

Biological psychiatry and psychosocial insights

Biological research has established itself as the cutting edge of contemporary psychiatry. However, through its own limitations it has at the same time actually created an increased awareness of psycho-social aspects of biology.

The condition of depression is a useful vehicle for exploring progress made in determining biology and regenerating psychosocial insights.¹ A biology of depression has been understood over the years through a number of pivotal findings related to diminished neurotransmitter breakdown products (in both urine and cerebrospinal fluid), cortisol levels and sleep architecture. The development of drugs for the treatment of depression demonstrates the power of serendipity. Through observations of mood changes in non-psychiatric patients being treated for hypertension (using reserpine) and tuberculosis (using isoniazid) an understanding of neurotransmitter functioning and related enzymatic processes contributed to the "monoamine hypothesis" that has influenced our thinking and determined the pharmacology of intervention.

Current insights, through our ability to view the brain at both a structural and functional level, have taken us beyond what may now seem to be simplistic explanations. The discovery of volume depletion in the hippocampus of depressed individuals has potentially seen the beginnings of a new era of understanding of the biology of depression. It is known that the hippocampus is rich in glucocorticoid receptors, and that elevated cortisol leads to cell apoptosis in this region. Further, the capacity for neurogenesis in this area has been determined through research on patients with active and treated Cushing's syndrome. Most recently, animal studies have demonstrated that ablation of the hippocampal area leads to inhibition of expected behavioural responses to antidepressants.²

The discovery of a substance known as brain derived neurotrophic factor (BDNF) which has an ability to reverse hippocampal brain apoptosis i.e. promote neurogenesis, adds to an emerging picture: antidepressants promote release of BDNF through gene stimulation and far from being simply monoamine manipulators, are actually gene stimulators. This would appear to be a most interesting and exciting insight. However, one must be wary of biological reductionism. If we have learnt one thing, the brain is a complex organ governed by multiple interactive systems. We may simply have a more advanced understanding of a piece of the puzzle.

Biological findings have shifted theories of causation in a novel direction: However, intriguing data caution against an absolutist approach. To illustrate the point, what sense is one to make of recent findings that the "placebo response" is an actual response

both clinically and biologically? Changes in regional cerebral blood flow³ and brain glucose metabolism⁴ occur in response to an inert substance, different to those occurring in response to active substance, but with the same outcome. The emergence of data which concludes that psychotherapy (interpersonal) is as effective as pharmacotherapy⁵ poses further questions e.g. is talking a biological intervention?

Social elements are critical components of our understanding of depression, and they include life stress and socio-economic status. The theory of depression as a biologically mediated response to stress is supported by research consistently documenting the association of depression with earlier life stress.⁶ Furthermore, an enduring finding related to an inverse relationship between socio-economic status and depression⁶ emphasizes social aspects of aetiology.

In the exploration of biological aspects it is prudent to retain focus on the psychosocial components that shape and influence biology.⁷ As a holistic discipline, the reawakening of psychosocial thinking through biological endeavors signals a critical step forward and strengthens the bio-psychosocial ethos of our discipline.

References

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