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The Importance of Psychoneuroimmunology for Social Workers

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Abstract

A wealth of information regarding how the immune system can influence the brain and result in changes in mood and behavior has accumulated. Inflammation is a causal factor in some cases of major depression and psychotic disorders, and predicts whether trauma will result in Post Traumatic Stress Disorder (PTSD). Fortunately, studies in the area of psychoneuroimmunology have also suggested ways to decrease inflammation. Knowledge of this information is vital for social workers so that the impact of their interventions can be maximized. Moreover, for macro-practice social workers the information underscores the importance of access to nutritional food, access to safe places for exercise, and the time for food preparation and exercise, which should be considered as social justice issues.

Key Words: psychoneuroimmunology, inflammation in depression, inflammation in psychosis, microbiome, vaccinating against PTSD, yoga and distress

The Importance of Psychoneuroimmunology for Social Workers

The emerging field of psychoneuroimmunology investigates how the immune system alters behavior, mood, the brain, and hormones. New data suggest that inflammation plays a causal role in at least some cases of major depression, Post Traumatic Stress Disorder (PTSD), psychosis, as well as more ephemeral variations in mood. Inflammation refers to "the local accumulation of fluid, plasma proteins, and white blood cells that is initiated by physical injury, infection, or a local immune response" (Murphy, 2012, p. 805). This paper will review the evidence for inflammation playing a role in a broad range of behavior. This paper will also review the findings regarding those life style changes that are associated with a decrease inflammation. Social workers are in a unique position to assist clients in making these salubrious life-style changes.

Before getting started, a little preliminary information on the immune system may help to clarify later discussion. The immune system, that is the events orchestrated by white blood cells (leukocytes), is divided into two branches: the innate system and the adaptive system. The innate system involves the types of white blood cells that are involved in creating inflammation. Although this system is vital for fighting bacterial infections, it needs to be quickly downregulated when the pathogen is cleared because these cells release reactive oxidative species that can destroy a pathogen but also the host's cells. In contrast to the innate system, the adaptive system is recruited with vaccination and involves antibody production. The leukocytes involved in the adaptive system only target one specific protein (usually from a pathogen) and are less involved in creating inflammation. Work by Steven Cole (2014) suggests that in some ways the adaptive and innate systems are inversely related. Cole gathers people's white blood cells and then investigates which genes are being expressed and used to make proteins. For those persons whose fight/flight systems are strongly activated, proteins involved in creating inflammation are upregulated and proteins of the adaptive system needed for fighting cancer and viruses are downregulated. This pattern is also seen in those who are socially disconnected (Moleni et al., 2015).

Conversely, Cole found that people who have higher levels of psychological well being, which included having a sense of purpose, were stronger on the adaptive arm of the immune system and less strong on innate/inflammation arm (Fredrickson et al., 2015; Frederickson et al., 2013). When the word "inflammation" is employed in this paper, it generally refers to events orchestrated by the innate immune system.

Depression

Findings Suggesting that Inflammation Plays a Role in Depression

Inflammatory markers in blood are on average higher in those with depression compared to healthy individuals (Raison, Capuron, & Miller, 2006; Schiepers, Wichers, & Maes, 2005) and this was confirmed in a recent meta-analysis (Köhler et al., 2017). Setiawan et al. (2015) imaged activated white blood cells in brain and found that those with depression, on average, had more activated white blood cells. Other studies have reported on the percentage of the sample of depressed individuals showing elevations in inflammatory markers. Raison et al. (2013) found elevations in 45% of their depressed sample and Tartter, Hammen, Bower, Brennan, and Cole (2015) reported elevations in 1/3 of depressed persons.

Several studies have found that genetic variations associated with enhanced inflammatory capacity interact with childhood maltreatment or significant life stressor to increase the risk for the emergence of depression (Cohen-Woods et al., 2018; Kovacs et al., 2016). Animal work is consistent with strong inflammatory capacity being a risk factor for development of depressed behavior following the occurrence of a social stressor. Hodes et al. (2014) stimulated the white blood cells of their animals to determine which animals exhibited a strong inflammatory response and which animals did not. They then subjected their animals to social defeat. Those animals with the stronger inflammatory responses exhibited higher levels of depression in response to the social defeat. Moreover, when the strongly releasing leukocytes were transferred to the resilient animals, the resilient animals appeared more depressed after social defeat.

Raison et al. (2013) used an antibody to a major pro-inflammatory hormone to treat depression. For those with evidence of inflammation, the antibody resulted in a significant decline in depressive symptoms, although those depressed individuals with elevations in inflammatory factors often fail to respond to traditional antidepressants (Kiraly et al., 2017). Various other anti-inflammatories (aspirin, COX-2 inhibitors, ketamine) have demonstrated efficacy in ameliorating depression (Hodes, Kana, Menard, Merad, & Russo, 2015).

The Manipulated Variable Findings

Inflammation can be created by injecting the wall of a bacterium (lipopolysaccharide or LPS) under the skin. In work with animals, LPS injection results in depressed behavior (Dantzer, O'Connor, Lawson, & Kelley, 2011; Dantzer et al., 1998). A number of researchers have injected LPS into human subjects and noted the following: increases in depressed mood, anxiety, and memory problems (Kullman et al., 2013; Yirmiya et al., 2000); more activity in anxiety centers of the brain when an individual views angry faces (Inagaki, Muscatell, Irwin, Cole, & Eisenberger, 2012); a lowered threshold for physical pain (Wegner et al, 2014); less activity in brain reward circuitry in response to money (Eisenberger et al., 2011); and more activation in social pain areas of the brain in response to social exclusion, although this heightened brain activation was only noted in females with induced elevations in inflammatory markers (Eisenberger, Inagaki, Rameson, Mashal, & Irwin, 2009);

Interferon alpha, an inflammatory hormone, has been used as a treatment for melanoma and hepatitis. Across studies, 20 to 50% of persons receiving IFN-alpha become depressed (Raison, Dantzer et al., 2010).

The Relationship between Stress and Inflammation

A wide variety of types of stress are associated with inflammation. Lonely people (Cole et al., 2007) and caregivers of Alzheimer's patients (Kiecolt-Glaser et al., 2003; Miller et al., 2008; von Kanel et al., 2006) exhibit elevations in blood markers of inflammation. In a study of British civil service workers, all of whom had access to health care, those who earned less and had less control over their work environments exhibited higher blood inflammatory markers after controlling for diet and smoking

(Steptoe et al., 2003). Similarly, those in low status jobs exhibit higher levels of inflammatory markers (Powell et al., 2013) as do those of higher social status who experience financial stress (Sturgeon et al., 2016)

By inducing stress in subjects, researchers have shown that there is a causal connection between stress and inflammation. In an animal model of major depression, researchers have subjected rodents to inescapable shock. Subsequent to shock, the animals exhibit signs of depression (less activity, less consumption of sweetened liquid) and elevations in brain levels of inflammatory hormones (Maier & Watkins, 1998; Wohleb, Franklin, Iwata, & Duman, 2016). However, if a chemical sponge for an inflammatory hormone is placed into the brain, the rodents behave normally (Maier & Watkin, 1998).

Researchers have also stressed people and then measured markers of inflammation. Pace et al. (2006) used the Trier Social Stress Test during which people give a speech about an embarrassing moment to a hostile audience. This procedure reliably elevates markers of inflammation in blood. Researchers have also had people play an interactive cyberball game on the computer from which they are suddenly excluded. The socially excluded exhibit activation of stress/pain centers in brain (Eisenberger, Lieberman, & Williams, 2003). Moreover, the level of brain activation to the cyberball social exclusion correlates with magnitude of inflammatory activation as measured during the Trier social stressor test (Slavich, Way, Eisenberger, & Taylor, 2010). There is reason to believe that the inflammatory factors are causing the activation in the brain's stress/pain centers in the cyberball task. If acetaminophen, which decreases inflammation, is provided before playing the cyberball game, then activation in the stress/pain areas of the brain fails to occur (DeWall et al., 2010).

Vulnerability to Post Traumatic Stress Disorder

Prior levels of inflammation may be a risk factor for PTSD given subsequent occurrence of trauma. Work with people has confirmed this hypothesis (Eraly et al., 2014). Moreover, consistent with inflammation playing a causal role in the emergence of PTSD, an early or steeper rise in inflammatory factors after trauma predicts later symptoms of PTSD (Pervanidou et al., 2007; Sumner et al., 2018).

A study by Reber et al. (2016) raised the possibility of being able to vaccinate against PTSD.

Exposure to a particular type of bacteria can elicit an anti-inflammatory response and can activate neurons called anti-panic neurons in the brain (Lowry et al., 2007). Reber et al. vaccinated mice with this bacteria and then subjected them to intrusion by a predator. The vaccinated animals, compared to the sham vaccinated, displayed little anxiety and were far less submissive. Using the same vaccination procedure, Fox et al. (2017) found that the vaccinated animals were faster to extinguish a conditioned fear.

The anti-panic neurons that are activated by the bacteria can also be activated by heat (Hale et al., 2017). Janssen et al. (2016) tested heat as an antidepressant, increasing people's body temperature to 101 degrees which required about 20 minutes of heat application. This intervention ameliorated depression for about six weeks.

Psychosis

The proximal cause of hearing voices is caused by too much dopamine release. However, Grace and Gomes (2018) argue that defects in the dopamine system have not been found in those with psychosis. In contrast, there is a great deal of evidence implicating disturbances and loss of neurons located in the hippocampus that control dopamine release (Grace, 2016). The activity of these particular neurons, called fast-spiking GABA interneurons, is driven by NMDA receptors on these neurons. Inflammation and associated reactive oxidative species released during inflammation can interfere with NMDA signaling and dampen the activity of the fast-spiking GABA interneurons or result in their death (Chung, Fish, & Lewis, 2016; Do, Cabungcal, Frank, Steullet, & Cuenod, 2009; Grace, 2016; Miller & Goldsmith, 2017). (The details of this story are reviewed in Littrell, 2015, Chapter 6).

Much evidence is available attesting to brain inflammation being associated with psychosis. Proteins associated with inflammation have been found in the blood of those with schizophrenia (Yee et al., 2017). In a location more proximal to the brain, inflammatory factor elevations have been noted in the cerebrospinal fluid in many studies (Müller, Weidinger, Leitner, & Schwarz, 2015; Wang & Miller, 2017). Van Rees et al. (2018) took blood from first-episode, drug naïve persons with psychosis and mixed them with brain white blood cells (called microglia). The blood of those with psychosis resulted in activation of the microglia, although blood from controls did not. Consistent with the idea that the

presence of substances that can activate white blood cells resulting in symptoms of psychosis, relapses to hearing voices is often occasioned by urinary tract infections (Miller et al., 2013). Finally, it is now possible to image (see) activated white blood cells in the brain. Several such imaging studies have found evidence of activated white blood cells in brain in non-medicated persons with psychosis (Bloomfield et al., 2016; Doorduin et al., 2009; Van Berckel et al., 2008), however, there are failures to replicate (Miller & Goldsmith, 2017).

A history of trauma has been noted in those for whom psychosis later emerged (Aas et al., 2017, Heins et al., 2011). As discussed earlier, stressful events do elevate inflammatory factors in the brain (Jiang et al., 2013). Thus, inflammation may explain the link between trauma and psychosis. Inflammation might also explain the many studies showing that an inflammatory state in the mother during gestation is associated with elevated risk for psychosis in the offspring (Müller et al., 2015) and the fact that variants of genes coding for immune system proteins elevate the risk for psychosis (Müller et al., 2015; Pouget, 2018). Finally, in those treated with an inflammatory hormone (interferon alpha) for conditions such as hepatitis C or melanoma, although the emergence of depression was more common, in one study 0.1% developed psychosis and in another 0.4% developed psychosis. In both samples psychosis resolved with the discontinuation of the inflammatory hormone therapy (Miller & Buckley, 2016).

In an attempt to prevent the emergence of psychosis in those youth at risk, several interventions have been attempted. Antipsychotic medications were not effective. However, omega-3s, whose anti-inflammatory properties are well-recognized (Serhan, 2017), did reduce the emergence of psychosis in one cohort at 6.7 year follow-up (9.8% versus 40%) (Amminger, Schafer, Schlogelhofer, Klier, & McGorry, 2015), although not in another cohort at one year follow-up (McGorry et al., 2017). Moreover, higher blood levels of omega-3 were predictive of an absence of psychosis at seven-year follow-up (Mossaheb et al., 2017). Anti-inflammatory approaches have been successful in reducing symptoms of psychosis whether in those carrying a diagnosis of bipolar (Husain, Strawbridge, Stokes, &

Young, 2017) and schizophrenia (Melbourne, Feiner, Rosen, & Sharma, 2017; Miller & Buckley, 2016; Sommer et al., 2014).

A particularly poignant illustration of the profound impact of brain inflammation on behavior is the story of Susannah Cahalan, a journalist. Susannah (2012) describes her journey into psychosis in her book, *Brain on Fire: My month of madness*. Susannah details her symptoms of mania, paranoia, production of verbal word-salad, and catatonia through which she cycled. Successful treatment was only achieved when confirmation of brain inflammation was made and addressed. In terms of explanation for why Susannah experienced inflammation, Susannah had had a bout of melanoma earlier which resulted in her white blood cell's production of antibodies to the NMDA receptors in her brain. In fact, antibodies to NMDA receptors are found in 1-10% of those with diagnoses of schizophrenia (Müller et al., 2015; Pollack, McCormack, Peakman, Nicholson, & David, 2014; Teixeira, Rocha, & Zhang, 2017). Also of relevance, the recognition that inflammation in the brain can manifest behaviorally in psychosis explains why symptoms of psychosis are found in those with brain inflammatory states associated with viral infections and various autoimmune diseases (Müller et al., 2015).

Perhaps in the near future, the recognition that brain inflammation can manifest as symptoms of schizophrenia and mania, as occurred for Susannah Cahalan, will call into question the diagnostic categories in the *Diagnostic and Statistical Manual* of the American Psychiatric Association. In fact, many have questioned whether Bipolar Disorder I and Schizophrenia share a common underlying causation and should be considered variations of the same disorder (Berk et al, 2011; Clementz et al., 2016; Guloksuz & van Os, 2017; Kuswanto et al., 2016).

What Can Be Done to Lower Inflammation?

Enhancing Vagal Tone

The Parasympathetic Nervous System (PNS) is a branch of the nervous system that controls internal organs and the vasculature. The PNS also sends projections to the lymph nodes, where white blood cells reside. The PNS can downregulate inflammation (Chavan, Pavlov, & Tracey, 2017; Pavlov & Tracey, 2017). A measurable read out of the strength of the PNS (called vagal tone) is heart rate

variability. Greater heart rate variability is better because it reflects better coordination between breathing and heart rate (Porges, 2007, 2011). Moreover, heart rate variability correlates positively with capacity for social engagement and better control over reactions to stress (Quintana, Kemp, Alvares, & Guastella, 2013).

There are many ways to increase vagal tone: meditation and yoga (Kiecolt-Glaser et al. 2010; Pace et al., 2009); cheap biofeedback equipment (Lehrer et al., 2003; 2006; Lehrer & Gevirtz, 2014) and, similarly, a phone app with breathing instruction called "the stress doctor" marketed by azumio (Littrell, 2015, p. 164); aerobic exercise (Coats et al., 1992; Hansen, Johnsen, Sollers, Stenvik, & Thayer, 2004); and time spent with trusted companions (Grippo et al., 2007; 2009; Kemp et al., 2012) or engaging in pro-social care-giving (Eisenberger & Cole, 2012; Quintana et al., 2013) which release oxytocin which, in turn, increases vagal tone.

Healthy Life Style

Exercise can prevent the rise of inflammatory factors elicited by stress (Hamer & Steptoe, 2007; Mathur & Pedersen, 2008), has anti-depressant effects (Babyak et al., 2000; Hoffman et al., 2011) and improves cognition in those with psychosis (Pajonk et al., 2010). Getting adequate sleep is important because sleep deprivation is inflammatory (Irwin, Wang, Campomayer, Collado-Hidalgo, & Cole, 2006; Zielinski et al., 2017). Yoga ameliorates mental distress and decreases inflammation (Balasubramaniam, Telles, & Doraiswamy, 2013; Black et al., 2013; Khalsa, 2013; Kiecolt-Glaser et al., 2010) and increases the expression of proteins needed for fighting viruses and cancer (Cole, 2014).

Dietary Interventions

Omega-3s are a type of fat richly abundant in seafood as well as other foods. There are receptors for omega-3 metabolites on white blood cells which slow down inflammation (Serhan, 2017). Omega-3s increase vagal tone (Hansen, Johnsen, Sollers, Stenvik & Thayer, 2004; La Rovere & Christensen, 2015) and promote a healthy gut environment (Forsythe, Sudo, Dinan, Taylor, & Bienenstock, 2010). Sublette, Ellis, Geant, & Mann (2011) evaluated the studies that employed omega-3s as a treatment for depression concluding that they do work. However, the optimal balance is greater than 60% EPA and less than 40%

DHA. In terms of other things to eat, curcumin, found in the Indian spice turmeric, is anti-inflammatory (Aggarwal, 2010; Lopresti et al., 2014; Shehzad, Rehman, & Lee, 2013) and does reduce symptoms of depression (Ng, Koh, Chan, Ho, 2017).

The research on omega-3s and mental health is new and funding for studies is less available than for pharmaceutical treatments. Thus, strong evidence is lacking. A Cochrane Review by Appleton, Sallis, Perry, Ness, and Churchill (2015) concluded that there was not yet sufficient high quality evidence to determine efficacy in treating major depression. Another Cochrane Review by Montgomery and Richardson (2008) also concluded there was insufficient evidence for making recommendations on omega-3s for bipolar disorder as did a Cochrane Review by Irving, Mumby-Croft, and Joy (2011) on omega-3s supplementation for schizophrenia.

The Microbiome

Attention has been focused recently on the microbes which live within us and outnumber the cells which belong to us (Cryan & Dinan, 2012). The species of bacteria that one harbors in the gut influence both mood and behavior. In a particularly convincing demonstration, Bercik et al. (2011) transferred the fecal contents from an intrepid mouse strain into the digestive tracks of a timid strain of mice as well as the reverse transfer. They showed that exploratory behavior ranging from timid to bold was determined by the type of fecal transfer. The timid mice were emboldened by the transplant of the intrepid mice feces. The intrepid mice became timid upon receipt of the feces from the timid mice. Not only did the fecal transplants impact the exploratory behavior of the mice, but there were commensurate changes in growth factors in relevant areas of the brain. Similar experiments have shown that fecal transplants from obese mice can make the thin mice obese (Collins, Kassam, & Bercik, 2013; Turnbaugh et al., 2006).

Particular gut microbiota correlate with behavioral characteristics in humans and mice. Recovery from psychosis is more likely in those with microbiota compositions more similar to those without a mental health diagnosis (Schwarz et al., 2017). Those with depression have an elevated occurrence of leaky gut (Maes, Kubera, Leunis, & Berk, 2012) and differences in microbiota composition (Jiang et al.,

2015). In work with hamsters which were engaged in an aggressive conflict, gut microbiota composition predicted which animal would win the conflict (Partrick et al., 2018).

What changes the gut microbiome? Stress encourages colonization by inflammatory microbiota species (Bangsgaard-Bendtsen et al., 2012; Rieder, Wisniewski, Alderman, & Campbell 2017; Watanabe, Arase, Nagaoka, Kawai, & Matsumoto, 2016), decreases the tight junctions which prevent microbes from exiting the gut and moving into circulation (thereby resulting in systemic inflammation) (Dinan & Cryan, 2012; Kiliaan et al., 1998), and decreases species diversity in the gut and reduces *Latobacillus* (which will be discussed later) (Partrick, et al., 2018). Exercise encourages anti-inflammatory species in the gut (Cook et al., 2016).

Diet has a major impact on the types of microbes found in the gut. Chassaing et al. (2017) showed that emulsifiers, which are added to many foods (e.g., bread, ice cream) to extend shelf life, change the composition of the microbiome and result in systemic inflammation. High fat diet does result in changes in microbiota and low-grade inflammation (Breitin, Gewirtz, & Chassaing, 2018). Consumption of fiber (for example, in apples) encourages anti-inflammatory varieties of microbiota (Macia et al., 2015; Tan et al., 2016); and yields a thicker protective mucosal layer in the gut creating a barrier so that bacteria cannot move into the blood stream (Breitin et al., 2018).

Some foods, called probiotics, actually introduce new bacteria into the gut. Yogurt and fermented milk products contain bacteria (e.g., *Lactobacillus casei*). Work with rodents showed that consumption of fermented milk products can prevent the stress-induced increase in inflammatory factors in brain (Ait-Belgnaoui et al., 2012) and can attenuate the stress-response of leaky gut and subsequent rise in the stress hormones cortisol and adrenaline (Ait-Belgnaoui et al., 2014). Research with people has shown that for those women who are high on a neuroticism scale, those who consumed more fermented milk products subsequently scored lower on a measure of social anxiety (Hilimire, DeVylder, & Forestell, 2015). Benton, Williams, and Brown (2007) found that for those with highest scores on depressed mood scale, probiotic consumption resulted in improved mood compared to placebo. Tillisch et al. (2013) had women consume fermented milk products for 4 weeks and then imaged their brains as they looked at

angry and fearful faces. Those women who had consumed fermented milk products exhibited less activity in areas of the brain involved in processing negative emotions (amygdala and the insula).

Presently, only a few randomized, placebo-controlled trials of probiotics have been conducted on clinical samples. In a randomized, double-blind, placebo-controlled study of persons with major depression, Akkasheh et al. (2016) found greater reduction of Beck depression scores as well as greater reduction on an inflammatory index in those consuming fermented milk products for 8 weeks. In a meta-analysis of 10 placebo-controlled, randomized, double-blind trials of fermented milk products compared to placebo including persons with depression and those without, Ng, Peters, Ho, Lim, and Yeo (2018) failed to find a difference in depression scores for the full sample. However, a sub-analysis of those with mild to moderate depression found lower depression scores for the probiotic group. There are fewer studies of probiotics in those with psychosis, although a negative trial of probiotics for those with schizophrenia was reported by Dickerson et al. (2014). A later study found that probiotics reduced the rate of rehospitalization in those with acute mania (Dickerson et al., 2018). Little information is yet available with regard to dosing, optimal delivery methods, and optimal species of bacteria to include in the probiotic.

Caveats

How strong is the case for inflammation being linked with behavioral outcomes? The manipulated variable research with animals and people provides the strongest support. With regard to questions such as how many individuals carrying particular diagnoses can be considered to be in inflammatory state, definitive answers are elusive. First, the lack of reliability of *DSM* diagnoses are legendary and have been criticized by leaders in our field (see Kirk & Kutchins, 1992). Failure to correctly classify individuals will mitigate finding relationships with other variables. Second, agreement on optimal measures of inflammation are lacking. Indeed, Del Guidice and Gengestad (2018) recently offered a well-reasoned critique on the use of often relied upon indicators of inflammation (viz., interleukin 6 and CRP) suggesting that often these measures reflect anti-inflammatory processes. Third, the categories in the *DSM* rely on symptoms, many of them requiring subjective judgements of the client,

not measures of what actually is happening in the brain/body. Perhaps, in the future, the categories in the *DSM* will be reorganized.such that depressive behaviors associated with inflammation will be categorized as a disorder distinct from depressive behaviors associated with some other underlying cause, whatever that might be. However, before this can occur, a consensus on the best method for diagnosing brain inflammation (brain imaging, spinal tap, blood markers, various types of antibodies) must emerge.

The reader may have noticed that many ways of influencing inflammation were discussed in the section on interventions. Unfortunately, it is not yet known which are the most powerful influences and whether, for example, consuming more omega-3s can offset a high rate of consumption of foods containing preservatives. Studies evaluating foods must ensure that the nutrient is absorbed properly. For example, curcumin absorbs better when consumed with black pepper (Aggarwal, 2011). Beyond this, the recent Cochrane Review failing to find a beneficial effect of omega-3 consumption on cardiovascular mortality, albeit finding some reduction in combined heart and stroke events (Abdelhamid et al. 2018) raises additional issues. Many of the studies in the Abdelhamid et al. review considered omega-3 capsules. Would the same results have been found if the omega-3 were consumed in food sources such as fresh fish, flaxseeds, chia seeds, or walnuts? In the future, there may be studies offering more definitive statements regarding optimal ways to consume beneficial nutrients for best absorption and which are the most important foods to eat and which foods should be avoided. While strong scientific evidence is lacking regarding any one particular influence on inflammation, it should be noted that the ways to decrease inflammation reviewed here are generally consistent with the "heart healthy diet" and the admonition to "move more" as recommended by the American Heart Association. What is new here is that a healthy life-style is now recognized as impacting mental health as well as physical health. Questions regarding the strength of these life style changes on mental health are not yet available.

How Social Workers Can Use this Information?

Direct practice social workers do work with clients who are sometimes experiencing considerable distress and dysfunction. Generally, the focus is on optimizing talk therapy interventions and compliance with treatment recommendations. The findings presented here suggest that ignoring diet and sedentary

behavior can undermine the positive effects of psychosocial interventions. Thus, knowledge regarding the impact of life style choices can enhance direct practice social work interventions. For macro-practice social workers, the findings presented here underscore the importance of access to nutritional food, access to safe places for exercise, and the availability of time for food preparation and exercise. Fortunately, a literature has emerged on interventions for increasing access to good nutrition and evaluating these interventions (see Fowler & Giger, 2017; Society for Public Health Education, 2015; Taillie et al., 2017). Factors such as capacity for establishing a healthy life style should be included in measures of social justice. Moreover, the public needs to demand that the food industry comply with health promoting standards.

Social workers do help clients identify meaning in life and they help clients strive toward meaningful relationships with others. When people feel connection to others and feel their lives have a purpose, then inflammation is decreased (Fredrickson et al., 2015; Frederickson et al., 2013; Moleni etal., 2015). Changes in diet, exercise patterns, vagal tone provide measurable, achievable goals that, when realized, can decrease inflammation and thereby ameliorate disorders and foster resilience. Social workers are in the business of helping clients make life style changes. The information presented here provide a rough guide social workers in knowing which life style changes to encourage.

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