# ACQUIRED VULNERABILITY: COMORBIDITY IN A PATIENT POPULATION OF ADULT OFFSPRING OF HOLOCAUST SURVIVORS

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Objective: *Offspring of traumatized individuals tend to be more susceptible to PTSD when exposed to a traumatic challenge. Our study examines patterns of comorbidity in offspring of Holocaust survivors who were "challenged" by the natural onset of anxiety and depression.* Method: *Eighteen offspring of Holocaust survivors who presented to the clinic with chief complaints of anxiety and depression were compared to matched controls. Six separate chi square analyses were performed to evaluate the frequency of different psychiatric diagnoses and comorbidity in both groups.* Results: *Adult offspring of Holocaust survivors had significantly more specific comorbidity of anxiety and depressive disorders and more diagnoses of atypical depression compared to controls.* Conclusions: *Individuals who come from traumatized families may be prone to exhibit comorbidity after being afflicted by routine psychiatric disorders. Thus, comorbidity may constitute an indicator of psychiatric vulnerability which most likely is mediated by the hypothalamic pituitary axis. Mechanisms of intergenerational traumatization as a philogenetically programmed survival mechanism in mammals will be presented. These findings have forensic implications in further understanding the "egg shell" plaintiff.*

Early attempts to identify a "complex" of symptoms characteristic for children of Holocaust survivors (1) have not been replicated. Solkoff (2) pointed out the methodological flaws of early studies in this population. Recent systematic studies have demonstrated an absence of specific psychopathology in nonpatient offspring of Holocaust survivors (3), but a significant protracted course in Israeli combat soldiers with PTSD who were also offspring of Holocaust survivors (4) was found.

The purpose of our study was to reveal any unusual diagnostic pattern or any specific comorbidity in adult offspring of Holocaust survivors, selected from a patient population that presented with initial chief complaints of anxiety and depression. Comorbidity was defined as the co-occurrence of two or more DSM-IV diagnostic entities in the same individual. To our knowledge, this is the first study to examine comorbid patterns as an indictor of vulnerability in subjects who came from a traumatized family background. Defining such vulnerability could prove useful in establishing guidelines for the assessment of damages in personal injury cases that involve individuals who come from traumatized backgrounds.

## METHOD

Thirty-six outpatients seen in a general psychiatric practice between 1987 and 1994 were selected. All 36 of these individuals had sought treatment for chief complaints of anxiety and depression. This is a retrospective study based on chart review. All 36 patients had been initially interviewed by one of the authors (A.N.) and were diagnosed by DSM-IV criteria. Patients who presented DSM-IV symptoms of major depression associated with inverted functional shift (hyperphagia, hypersomnia), anxiety (of a generalized type) and a tendency to overreact to personal rejection with intermittent dysphoria lasting for at least two weeks (a "crash-like response"), were assigned a diagnosis of "atypical depression."

Eighteen patients (seven males and eleven females) were American-born offspring of Holocaust survivors. At least one of the parents of each of our Holocaust subjects had been in a Nazi concentration camp, and all parents had immigrated to this country from Europe. Eighteen control patients (seven males and eleven females) were offspring of Jewish non-immigrant parents. The range of age for the sample was between 35 and 52 years of age; mean ages for the subject and control groups were 40.5 and 43.6 years, respectively. T-test analysis showed no significant differences between these two groups ($T=-1.42$, $p=0.166$). Groups were matched on education (at least two years of college), socioeconomic status and marital status.

The criteria of inclusion into the study were: 1) Ashkenazi Jewish ethnicity; and 2) The initial presentation to the clinic with chief complaints of anxiety and depression. In our clinic, all charts of children of Holocaust survivors are tagged with the code: "2-G" (Second Generation).

During the initial selection process, the selecting clinician was blinded for the formal diagnosis (which is written on the last page of the initial evaluation). Chi-square analyses were performed to compare the diagnostic frequencies of depressive and anxiety disorders in the control and target groups. Six separate analyses were performed to evaluate frequencies of generalized anxiety disorder, major depression, atypical depression, dysthymic disorder, any comorbidity, and individuals with a specific comorbidity of anxiety and depressive disorders. An additional chi square analysis was performed to evaluate the frequency of diagnosis by sex of subject.

## RESULTS

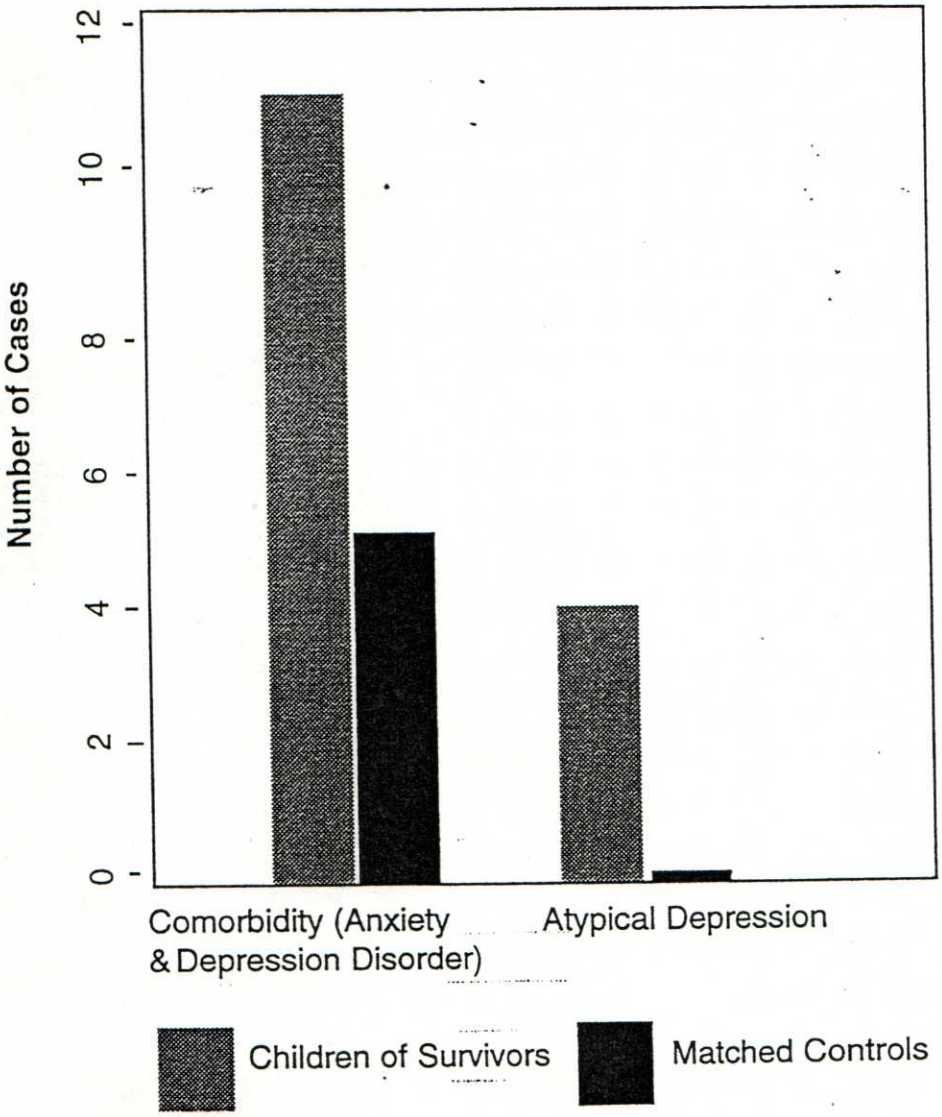
No significant relationship was found between diagnosis and sex of subject: ($x^2(4)$ --5.14, p=0.273). However, adult offspring of Holocaust survivors had significantly more diagnoses of atypical depression and exhibited more specific comorbidity of anxiety and depressive disorders than controls (see Figure 1). No such differences were evident in non-specific comorbidity, generalized anxiety disorder, dysthymic disorder, or major depression alone (see Table 1).

## DISCUSSION

Our findings are consistent with the general concept that children of Holocaust survivors come from heterogenous backgrounds and that they may constitute a heterogenous group (5). When afflicted by psychopathology (that is not specific for children of Holocaust survivors), these patients may present a broader range of clinical manifestations, and thus, may show comorbidity more often than other patient populations. The presence of atypicality may also suggest that this is a more anxiety prone cohort (6).

Our findings may then suggest that patients who come from traumatized families may more often present a broader spectrum of clinical pres-

**Figure 1: Children of Holocaust Survivors and Matched Controls: Group Differences**



entation which includes anxiety as a sole entity, atypical symptoms of depression, as well as typical symptoms of depression and anxiety.

**Table 1. Results of Chi-Square Analyses of Diagnoses and Comorbidity**

| | Number of subjects (N=36) | | | |
|---|---|---|---|---|
| | 2-G* | Controls | Chi-square | Signif. |
| GAD | 10 | 9 | 0.112 | 0.738 |
| Dysthymia | 5 | 7 | 0.500 | 0.480 |
| Major Depression | 3 | 5 | 0.643 | 0.423 |
| Comorbidity (Any) | 12 | 9 | 1.029 | 0.310 |
| Comorbidity (Anx + Depress. Dis.) | 11 | 5 | 4.050** | 0.044 |
| Atypical Depression | 4 | 0 | 4.500** | 0.034 |

*2-G = second generation

**p<.05

The subject of psychiatric vulnerability needs to be further explored. The current available literature on trauma points to the difficulties in defining psychological vulnerability (7). At least one previously published study (4) found that certain individuals who come from traumatized families show a prolonged recovery, in comparison to controls after "challenged" by a traumatic events (combat trauma). Likewise, Nader (8) showed that children whose parents have experienced significant traumata in life were more likely to present symptoms of PTSD after witnessing a sniper attack on a Los Angeles school ground. Our findings suggest that "the challenge" does not have to be a traumatic event ("trauma specific") but may constitute just the natural occurrence of psychopathology ("trauma nonspecific").

## THE MECHANISM OF TRANSMISSION

The mechanism by which offspring are affected by the parents' traumatic experience seems to be subtle, complex, and not entirely elucidated yet. Different models have been proposed (3) but have largely addressed the psychodynamic and psychological aspects of transmission. These contributions are based on previous work on nongenetic familial transmission of traits (9). We are proposing a mechanism of transmission that involves three major concomitant processes: a) verbal and nonverbal transmission of information; b) transmission of acquired traits (10); and c) (intrafamilial) microtraumatization/ dysregulation (i.e., biological dysregulation) (11).

### Transmission of Information

Transmission of information makes the historical facts available to the offspring either in overt or covert manners. The verbal account of history may be partial or complete. In many cases, nonverbal communication may have a complementary role in the grasp of the trauma by offspring, in addition to verbal communication of events. Nonverbal communication consists of clues which create a context and a mood in which the history of traumatic events is communicated.

### Transmission of Acquired Traits

Transmission of acquired traits refers to a nongenetic vertical transmission of certain adaptive and maladaptive traits of the parents which were acquired as part of their way of coping with the trauma. This is further analysis of transmission of trauma based on Kendler's work on transmission of traits (9).

### Intrafamilial Microtraumatization/Dysregulation

Intrafamilial microtraumatization/dysregulation refers to an acquired biological vulnerability which lowers an individual's threshold for the onset of psychiatric disorders. We hypothesize that unpredictable familial environments, acting as a "dysregulating factor," result in repeated brief states of arousal, which via kindling lead to peaks in stress hormones and

their metabolites. These in turn, may have toxic effects on different CNS and peripheral sites, which leads to an impaired autonomic modulation (12). By means of the more recently revealed anatomical connections between the nucleus of the solitary tract and the dorsal motor nucleus of the vagus with the amygdala (constituting together a major area of autonomic control) (13), cortical events are connected directly to the vagal system, which in turn, detects psychophysiological changes and may modify the threshold of proprioception. Such changed thresholds may further explain a measurable variability in autonomic responses, i.e., respiratory sinus arrhythmia, in individuals vulnerable to stress as demonstrated by Porges (14-16). This model is paralleled by psychological mechanisms described in previous contributions (17, 18). The complexity of intergenerational transmission (19) may also explain why a number of previous contributions, by describing different facets of the same process, have produced inconsistent, and at times, conflicting research findings (2).

Our model is further supported by Soumi's work on primates (20). Soumi also identified three major mechanisms that function concomitantly: a) cross generational transmission via observation or learning (i.e., monkeys seem to be able to pass on specific fears to their offspring); b) cross generational transmission via effects on maternal behavior (i.e., offspring of stressed out mothers tend to overreact both behaviorally and physiologically to their own encounters with stress). According to Soumi and Levine (20), in animals this can occur when trauma and its sequela compromise an individual's capability as a parent; c) cross generational transmission via parental mechanisms (biological changes that occur secondary to maternal stress during gestation with behavioral consequences on the offspring (i.e., maternal glucocorticoids etc.).

Based on Soumi's work, we hypothesize that "intergenerational transmission of trauma" in humans is a philogenetically based mechanism of adaptation, a biologically based survivor mechanism by which offspring are prepared and preinformed about dangers. Such a mechanism, most likely present in animals, may have adapted and reshaped to include verbal

capabilities in humans, thus creating a complex multilayered parent-child communication and bonding system. The biological arm of the mechanisms of transmission (acquired biological vulnerability) seems to be supported by Yehuda's recent findings (21), according to which offspring of Holocaust survivors with low normal cortisol levels show an exaggerated elevation of cortisol when challenged by traumatic events. In a similar vein, Carlson reported that Romanian orphans who were deprived of maternal attention showed cortisol abnormalities which correlated with the extent of developmental retardation. These children also had a blunting of cortisol elevation in response to stress (22). The mechanism of intergenerational transmission of trauma and stress may further constitute a process by which new psychopathology is generated by environmental events in individuals with minimal genetic/familial loading for psychiatric disorders. It may explain one of the mechanisms by which at least some psychiatric disorders have become more prevalent since the beginning of the century (23). Intergenerational transmission of trauma may further explain how environmental events can modify deep seeded biological states (i.e., "nurture" modifying "nature"). Finally, it may constitute the sociobiological substrate for some psychodynamic processes of early childhood trauma and its consequences, noted empirically by psychoanalysts over the past century (24).

If these findings can be replicated, then it would constitute further proof that a complex of subtle and persistent environmental changes (for instant chaotic, unpredictable parental behavior) may act as a "dysregulating factor" on the hypothalamo-pituitary adrenal system to produce latent biological abnormalities. This would then further support the need for the assessment of psychiatric vulnerability by means of specific biological markers.

## FORENSIC CONSIDERATIONS

Forensic implications are multiple and significant: Individuals whose parents were victims of trauma may constitute a subgroup of potential "egg shell" plaintiffs. The origins of the "egg shell skull" doctrine can be

found in English common law. In 1901, in the case of *Dulieu v. White* (25), a preexisting condition of the plaintiff, namely pregnancy (not a disease), which was unknown to the defendant, did not bar the plaintiff's compensation for damages. Dulieu, the plaintiff, was working in a tavern when the defendant, who was driving a carriage, passed the tavern, lost control of his horses, causing the carriage to collide into the tavern. The court decided in favor of the plaintiff that shock and fright caused by the collision resulted in the premature birth of a brain-damaged infant.

The egg shell skull theory advocates that: "The defendant greets the plaintiff as he or she finds the defendant." Thus, both a preexisting condition within the plaintiff and conduct by the defendant operate on the condition so as to produce aggravated injury. According to the 1965 restatement of tort (26) in cases of the "thin" skull principles: "The negligent actor is subject to liability for harm to another although a physical condition of the other which is neither known nor should be shown to the actor makes the injury greater than that which the actor as a reasonable man should have foreseen as a probable result of his conduct."

The egg shell skull theory has been tested repeatedly in American civil cases for various physical disorders (27-30), as well as psychiatric disorders such as behavioral and personality changes (31, 32), psychosis (33), phobia (34), schizophrenia (35), childhood schizophrenia after assault (36), and aggravation of dissociative disorder after sexual harassment (37). More recently the "egg shell theory" has been applied in criminal cases as well (38, 39). Finally, in a recent much publicized case of an au pair shaking an infant, which resulted in the infant's death, the jury found the defendant guilty based on the "egg shell skull" theory (40). However, the judge in this case felt that the defendant lacked the intent to cause death or even injury to the child and thus reduced the second degree murder verdict to involuntary manslaughter (41).

The above presented egg shell skull cases further highlight the importance placed on the concept of vulnerability in recent legal case history. Further understanding on the origins of vulnerability (for instance, predis-

posed versus acquired) could prove to be helpful in future developments on the "egg shell" concept.

Our experience shows that often "egg shell" individuals may not present any preexisting psychopathology and may even show an initial defensive resilience to psychiatric disorders. However, after the onset of psychopathology (mainly depressive or anxiety disorders) as part of a personal or work-related injury, these individuals may present: a) prolonged temporary disability (due to added comorbidity); b) more severe symptomatology; and c) more significant than expected residual symptomatology.

In such individuals, an attempt has to be made to clearly identify the relationship between the prodromal phase of a psychiatric disorder and the full onset of the disabling phase of the disorder. In psychiatry, prodromal symptoms are often confusing. We recommend that whenever possible, prodromal symptoms may not be considered as indicative of a specific psychiatric illness. This is because often such prodromal symptoms may constitute long-term premorbid personality traits for which the patient's activity and lifestyle have become congenial. The relationship between premorbid traits and early symptoms is often difficult to delineate. For instance, most psychiatrists have encountered patients who develop the first disabling manic episode after a significant trauma. However, many of these patients may present preexisting prodromal symptoms that are not disabling whatsoever. Such prodromal symptoms may even temporarily render these individuals excessively productive and successful. If such a person is examined prior to the onset of the disabling mania, often it is impossible to differentiate such behavior from that of a highly energetic and creative individual (premorbid personality). Therefore, we recommend the following guidelines:

1) A general principle should be observed according to which individuals who have no clear disabling symptoms and did not qualify for a diagnosis should not be considered as having a disorder until DSM-IV criteria are met.

2) If repeated injuries have had a cumulative effect and "acquired vulnerability" to stress is suspected, but no diagnosable symptomatology is present, the repeated stressors may not have reached the "critical" threshold to qualify for an injury.

3) If one specific event falls at the end of a series of repeated stressors and results in the onset of symptoms, then the event in question may be seen as a causative factor of a "psychiatric injury." In general, if no symptoms are present and no psychiatric condition can be diagnosed, then no demonstrable injury has occurred. However, a case by case, careful assessment of causation is recommended.

All such forensic considerations are important in damage assessment and cost of future medical care.

## CONCLUSIONS

The concept of acquired vulnerability has been grossly neglected in the recent psychiatric literature. Studies on intergenerational aspects of trauma may shed more light on early childhood factors at play in rendering individuals more vulnerable to stress. Psychiatric comorbidity, like the coexistence of anxiety and depression, may be a mean of assessing such vulnerability. At least three different mechanisms may be at play in intrafamilial traumatization ("transmission of trauma"). In American jurisprudence, there is a significant tradition in considering physical and psychological vulnerability in verdicts and damage assessment.

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