modifications of the manner in which the DBT protocol was carried out. It may be that adherence to specific aspects of the DBT protocol are necessary for a successful outcome.

To examine specific hypotheses as regards how the prescribed treatment process of DBT leads to positive outcome, Shearin and Linehan used the short-form questionnaire version of the Structural Analysis of Social Behaviour (SASB) to test 4 key assumptions of the DBT model (96,108). The first assumption, that DBT requires a synthesis of opposites, was operationalized in SASB terms as therapist behaviour vacillating between instructing, controlling, and giving autonomy. A concrete example of this provided by the authors was how the therapist might deny a patient's request to be hospitalized after assessing it as inappropriate (therapist controlling), but providing the patient with instructions on how they might arrange to be hospitalized on their own (therapist giving autonomy). In SASB terms, the patterns of both control and autonomy-giving were evident in the weekly ratings made by patients and therapists. Other DBT assumptions included that the therapist be nonpejorative, provide modeling, and that the patient would perceive the therapist more warmly following reductions in symptomatic behaviours. The results of the SASB ratings also supported these remaining 3 hypotheses. By empirically examining the process, this study confirms some of the theoretically specified components of the DBT model (for example, complex communication and a nonpejorative therapeutic stance). Such findings may prove helpful to guide generalization of this approach to other settings.

Studies That Examined Personality Disorders in General. Winston and colleagues have developed protocols to carry out trials for specific models of brief dynamic psychotherapy (102,104). In one study, they examined the impact of 2 types of psychotherapy in "non-acting out" personality disorders (compulsive, avoidant, dependent, passive-aggressive, histrionic, or a mixture of these) (103). They found no difference between brief dynamic psychotherapy and behavioural therapy, although both therapy conditions did better than a wait-list control group. In a second similar study, they found that cluster C and some cluster B (that is, histrionic) personality disorders responded equally well to 2 types of psychotherapy and did clinically and statistically better than a wait-list control (104). These findings are not surprising because many prior psychotherapy studies have failed to demonstrate differential levels of effectiveness among treatments and because patients included in these studies were generally less disturbed and displayed a more neurotic variety of personality pathology. Nonetheless, this work represents an important foothold in the arena of psychotherapy trials with personality disorders, and, importantly, it focuses attention on the less prominent personality disorders.

Overview of the Psychosocial Evidence. Overall, the psychosocial treatment outcome studies demonstrate positive outcomes. Inspection of Table 3 shows some interesting trends. First, it appears that the psychosocial interventions generally have their most positive outcome on symptomatic behaviour, which is the area most directly targeted by the behavioural intervention employed. For symptoms, results are sometimes mixed. This may happen because the interventions are mainly directed at symptomatic behaviour rather than symptoms specifically. It may also happen because behavioural psychosocial treatments, in molding and changing behaviour, actually mobilize anxiety. Therefore, gains in symptomatic behaviours may occur with exacerbations in symptomatic dysphoria, at least during the period of change.

A consistent finding throughout, however, is that while patients improve, usually both in terms of clinical and statistical significance, they do not reach a level of "normalcy." While there are no clear criteria for personality disorder remission, the evidence from psychosocial studies suggests that some level of personality pathology persists. Finally, in contrast to the voluminous literature on the psychotherapeutic treatment of personality disorders, the relatively limited number of controlled trials for treatment outcome is striking. It has been argued that methodological approaches such as randomized clinical trials (RCT) limit the individual variation that typifies psychotherapy in practice, thus limiting their utility in providing ecologically valid evidence (109). This may be more generally reflected in psychotherapy trials in which differences between active treatments are more the exception than the rule when it comes to tests of psychosocial intervention. Conversely, when clinical trials are complemented with an empirical examination of process variables, as previously described in the case of the DBT, results that are pertinent to generalization can potentially be obtained.

Conclusion

There were several ways by which we attempted to organize, order, and classify the various investigations and the variables scrutinized within those studies. Some arbitrariness was inevitable. For example, how to classify a follow-up study that occurred years after the index treatment which lasted only several weeks? Is this really a test of a treatment, or does it perhaps best fit within the realm of naturalistic studies? Similarly, some might argue that there is a degree of arbitrariness among the domains of symptoms, symptomatic behaviour, and functioning. How can the distinctions be specified better, and how do they relate to the core of personality and, hence, personality disorder? More important for the question of treatment outcome, how does change in one domain affect the other domains? For lasting change to occur, is it best to start with symptoms or with behaviours? More appropriately, in which cases is it best to start with one or the other?

Clearly, there are several classes of dependent variables. While we are not firmly committed to these distinctions as we have formulated them, we do believe that further progress in the treatment of personality disorders will require more systematic specification of outcome variables as well as interrelationships between different domains of variables and to syndromal concepts. Furthermore, such domains occur defacto in the DSMs, where certain personality disorders are more symptom-based, while others reflect the extremes of dimensions of personality traits that can also exist within normal limits. Stone has made this point by noting that borderline, schizotypal, and antisocial personality disorders are symptom-based, while, in contrast, the remaining personality disorders are based on personality traits (110). In this light, it is not surprising that symptom-based personality disorders tend to occupy centre stage in the treatment outcome literature in general and even more so in pharmacology studies in which the focus is on treating symptoms. A related conceptualization is that personality traits are stable and become disorders when amplified (111).

What are we to conclude from the treatment outcome studies? In the pharmacology literature, there is a dearth of placebo-controlled studies for treating *symptomatic aspects* of personality disorders. With that said, there appears to be relative efficacy for symptoms targeted by specific classes of medication. Psychosocial interventions, however, have demonstrated significant reductions in symptomatic behaviours. As regards the syndrome versus symptom distinction, Soloff has aptly noted that one does not medicate a personality disorder but, rather, the associated symptoms (112). To a certain degree, this also holds true for psychosocial interventions in which behaviours are more the target for change than the syndrome. In both cases, it might be assumed that the underlying goal is to alter the syndrome, yet we presently have no evidence to assess progress on the latter. Of interest, and not yet answerable, is how affecting specific symptoms and symptomatic behaviours might impact the constellation of characteristics that we taxonomically identify as a personality disorder. Finally, the majority of treatment outcome studies were conducted in a relatively brief amount of time for what are considered to be enduring disorders. Given the enduring aspects of personality disorders, longer-term interventions would seem critical to impact personality disorders at the syndromal level.

We began this paper with 3 basic questions in mind. The first question concerned the changeability of personality disorders. The evidence from the naturalistic studies suggests quite clearly that, in the long-run, change does occur. As regards the nature of change, the answer to the second question appears to be that people become less symptomatic and reduce symptomatic behaviours but still do not quite make it to normalcy, or, if we were dealing with Axis I, would be termed full remission. For the third question, it does seem that treatments can help this process along substantially. More work is needed, however, to demonstrate the lastingness of treatments. Presently, the evidence suggests that there is good reason to proceed.

Clinical Implications

- Contrary to DSM assumptions about Axis II, treatment outcome of personality disorders is a valid concept.
- The majority of studies of many aspects of outcome suggest that treatment outcomes are positive.
- Dependent variables assessing treatment outcome can be systematically grouped by symptoms, symptomatic behaviours, and functioning.

Limitations

- Comparison of results across studies is difficult because of variations in dependent variables.
- Outcome variables are not well specified to syndrome, which suggests that remission has not been adequately considered.
- The majority of treatment outcome research focuses on BPD, which leaves other personality disorders less recognized.

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Résumé

Objectif : Examiner les résultats du traitement des troubles de la personnalité.

Méthode : Après avoir procédé au dépouillement de la documentation concernant les études sur les troubles de la personnalité, on n'a conservé que celles ayant principalement trait au deuxième niveau. Parmi ces études, on a distingué les études naturalistes de celles qui portaient spécifiquement sur les résultats de traitement. Ces dernières ont été examinées du point de vue de l'intervention, des variables dépendantes et des résultats du traitement.

Résultats : Contrairement aux hypothèses contemporaines sur le deuxième niveau, on a repéré un nombre important d'études portant sur les résultats de traitement. Les tendances au regard des hypothèses qui sous-tendent les méthodes psychosociales et pharmacologiques ont été cernées sous l'angle des variables dépendantes.

Conclusion : On a repéré des traitements efficaces pour soulager les symptômes et modifier les comportements symptomatiques qui accompagnent les troubles de la personnalité. On a examiné les répercussions de ces résultats sur la possibilité de rémission de tels troubles.

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