Mothers of Children with Inflammatory Bowel Disease: A Controlled Study of Adult Attachment Classifications and Patterns of Psychopathology

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ABSTRACT

We inquire into parental correlates of illness expression in three pediatric diagnoses: Inflammatory Bowel Disease, cancer and renal disease. Children with cancer and renal disease were the comparison groups, using valid pediatric measures for comparison across diagnostic categories in chronic illness. We found compromised parental support in families with IBD children, comparing relations among child’s medical adjustment, parental attachment and psychopathology profiles. Higher rates of insecure attachment are found in mothers of children with IBD; these mothers exhibit increased psychiatric symptoms. The results emphasize: 1) supporting the parent-child relationship, 2) parental well-being, and 3) possible precipitants of gene-regulated onset of IBD contributing to illness severity and course. We offer a theoretical model considering four factors for IBD gene-regulated onset. This preliminary study should encourage longitudinal studies of attachment in chronic illness, particularly IBD.

Ulcerative colitis (UC) and Crohn’s Disease (CD), together known as Inflammatory Bowel Disease (IBD), are painful and debilitating conditions characterized by chronic intestinal irritation. Symptoms include persistent diarrhea, nausea, vomiting, low grade fever, joint pain, weight loss, and incapacitating abdominal pain (1, 2). IBD often presents in adolescence (3, 4) and symptom profiles range from mild to severe, with a clinical course ranging from episodic to nearly continuous.

Familial tendencies suggest a genetic component to IBD, such as an increased incidence of IBD in the first degree relatives of probands (5-7). There is approximately 75-80% same-disease concordance within a family; concordance rate in monozygotic twins is higher for Crohn’s Disease than Ulcerative Colitis (8-10). Family genome-screening studies have identified links to IBD on specific chromosomal regions; some regions are disease specific (11-14). The IBD2 locus on chromosome 12 has been identified as the sole UC-specific region identified to date (7). A major breakthrough came with the detection of NOD2 (now called CARD15), the first CD-specific susceptibility gene (14).

Although genetics play a key role in IBD onset, researchers have elaborated on the concomitant influence of environmental factors, including regulation of gene onset. Environmental influences affect the severity, course, and possible genomic expression of structural genes (15-18). Laharie et al. (19) investigated conjugal cases of IBD and the likelihood of IBD in the offspring of these couples. The authors divided the couples into three groups: 1) both couples had IBD symptoms before living together, 2) one spouse had symptoms before and one

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**Repeated measures analysis of variance was not employed because many of the MCMI-II scales are comprised of partially overlapping sets of items, and are thus not independent. This violates a basic assumption of repeated measures ANOVA, and renders participants with an elevated score on one scale more likely to obtain elevated scores on additional scales. Readers are encouraged to keep this in mind when interpreting the presented pattern of results.

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spouse had symptoms after living together, and 3) both spouses developed symptoms after living together. The number of offspring in the third group was significantly greater than would be found by chance. This finding supports the role of environmental factors in IBD etiology, as well as possible assortative mating based on personality and/or attachment factors underlying the physical manifestations of IBD. Other environmental factors such as acute daily stress are significantly correlated with symptoms (20). Szajnberg et al. (18) reported severe stress in the one year prior to IBD illness onset in children.

Strained or broken relationships with loved ones are particularly potent psychosocial determinant of symptom severity, relapse, and perhaps illness onset. Engel (21) reported that real or imagined loss of a loved one frequently precedes the development of ulcerative colitis. In an extension of this finding, Szajnberg et al. (18) found insecure adult attachment classifications in 78% of the mothers of children with newly diagnosed IBD, as assessed by the Adult Attachment Interview (22). This figure is more than 50% higher than in normative samples (23). Insecurely attached mothers are less supportive and either affectively aloof or enmeshed than securely attached mothers (24-26); this suggests that an unsupportive family environment places children with IBD at risk for more severe episodes of illness. Moreover, children of insecurely attached mothers are more negative and angry, as well as less enthusiastic (25, 26), and more likely to be insecurely attached (e.g., 26). These characteristics may render children more vulnerable to separation anxiety and depression (27, 28), and less able to utilize family members in order to cope with a debilitating illness such as IBD.

A brief note about adult attachment classification. This interview has been used and validated across thousands of adults in several cultures, including Israel. The interview results in classification as Securely attached (approximately 65% of a population), and two types of Insecurely attached – Dismissive/Defended and Angry/Enmeshed (approximately 35% of a population). There is a very infrequent form of Unresolved “mourning” classification found within one year of the death of a loved one, or after severe trauma. Consistent with the theory of attachment, both Securely and Insecurely attached individuals have working models of achieving and maintaining closeness; attachment theorists have avoided calling Insecurely attached individuals as “abnormal,” reserving such a term for those rare individuals who have no successful working models of attachment. Nevertheless, those who are Insecurely attached have greater anxiety about achieving/maintaining closeness and therefore may be at-risk for difficulties.

This current controlled study inquires into the parental correlates of illness expression. It clarifies and extends findings suggesting compromised parental support in families with IBD children. Thus, the relations between child adjustment, parental attachment status, and parental psychopathology profiles were contrasted in families of children with IBD, and in families of children with either cancer or renal disease.

This design intends to address two shortcomings in the literature on IBD. First, a control group was employed to address the possibility that raising a child with a chronic, possibly life-threatening disease may have caused the high rate of insecure attachment classifications of mothers of IBD children. Second, indices of maternal psychopathology were included in addition to Adult Attachment Interview assessments, to clarify potential parental contributions to child adjustment and illness expression.

We hypothesize a four-factor multigenomic model from which researchers and physicians may work in order to understand the onset and potential exacerbation of IBD symptoms.

METHODS

PARTICIPANTS

Child-onset IBD patients and chronically ill controls within two years of illness diagnosis were recruited from the Department of Pediatrics of a major Midwest hospital. All recruited families agreed to participate. This represents a complete population study at this Children’s Hospital, the only major pediatric hospital in the area. Therefore, the sample represents all or nearly all hospitalized pediatric IBD patients within the community. A total of 21 IBD patients (16 with Crohn’s Disease, 5 with Ulcerative Colitis) and 20 age-matched controls (14 with cancer and 6 with hemodialysis-dependent renal disease) were included. Because of the measures (below) designed by Stein and Jessop, researchers are now able to compare disease course and psychiatric/psychological status across pediatric diagnostic categories. Cancer and Renal disease case were selected as they are serious chronic illnesses, like IBD, and are prevalent in our pediatric regional hospital. Control children were selected for comparable age, gender, length of illness, family size, and socio-economic status. The sample was primarily Caucasian, with one African American family in the IBD group. Descriptive characteristics of the
sample are presented in Table 1. There were no significant differences on any demographic measures.

<table>
<thead>
<tr>
<th>Table 1. Descriptive Characteristics of Sample by Group</th>
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<tr>
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<tr>
<td>Child’s age at assessment</td>
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<tr>
<td>Duration of illness (months)</td>
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<tr>
<td>Child’s KBIT* IQ</td>
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<tr>
<td>Mother’s age at assessment</td>
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<td>Family income (thousands)</td>
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*Inflammatory Bowel Disease. *Kaufman Brief Intelligence Test.

**PROCEDURES**

One interviewer conducted the adult interviews, while a second interviewer conducted the child interviews. Both interviewers were blind to the diagnostic status of participants, although some of the mothers did reveal the child’s diagnosis during the course of the interview.

**PARENT MEASURES**

*Adult Attachment Interview (AAI): (22).* The primary caretaker of the child (the mother in all cases) completed the AAI, a one to two hour semi-structured interview that assesses the respondent’s state of mind regarding their childhood attachment experiences and current attitude towards significant others, including their child. Participants are asked to describe in detail their childhood relationships with parents, to elaborate on experiences of separation, feelings of rejection, and significant losses throughout their lives. They are asked to outline their current relationships with parents, offer interpretations of their parents’ behavior when the respondent was a child, elaborate on the impact of their childhood experiences on their current functioning, and elaborate on their concerns and behaviors in parenting their own children. Interviews were audio taped, then transcribed and edited with all references to pediatric diagnosis removed. Transcriptions were scored by a trained rater blind to the child’s diagnostic status.

Based on interview transcripts, adult attachment classifications are derived from rater-coded childhood experiences of parental warmth, rejection, neglect, pressure to achieve, and parent-child role reversal (29). The respondents’ current state of mind regarding their attachment experiences is then rated on several dimensions, including: coherency, idealization of parents, inability to recall parental behaviors, speech passivity, and angry preoccupation with parents. Based on their current state of mind regarding childhood experiences with parents and attachment, respondents are then classified as either Secure, or Insecure Dismissive, or Insecure Preoccupied with respect to attachment (29).

**SECURE** AAI classifications are rated when: descriptions of childhood events are coherent and they acknowledge importance of early experiences on development and current functioning. **INSECURE Dismissive** AAI classifications are rated when: descriptions of childhood events are incoherent, when they exhibit idealization of parents or difficulty recalling attachment-relevant experiences, and when they de-emphasize the importance of these experiences on current functioning. **INSECURE Preoccupied** AAI classifications are rated when: descriptions of childhood events are incoherent, but differ from dismissing individuals by either expressing ambivalence or passivity regarding their relationships with parents or expressing continued anger toward parents.

**Impact on the Family Scale.** The IOF (30) is a 24-item parent-report checklist that assesses the impact of children’s illness on family functioning. The scale yields four factors, including Financial (e.g., the illness is causing financial problems for the family), Social/Familial (e.g., we see family and friends less because of the illness), Personal Strain (e.g., nobody understands the burden I carry), and Mastery (e.g., learning to manage my child’s illness has made me feel better about myself). Internal consistency is acceptable for all factors (30). This scale is the counterpart to the Clinician’s Objective Burden of Illness Scale (COBI: see below).

*Millon Clinical Multi-Axial Inventory-II.* Mothers completed the MCMII-II (31), a 175-item inventory that yields continuous scores on 20 clinical scales, most of which correspond to Axis I and Axis II disorders represented in the Diagnostic and Statistical Manual of Mental Disorders (32). While formal diagnoses should not be rendered based on elevated scale scores in the absence of corroborating behavioral evidence of psychopathology (33), the MCMII can help formulate hypotheses about symptoms (Axis I), and personality traits (Axis II).

**PHYSICIAN MEASURES**

*Clinician’s Objective Burden of Illness Index.* The COBI (34) is a 44-item questionnaire designed to compare the severity of physical illness across diagnostic categories. Questions address the degree of symptom severity in areas such as seizures, bladder and bowel control, need for surgery, prognosis, age-appropriate self-care, need for medication, route of administration, need for wound
care, need for physical therapy, etc. Scores correlate with need for hospital care and the frequency of urgent care. The instrument was included in order to obtain an independent estimate of illness severity among IBD, cancer and renal patients.

**CHILD MEASURES**

*Kaufman Brief Intelligence Test.* The K-BIT (35) is an abbreviated intelligence test that was administered to rule out disparities in IQ as an alternative explanation for potential group differences in illness expression. The K-BIT requires roughly 30 minutes to administer and score. Test-retest reliability coefficients for Composite IQ scores averaged .94 in the standardization sample, and correlations with Full Scale IQ scores obtained from the Wechsler Intelligence Scale for Children – Revised (36) averaged .80.

All subjects asked agreed to participate and complete data was obtained on all subjects.

**RESULTS**

**CHILD ILLNESS SEVERITY**

Children in both the IBD and control groups were first compared on indices of illness severity in order to assess the impact of any group difference on the outcome measures. On the IOF, where data were missing for one IBD participant, a series of one-way ANOVAs revealed no significant differences on any of the four factors (all $F's(2,38) < .53$, all $p's > .50$). Thus, the subjective impact of child illness on family members did not differ significantly across groups according to maternal report.

On the COBI, where data were missing for two IBD patients and four controls, a one-way ANOVA revealed a significant group difference ($F(1,33) = 8.04$, $p < .008$). Participants in the IBD group scored significantly higher ($M = 100.74$, $SD = 37.63$) than controls ($M = 57.19$, $SD = 52.99$). Thus, according to physician report, IBD participants were more symptomatic than children in the Cancer/Renal group. This is despite subjective ratings by parents that showed no differences between the diagnostic groups.

**MATERNAL ATTACHMENT**

First, attachment classifications were dichotomized into *secure* and *insecure* groups. As is customary in the attachment literature, the insecure was comprised of both preoccupied and dismissing mothers.

In the IBD sample, 5 mothers were classified as secure (24%) and 16 as insecure (76%). In the Cancer/Renal group, 11 were classified as secure (55%) and 9 as insecure (45%).

As predicted, the IBD mothers’ group had a higher proportion of insecurely attached ($z=2.57$, $p<.049$).

A clarification about the rate (45%) of insecure attachment in CA/renal group: this is comparable to those found in normative populations across thousands of adults in several cultures. This may be of concern, but does not differentiate the CA parents from adults studied with the AAI in studies over many years.

**MATERNAL MCMI-II PROFILES**

Relations between child diagnostic status, maternal attachment classification, and maternal MCMI-II profiles were compared by conducting a series of one-way ANCOVAs. In each analysis, MCMI-II scale scores served as criterion variables, with COBI scores employed as a covariate to control for the possible impact of child illness severity on maternal psychiatric symptoms. Table 2 outlines results for both the clinical syndrome scales, which correspond most closely with Axis I psychopathology, and the personality scales, which correspond most closely with Axis II psychopathology. Complete MCMI-II data were missing for 4 mothers in the IBD group and 2 mothers in the Cancer/Renal group.

As Table 2 reveals, significant Maternal Attachment Classification vs Child Diagnostic Status interactions were uncovered for 2 of the 9 Clinical Syndrome Scales (Alcohol Dependence and Major Depression), and for 6 of the 13 Personality Scales (Avoidant, Antisocial, Passive-Aggressive, Self-Defeating, Schizotypal and Borderline). Although several main effects of attachment classification were also revealed, they will not be interpreted due to the significant interaction effects, where in each case *insecurely attached mothers of IBD children obtained higher scores than mothers of the other two groups*. The only main effect that was significant in the absence of an interaction was for the Somatoform scale, where mothers of control group children scored significantly higher than mothers of IBD children. This finding should be interpreted cautiously, as it represents the only significant diagnostic group effect among 22 comparisons.

**DISCUSSION**

This controlled study clarifies and specifies the previous reports of higher rates of insecure attachment in mothers of children with IBD, supporting our first hypoth-
It supports our second hypothesis, that insecurely attached mothers of children with IBD would exhibit increased psychiatric symptoms compared to mothers of children with either cancer or renal disease. However, none of the group means fell in the clinical range on any of the MCMI-II scales (i.e., base rate scores 75). Thus, although insecurely attached mothers of IBD children were more symptomatic according to self-report, they may not be diagnosable with psychiatric disorders.

We paid special attention to comparisons of the Cancer/Renal control group verses the IBD group on COBI (clinician) and IOF (parent) illness severity scores. Do cancer/renal patients serve as a valid control group for IBD? Higher scores on the COBI were obtained by IBD participants, which suggest greater physician-rated illness severity. The nature of IBD requires that afflicted children follow a strict medication regimen, engage in dietary restrictions, and monitor their bowel habits continually. In addition, parents must attend closely to their children, who frequently miss school with debilitating abdominal pain. While cancer and renal disease patients are likely to require similar levels of care before, during and after treatment, they often experience higher functioning at other times. All patients were in remission, but the COBI/IOF measures permit comparability across disease categories (whether remission or not). Because of these measures, we appear to be seeing group and not phase differences. But, this is an empirical question that should be investigated further. Moreover, differences in maternal MCMI-II profiles were found after variance attributable to COBI scores was statistically removed. As a result, the finding that clinicians’ assessments of illness impact was higher in the IBD group compared to the Cancer/Renal group was not surprising. However, parents of IBD children did not rate their experience of the children’s illness as more stressful than parents of children with cancer or renal disease. This result provides evidence that a pediatric cancer/renal control group is an acceptable control group with which to compare IBD children.

Most research on psychosomatics has been done with adults; however, research on IBD in children and adolescents has been increasing over the past decade (1-4). Very little is known about the relation between interpersonal relationships, specifically the relation between attachment and pediatric psychosomatic illness. In order to shed light on this phenomenon, it is important to have a working theoretical model. When developing a model of psychosomatic illness, we suggest a developmental systems transactional approach and include the investigation of biological, social and

### Table 2. Relations between Maternal Attachment Classification, Child Diagnostic Status and Maternal MCMI-II Scale Scores.

<table>
<thead>
<tr>
<th>Maternal Attachment Classification</th>
<th>Child Diagnostic Status</th>
<th>Interaction Effect</th>
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<tbody>
<tr>
<td>MCMI-II Scale</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinical Syndrome Scales</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>0.04</td>
<td>1.12</td>
</tr>
<tr>
<td>Somatoform</td>
<td>0.01</td>
<td>4.22*</td>
</tr>
<tr>
<td>Bipolar: Manic</td>
<td>0.01</td>
<td>0.39</td>
</tr>
<tr>
<td>Dysthymia</td>
<td>0.43</td>
<td>0.87</td>
</tr>
<tr>
<td>Alcohol Dependence</td>
<td>3.80</td>
<td>0.66</td>
</tr>
<tr>
<td>Drug Dependence</td>
<td>2.32</td>
<td>0.67</td>
</tr>
<tr>
<td>Thought Disorder</td>
<td>1.49</td>
<td>0.01</td>
</tr>
<tr>
<td>Major Depression</td>
<td>1.63</td>
<td>0.51</td>
</tr>
<tr>
<td>Delusional Disorder</td>
<td>0.30</td>
<td>0.01</td>
</tr>
<tr>
<td>Personality Scales</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schizoid</td>
<td>0.16</td>
<td>0.74</td>
</tr>
<tr>
<td>Avoidant</td>
<td>4.78*</td>
<td>0.01</td>
</tr>
<tr>
<td>Dependent</td>
<td>0.66</td>
<td>1.39</td>
</tr>
<tr>
<td>Histrionic</td>
<td>0.28</td>
<td>1.55</td>
</tr>
<tr>
<td>Narcissistic</td>
<td>0.91</td>
<td>0.69</td>
</tr>
<tr>
<td>Antisocial</td>
<td>3.45</td>
<td>0.80</td>
</tr>
<tr>
<td>Aggressive-Sadistic</td>
<td>3.00</td>
<td>0.01</td>
</tr>
<tr>
<td>Compulsive</td>
<td>0.02</td>
<td>3.59</td>
</tr>
<tr>
<td>Passive-Aggressive</td>
<td>5.81*</td>
<td>0.06</td>
</tr>
<tr>
<td>Self-Defeating</td>
<td>6.53*</td>
<td>0.22</td>
</tr>
<tr>
<td>Schizotypal</td>
<td>9.96</td>
<td>1.31</td>
</tr>
<tr>
<td>Borderline</td>
<td>10.26**</td>
<td>1.37</td>
</tr>
<tr>
<td>Paranoid</td>
<td>0.78</td>
<td>0.09</td>
</tr>
</tbody>
</table>

attachment classifications.

Given the relationship between parental attachment status and childhood IBD, family or individual intervention and education may attenuate illness expression. Parent work should focus on attachment-related themes in those who show risk-factors for insecure attachment: improved parental responsiveness and availability.
cognitive vulnerabilities (37).

Alexander and French (38) hypothesized three factors necessary for adult onset of psychosomatic illness (specifically, ulcerative colitis, asthma, allergic dermatitis, duodenal ulcer, essential hypertension, rheumatoid arthritis and hyperthyroidism). First, they proposed that a physiological factor was needed to contribute to illness onset, e.g., elevated pepsinogen or helicobacter pylori in duodenal ulcer. Second, they hypothesized that recent increased life stress was a necessary agent for disease onset. Third, they identified disease-specific intra-psychic conflict as a necessary factor for the onset of a psychosomatic illness. However, their study, performed in the early 1950s, demonstrated no statistically significant findings. Unfortunately, statistical methods available at the time were inadequate to draw conclusions.

We present a four-factor model for child-onset IBD, using Dobzhansky's concept of regulatory and structural genes: 1) A multigenomic factor; 2) Peripubertal developmental vulnerability (a critical period); 3) Recent increased life stress; 4) Attachment vulnerability in the primary caretaker. While this model is speculative, it provides an approach to empirically-designed studies such as has been done with childhood asthma for testing.

1. The multigenomic factor. Dobzhansky and Gould (16) postulated variable genomic expression or penetration. Ginsburg, Szajnberg and Buck (39), citing animal and twin behavioral research, demonstrated the mechanism for variable genomic expression in MZ twins primed via affect: regulator genes can be turned on/off by environmental events.

We postulate a comparable set of mechanisms in the model postulated below: insecure maternal attachment can up-regulate pre-existing, quiescent structural gene(s) for IBD expression, whereas secure attachment will not. If there were variable loading – for instance, hypothetically five genes associated with IBD onset – then someone with one or two genes would have necessary, but not sufficient loading for expression. Someone with sufficient genetic loading can develop IBD with less input from the remaining three factors (peripubertal age, increased life stress, attachment). This multigenomic model is testable.

Controlled studies and accounts in the popular press discuss causal associations between the path of serious physical illness and psychological state, for instance, in differential breast cancer survival between women in support groups verses those who are not (40). In childhood physical illness, Lavigne and Faier-Routman's (17) meta-analytic study shows differential psychological symptomatology among different pediatric illnesses, with IBD and neurological disorders showing greater psychological morbidity. This disease-specificity of psychological difficulty runs counter to earlier studies by Stein and Jessop (34), which, however, did not include IBD in their cohorts. Mrazek and colleagues (41) performed an elegant and powerful study of expectant mothers with asthma and onset of wheezing in their infants. Mothers with insecure attachment were more likely to have infants who developed wheezing. Fritz and colleagues (42) and Burke and Engstrom (43) have extended work on asthma in children as an interaction between soma and psyche (42-45).

2. Peripubertal developmental vulnerability factor. This factor is based simply on an empirical observation that most children experience the onset of IBD around the time of puberty. However, we build on the concept of "critical periods" first described in the animal literature (46). Scott and Fuller (46), in landmark animal behavior studies with dogs, demonstrated that the absence of socialization to people or other animals by 12 weeks will disrupt dog behavior. We can hypothesize in IBD, that the normative endocrinological events of peripubert, may also prime certain vulnerable genes for IBD expression, particularly if these may have been primed by previous factors of recent life stress and attachment disorder (a more chronic, diffuse form of life stress). Attachment insecurity may be a form of life stress termed strain or cumulative (47).

3. Life stress factor. Both physical and psychological stress can exacerbate bowel symptoms. One study of the rat colon provides supporting evidence for the interaction between vulnerability to stress due to previous inflammation and subsequent inflammation. In this study, rats with a history of inflammation in the colon experienced an increase in inflammation and a reduction of function when stressed with a restraint procedure compared to the stressed control rats (48). This study illustrates the need to consider the complex interactions between a vulnerable diathesis and stressors that precipitate reactions in the colon. However, studies illustrating the effects of stress on the colon are not unique to animals, many human studies also illustrate similar effects.

In a study of patients with Ulcerative Colitis, Levenstein and colleagues (49) found that high levels of perceived stress over long periods tripled the risk
of exacerbation of symptoms; whereas short-term stress did not increase experience of symptoms. The stress associated with maintaining an insecure working model of attachment occurs across the lifespan without intervention. Therefore, this highlights the need for researchers to include attachment within the biopsychosocial model of disease in order to understand how an incoherent working model and stress in relationships can contribute to vulnerability to symptom exacerbation and relapse. The study also reported a trend toward significance for the relation between relapse and number of stressors in the month prior to relapse. The results from these studies, among others, point to the clear relation between stress and disease vulnerability in IBD; however, the exact relation between type and level stress and colon symptoms requires further research and clarification.

In our first study, not only were life stress scores extremely high, but also some parents downplayed stress. For instance, one mother said there was no “unusually” high stress. The past year, she later reported, the child discovered her father dead in bed when she was dropped off alone on a weekend custody visit. In addition, the child’s brother was almost killed in a drive-by shooting.

4. Attachment vulnerability factor. Attachment vulnerability, along with the previous factors, may be a mediating variable for genomic regulation of illness onset. Attachment influences both social relationships as well as cognitive models of the self and interpersonal relationships (50). Therefore, being insecurely attached and having a genomically-vulnerable GI tract may be all that is necessary to set into motion the onset of IBD.

Our postulated model can be used empirically to assess the factors associated with the illness onset. In addition, we suggest that insecure attachment may be an aggravating factor in illness maintenance/relapse. To the degree that IBD itself puts stress on a family, or a parent-child system, then insecure attachment is more vulnerable social system being further stressed and reciprocally may stress the ill child. Further research using this model can shed light on the focus areas for which specific therapeutic intervention can be provided over and above physical illness stabilization. This model includes genetic predisposition, “critical period” (peripubertal onset), life stress and attachment, with variable loading of the factors. This model can be tested empirically.

Finally, if attachment vulnerability is a factor in IBD illness onset, then families who have predisposing histo-

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46. Scott JP, Fuller JL. The genetics and the social behavior of the dog. Chicago, Ill.: University of Chicago, 1984.