COMMENTS AND RESPONSES

Clinical Depression Versus Distress Among Patients With Type 2 Diabetes: Not Just a Question of Semantics

Response to Fisher et al.

Fisher et al. (1) showed several ambiguities in the current meaning of depression. Individuals who feel saddened or unhappy by virtue of life's vicissitudes meet criteria for major depressive disorder (MDD) if these moods are associated with decreased energy or interest. Such imprecision is the result of a historical anomaly: In 1980, the Diagnostic and Statistical Manual of Mental Disorders (DSM)-III amalgamated a collection of depressive disorders and syndromes (2). Thus, present MDD represents different phenotypes that go from the melancholic patient to individuals without low mood but with dysphoria associated with high interpersonal rejection sensitivity (2–4). Furthermore, predepressive personality characteristics (neuroticism and the tendency to respond intensely to adverse stimuli) also qualify high in the commonly used rating scales for MDD, as in the Center for Epidemiological Studies Depression Scale.

MDD is coming into increasing disfavor among psychiatrists; possibly, in the near-next edition of DSM (DSM-V), MDD will be substituted by distinct depressive phenotypes (2). Nevertheless, how can we surpass this challenge nowadays? The biological consequences of several subtypes of depression or distress (DoD) are characterized by a deregulated "stress-response," in which the activation of the hypothalamic-pituitary-adrenal axis and the sympathetic-adrenal-medullary axes impairs immunity and produces an increase in acute-phase proteins and a persistent low-grade inflammation syndrome (5).

Pharmacological actions to improve this pathological stress response could reduce patients' comorbidities. Additionally, distressed people can be managed with common antidepressive/anxiolytic maneuvers, although the dysphoric subgroup (atypical depressive patients without low mood) is highly resistant to classic antidepressants drugs and may require antiglutamatergic/dopaminergic augmentation strategy (6). Predepressive neuroticism (a hallmark for distress reactions) may resist any existing physical or psychological therapy.

Psychosocial stress increases salivary α-amylase via adrenergic mechanisms (7). DoD patients may have elevations in the salivary amylase. Since for the individual patient severe depressive symptoms change over time, fulfilling criteria for major depression, minor depression, dysthymia, and subsyndromal states, a biological marker that objectively shows important psychological suffering is of extreme importance. Hence, studies relating DoD and diabetes will be improved if they include a biological marker of psychosocial stress like measurements of salivary α-amylase.

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References

1. Fisher L, Skaff MM, Mullan JT, Arean P, Mohr D, Masharani U, Glasgow R, Laurencin G: Clinical depression versus distress among patients with type 2 diabetes: not just a question of semantics. *Diabetes Care* 30:542–548, 2007
2. Shorter E: The doctrine of the two depressions in historical perspective. *Acta Psychiatr Scand* 433:5–13, 2007
3. Parker G, Roy K, Mitchell P, Wilhelm K, Malhi G, Hadzi-Pavlovic D: Atypical depression: a reappraisal. *Am J Psychiatry* 159:1470–1479, 2002
4. Zimmerman M, McGlinchey JB, Young D, Chelminski I: Diagnosing major depressive disorder IX: are patients who deny low mood a distinct subgroup? *J Nerv Ment Dis* 194:864–869, 2006
5. Vale S: Psychosocial stress and cardiovascular diseases. *Postgrad Med J* 81:429–435, 2005
6. Vale S, Espejel MA, Dominguez JC: Amantadine in depression (Letter). *Lancet* 21:437,1971
7. Ehlert U, Erni K, Hebisch G, Nater U: Salivary alpha-amylase levels after yohimbine challenge in healthy men. *J Clin Endocrinol Metab* 91:5130–5133, 2006