Survivors of Childhood Trauma: Possible Approaches to Therapy

TO THE EDITOR: The clinical case conference by Andreea L. Seritan, M.D., Glen O. Gabbard, M.D., and Lloyd Benjamin, M.D., published in the Oct. 2006 issue of the *Journal* (1), is much appreciated. Several aspects of the case conference deserve comment.

"Ms. A" is a child survivor of the Holocaust (2, 3) who was hidden with her family, not separated from them. She did not have to assume a gentile identity nor was she adopted. Furthermore, she is not a death camp survivor, one of the "living skeletons" liberated from such places as Auschwitz, Buchenwald, and Dachau. Nevertheless, the deaths of her mother and brother were incalculable losses, sustained while she lived in constant danger of being caught and killed by Nazis. She was also suspected of being sexually abused, possibly as part of the "cost" of being protected.

The indelible impact of such genocidal persecution could not be ameliorated in short-term therapy, and while I do not believe any such claims were made, this point was not clearly delineated. Furthermore, there was no posttermination follow-up to determine whether the patient's trauma was "worked through" (1, p. 1705) as opposed to "opening up more than the patient can handle"(1, p. 1708).

Despite Dr. Seritan's sensitivity, did she inadvertently get drawn into a masochistic enactment that could have re-traumatized the patient by inflicting another loss upon her? Ms. A was appropriately informed that Dr. Seritan was moving in 6 months and had reservations about the treatment. But was Dr. Seritan so impressed by the patient that she proceeded nonetheless? Perhaps the wish to write about the patient also influenced her. Nonetheless, the patient's resilience, ego, strength, and presentation were positive factors not to be minimized.

However, Ms. A's persona and idealizing maternal transference not only recreated the long-lost mother who suddenly disappeared, but also concealed the terrified child who had to be invisible to the Nazis, invisible to her stepmother, and perhaps somewhat invisible to her lovely therapist with a European accent. In the patient's unconscious mind, the therapist could have been a Nazi also.

Dr. Seritan read about the Holocaust and consulted knowledgeable supervisors. While Dr. Gabbard correctly points out that empathic attunement is more important than factual knowledge, our research (2, 3) suggests that clinicians working with survivors of historical trauma are well-served by knowing about the actual circumstances that their patients' endured. The reconstruction of the traumatic period and the opportunity to develop a coherent narrative—often for the first time—may be one of the important achievements in therapy. Perhaps Dr. Seritan alluded to this when she states, "I pointed out to Ms. A how much work she had done and how much she had accomplished."

References

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Dr. Brenner reports no competing interests.

Drs. Seritan and Gabbard Reply

TO THE EDITOR: We would like to thank Dr. Brenner for his interest in our case conference.

We certainly agree that the impact of the tremendous trauma of being a child survivor of the Holocaust cannot be ameliorated in brief therapy, nor was this claim made in our article. Rather, our case conference depicts one of many possible approaches to the impact of horrific childhood trauma when the therapist is faced with a time-limited treatment. The impetus to write about this extraordinary woman came from deep respect for her experience and resilience and the desire to share her story of survival with others. Contrary to Dr. Brenner's suggestion, the decision to begin treatment with her was completely unrelated to a wish to write something about her.

The missed opportunity of working through the negative transference in the course of this brief therapy was pointed out in our article (p. 1708). Dr. Gabbard further illustrates how the therapist and the patient partnered in shaping the therapy, with the re-creation of the good mothering experience as the predominant theme in the transference-countertransference dimensions of the treatment.

We share Dr. Brenner's view that the development of a coherent narrative is a major goal of working with such patients. Although this task was incorporated in the brief therapy reported in our case conference, it was not the centerpiece of Ms. A's treatment. The main agenda was dealing with loss. As our concluding remarks underline, the patient's work throughout her therapies has allowed her to remain a strong presence in her family, stay socially active and involved in her community, and continue to grow. Her story of courage and dignity, despite terrible childhood trauma, was our most important message.

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Author disclosures accompany the original article.

Serotonin Transporter, Stressful Life Events, and Depression Severity

TO THE EDITOR: Recently, Gil Zalsman, M.D., and colleagues reported that lower expressing alleles ($S+L_G=S'$) of the 5-HTTLPR polymorphism "independently predicted greater depression severity and predicted greater severity of major depression with moderate to severe life events compared with the higher expressing L_A allele" (1, p. 1588). We call into question the validity of these conclusions for several reasons.

First, the interaction analysis included 79 patients whereas the original cohort included 191 subjects. From Figure 1 and the data presented in the results section, we conclude that the frequencies of the L′L′, L′S′, and S′S′ genotypes in the group retained for the interaction analysis are 68.3% (54/79), 8% (6/79), and 24% (19/79), respectively. These proportions are

highly different (χ^2=46, df=2, p=0.000) from those reported in the entire cohort of depressed patients as presented in Table 1 of the article. No explanations for these differences or discussion of their consequences are provided. Additionally, these numbers indicate that some of the gene-by-environment strata have a very low cohort size (<5), which leads us to question the robustness of the results presented.

Second, simple calculations allowed us to make conservative estimates of the lower boundary of the Hamilton Depression Rating Scale (HAM-D) values for the S′L′ genotype for low (19.08) and high (22.74) stressful life events (available upon request). From these calculations and information provided in the text and in Figure 1, we concluded that patients with the S′S′ genotype have lower (at low stressful life events) or equivalent (at high stressful life events) HAM-D scores compared with those with the L′L′ genotype. Irrespective of stressful life events, patients with the L′S′ genotype have HAM-D scores that are at least equivalent (most likely higher if cohort sizes are not too unbalanced) when compared with patients with the L′L′ genotype. For these reasons, we question the rationale of grouping the S′S′ and S′L′ genotypes in a dominant model as shown in Figure 1 of the article by Dr. Zalsman and colleagues. Our estimates suggest that the reported interaction is most likely because of the fact that patients with the S′S′ genotype and low stressful life events have low HAM-D scores compared with patients with the L′L′ genotype exposed to low stressful life events while, at high stressful life events, patients with S′S′ and L′L′ genotypes have equivalent HAM-D scores. Finally, the significant effect of genotype on the severity of depression is primarily because of a small group (N=6) of patients with the S′L′ genotype, since patients with the S′S′ genotype have in fact the lowest severity scores, thus invalidating the claim that the lower expressing alleles of the 5-HTTLPR polymorphism in the serotonin transporter gene "predicted greater depression severity" (1, p. 1588). Disclosing the exact HAM-D values (SD) and number of patients in the six gene-by-environment strata and discussing the reasons for excluding more than one-half of the cohort would help readers to better interpret the results of this study.

Reference

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Dr. Joober receives financial support from the Canadian Institutes of Health Research and is on the advisory panel at Janssen-Ortho.

Drs. Sengupta and Schmitz report no competing interest.

Supported in part by a grant from the Fonds de la Recherche en Santé du Québec and the Canadian Institutes of Health Research (Dr. Joober).

Drs. Zalsman and Mann Reply

TO THE EDITOR: We thank Drs. Joober, Sengupta, and Schmitz for their comments on our recent study, but we disagree with their conclusions and calculations. They raise the question whether a selection bias operated in selecting a subcohort of 79 subjects for the gene-by-environment interaction analysis. The sub-cohort of cases that was used for the gene-by-environment analysis consisted of those subjects who completed the St. Paul-Ramsey Rating Scale for Life Events. Selection effects are always a concern in association studies, but three other published studies report results consistent with our finding that depression is sensitive to life events in the group with the low expressing alleles of this polymorphism (2–4), despite studying populations of different ages (children, adolescents, and young adults) from different countries (New Zealand, United States, and United Kingdom) and using different measures to assess life events or stress.

The second concern raised by the authors is linked to the source of the observed interaction. They attribute reported gene-by-environment to their speculation about our data that patients with the S′S′ genotype and low stressful life events might have had lower HAM-D scores compared with patients with the L′L′ genotype, while at high stressful life events, both groups might have "equivalent depression scores." To clarify this, we present the HAM-D scores (SD) and number of patients in the six gene-by-environment strata (Table 1). As can be seen from the table, under high stressful life events, the L′L′ group actually has lower depression scores, both compared with the S′S′ and S′L′ groups and with the L′L′ group under low stressful life events. In contrast, the S′S′ group under high stressful life events almost doubles the depression scores of the S′S′ group under low stressful life events, whereas the L′L′ group has comparable depression scores regardless of stressful life events.

TABLE 1. Mean HAM-D Scores and 5-HTTLPR Genotype in Depressed Patients Experiencing High and Low Recent Stressful Life Events[a]

Life Event Stress Level	Genotype					
	S′S′		S′L′		L′L′	
	Mean	SD	Mean	SD	Mean	SD
Low (N=28)[b]	10.8	8.8	20.1	7.9	21.3	5.5
High (N=51)[c]	19.5	5.9	20.2	5.5	17.6	3.9

[a] 5-HTTLPR- serotonin transporter promoter linked polymorphism; L′=L_A high expressing allele; S′=S+L_G low expressing alleles.
[b] Number of patients in S′S′ genotype=5; number of patients in S′L genotype=13; number of patients in L′L′ genotype=10.
[c] Number of patients in S′S′ genotype=14; number of patients in S′L′ genotype=22; number of patients in L′L′ genotype=15.

Although our cohort sizes were modest, the results of our study are consistent with the other cited studies (2–4). Independent replications validate our conclusions more convincingly than a single study with a larger cohort size.

References

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