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Research report

Discriminating melancholic and non-melancholic depression by prototypic clinical features

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ABSTRACT

Background: Melancholia is positioned as either a more severe expression of clinical depression or as a separate entity. Support for the latter view emerges from differential causal factors and treatment responsiveness but has not been convincingly demonstrated in terms of differential clinical features. We pursue its prototypic clinical pattern to determine if this advances its delineation.

Methods: We developed a 24-item measure (now termed the Sydney Melancholia Prototype Index or SMPI) comprising 12 melancholic and 12 non-melancholic prototypic features (both symptoms and illness correlates). In this evaluative study, 278 patients referred for tertiary level assessment at a specialized mood disorders clinic completed the self-report SMPI as well as a depression severity measure and a comprehensive assessment schedule before clinical interview, while assessing clinicians completed a clinician version of the SMPI items following their interview. The independent variable (diagnostic gold standard) was the clinician's judgment of a melancholic versus non-melancholic depressive episode. Discriminative performance was evaluated by Receiver Operating Characteristics (ROC) analysis of four strategies for operationalising the SMPI self-report and SMPI clinician measures, and with the former strategies compared to ROC analysis of the depression severity measure. The external validity of the optimally discriminating scores on each measure was tested against a range of clinical variables.

Result: Comparison of the two self-report measures established that the SMPI provided greater discrimination than the depression severity measure, while comparison of the self-report and clinician-rated SMPI measures established the latter as more discriminating of clinically diagnosed melancholic or non-melancholic depression. ROC analyses favoured self-report SMPI distinction of melancholic from non-melancholic depression being most optimally calculated by a 'difference' score of at least four or more melancholic than non-melancholic items being affirmed (sensitivity of 0.69, specificity of 0.77). For the clinician-rated SMPI measure, ROC analyses confirmed the same optimal difference score of four or more as highly discriminating of melancholic and non-melancholic depression (sensitivity of 0.84, specificity of 0.92). As the difference score had positive predictive values of 0.90 and 0.70 (for the respective clinician-rated and self-report SMPI forms) and respective negative predictive values of 0.88 and 0.70, we conclude that the clinician-rated version had superior discrimination than the self-report version. External validating data quantified the self-rated and clinician-rated Index-assigned non-melancholic patients having a higher prevalence of anxiety disorders, a higher number of current and lifetime stressors, as well as elevated scores on several personality styles that are viewed as predisposing to and shaping such non-melancholic disorders.

Limitations: Assigned melancholic and non-melancholic diagnoses were determined by clinician judgement, risking a circularity bias across diagnostic assignment and clinical weighting of melancholic and non-melancholic features. The robustness of the Index requires testing in primary and secondary levels of care settings.

Conclusions: The clinician-rated SMPI differentiated melancholic and non-melancholic depressed subjects at a higher level of confidence than the self-report SMPI, and with a highly acceptable level of discrimination. The measure is recommended for further testing of its intrinsic and applied properties.

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1. Introduction

There has been a longstanding view positioning melancholia as a distinct 'type' of depression that appears more quintessentially biological, and which has been variably termed 'endogenous', 'endogenomorphic', 'autonomous', 'vital', 'Type A' as well as 'melancholic' depression (Jackson, 1986; Parker et al., 2010; Parker and Hadzi-Pavlovic, 1996a; Taylor and Fink, 2006). The arguments in favour of its distinct status have included (Parker et al., 1996, 2010) a somewhat distinctive pattern of symptoms and signs, a greater relevance of genetic and other biological – as against psychosocial – determinants, concomitant evidence of biological dysfunction particularly involving the hypothalamic–pituitary–adrenal axis, a differentially stronger response to physical treatments such as antidepressant drugs and electroconvulsive therapy than to psychotherapy, and a low placebo response rate.

The longstanding binary view positioned such a depressive 'type' as distinct from a second 'type' – variably termed 'neurotic' or 'reactive' depression in terms of clinical symptoms and preferential causes. Despite some consistency in the clinical ('endogeneity') symptoms long listed as having some specificity to melancholia, the advent and application of differing multivariate analytic approaches in the 1950's – whether factor, cluster and (later) latent class analysis (Parker and Hadzi-Pavlovic, 1996a) – failed to deliver support for a clear-cut symptom-defined binary solution.

Reasons for failing to so differentiate melancholia would include it actually not being a distinctive depressive type and simply being a more severe expression of depression—essentially the unitary or dimensional view. Alternatively, it could be that melancholia is a differing 'type' but lacking a pristine clinical boundary so disallowing clear clinical delineation and/or that its putative clinical symptoms and signs are limited in terms of their specificity and capacity to define melancholia. For example, and as we have quantified (Parker et al., 1996), none of the historically-weighted endogeneity symptoms show absolute specificity and, at best, show modest differential prevalences. Even if a symptom demonstrates discriminatory potential, identifying how it is best operationalised and measured is rarely straightforward. If not absolute, deciding whether to impose a cut-off for its 'presence' along dimensions of severity, persistence or some other parameter is problematic. Further, age, gender and duration of episode may impact on symptom ratings, while response biases (e.g., excessive subjective weighting versus denial and minimisation) influence self-reporting—just as assessment by external observers can be influenced by rating biases.

Finally, if melancholic depression is a 'circuit disorder' involving disruption of neurocircuits, then the actual site or dynamics of the disruption may account for certain symptoms (e.g., abulia, psychomotor agitation) being distinctive in some individuals and minimal or even absent in others. Thus, even if melancholia is a discrete condition, its symptom markers are limited by multiple factors that must confound any analytic study seeking to delineate it simply on the basis of symptoms with any precision.

Historical approaches to defining and classifying melancholia over recent decades have involved relatively few strategies. First, and most commonly, limiting definition to a prescribed number of symptoms (as occurs in DSM-IV). Second, melding clinical symptoms with non-symptom correlates of melancholia. The latter approach has only a few examples. One was the Newcastle Index (Carney et al., 1965) which weighted items such as 'adequate personality', 'no adequate psychogenesis' and previous episodes in addition to symptoms. Another was the DSM-III-R classification of melancholia which included items such as absence of any pre-morbid personality disturbance, previous episodes with good recovery and previous good response to somatic therapies in addition to symptoms.

Narrower strategies have been evaluated. First, weighting and measuring signs of psychomotor disturbance (PMD), with the view that such observable signs are surface markers of underlying neuropathological processes in melancholia, a model reflecting PMD's longstanding position as a central marker of melancholia (Berrios, 1988). Following on Widlöcher's (1983) development of a refined measure, we developed the observer-rated CORE measure (Parker and Hadzi-Pavlovic, 1996b) of PMD—with that measure so named to capture its reference to 'core' signs of melancholia. Limitations to rating signs validly include the reality that not all patients present at the nadir or depths of their depressive episode and that the motor signs of PMD are seemingly less overt or severe in younger melancholic patients.

In the last few years, we have favoured diagnostic measurement melding clinical features and non-symptom correlates, and offer several reasons. First, the approach concedes limitations (just detailed) to relying on any symptom set alone. Second, it reflects a number of the longstanding prototypic ascriptions to the concept of melancholia—with even its synonym 'endogenous depression' proceeding beyond symptoms. Third, it is consistent with the approach to defining many medical conditions (e.g., Parkinson's disease) and where diagnosis is based on a range of antecedent and course of illness factors in addition to symptoms. Fourth, we have already demonstrated (Parker et al., 2010) that adding course of illness and context variables to refined symptoms actually improves delineation of melancholic and non-melancholic depression made by symptom definition alone—and in that report made an analogy to navigational strategies that rely on multiple reference points to improve precision. Further, it acknowledges the likely reality that melancholia is 'fuzzy', and suggests that definition might better be weighted to prototypic delineation rather than to seeking absolute definition.

We therefore developed (Parker et al., 2012) the SERDEX measure (SElf-Report of Depressive EXperiences) which lists 12 items weighted to melancholic depression in a left-hand column and 12 items weighted to the non-melancholic depressive conditions in a right-hand column. Individuals are invited to tick any item from either column that they regard as 'characteristic' in terms of their depressive experience, whether (dependent on the clinical or study objective) experienced currently or over time. The listed items assess symptoms historically favoured as most differentiating of melancholic and non-melancholic depression, but also assess premorbid interpersonal functioning, distal and proximal stressors, the context and impact of proximal stressors on inducing and maintaining the depression, and trait emotional dysregulation levels. Each item was selected and often progressively refined in its definition by considering its utility in previous studies undertaken by our research group over the last twenty years (e.g., Parker and Hadzi-Pavlovic, 1996a; Parker et al., 2010) and with all having been tested empirically to quantify their differentiating potential. For example, while early morning wakening is commonly listed as a symptom of 'endogenous' or 'melancholic' depression, we have never quantified it as having distinctive differentiation across melancholic and non-melancholic depression and it was therefore not included. After ticking relevant *items*, respondents are then requested to judge whether their 'profile' or clinical *prototype* is best captured by Description A (left-hand column descriptors), Description B (right-hand column), is somewhat closer to A than to B, is somewhat closer to B than to A, or is an equal mix or A and B descriptors—with this second 'prototypic' measurement component seeking to determine overall 'pattern' correspondence to melancholic or non-melancholic depression. For the present study we developed an equivalent clinician-rated version of the measure.

We reported the properties of the initial self-report measure in an earlier paper (Parker et al., 2012) with that development study involving a sample of 141 unipolar depressed patients assessed at