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THE DEVELOPMENT OF BORDERLINE PERSONALITY DISORDER— A MENTALIZING MODEL

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This paper describes a mentalization-based model of the development of borderline personality disorder (BPD). The model takes into account constitutional vulnerability and is rooted in attachment theory and its elaboration by contemporary developmental psychologists. The model suggests that disruption of the attachment relationship early in development in combination with later traumatic experiences in an attachment context interacts with neurobiological development. The combination leads to hyper-responsiveness of the attachment system which makes mentalizing, the capacity to make sense of ourselves and others in terms of mental states, unstable during emotional arousal. The emergence of earlier modes of psychological function at these times accounts for the symptoms of BPD. The model has clinical implications and suggests that the aim of treatment is not only to encourage development of mentalizing but also to facilitate its maintenance when the attachment system is stimulated.

It has become clear that the biological and psychosocial pathways to borderline personality disorder (BPD) are extremely complex. So far no model has been advanced that is able to integrate all the available data. Consideration has to be given to the role of genetics and constitutional vulnerabilities, neurophysiological dysfunctions of affect regulation and the stress response, evidence concerning the limbic system, executive control and frontal cortex dysfunction, psychosocial histories of childhood maltreatment and abuse found in a significant proportion of cases, and the disorganization of aspects of the affiliative behavioral system, most particularly the attachment system, found in almost all individuals with a diagnosis of BPD. Our suggestion is that these areas can be brought together within a model that focuses on the development of mentalization. We offer a selective review of evidence that is suggestive of the developmental model we propose and is at least consistent with our proposition. Such selective

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overviews of research have major limitations if considered to be of probative value. The aim of this paper is to illustrate our clinical model with evidence rather than claim that the evidence is in any sense "proof" of the approach we have taken.

Our premise is that unstable or reduced mentalizing capacity is a core feature of borderline personality disorder. This is not an insignificant proposal because it impacts directly on treatment—if a treatment is to be successful it must either have mentalization as its focus or at the very least stimulate development of mentalizing as an epiphenomenon.

MENTALIZING

Mentalization is the capacity to make sense of each other and ourselves, implicitly and explicitly, in terms of subjective states and mental processes. Understanding other people's behavior in terms of their likely thoughts, feelings, wishes, and desires is a major developmental achievement that, we believe, biologically originates in the context of the attachment relationship. Our understanding of others critically depends on whether as infants our own mental states were adequately understood by caring, attentive, nonthreatening adults. Consequently there is ample opportunity for the process to be disrupted.

The capacity to understand self and others as being guided by aims and intentions is considered to be a key developmental achievement and the disruption of this is seen to be a major aspect in the psychopathology of BPD. The most important cause of such disruption is psychological trauma early or late in childhood which undermines the capacity to think about mental states or the ability to give narrative accounts of one's past relationships. Even the capacity to identify the mental states associated with specific facial expressions may be impaired. This reduced capacity for mentalizing may be speculatively attributed to one or more of at least four processes: (1) the vulnerable child's defensive inhibition of the capacity to think about others' thoughts and feelings in the face of the experience of the genuine malevolent intent of others; (2) early excessive stress which distorts the functioning of arousal mechanisms, resulting in the inhibition of orbito-frontal cortical activity (arguably the location of one of the neural systems involved in mentalizing) at far lower levels of threat than would be normally the case; (3) the fact that any trauma arouses the attachment system, leading to an intensified search for attachment security and a deactivation of reflective capacity. Where the attachment relationship is itself traumatizing such arousal is exacerbated because, in seeking proximity to the traumatizing attachment figure, the child may be further traumatized. Such prolonged activation of the attachment system may have specific inhibitory consequences for mentalization; (4) the child, in "identifying with the aggressor" as a way of gaining illusory control over the abuser may internalize the intent of the aggressor in an alien (dissociated) part of the self. While this might offer temporary relief, the destructive intent of the

abuser will in this way come to be experienced from within rather than outside of the self, leading to unbearable self-hatred.

NEUROBIOLOGY OF MENTALIZING

Brain abnormalities identified in borderline patients are consistent with the suggestion that a failure of representation of self-states is a key dysfunction in BPD. Some evidence suggests that the anterior cingulate cortex plays a key role in mentalizing the self, at least in the domain of emotional states (Frith & Frith, 2003). Lane (2000) has proposed more specifically that implicit self-representations (i.e., phenomenal self-awareness) can be localized to the dorsal anterior cingulate, whereas explicit self-representations (i.e., reflection) can be localized to the rostral anterior cingulate. Activation of the medial prefrontal cortex has been demonstrated in a series of neuroimaging studies in conjunction with a wide range of mentalization inferences, in both visual and verbal domains (Gallagher et al., 2000). It appears that the prefrontal cortex is involved when mentalizing interactively in a way that requires implicitly representing the mental states of others. The mesial prefrontal cortex, the parieto-temporal junction, and the temporal poles constitute a network of areas that are invariably active when mentalizing activity is taking place (Gallagher & Frith, 2003). The same area of the brain is involved in other tasks which have been clinically described as challenging to patients with borderline problems, including assessing social trustworthiness (Winston, Strange, O'Doherty, & Dolan, 2002), interpreting the meaning of facial expressions (Critchley et al., 2000), making moral judgements (Greene & Haidt, 2002), and tasks that entail attending to one's own emotions (Gusnard, Akbudak, Shulman, & Raichle, 2001). It has been argued that exposure to stress impairs prefrontal cortical function and the impairment may be catecholamine mediated (Arnsten, 1998). In line with this suggestion is the observation that N-acetyl-aspartate (NAA), a marker of neural integrity, is lowered in the anterior cingulated region of the medial prefrontal cortex of maltreated children and adolescents (De Bellis, Keshavan, Spencer, & Hall, 2000).

GENETICS AND CONSTITUTIONAL VULNERABILITY

The results of twin studies show that the heritability of traits delineating personality disorder are 35%–56% (Jang, Livesley, Vernon, & Jackson, 1996). Livesley, Jang, & Vernon (1998) have suggested a four factor structure of the inherited components of BPD. The large first factor contained: general tendency towards labile affects, unstable cognitive functioning, unstable sense of self, and unstable interpersonal relationships. This strongly resembles the clinical picture of BPD. The fact that the genetic structure strongly resembles the phenotypic structure suggests that the pattern of traits in BPD is highly heritable. It has also been suggested that children who go on to develop BPD are partly vulnerable because they bring hard-to-manage temperaments to the parent-child relationship (De-

pue & Lenzenweger, 2001). In general, we believe that in addition to constitutional vulnerability, suboptimal environmental conditions at several developmental stages combine to create a vulnerability to BPD.

Current evidence suggests that genes have both main effects (Torgersen, 2000; White, Gunderson, Zanarini, & Hudson, 2003) and interactive effects with anomalous environmental influences (Caspi et al., 2002, 2003). A suggestive finding from the Dunedin study, which offers evidence on environmental risks while controlling for genetic influence by using only monozygotic twin pairs, indicated that compared with children of mothers with depression or antisocial PD, only the children of depressed *and* antisocial mothers had significantly higher levels of antisocial behavior and rates of DSM-IV conduct disorder, even after controlling for characteristics of maternal depression (Kim-Cohen, Caspi, Rutter, Tomas, & Moffitt, 2006). Given that antisocial PD is highly comorbid with BPD in women and that in this sample, women with antisocial traits also tended to have a history of suicidality, there is some reason to believe that the findings partially reflect maternal borderline traits. Experiences elevating the risk were factors such as multiple caregiving, abuse, including physical maltreatment, high levels of maternal hostility, and exposure to domestic violence.

FRAGILITY OF AFFECTIVE REGULATORY PROCESSES

Clinicians working with patients with BPD universally accept that failure of affect regulation is an important feature of the condition and this has been confirmed in numerous research studies (Sanislow et al., 2002; Zanarini, Frankenburg, Hennen, & Silk, 2003). Borderline patients experience more negative affect and negative experience has high salience for them. Importantly for a mentalizing model the acquisition of emotional regulation skills begins in infancy and continues through childhood and adolescence to adulthood. To achieve normal self-experience the infant needs his emotional signals to be accurately or contingently mirrored by an attachment figure. This is far more than acceptance and validation of the experience by a significant adult and must support the child's sense of agency as an initiating being (Ryan, 2005). Illusory control of the interaction generates a sense of agency and pleasure in the infant (Watson, 1984). Loss of contingency has temporary "catastrophic" consequences for self-organization and affect regulation (Braungart-Rieker, Garwood, Powers, & Wang, 2001; Ellsworth, Muir, & Hains, 1993; Haley & Stansbury, 2003; Weinburg & Tronick, 1994, 1996). In mirroring the infant, the caregiver must achieve more than contingency (in time, space, and emotional tone). The mirroring must be "marked" (e.g., exaggerated), in other words slightly distorted, if the infant is to understand the caregiver's display as part of her or his emotional experience rather than an expression of the caregiver's (Fonagy, Gergely, Jurist, & Target, 2002; Gergely, 2004). This will enable the infant to internalize the representation of the reflection of her or his experience and thus generate a representational system for internal states (a kind of social biofeedback system; Gergely & Watson, 1996).

There is evidence to suggest that the absence of marked contingent mirroring is associated with the later development of disorganized attachment (Gergely, Koós, & Watson, 2002; Koós & Gergely, 2001, 2001a). The disorganized pattern in infancy is marked by infants' incoherent and ineffective attempts to self-regulate upon reunion with their caregiver following a brief period of separation. Infants whose attachment is disorganized exhibit behaviors like freezing, rocking (dissociation), and self-harm (e.g., head-banging) following brief separation from the caregiver (Lyons-Ruth & Jacobovitz, 1999). They go on to develop oppositional, highly controlling behavioral tendencies in middle childhood (Green & Goldwyn, 2002; van Ijzendoorn, Scheungel, & Bakermanns-Kranenburg, 1999) and dissociative features in adolescence and adulthood. Given all these features it is not surprising that disorganized attachment patterns have been linked to BPD. But the pattern also appears to be connected to limited self-control as well as affect dysregulation (Kochanska, Coy, & Murray, 2001; Kochanska & Murray, 2000) which is a feature of BPD.

Neuroscientific studies highlight evidence of structural and functional deficit in brain areas central to affect regulation perhaps, in part, consequent on disrupted attachment patterns. Herpertz and colleagues (2001) have shown decreased amygdala volume and disrupted amygdala activity possibly mostly associated with processing of negative affect. Evidence of disrupted occipito-frontal cortex serotonergic functioning that may link to impulsivity has also emerged and neurotransmitters have been implicated in the development of BPD, in particular those believed to be involved in modulating affective states such as impulsive aggression which is an important component of the behavior disturbance found in BPD (Coccaro, Berman, Kavoussi, & Hauger, 1996). It seems that impulsiveness, autoaggression, and outwardly directed aggression are all associated with dysfunctions of the serotonergic system. Reduced serotonergic activity may inhibit a person's ability to modulate or control destructive urges although the causal pathway remains unclear.

ATTENTION AND SELF-CONTROL

Recent studies of attention have identified difficulties for borderline patients in inhibiting ideas that are peripheral to a set task. They remember more words that they are instructed to forget, especially when they are of emotional significance to them. In essence they have problems when doing things that require controlled processing of information, which might account for their impulsivity and erratic behavior. Attentional problems also imply that they may have difficulty when under stress in directing attention appropriately to their interpersonal and social context. Self-control is learned in part via the caretakers' regulatory activity and joint attention with the caregiver serves a self-organizing function. Fearon and Belsky (2004) have suggested that early attachment relationships are possible organizers of the attentional system and a positive relationship has been found between the quality of attachment and attentional performance.

Children with secure attachment appear to be protected from the effects of cumulative social risk when compared to insecurely attached children. In related work Kochanska, Aksan, and Carlson (2005) suggest that self-control and the internalization of a capacity to regulate is rooted in mutual responsiveness in mother-child dyads. High levels of responsive interaction between 26–41 months predicted greater self-control, the internalization of maternal rules, and a lessened need for maternal control and coercion. Children manifested more effortful control and were able to follow both do and don't commands better given mother-child mutually responsive orientation.

There is considerable accumulating evidence that self-control and the capacity to direct attention are linked (Posner & Rothbart, 1998, 2000). Clinical observation consistently indicates that patients with BPD have difficulty in inhibiting behavior and/or delaying responses; for example, suddenly walking out or making peremptory demands. Work on cortical localization of self-control invariably points to the pre-frontal cortex (Barkley, 1997; Metcalfe & Mischel, 1999). Brendel, Stern, and Silbersweig (2005) investigated the go/no-go task in the context of emotional states thereby identifying the function of the systems subserving the interaction of emotion and behavioral control. They detected dysfunction in the medial OFC providing further evidence that borderline patients have neural systems that do not successfully modulate behavioral responses when emotionally aroused. Patients with BPD have also been observed to make significantly more punishment-reward commission errors on a go/no-go task than healthy subjects (Leyton et al., 2001).

PET scan studies have shown that making choices between small likely rewards and large unlikely rewards entails activity in the right inferior and orbital pre-frontal cortex (Rogers et al., 1999). These pre-frontal regions are known to have rich inter-connections with limbic structures likely to be involved in drives, rewards, and motivation. As these structures are also well-connected to dorso-pre-frontal cortical areas that serve a broad range of cognitive processes independent of social or emotional salience, the orbital and inferior pre-frontal cortex may be ideally suited to coordinate the probabilities of outcomes with their emotional reward value. Further, and once again in line with neurotransmitter dysfunctions we have noted above, low serotonin activity is associated with impulsiveness (Linnoila & Virkkunen, 1992) while enhanced serotonergic activity appears to enhance delayed gratification (Bizot, Le Bihan, Puech, Hamon, & Thiebot, 1999).

INTEGRATION OF BIOLOGICAL AND CONSTITUTIONAL VULNERABILITY INTO A MENTALIZING DEVELOPMENTAL PERSPECTIVE

The mentalizing theory of borderline personality disorder is rooted in Bowlby's attachment theory and its elaboration by contemporary developmental psychologists whilst paying attention to constitutional vulnerabili-

ties. We have already indicated that there is suggestive evidence that borderline patients have a history of disorganized attachment which leads to problems in affect regulation, attention, and self-control. Building on the accumulating evidence from developmental psychopathology discussed above, the mentalization theory of borderline personality disorder suggests that individuals either constitutionally vulnerable and/or exposed to influences that undermine the development of cognitive capacities necessary for mentalization such as neglect in early relationships (Battle et al., 2004) where the contingency between their emotional experience and the caregiver's mirroring is noncongruent (Crandell, Patrick, & Hobson, 2003), develop with an enfeebled ability both to represent affect and effortfully control attentional capacity (Posner et al., 2002). Early trauma may also cause changes in the neural mechanisms of arousal leading to a relatively ready triggering of the arousal system underpinning posterior cortical activation in response to relatively mild emotional stimuli. This triggering simultaneously takes the frontal mentalizing parts of the brain "offline" (Arnsten, 1998).

Social cognitive capacities develop in the context of primary caregiving relationships and as such are relatively vulnerable to environmental disturbance exemplified by severe neglect, abuse, and other forms of maltreatment. The relevant attachment literature has recently been expertly reviewed by Levy. Nine studies have examined attachment patterns with patients diagnosed with BPD using the best available assessment of adult attachment, the Adult Attachment Interview; two further studies used rating scales and over a dozen used self-report measures. While the relationship of BPD diagnosis and specific attachment category is not obvious, there is little doubt that BPD is strongly associated with insecure attachment (6–8% of BPD patients are coded as secure) and there are indications of disorganization (unresolved attachment and cannot classify category of attachment) in interviews and fearful avoidant and preoccupied attachment in questionnaire studies (Levy, 2005). Summarizing across several studies, it appears that early attachment insecurity is a relatively stable characteristic of the individual, particularly in conjunction with subsequent negative life events (94%; Hamilton, 2000; Waters, Merrick, Treboux, Crowell, & Albersheim, 2000; Weinfield, Sroufe, & Egeland, 2000). Given evidence of the continuity of attachment from early childhood at least in adverse environments and the two longitudinal studies following children from infancy to early adulthood which reported associations between insecure attachment in early adulthood and BPD symptoms, the extent to which childhood attachment may affect mentalization may be relevant to the development of BPD. The quality of children's primary attachment relationship has been shown by a number of studies to predict mentalizing ability (e.g., Fonagy & Target, 1997; Harris, 1999; Meins, Fernyhough, Russell, & Clark-Carter, 1998; Ontai & Thompson, 2002; Raikes & Thompson, 2006; Steele, Steele, Croft, & Fonagy, 1999; Symons, 2004; Thompson, 2000) although a link with emotional understanding aspects

of mentalizing rather than theory of mind components is more consistent. Overall it seems likely that this relationship is mediated within a family via the coherence and mentalizing nature of the general discourse in the home (e.g., Dunn, 1996; Dunn, Brown, Somkowski, Telsa, & Youngblade, 1991; Nelson, 2005; Ruffman, Slade, & Crowe, 2002).

FAMILY FACTORS

Family studies have identified a number of factors that may be important in the development of BPD; for example, a history of mood disorders and substance misuse, but few of the studies point to the specific features of parenting that create a vulnerability for borderline PD. Classically, parents of patients with BPD were considered to be over-involved and over-protective in the patient's welfare. But Gunderson, Kerr, and Englund (1980) failed to identify over-involvement in families of patients with BPD and suggested that a more common pattern was for the parents to be involved with one another to the exclusion of their children. More recent evidence suggests that neglect, emotional under-involvement, and invalidation by caretakers are important. Prospective studies in children have shown that parental emotional under-involvement contributes to difficulties in socializing and perhaps with risk for suicide attempts (Johnson et al., 2002). Most of the findings suggest that BPD individuals (at least while symptomatic) see their relationships with their mothers as conflictual, distant, or overprotective; their fathers as less involved and more distant suggesting that problems with both parents are more likely to be the common pathogenic influence in this group than problems with either parent alone. Whilst these findings should be replicated with recovered patients, the general point about biparental difficulties is given further support from studies of abuse.

Physical, sexual, and emotional abuse all occur in a family context and high rates are reported in BPD. Zanarini reported that 84% of borderline patients retrospectively reported experience of biparental neglect and emotional abuse before the age of 18 with emotional denial by the caretakers of their experiences being a predictor of BPD (Zanarini, Frankenburg, Reich, Marino, Lewis, Williams et al., 2000) suggesting that these parents were unable to take the experience of the child into account in the context of family interactions. Overall researchers have concluded that abuse alone is neither necessary nor sufficient for the development of BPD and that predisposing factors and contextual features of the parent-child relationship are likely to be mediating factors in its actual development. Parental responses play an important role in the pathogenetic effects of abuse with parental responsiveness (believing the reports, protecting, and not expressing high levels of anger) following reports of abuse promoting more rapid adjustment (Everyson, Hunter, & Runyon, 1989) and lack of emotional responsiveness, low support, and inadequate validation possibly potentiating the effects. Thus caregiver response to the abuse may be more

important than the abuse itself in long-term outcome (Horwitz, Widom, McLaughlin, & White, 2001).

The contextual determinants for the sequelae of maltreatment resonates with findings suggesting that emotional discussion in family interactions may be a key factor in determining capacity to understand mental states. Maternal disciplinary style (Ruffman, Perner, & Parkin, 1999; Vinden, 2001) and the inclination of mothers to take the psychological perspective of their child, including maternal mind-mindedness and reflective function in interacting with or describing their child influences the development of emotion understanding and ToM (Fonagy, Steele, Steele, & Holder, 1997; Fonagy & Target, 1997; Meins et al., 2002, 2003; Peterson & Slaughter, 2003; Sharp, Fonagy, & Goodyear, 2006; Slade, 2005). Other relevant features of the emotional climate within the family (e.g., Cassidy, Parke, Butkovsky, & Braungart, 1992) are likely to be disrupted by maltreatment, especially the child's opportunity to suspend reality and freely play, which also greatly facilitates the emergence of mentalization (Harris, de Rosnay, & Pons, 2005; Jenkins & Astington, 2000; Youngblade & Dunn, 1995). Children who frequently engage in pretence score high on false belief tests (Taylor et al., 1998).

The New York Children in the Community Study (Johnson, Cohen, Brown, Smailes, & Bernstein, 1999) included 738 youths recruited from upstate New York and assessed repeatedly between 1975 and 1993. It reported that childhood abuse substantially increased the risk of cluster B personality disorder in general and borderline personality disorder in particular. In a follow-up report to this study (Johnson, Smailes, Cohen, Brown, & Bernstein, 2000), these researchers demonstrated that emotional, physical, and supervision neglect were all associated with increased risk for PDs. The emphasis on neglect is consistent with the higher prevalence of neglect compared to either physical or sexual abuse but has been relatively ignored by aetiological theories. It appears that supervision neglect is particularly likely to be associated with borderline, paranoid, and passive-aggressive PDs. Interestingly, cognitive neglect was not associated with any PD symptoms. Supervision neglect includes items such as allowing the child to go out as he or she pleases, being tolerant of the child using cannabis, and so on. The prevalence of supervision neglect among parents of any cluster BPD was 30% and the odds ratio for BPD was 7.3. The contribution of neglect remained significant after abuse was controlled for.

There is now evidence to suggest that early attachment trauma and neglect undermines the development of the capacity to think about mental states although much of this is indirect. Maltreated children are unable to show empathic responses to other children in distress, exhibit emotionally dysregulated behavior, and make proportionately fewer references to internal states. Maltreating mother-child dyads discuss emotions less frequently than nonmaltreating dyads and it is in the area of emotional understanding of self and others that there is the strongest evidence for the

adverse impact of maltreatment. Delayed emotional understanding occurs in maltreated children although this difference is often reduced when groups are well matched socially and intellectually. However, the delay may be linked to specific social deficits in these children indicating the centrality of mentalizing as a key component of social functioning. Delayed theory-of-mind understanding has also been reported in maltreated children but, again, it remains unclear whether the problems result from maltreatment itself or whether they are a function of broader intellectual delays.

The MBT model suggests that individuals with BPD, while able to mentalize, are more likely to abandon the capacity under high emotional arousal; for example, in response to maltreatment, because mentalization was not well established during the first decade of life, in part, as a consequence of early maltreatment and its associated problems. In two studies we were able to demonstrate the relationship of low mentalization, maltreatment, and borderline diagnosis. In a sample of 86 individuals with PD diagnosis we found that 97% of patients who had a history of maltreatment and low reflective function met criteria for BPD (Fonagy et al., 1996). In a more recent study, we found that of 3 groups of patients matched for gender, age, education, and Axis-I diagnoses, those who also met criteria for BPD scored lower on Baron-Cohen's Reading the Mind in the Eyes Test (Baron-Cohen & Cross, 1992; Baron-Cohen, Jolliffe, Mortimore, & Robertson, 1997) than a group without Axis-II diagnoses or a group with Axis-II but not cluster B diagnoses (Fonagy, Stein, Allen, & Vrouva, submitted). Structural equation modelling (SEM) revealed that those with a history of adversity who had low scores on the Eyes task were also more likely to be the ones with BPD diagnoses. The SEQ model in which low mentalizing score was placed in the model as an outcome of BPD diagnosis fitted far more poorly suggesting that low mentalizing is consequent on maltreatment history but not all those with low mentalizing associated with maltreatment acquire a BPD diagnosis.

Taking all this into account, the mentalization-based approach predicts that it is not the fact of maltreatment but more the family environment that discourages coherent discourse concerning mental states and it is this that is likely to predispose the child to BPD. Studies that have examined the family context of childhood trauma in BPD tend to see the unstable, nonnurturing family environment as the key social mediator of abuse (Bradley, Jenei, & Westen, 2005) and under-involvement the best predictor of suicide (Johnson et al., 2002) and personality dysfunction (Zweig-Frank & Paris, 1991). In our formulation, we consider parental emotional under-involvement with children as most likely to impair the appropriate development of social cognition. In the MBT model, we do not attribute a central role to trauma, but we expect that in individuals made vulnerable by early inadequate mirroring and disorganized attachment to highly stressful psychosocial experiences in an attachment context, trauma will play a key role in shaping the pathology of BPD and will contribute to

directly causing it by undermining the capacity for mentalization. We consider that the impact of trauma is most likely to be felt as part of a more general failure of consideration of the child's perspective through neglect, rejection, excessive control, unsupportive relationship, incoherence, and confusion. These can devastate the experiential world of the developing child and leave deep scars which are evident in their social-cognitive functioning and behavior.

This aspect of our formulation therefore converges with that advanced by Marsha Linehan concerning the assumption of invalidating family environments and creatively developed further by Fruzzetti, Shenk, and Hoffman (2005) and Fruzzetti, Shenk, Lowry, and Mosco (2003). These workers report that parental invalidation, in part defined as the undermining of self-perceptions of internal states, was not only associated with the young person's reports of family distress, their own distress, and psychological problems but also with aspects of social cognition, namely their ability to identify and label emotion. Along with other aspects contributing to the complex interaction described as invalidating, this amounts to a systematic undermining of a person's experience of their own mind by the replacement of their mind with another or a failure to encourage discrimination between their own feelings and experiences and those of the caregiver.

MENTALIZING, PHENOMENOLOGY OF BPD, AND SOME CLINICAL CONSEQUENCES

The phenomenology of BPD is the consequence of this inhibition of mentalization, and of the reemergence of modes of experiencing internal reality that antedate the development of mentalization. In addition, there are background indications of the disruption of self-organization manifesting as the constant tendency to reexternalize the self-destructive poorly integrated, alien (nonself) experiences within the self (what we believe psychodynamic clinicians have long recognized as "projective identification"). Individuals with borderline personality disorder are "normal" mentalizers except in the context of attachment relationships but they tend to misread minds, both their own and those of others, when in intense interpersonal encounters, often when emotionally aroused. As a relationship with another moves into the sphere of attachment the ability to think about the mental state of the other can rapidly disappear. When this happens, prementalistic modes of organizing subjectivity emerge, which have the power to disorganize these relationships and destroy the coherence of self-experience that normal mentalization sustains through narrative.

As a consequence, mentalization gives way to prementalistic ways of representing subjectivity: (1) psychic equivalence, (normally described by clinicians as concreteness of thought) in which alternative perspectives cannot be considered; there is no experience of "as if" and everything appears to be "for real." This can add drama as well as risk to interpersonal experi-

ence and the exaggerated reaction of patients is justified by the seriousness with which they suddenly experience their own and others' thoughts and feelings. (2) pretend mode in which, conversely, thoughts and feelings can come to be almost dissociated to the point of near meaninglessness. In these states patients can discuss experiences without contextualizing them in any kind of physical or material reality. Attempting psychotherapy with patients who are in this mode can lead the therapist to lengthy but inconsequential discussions of internal experience that have no link to genuine experience. (3) Finally, early modes of conceptualizing action in terms of that which is apparent can come to dominate motivation. Within this mode there is a primacy of the physical; experience is only felt to be valid when its consequences are apparent to all. Affection, for example, is only "real" when accompanied by physical expression.

A particularly disruptive feature of borderline cognition is the apparently unstoppable tendency to create unacceptable experience within the other through the externalization of the abuser which has been internalized by the traumatized individual as an alien part of the self. This can create a terrified alien self in the other—therapist, friend, parent—who becomes the vehicle for what is emotionally unbearable. Moreover, the need for this other can become overwhelming and an adhesive, addictive pseudo-attachment to this individual may develop. The alternative to such projective identification is to attack or destroy the self by self-harm and suicide.

THERAPEUTIC IMPLICATIONS

Borderline patients are uniquely vulnerable to therapist interventions (Fonagy & Bateman, 2006). Patients can easily be thrown in to pretend mode in which they take on the perspective of the therapist and use it as part of themselves or alternatively are precipitated into confusion as their mentalizing capacities collapse. A complex formulation too early in therapy runs the risk of inducing pretend mode in vulnerable borderline patients and we would suggest therapists need to be alert to such problems, which can be difficult to identify. MBT tries to formulate a process rather than actual relationship patterns in an attempt to reduce the risk of inducing pretend mode.

As a result of some of these dangers with borderline patients, some therapy techniques are actively avoided in MBT (Bateman & Fonagy, 2006). First, we suggest that therapists avoid allowing excessive free association, a technique possibly more useful for neurotic patients. Second, we do not encourage active fantasy about the therapist. The use of fantasy and free association is not a major aspect of MBT because the development of insight is not a primary aim of MBT. Working with fantasy is a technique used in insight-orientated therapy as a way of understanding unconscious thinking. MBT is more concerned with preconscious and conscious aspects of mental function within the interpersonal domain. Fantasy itself is

too distant from reality and we do not, therefore, encourage elaboration of the patient's fantasies about the therapist because it is likely to be iatrogenic and to invoke pretend mode rather than increase elaborated representations linked to reality. Alternatively, fantasy experienced in psychic equivalence mode becomes reality and is experienced as real, losing its "as if" quality. Third, recognizing that patients operate in psychic equivalence mode also implies that their understanding is characterized by a conviction of being right. This makes entering into Socratic debates mostly unhelpful. Fourth, patients commonly assume that they know what the therapist is thinking. This is to be accepted initially. Problems for the therapist will arise if he claims primacy for introspection; i.e., saying that he knows his own mind better than the patient. This will lead to fruitless debate. Finally, in contrast to many therapies which actively withhold self-disclosure, we suggest that tactful disclosure about what you are feeling is essential if the patient is to discover his own state of mind by contrasting it with yours. In addition, explicit statements about the therapist's experience or contribution to any interaction in a manner that furthers the joint understanding of the relationship are necessary to ensure clear "marking."

MBT has concerns that too much identification of relationship patterns might reduce the development of the patient's ability to seek his own understanding and that patterns tend not to be exclusive to the group We focus on the mental resources that are available to deal with recurrent patterns of behavior and relationships rather than identification of the patterns themselves. This emphasis is nontrivial clinically. Mentalizing therapists do not get involved in discussing the structure or nature of the relationship that the patient brings, but focus more on the patient's capacity to think about the relationship. For example, the MBT therapist addresses the rigidity of schematic representations or roles rather than the roles or schemas themselves; the MBT therapist tries to enhance and facilitate flexibility and generate alternative perspectives. We suspect that this process may be one effective component of a number of psychotherapeutic approaches—while ostensibly focusing more on teasing out the actual roles, it is the action of teasing out rather than the understanding that the patient arrives at as a consequence of the work that is crucial. From a mentalizing perspective the problem lies in the capacity to process roles and experiences when they are activated within specific contexts.

A mentalizing understanding of BPD is possibly distinct, theoretically, from a number of other approaches. Yet in clinical practice, mentalizing may be an aspect of psychotherapy that unifies numerous effective approaches to the treatment of this challenging group of patients. Perhaps one of our primary distinctions is the organization of treatment and the training of therapists to ensure a felicitous context for a focus on mentalizing itself and our recommendation to concentrate on techniques that enhance mentalization whilst avoiding interventions that might reduce the patient's fragile capacities.

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