

The WAGER Vol. 7(44) - The Role of Parental Bonding in Problem Gambling

October 30, 2002

Research suggests that parental bonding can influence a child's development and might influence risk for the development of psychopathology in adulthood (Bowlby, 1977). Results from the National Comorbidity Study suggest that measures of parental bonding, including lack of care and overprotection, are associated with the subsequent development of a variety of mental disorders in adulthood (Enns, Cox, & Clara, 2002). In a recent study, Grant and Kim explored the relationship between parental bonding and pathological gambling (PG); their research is the focus of this week's WAGER (Kim and Grant, 2002).

Kim and Grant recruited 33 DSM-IV identified pathological gamblers (M = 19, F = 14, age range = 27-72 years, mean age = 46.8) from a sixteen week outpatient paroxetine treatment study of PG to complete a parental bonding questionnaire, the Parental Bonding Instrument (PBI; Parker, Tupling, & Brown, 1979).¹

It is unclear whether the paroxetine study had more than 33 participants who met the inclusion criteria; this precludes the calculation of a response rate for the parental bonding study. The PBI measures individuals' perceived parental rearing practices up to the age of 16 and requires participants to score several parental behaviors and attitudes on a four point scale. The PBI items represent two dimensions of parental behavior: parental care (expression of affection) and parental protection (encouragement or suppression of the child's exploration of the environment). The PBI is best understood through the interaction of its two dimensions of parental behavior. Examining either dimension independently could easily lead to erroneous conclusions about quality and parenting style. For example, while common sense suggests that higher parental care scores are always beneficial, the degree of benefit depends on parental protection scores; the PBI suggests that when a high level of care is coupled with overprotection, an "affectionate constraint" parental style results. One might imagine a child feeling smothered by a parent who reportedly loves them so much that they are not allowed to do anything. The PBI suggests that high parental caring scores coupled with low parental protections scores result in an "optimal parenting" style

(Ainsworth, Blechar, Waters, & Wall, 1978; Mak, 1994).

Table 1 presents the distribution of parental patterns among PGs in this study. The largest group of pathological gamblers (39-43%) reported neglectful parenting; far fewer reported optimal parenting. Studies that have included normal controls have found rates of optimal bonding between 40-60% (Leon & Leon, 1990; Torgerson & Alnaes, 1992); Kim and Grant found the rate of optimal bonding for pathological gamblers to be only 17-30%.

Table 1. Parenting Patterns in Pathological Gamblers (N=33)

	High Protection	Low Protection
High Care	Affectionate Constraint Maternal: 3.0% Paternal: 13.3%	Optimal Parenting Maternal: 30.3% Paternal: 16.7%
Low Care	Affectionless Control Maternal: 27.3% Paternal: 26.7%	Neglectful Parenting Maternal: 39.4% Paternal: 43.3%

Unfortunately, direct comparisons of the parental patterns of PGs to the general population are not possible because Grant and Kim did not recruit or test control subjects. The results described in Table 1 would be more informative if we were able to ascertain how, or even if, these parental patterns are significantly different from the general population. Additionally, Grant and Kim did not report response rates; we therefore do not have information on how many patients declined to be involved. This small sample of treated PGs might not have been representative of the population from which they were drawn. Further, the experimental design precludes any statements of causation between parental style and pathological gambling and cannot rule out the possibility that other factors may contribute simultaneously to the inadequacy of parental bonding and to the psychopathology of the child; the relationship between parental bonding and psychopathology may be spurious. For example, parental bonding and offspring psychopathology might better relate to shared genetic mental health vulnerabilities or to environmental factors than to each other. A final caveat to the study is the possible inaccuracy of self-report, particularly dating back to ones' childhood. Memory can be poor and the life experiences since childhood could color individuals' perceptions.²

Nevertheless, Grant and Kim improve our understanding of the etiology of PG and utilize a unique approach to the study of this topic. Importantly, they encourage us to examine factors throughout the life-course that may contribute to PG. Further research is needed to firmly establish whether parental bonding is a

casual factor in PG and, if so, through what mechanisms parental bonding influences the development of PG and other mental health disorders.

Comments on this article can be addressed to Rachel Kidman.

Notes

1. Participants were excluded from the study if they had other Axis I disorders, recent substance abuse histories, or severe personality disorders.

2. It is worth noting that the way people remember their childhood might be important even if it is inaccurate.

References

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