The biopsychosocial model, stress, and the efficacy of mindfulness therapy

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Abstract

A thorough review of the Biopsychosocial Model, stress, and the efficacy of mindfulness therapy is warranted and would provide a theoretical foundation upon which to uncover methods of treating stress. While the biomedical model of health has dominated the thinking and actions of healthcare practitioners for the past 300 years (Slife & Wendt, 2006), it tends to reduce illness to what Taylor (2009) calls "low-level processes," placing emphasis on the biological processes such as disordered cells and chemical imbalances rather than psychological or environmental processes. In addition, this model only takes into consideration one factor for illness. An examination will be made into the Biopsychosocial Model, stress, and the efficacy of Mindfulness Therapy in treating stress. Understanding how the body reacts to stress, and the way Mindfulness affects perceived stress will pave the way for future studies and understanding of the variables that affect cortisol levels and inflammation in the body.

The Appropriateness of Psychology in Utilizing the Biomedical Model

The biomedical model of health has dominated the thinking and actions of healthcare practitioners for the past 300 years (Slife & Wendt, 2006). It is used by physicians in the diagnosis of disease and has been emulated by the American Psychological Association (APA) in defining evidence-based practice (Taylor, 2009; Slife & Wendt, 2006).

The biomedical model defines an individual's health as the freedom from disease, pain, or defects, and focuses on physical bodily processes such as pathology, biochemical imbalances, and neurophysiological abnormalities (Taylor, 2009). The model views psychological and social processes as separate from the pathophysiology of disease (Taylor, 2009).

The biomedical model has some advantages for studying some disease processes, but has various drawbacks. First, it reduces illness to what Taylor (2009) calls "low-level processes," placing emphasis on the biological processes such as disordered cells and chemical imbalances rather than psychological or environmental processes. Second, this model only takes into consideration one factor for illness as opposed to considering that biological processes are but one factor in the pathophysiology of illness development. Third, it is based on a dualistic approach, separating the mind from the body. Last, it places much more emphasis and importance on illness as opposed to health.

The biomedical model poses problems for the explanation of some illnesses. It fails to explain why under the same stressors and exposure, some will develop an illness while others do not (Taylor, 2009). Additionally, it does not explain how having genetic markers for a certain illness or disease does not guarantee the onset or development of that illness or disease (Daruna, 2004; Taylor, 2009). There are many factors that have an impact on the development of illness. Biological process is but one factor. There is a strong body of evidence that indicates that psychological stressors, environmental factors, and social influence can all play a role in the development of illness (Daruna, 2004; Taylor, 2009).

As a result of the difficulties that the biomedical model faces, there has been a shift towards the adoption of a biopsychosocial model of health and illness (Taylor, 2009). The biopsychosocial model maintains that illness is a result of numerous factors to include biological, psychological, and social that all provide influence at the macro and microlevels (Taylor, 2009). Additionally, the biopsychosocial model does not separate the mind from the body, positing that both the mind and body can have profound effects on the health of an individual. Moreover, this model takes into account both health and illness, which results in the view that health is achieved through a combination of biological, psychological, and social needs being met.

In order to explain the interaction of the microlevel biological variables and macrolevel psychological and social variables, a systems theory is adopted. The systems theory posits that every level of an organism is linked hierarchically, and a change at one level will bring about change in all other levels, Taylor, 2009; Daruna, 2004). Thus a change at the cellular level will...
affect an organism at the psychological and social level, and a change at the psychological and social level will bring about change at the cellular level (Taylor, 2009). The interrelated nature of an organism is extremely complex and researchers have barely scratched the surface to explain the exact nature of the interactions that take place (Daruna, 2004). This interaction requires collaboration and interdisciplinary thinking across many fields, as well as multivariate testing approaches and statistical methods (Taylor, 2009).

The implications for the biopsychosocial model in clinical practice are many. It must take into account that there are combinations of biological, psychological, social, and environmental factors in the diagnosis of individuals, which may necessitate an interdisciplinary approach to diagnosis (Taylor, 2009; Daruna, 2004). Additionally, any recommendations for treatment must also take into account these factors, which will help to individualize treatments to the specific needs of the individual, increasing the efficacy of the treatment (Taylor, 2009; Daruna, 2004). Moreover, the biopsychosocial model places a strong emphasis on the relationship between the patient and practitioner (Taylor, 2009; Daruna, 2004). Having a strong effective relationship can improve patient services, increase treatment efficacy, and reduce the amount of treatment time for improved symptomology (Taylor, 2009).

The APA has attempted to follow in the footsteps of the biomedical model, which states that “evidence-based practice is the integration of best research evidence with clinical expertise and patient values.” The biomedical model has longed used RCTs as its basis for testing for the efficacy of treatments with patients, tends to reduce human beings to their mechanical parts, and separates the mind and the body. When they test a certain drug and its effect on a specified condition, they have already made the assumption that it is the drug that is having the effect on a specific condition. The AMA has only recently started to concede that there is a huge psychological component to disorders and conditions and that these can have a huge impact on an individual’s health and well-being.

The question at hand is whether the field of psychology should be following in the footsteps of medical science. It seems that psychology in its attempt to justify itself and be considered a pure science has ultimately regressed in the ability to accomplish what the forefathers and foremothers intended. William James (1842-1910) considered all aspects of an individual as important, both sensory and non-sensory (Slife and Wendt, 2006). Psychologists have unintentionally removed the human component from psychology. Very few fit into textbook definitions, and until researchers start looking outside the box, the field will remain stagnant. There are multiple facets to all disorders, and textbook definitions and RCTs are only part of the overall picture. Slife and Wendt (2006) point out, psychologists must look at all of the tools in their toolbox and pick those tools for the job at hand. There is no such thing as a one tool fits all. This is why Psychology best fits into a Biopsychosocial Model as it encompasses all aspects of an individual.

The Effects of Stress and Anxiety on Depression and Immunomodulation

If an understanding is to be gained in how the Biological, Social, and Psychological components work on the overall well-being of an individual, an examination needs to be made in the role our emotions play in overall health. In particular, how do negative emotions such as stress and anxiety affect inflammation and overall immune response. Does the psychological state of an individual play a role in the health of an individual, and to what extent can an individual perceptions effect the overall health and well-being?

O’Donovan et al. (2010) posits that research has been lacking in regards to the effect that anxiety has on depression, immune response, and inflammation. According to O’Donovan et al. (2010), this is extremely important because anxiety may be a better predictor of inflammation than depression. O’Donovan et al. (2010), posit that a key role in understanding the relationship that exists between anxiety and inflammation is in the cognitive biases individuals may have towards threat-related information. There is increasing evidence that when an individual is exposed to either real or imagined psychological threats, various biological systems are activated.

Of particular interest is the activation of the hypothalamic–pituitary adrenal (HPA) axis, which regulates inflammation responses in the body through the release of the hormone cortisol (Slavich et al. in O’Donovan et al, 2010). O’Donovan et al. (2010) posit that anxious individuals who experience continual and exaggerated perceptions of threat can result in that individual having a chronic activation of the biological stress response. This in turn can lead to a reduced secretion of cortisol in the morning and an increase in
inflammation (Miller et al., 2007; Kiecolt-Glaser et al., 2003 in O’Donovan et al., 2010).

O’Donovan et al. point out that early research was based on a generality model based on Selye (1956). The generality model made the assumption that physical and psychological stimuli resulted in common biological reaction patterns. More recently, Kemeny (2003) and Moons et al., (2010) proposed an integrated specificity model, which hypothesizes that the biological response patterns are specific depending upon the specific psychological state. Utilizing the integrated specificity model, O’Donovan et al. examined the relationship between cortisol levels and anxiety. Although anxiety and depression are often present in a comorbid condition, and that both anxiety and depression overlap with neuroticism, O’Donovan et al. (2010) hypothesized that clinical anxiety will have a specific effect on cortisol levels and inflammatory markers independently of depressive symptoms and neuroticism.

Utilizing an experimental design, the study included 27 participants in the experimental (anxious) group and 29 participants in the control (non-anxious) group. Participants were assigned based on their score on the Hospital Anxiety and Depression Scale (HADS-A), with the experimental group scoring in the clinical range and the control group scoring low on this scale. All of the participants were white with 68% being female. Participants who had chronic illness, acute illness within the previous two weeks, possible current infection, alcoholism, use of medication, anesthesia in the previous three months, and night shift work in the previous two weeks were excluded.

Data was collected on participants for neuroticism, utilizing the 12-item Eysenck Personality Questionnaire-Revised, Short Form, health status through self-report measures, salivary cortisol levels, utilizing a high sensitivity salivary cortisol enzyme immunoassay, and pro-inflammatory cytokine interleukin-6 (IL-6) and systemic inflammatory marker C-reactive protein (CRP) levels through blood samples. All biological samples were taken in the morning between the hours of 7:30am and 9:30am, and participants were required to follow a protocol of fasting, not smoking, no exercise, and avoidance of alcohol for a specified period of time prior to the experiment. Following the biological sample collection, participants completed the questionnaires.

Continuous variables were assessed using Pearson’s correlations, while Student’s t-tests were used to assess the relationships in cortisol, IL-6 and CRP between the two groups. O’Donavan et al., in order to test whether differences were independent of potential covariates, then reran their model, controlling for age, gender, depression and neuroticism using analysis of covariance. The results showed that there were lower levels of morning cortisol and higher levels of the pro-inflammatory cytokine IL-6 among the clinically anxious group. This difference was apparent even when they controlled for depression and neuroticism, which indicated some specificity in the relationship between anxiety and IL-6. There was no difference in the levels of CRP among the two groups. One hypothesis for this is that there are differences within the psychosomatic pathway that leads to the production of CRP as opposed to IL-6. This also provides evidence in support of specificity in regards to negative emotional states and the neural and hormonal responses that lead to inflammation. O’Donavan et al., posit that cognitive biases towards what an individual perceives as threatening may be exaggerated among anxious individuals, which leads to prolonged activation of the biological stress response. This in turn results in inflammation (O’Donavan et al., 2010).

Although the study sample was small, it did shed light on two areas. First, it exposed the potential vulnerability to inflammation among individuals who are clinically anxious. Second, it provided evidence to support varying biological response mechanisms based upon perceived threat by the individual. This study provided a base for expounded research on perceived threat and inflammation response.

Understanding the links between anxiety, depression, and the immune system will help provide efficacious treatments for these disorders and well as provide potential prophylactic practices to reduce onset (Test, 2010). Understanding these mechanisms lead us to examining ways in which to reduce perceived or real stress and resulting depression. Through the mitigation of stress and anxiety, a reduction may be seen of overall depression and an increase of overall well-being can occur.

The Efficacy of Mindfulness Therapy to Mitigate Stress and Depression

As can be seen, evidence supports the effects of anxiety, stress, and depression on the immune system and inflammation. One method that has been gaining attention to mitigate the stress and anxiety is Mindfulness Therapy. The question remains, however, as to the efficacy of this therapy. A variety of articles can be found on “mindfulness therapy to treat
depression”, especially as more researchers work toward determining it’s efficacy in treating different physical and mental health problems. To discern the efficacy of Mindfulness as a treatment method for addressing anxiety and depression, a review of the literature was conducted in order to compare the data.

A recent article was published in the Archives of General Psychiatry on “Antidepressant Monotherapy vs. Sequential Pharmacotherapy and Mindfulness-Based Cognitive Therapy, or Placebo, for Relapse Prophylaxis in Recurrent Depression”. The researchers operationalized the definition of relapse prophylaxis. The findings of their sample from individuals in depression remission while on antidepressant medication. They utilized treatment options of mindfulness-based cognitive therapy (MBCT) and antidepressant medication, which were compared to a control group on placebo. The experiment tested three condition; the efficacy of using MBCT for relapse prophylaxis in recurrent depression, the efficacy of using antidepressant medication for relapse prophylaxis in recurrent depression, as well as the control group placed on placebo. This study demonstrated that MBCT offers protection against depression relapse or recurrence for people that had previously been treated with antidepressants, in par with maintenance pharmacotherapy with antidepressants. While this therapy should not be used as a substitute for other treatments (Thomas, 2011), it provides evidence that MBCT may be effective as a prophylaxis to prevent depression relapse in people that had previously been on antidepressants (Segal et al., 2010).

A meta-analysis on the effects of mindfulness-based therapy on anxiety and depression was conducted by Hofmann, Sawyer, Witt, & Oh (2010). The results of this analysis concluded that mindfulness-based therapy was a promising intervention in treating anxiety as well as mood problems. The studies analyzed only dealt with mindfulness-based therapy modeled the same as Mindfulness-based Stress Reduction (MBSR) and Mindfulness-based Cognitive Therapy (MBCT), such as in duration and as an in-person experience (Hofmann et al., 2010). The authors evaluated the quality of each included study (Hofmann et al., 2010). This would help them ensure the validity of the studies they included. The authors began the analysis with a critical view of the efficacy of mindfulness-based therapy, and were surprised at the effects being robust and strong as they were (Hofmann et al., 2010). Overall this meta-analysis provides a helpful resource for writing about the topic of mindfulness therapy for depression.

Kuyken et al., (2010) conducted a study on the mediating factors in the treatment effects of Mindfulness-based Cognitive Therapy. Kuyken et al., (2010), posed the question of whether Mindfulness-based Cognitive Therapy effects are mediated by the enhancement of mindfulness and self-compassion across treatment, and/or by alterations in post-treatment cognitive reactivity. This was embedded in a randomized controlled trial comparing the MBCT with maintenance antidepressants (mADM). They clearly defined their sample size of individuals in full or partial remission of depression. They clearly defined their treatment options of mindfulness-based cognitive therapy (MBCT) while mADM was omitted, and the use of mADM. Variables included the severity of depressive symptoms, mindfulness and self-compassion via self-reporting instruments; and cognitive reactivity. This study offered valid explanation as to how MBCT works while also providing opportunity for future study to test hypothesis about the mechanisms of change of MBCT. Overall, this article provides a credible resource for explanation of how mindfulness-based cognitive therapy works.

Implications for Further Research

The link between stress and the reaction to it are very complex. What each person experiences in quite different, and includes both physiological and psychological factors (Kut et al., 2007). The effect that stress has on overall immune and inflammatory response is well established (Chapman, Tuckett, & Song, 2008; Coe & Laudenslager, 2007). There is a need, however, to understand the mechanisms involved that make individuals more susceptible to stress and more efficacious ways to treat it.

Understanding the role that the Biopsychosocial model and Mindfulness Therapy play in pain in the mediation of inflammation that stress can cause is of extreme importance to the potential for developing effective programs for high risk individuals. Self-esteem and perceived stress both correlate with higher depression and lower dispositional hope among individuals (Strom, 2006), and may be pivotal factors in how an individual copes with their situation and how this in turn affects immunomodulation. In addition, understanding how Mindfulness affects perceived stress will pave the way for future studies and understanding of the variables that affect overall stress levels.
References


