

Journal of Psychiatry and Psychiatric Disorders

Volume 1, Issue 5

Short Communication

The Diathesis-Stress Model: Psychosocial Stressors, Trauma and Diabetes

Faisal A. Islam* and Zia Choudhry

Department of Mental Health and Clinical Research, International Maternal and Child Health Foundation, NPO,
Montréal, Québec, Canada

***Corresponding Author:** Faisal A. Islam, Department of Mental Health and Clinical Research, International
Maternal and Child Health Foundation, NPO, Montréal, Québec, Canada, Tel: +685269467; E-mail:
faisal.islam218@gmail.com

Received: 18 July 2017; **Accepted:** 26 July 2017; **Published:** 31 July 2017

1. Introduction

Recent research indicates that post-traumatic stress disorder (PTSD), along with mood spectrum disorders, tends to affect the overall developmental course of Type II diabetes mellitus. Despite a paucity of pathophysiological understanding available for the observations in question, we can begin to extrapolate a possible mechanism of action for the associations. It has been reported that PTSD earthquake survivors in L'Aquila, Italy have modulated their respective dietary patterns from baseline [1]. Dysregulation in eating behaviors may be regarded as a maladaptive

response to the initial traumatic event. It is conceivable to assume that disturbed consumption patterns may increase an individual's likelihood of acquiring Type II diabetes mellitus some years down the line.

2. Keywords: Trauma; Diabetes; Post-traumatic stress disorder; Diabetes mellitus

3. Unraveling the HPA Axis

Individual stress response is essential for homeostatic preservation and therefore, survival of a given organism. The hypothalamic-pituitary-adrenal (HPA) axis is largely responsible for orchestrating the stress response in accordance with the overall degree of perceived threat from the injurious stimuli. In a nutshell, stressor(s) allow for the HPA axis to be duly alerted by means of an intrinsic feedback loop; concerted actions by the hypothalamus, pituitary gland and adrenals bring forth the release of a number of hormones and chemical signals that culminate in the body's 'flight-or-fight' response.

Epinephrine release leads to an elevation in blood pressure and heart rate. Furthermore, the release of cortisol results in transient hyperglycemia, which facilitates resourceful management of the body's energy reserves. In other words, energy is available for immediate use by the muscles that need it the most. Interestingly enough, elevated serum cortisol is also responsible for exerting a protective effect against PTSD development. [2] Based on this rationale, PTSD susceptible individuals, namely, combat-exposed veterans may be preemptively treated with high-dose cortisol [2]

Under unusual circumstances, stress hyperglycemia may serve as a harbinger for new onset diabetes. It would seem intuitive to simply measure a patient's cortisol levels as a method for determining the physiological effect of PTSD. Unfortunately, any attempts at establishing a basal range of cortisol in PTSD individuals have yielded inconclusive results [3]. However, it isn't unreasonable to envision repetitive (or prolonged) exposure to trauma as having a negative impact on the feedback loop. Psychophysiological factors associated with the stressful events may have potential long-term metabolic consequences. Murine models determined that 'chronic social defeat stress' or repeat exposure to a stressor over a period of 10 consecutive days have resulted in marked levels of depressive symptomatology coupled with obesity and insulin insensitivity [2].

4. Putting it All Together: The Diathesis-Stress Model

The Diathesis-Stress Model provides a conceptual framework for understanding the development of Type II diabetes mellitus and comorbid mood disorders within the context of key life events. The theoretical model posits that an individual's propensity ('diathesis') for acquiring a disorder is a function of the dynamic interplay that exists between the diathesis and ongoing stressors ('stress'). Personal attributes such as resilience can provide a buffer against the development of a psychopathology. Although, the exact mechanism and underlying influence of stressors for diabetes mellitus is not yet known, longitudinal studies have ascertained the role of 'high-level' stress on type II diabetes mellitus [4]. Depression, in and of itself, may also serve as an independent stressor that increases the likelihood of acquiring diabetes mellitus. The Diathesis-Stress Model can readily account for the role of PTSD in impacting diabetes mellitus. Intense or prolonged periods of stress can negatively affect the HPA axis, throwing the feedback loop off-course. Corticosteroid receptor sensitivity is also modulated by stress; variations in cortisol secretions often translate to real-world complications of diabetes [4]. Clinicians should utilize a multifaceted approach for uncovering the etiological basis for Type II diabetes mellitus with respect to psychosocial models. Even though previous studies evaluating the role of cortisol in PTSD have led to conflicting results, it may be prudent to consider investigating basal insulin levels in predisposed individuals. Furthermore, benign conditions such as stress hyperglycemia should be cautiously monitored in susceptible patients.

Future research may help delineate the subtleties involved in the mechanism of action for diabetic patients that have a positive history of PTSD.

5. Disclosure

The authors have no conflicts of interest to disclose.

References

1. Carmassi C, Bertelloni CA, Massimetti G, et al. Impact of DSM-5 PTSD and gender on impaired eating behaviors in 512 Italian earthquake survivors. *Psychiatry research* 225 (2015): 64-69.

2. Pfau ML, Russo SJ. Peripheral and central mechanisms of stress resilience. *Neurobiology of stress* 1 (2015): 66-79.
3. Gerardi M, Rothbaum BO, Astin MC, et al. Cortisol response following exposure treatment for PTSD in rape victims. *Journal of aggression, maltreatment & trauma* 19 (2010): 349-356.
4. Carvalho LA, Urbanova L, Hamer M, et al. Blunted glucocorticoid and mineralocorticoid sensitivity to stress in people with diabetes. *Psychoneuroendocrinology* 51 (2015): 209-218.



This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC-BY\) license 4.0](https://creativecommons.org/licenses/by/4.0/)