The role of biological factors in social withdrawal and avoidance of treatment for major depressive disorder: a path analysis

Susan J. Thomas
*University of Wollongong, sthomas@uow.edu.au*

Theresa A. Larkin
*University of Wollongong, tlarkin@uow.edu.au*

Peter R. Leeson
*University of Wollongong, pleeson@uow.edu.au*

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Abstract

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The role of biological factors in social withdrawal and avoidance of treatment for major depressive disorder: A path analysis

Susan Thomas*, Theresa Larkin, Peter Leeson

University of Wollongong, NSW, Australia

Despite the existence of effective treatments, individuals with depression usually avoid them, a phenomenon termed help-negation. The contributions of psychological factors including stigma have been widely researched however they do not adequately explain help-negation. Because biological processes are clearly involved in depression and withdrawal behaviours, we hypothesised that they contribute to treatment avoidance in depression. The stress hormone cortisol may mediate help-negation due to its involvement in cognitive and mood changes, withdrawal and suicide. Additionally, oxytocin, a neuropeptide, regulates neuroendocrine stress responses, and complex social behaviours. Blunted oxytocin function occurs in psychopathology involving social withdrawal, and may mediate help-negation. Testing models of relationships between biological variables, cognition and behaviour may lead to improved interventions and biomarkers of suicide risk. We quantified plasma oxytocin and cortisol levels in 30 individuals with major depressive disorder for which they were not seeking treatment, and 60 healthy controls and related these to psychopathology, perceived stress, psychosocial functioning, depressogenic cognitions, help-seeking intentions and attitudes to treatment. Path analyses were conducted to test hypotheses about relationships between the variables and model fit using established statistical indices.

As predicted, biological measures predicted treatment intentions, mediated by psychosocial factors. We present the path model, showing the strength and direction of relationships between the variables and their contribution to help-negation. Our study provides new information about biological factors involved in social withdrawal and treatment avoidance in depression. Our biopsychosocial model strengthens previous models based on psychosocial factors alone, of relevance to treatment uptake campaigns and early interventions.