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DOI 10.1002/wps.20126

Psychiatry and social nutritional neuroscience

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Cacioppo et al (1) elegantly outline potential contributions of social neuroscience to psychiatry. Their interdisciplinary approach could be enhanced by incorporating a nutritional perspective. Indeed, although the human brain represents only about 2% of human body mass, it accounts for ~20% of the total resting metabolic rate (2). As a consequence of the brain's intense energy requirements, metabolic aberrations can have substantial consequences for its function. In this commentary we focus on the progress and potential of social nutritional neuroscience, an area of growing interest and importance, for psychiatry.

In traditional terms, nutritional neuroscience focuses on the effects that various dietary components have on neurochemistry, neurobiology, behavior, and cognition. Social nutritional neuroscience takes a broader view that incorporates key bidirectional influences: social processes and behavior impact diet, both of which affect neurochemistry and neurobiology. These resulting dietary and biological changes may subsequently alter social and behavioral processes, ultimately creating a feedback loop. Studies addressing the dietary and biological consequences of depression help demonstrate the importance of viewing these relationships as a two-way street.

Depression can have a substantial effect on food intake. Appetite changes are a notable feature of major depressive disorder. In fact, one of the diagnostic criteria for this disorder in the DSM-

5 is diet-relevant: weight gain/loss or hyperphagia/hypophagia. Depression also influences dietary preferences (3): for example, some people increase comfort food intake when depressed (4).

Depression and diet can impact the same physiological systems. Mechanistic studies have shown how depression can modulate key pathways to inflammation including sympathetic activity, oxidative stress, transcription factor nuclear factor kappa B (NF- κ B) activation, and pro-inflammatory cytokine production (5). Diet affects inflammation and modifies brain function through these same processes (3,5). Both depression and stress also have well-documented negative effects on vagal activation. Because the vagus nerve innervates tissues involved in the digestion, absorption, and metabolism of nutrients, vagal activation can directly and profoundly influence metabolic responses to food, as well as inflammation, contributing to the lively interplay between the brain and the gut (5).

Diet and immune alterations may promote depression. Growing evidence suggests that people with poorer quality diets have a higher likelihood of being depressed than people with better quality diets (6). Furthermore, pro-inflammatory cytokine administration induces "sickness behaviors", i.e., behavioral changes that resemble the somatic symptoms evident in depression, like anhedonia and lethargy (7). Accordingly, depression may start a negative cascade whereby depression promotes dysregulated food consumption and physiological responses which, in turn, further enhance depression.

The bi-directional links among depression, diet, and biological responses are nicely illustrated by the relationship between depression and obesity, which is clearly linked to both diet and dysreg-

ulated physiological responses. For example, clinical depression and obesity often travel together (8). The risk for developing depression over time is 55% in obese persons, and depressed people have a 58% increased risk of becoming obese (8). In addition, a large prospective study showed that older depressed adults gained visceral fat over five years, while non-depressed adults lost visceral fat (9). Importantly, this association did not reflect changes in overall obesity, suggesting that depressive symptoms were specifically associated with changes in visceral fat, a central and important contributor to inflammation. Functioning as an endocrine organ, adipose tissue secretes a number of different peptide hormones and cytokines that influence brain function, metabolism and behavior (3).

While a poor diet increases risk for depression, a healthy diet may be protective. In a prospective study with over 10,000 participants, those who ate a Mediterranean diet that was rich in monounsaturated fats, fish, fruit, nuts, and vegetables had a lower risk for depression four years later than people who consumed diets with fewer of these foods and higher amounts of saturated fats (10). Healthy diets may also reduce anxiety symptoms, in addition to depressive symptoms (6).

After summarizing key issues in social neuroscience and its potential contribution to psychiatry, Cacioppo et al (1) concluded that neuroimaging and genetic research that focuses on specific component processes underlying social living is needed. In the field of social nutritional neuroscience, similar neuroimaging and genetic research would be valuable. In fact, an interdisciplinary approach that incorporates both social neuroscience and social nutritional neuroscience could foster unique and

provocative questions that would further our understanding of psychiatric disorders. A broader emphasis on the role of behavior as a key driver in nutritional neuroscience could also open up new vistas and prospects for future research.

Acknowledgement

Work on this commentary was supported in part by NIH grants CA158868, CA172296, and CA154054.

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DOI 10.1002/wps.20127