# ACUTE ENDOCRINE STRESS REACTIVITY AND RECOVERY IN WOMEN AT FAMILIAL RISK OF BREAST CANCER

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**Introduction:** To date, little research has investigated the effects of chronic stressors on responsiveness of endocrine parameters and recovery after acute psychological stress in humans. Here, alterations in the endocrine responses to an acute stressor in women at familial risk of developing breast cancer are reported.

**Methods:** Familial breast cancer risk was assessed using a standard risk estimation model. Women were then subjected to a well-established, widely used laboratory stressor comprising a speech task and mental arithmetic. Endocrine response patterns of women with or without increased familial risk were compared to a randomly assigned control group.

**Results:** Stronger responses in women at familial risk of breast cancer were seen in neuroendocrine activity of the major stress systems, the hypothalamo-pituitary-adrenal axis (plasma cortisol) and the sympathetic pathway (plasma epinephrine). For epinephrine, these alterations were evident through a delayed recovery pattern rather than increased reactivity. Cortisol showed both an increased reactivity as well as a delayed recovery in women at increased risk for breast cancer.

The two groups of women did not differ in the questionnaires used to assess psychological distress (Perceived Stress Scale PSS) and depressive symptoms (Beck Depression Inventory BDI). However, alterations in epinephrine response similar to those when looking at familial risk of cancer as the grouping factor were seen in groups divided based on the scores on the BDI. Here, women with higher but non-clinical BDI scores exhibited similar reactivity but slower recovery of the epinephrine response. This effect remained significant when statistically controlling for familial risk of breast cancer. It was further not mediated by higher scores on measures of perceived stress and stress symptomatology found in BDI-high women.

**Implications:** Results are in line with the theory of allostatic load. However, no mediating effect of self-reported distress on endocrine stress response alterations could be found. The observed enhanced reactivity and delayed recovery in cortisol and epinephrine stress response may indicate a potential pathway leading to detrimental health effects. Future research will have to determine if these response alterations are of predictive value. Such a delayed endocrine stress recovery may even be involved in the psycho-biological interactions leading to higher breast cancer incidence in this population.