

The syndemogenesis of depression

Concepts and examples

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Abstract

Syndemics, or the deleterious interaction of diseases and structural factors, is an essential and increasingly utilized theory with which to address health inequities and how they interact with disease. However, less research has been conducted on syndemogenesis, a process by which a specific disease or structural factor is more likely to exacerbate syndemic interactions. With the increasing incidence and prevalence of depressive symptomology in Westernized nations, and with depression coexisting syndemically with other illnesses, I use depression as a case study to highlight syndemogenic processes in action. Doing so will hopefully aid preventive efforts in these areas.

Keywords

depression, poverty, syndemogenesis

Introduction

In the past two decades, scholars have increasingly used the concept of syndemics as a lens to analyze health inequalities. Merrill Singer and colleagues (2017, 941) define syndemics as ‘interacting, co-present, or sequential disease and the social and environmental factors that promote and enhance the negative effects of disease interaction’. Syndemics theory has been used to illuminate the relationships, for example, among poverty, type 2 diabetes, and depression (Mendenhall 2012; Weaver and Mendenhall 2014); among tuberculosis, crowded housing, poverty, and HIV and AIDS (Littleton and Park 2009; Singer and Clair 2003); and among substance abuse, violence, and HIV and AIDS (Illangasekare et al. 2014; Singer 1994). More recently, Shir Lerman, Bayla Ostrach, and Merrill Singer (2017) edited a two-volume set on syndemics and stigma that clearly delineates the multiple ways that syndemics and stigma interact in various disease and structural contexts. Syndemic relationships operate on both the individual and the social levels, affecting interacting biological and structural relationships. Both relationship types contribute equally to syndemic relationships.

In his seminal book on syndemics, Singer (2009) defines syndemogenesis as the frequent and consequential role that certain diseases play in the development of syndemic cases. He later clarifies the term as a ‘syndemic-causing’ process and describes the process as digging deeper to address the root of other syndemics (Singer 2015, 208). While the significance of syndemics research has already been proven due to its emphasis on both biosocial and structural factors as parallel contributors to illness promulgation (Everett and Wieland 2013; Lerman, Ostrach and Singer 2017; Mendenhall 2012; Singer and Bulled 2016; Willen et al. 2017), research into syndemogenesis is equally essential due to its pinpointing of particular syndemic relationships which, by early detection and prevention, can mitigate other illnesses and syndemics. One value of this approach is the potential to identify specific syndemic interactions by tracing specific diseases across several syndemics and understanding the structural factors that support disease etiology.

I thus utilize Singer and colleagues’ (2017) definition of syndemogenesis as the ‘processes, pathways, and stages of syndemics development involving a disease-social context and disease-disease interactions’ to acknowledge the health or social conditions that underlie the syndemic-causing illness. Singer (1994, 2009) originally identified a syndemic that included substance abuse, violence, and AIDS (the SAVA syndemic), based on his research with Hispanics in Hartford, Connecticut (Emard 2017; Gideonse 2017; Singer 1994, 2009; Singer et al. 2017). I use the interactions between depression and several chronic diseases, namely diabetes, obesity, and HIV and AIDS, as cases-in-point to trace complex syndemogenic

interactions.¹² The interactions of these diseases with depression that I emphasize in this paper are both exacerbations and outcomes of health disparities. My intention is not only to broaden our understanding of syndemics research but also to inform future treatments and policy changes.

Given the ubiquity of depression as a health concern, it is critical to understand depression's relationship with other illnesses in order to proceed with appropriate treatment and policy development. Conceptualizing depression as a syndemogenic disease provides a framework through which to understand both the biological and the structural underpinnings of depression and its role in other syndemic associations.

Background: Depression

'Major depressive disorder' (henceforth 'depression') defines a collection of symptoms, such as fatigue and feelings of worthlessness, that affect approximately 350 million people worldwide; by 2020, depression will impose the second-largest burden of overall ill health worldwide (National Institutes of Mental Health 2017). Depression has become so common that psychiatrist Dan Blazer (2005, 3), quoting psychologist Anthony Marsella and colleagues (1985), has suggested that we live in an 'age of melancholy'. Depression is strongly correlated with both physical and other mental health conditions, such as type 2 diabetes (Cabassa et al. 2008; Lerman 2015; Weaver and Mendenhall 2014; Walders-Abramson 2014) and obesity (Chou and Yu 2013; Lerman 2017; Trainer et al. 2017).

Social inequality (e.g., poverty or racism) is a major contributing factor to the onset of depression (Belle and Doucet 2003; Flores et al. 2008; Galea et al. 2005). Depression is positively associated with both poverty (Galea et al. 2005; Hanandita and Tampubolon 2014; Lund et al. 2010) and with being the target of intimate partner violence (Beydoun et al. 2012; Johnson et al. 2014; Ludermir et al. 2010). The underlying factors of social inequality include inadequate financial or educational opportunities and a lack of a positive social support

¹ While HIV and AIDS were initially considered infectious diseases due to the looming death sentence a diagnosis warranted, they are now considered chronic diseases due to the introduction of antiretroviral therapies and associated decreased likelihood of disease transmission, and consequential extended lifetimes for those living with the diseases (Deeks, Lewin, and Havlir 2013; Mendenhall et al. 2017; Serrano-Villar et al. 2016).

² Limitations: While several common health conditions (diabetes, obesity, substance abuse, and HIV and AIDS) were discussed in association with depression, other health conditions correlated with depression, such as cancer, were not discussed due to lack of space.

network, which are in turn due to a wide range of structural factors, including homophobia, violence, sexism, and racism (Harrell et al. 2011; Mendenhall 2016; Stevens-Watkins et al. 2014; Williams, Sarker, and Ferdous 2018). Paul Farmer (2004) describes these adverse factors as the ‘building blocks’ of structural violence, which Farmer further defines as the experiences of people who live in poverty or other oppressed, marginalized states, such as genocide. Farmer’s description builds on that of Johan Galtung (1993, 106), who first defined structural violence as the ‘avoidable impairment of fundamental human needs’ (Ho 2007). These negative building blocks constitute the social inequalities that increase the incidence and prevalence of illnesses and form the foundation of structural violence (Mendenhall 2012).

While depression is associated with multiple illnesses, I focus in this article only on the well-documented syndemic relationships that depression has with diabetes, obesity, substance abuse, and HIV/AIDS. I now discuss each of these relationships in order to highlight depression’s role as a syndemogenic disease.

Depression and type 2 diabetes

There is abundant research indicating that individuals with type 2 diabetes (henceforth ‘diabetes’) are at increased risk for depression, and vice versa (Gask, Macdonald, and Bower 2011; Lerman 2017; Mendenhall 2016, 2012; Walders-Abramson 2014). In the United States, the prevalence rate of depression is two times higher in individuals with diabetes than in individuals without diabetes (Asamsama et al. 2015; Gask, Macdonald, and Bower 2011; Nawaz, Mane, and Ramakrishna 2015), and the risk of diabetes in individuals with depression is three times higher than in individuals without depression (Asamsama et al. 2015; Pan et al. 2011). Separate studies have found that mortality rates for some inpatients with both diabetes and depression has been estimated at 47 percent, compared with 14 percent in outpatients with neither disease, 23 percent in patients with diabetes only, and 22 percent in patients with depression only (Herrera 2013; Park, Katon, and Wolf 2013).

Untreated depression contributes to decreased diabetes self-care and participation in health care through behavioral pathways, including poor diet, decreased glucose monitoring, missed medical appointments, limited exercise, and increased likelihood of diabetes complications through inadequate diabetes management (Cabassa et al. 2008; Gask, Macdonald, and Bower 2011; Mendenhall et al. 2017; Weaver and Hadley 2011). Conversely, diabetes contributes to depression by impacting deteriorating social networks, draining financial resources, and requiring considerable lifestyle changes, such as taking daily medications and making significant diet changes (Egede and Ellis 2010; Katon, Maj, and Sartorius 2010). The severity and ensuing health complications of depression and diabetes, treatment resistance, increased

morbidity and mortality, and the cost to both the individual and society are all magnified when the illnesses coexist (Disdier-Flores 2010; Weaver and Hadley 2011). For example, food is a cohesive force: holidays, meetings, family meals, and casual gatherings often include food sharing (de-Graft Aikins 2006; Hopper 1981; Maclean et al. 2009; Mankekar 2005). When an individual cannot partake due to diabetes-related dietary limitations, the ensuing sense of otherness may provoke a reluctance to socialize, adding to a sense of social isolation. In my research among Puerto Rican women with diabetes who had to prepare diabetes-friendly meals for themselves and separate meals for the rest of their families, I found that they were twice as likely as women without diabetes to suffer from depression due to the loneliness associated with eating separate meals (Lerman 2015).

Depression shares several symptoms with diabetes (Lerman 2017; Mendenhall 2012; Held et al. 2010). For example, short sleep duration (under seven continuous hours per night) contributes to the development of diabetes (Knutson et al. 2006; Touma and Pannain 2011). During the slow-wave sleep process, metabolic changes contribute to glycemic homeostasis so that insufficient sleep leads to increased glucose levels and decreased insulin levels (Knutson et al. 2006; Touma and Pannain 2011). Since restless sleep is both a symptom and side effect of chronic depression (Pouwer, Nefs, and Nouwen 2013; Park, Katon, and Wolf 2013), decreased sleep is one mechanism by which depression may increase the risk of diabetes (Pouwer, Nefs, and Nouwen 2013).

Additionally, depression activates the hypothalamo-pituitary-adrenal (HPA) axis, which, in turn, triggers a series of neuroendocrine alterations that contributes to increased fat metabolism and insulin resistance and increases production of cortisol in the adrenal cortex; increased cortisol production in turn contributes to decreased cellular sensitivity to insulin and glucose absorption, although the exact pathways are still under investigation (Alonso-Magdalená, Quesada, and Nadal 2011; Chiodini et al. 2007; Everson et al. 2002). Living in a prolonged state of depression also contributes to increased risk for developing diabetes via the production of the hormone norepinephrine, which inhibits the production of insulin by glycogenolysis (promoting the breakdown of glycogen³ in the liver) and gluconeogenesis (production of glucose in the liver), thus contributing to increased levels of glucose in the blood (Alonso-Magdalená, Quesada, and Nadal 2011; Chiodini et al. 2007).

There has also been inconclusive research on the effect of antidepressants on glycemic control in individuals with diabetes. Some studies have found that selective serotonin

³ Glycogen is a form of stored energy produced in the liver and converted into glucose by the peptide glucagon and the hormone epinephrine (Lerman 2015).

reuptake inhibitors (SSRIs), the most common class of antidepressants, increase the risk for diabetes (Khoza et al. 2012; Mendenhall et al. 2017; Pouwer, Nefs, and Nouwen 2013), particularly by increasing the hormone cortisol, which, in turn, contributes to insulin resistance (Khoza et al. 2012; Vrshek-Schallhorn et al. 2013). Poor metabolic control has also been proven to reduce the positive reaction to antidepressants (Katon, Maj, and Sartorius 2010). Conversely, some research indicates that treating depression with SSRIs has not been shown to increase glycemic levels (Deuschle 2013; Egede and Ellis 2010). This has considerable implications for therapies for individuals with both diabetes and depression.

On the structural level, depression and diabetes are both associated with high crime rates, economic instability, and political inaction (Galea et al. 2005; Nawaz, Mane, and Ramakrishna 2015; Mendenhall 2012). For example, diabetes and depression syndemics in Puerto Rico were found to be rooted in poverty, crime, skyrocketing produce prices, and Puerto Rico's status as a US territory, which in turn contributed to limited funding for health insurance and steep taxes on foods imported from the US mainland (Lerman 2015). Likewise, Emily Mendenhall (2016) has identified a diabetes-depression syndemic among low-income groups in India, which she attributes to rural-urban migration, altered social networks and value systems, and gender-based violence, combined with shifts in the source, quantity, and quality of food and physical activity. Political inaction in the face of skyrocketing prices for healthy food, unemployment, and civil unrest (e.g., in the face of recent police brutality and ensuing protests) is a major stressor that contributes to a sense of helplessness and lack of control. With increasing unemployment and poverty rates, healthy food becomes unaffordable, contributing to the overconsumption of cheap, unhealthy food and to living in unsafe neighborhoods (Dhokarh et al. 2011; Lerman 2017). The combination of unemployment, poverty-derived depression, and the overconsumption of nutrient-deficient foods triggers the overproduction of cortisol, which increases the risk of developing diabetes (Nawaz, Mane, and Ramakrishna 2015; Park, Katon, and Wolf 2013; Pouwer, Nefs, and Nouwen 2013). Economic and occupational instability, ensuing family frictions, and the challenges associated with adapting to life with diabetes have also contributed to increased risk for developing depression. Having depression, in turn, contributes to a worsened diet, a lack of a stable and consistent social support network, and pharmaceutical and medical noncompliance, all of which worsen the risk and side effects of diabetes (Mendenhall 2015; Sánchez-Villegas et al. 2012; Weaver, Meek, and Hadley 2014).

Depression and obesity

Depression has a syndemic relationship with obesity (Brewis 2011; Lerman 2017; Trainer et al. 2017). Obesity, a condition characterized by excessive adipose (fat) tissue, has more than doubled worldwide since 1980, affecting more than six hundred million adults in 2014

(World Health Organization 2017). Obesity increases the incidence risk of type 2 diabetes, depression, stroke, cardiovascular disease, asthma, sleep apnea, and several types of cancer (Low, Chin, and Deurenberg-Yap 2009; Hossain, Kavar, and El Nahas 2007). Approximately 35 percent of adults in the United States have obesity (defined by body mass index [height/weight ratio] ≥ 30 , and/or skinfold thickness and waist circumference) (World Health Organization 2017). Obesity is due to complex interactions among several factors, with more than six hundred genes, socioeconomic status, supermarket placement within neighborhoods, neighborhood safety, and the economic inaccessibility of fruits and vegetables all contributing to obesity (Brewis 2011; Ulijaszek and Lofink 2006; Lerman 2017).

Individuals with obesity are twenty to fifty times more likely than individuals without obesity to suffer from an episode of depression lasting for more than a year and are five times more likely to gain weight than are individuals without depression (Brewis 2011; Lerman 2017; Murphy et al. 2009; Trainer et al. 2017). The social stigma of obesity often contributes to the depression of individuals with obesity and to the chronic psychological and physical depression stemming from obesity (Lerman 2017; Puhl and Heuer 2010; Trainer et al. 2017). For example, John Luque and colleagues (2015) found that Latinas reported depression as a risk factor for obesity due to the misconception of fat bodies as lazy bodies, and they emphasized the role of family involvement in successfully losing weight. Sarah Trainer and colleagues (2017) found that the structural stigma of obesity, such as being unable to fit into airplane seats or restaurant booths, and the interpersonal stigma of obesity, such as being passed over for promotions at work, both contributed to increased rates of depression among people with obesity and were motivating factors for people seeking bariatric surgery. With depression already a stigmatized condition, individuals struggling with obesity are also much less likely to access health care than are individuals with just obesity or depression, out of fear of being stigmatized not only by passersby but also by health care providers (Barney et al. 2006; Link, Yang, and Collins 2004). The fear of stigmatization from health care providers also prevents individuals from seeking out treatment and having regular check-ups (Alegría Drury and Louis 2002; Zhu, Norman, and While 2011).

The stress of obesity stigma has been shown to increase levels of cortisol, a hormone whose production increases with stress and is associated with deleterious health outcomes such as type 2 diabetes (Himmelstein et al. 2015; Lerman, Ostrach, and Singer 2017; Trainer et al. 2017), as shown in the previous section. For example, higher cortisol levels increase levels of insulin and drop blood sugar levels, which makes individuals crave high-sugar, high-fat foods (Lerman (2017). These sugary, fatty foods are also frequent comfort foods, which are commonly consumed under the perpetual stress stemming from poverty and unemployment (Greenhalgh 2015; Mankekar 2005) and which contribute to increased consumption and subsequent risk for obesity (Lerman 2017). In addition to cortisol, leptin, an adipose-

produced hormone that controls fat distribution and food intake, is a key physiological indicator of obesity; despite elevated levels of the hormone due to increased amounts of adipose (particularly above the obesity threshold), increased leptin resistance contributes to difficulty recognizing satiety (Bornstein et al. 2006; Milaneschi et al. 2012; Uliaszek and Lofink 2006). Conversely, decreased leptin levels have been found in people suffering from depression, particularly as leptin has been found to secrete antidepressant effects via enhanced neuroplasticity in the amygdala and the hippocampus, brain regions associated with happiness and well-being (Bornstein et al. 2006; Gong et al. 2013; Milaneschi et al. 2017). Keiko Asao and colleagues (2016) found that people who lived in poverty and were more likely to skip breakfast due to having depression were at increased risk of lower leptin levels than were people who did not have depression. Since leptin is produced in white adipose tissue, if the amounts of white adipose tissue are reduced too quickly via skipped meals, the ensuing decreased leptin levels can, in turn, contribute to increased risk of depression (Rigamonti et al. 2010). Leptin also regulates the HPA axis (Bornstein et al. 2006; Gong et al. 2013); increased leptin has been found to overstimulate the HPA axis, which places the body in a state of prolonged stress. As discussed in the section on depression and diabetes, prolonged stress due to HPA axis overstimulation also increases the vulnerability to diabetes (Bornstein et al. 2006; Gong et al. 2013).

Depression and obesity share several symptoms, such as sedentary behavior and irregular sleeping and eating patterns (Gong et al. 2013). Depression sometimes causes the sufferer to either lose interest in eating and to lose weight as a result, or to overconsume high-carbohydrate, high-sugar, high-fat foods and to gain weight as a result (Sánchez-Villegas et al. 2012; Wurtman and Wurtman 1995). Sweet, high-carbohydrate, high-fat foods trigger the production of serotonin, a hormone responsible for both elevated mood and suppressed appetite, by allowing the production of tryptophan, the building block of serotonin, at the expense of other hormones competing for access to the brain (Gibson 2006; Spring et al. 2008; Wurtman and Wurtman 1995). Food also holds emotional meaning: people eat comfort foods to feel better, especially if those comfort foods are linked to happier times (Barthes 2013; Gibson 2006). While individuals with depression may gain psychopharmacological advantages by consuming food that triggers the production of serotonin, those same foods also contain a lot of fat and cause substantial weight gain if consumed in large quantities, contributing to the development of obesity (Wurtman and Wurtman 1995).

There is some research implying that obesity is a side effect of antidepressants, particularly SSRIs, although the exact pathophysiology remains unclear (Lerman 2015; Patten et al. 2009; Serretti and Mandelli 2010). In my own research, I found that due to the medical brain drain from Puerto Rico to the US mainland (due, in turn, to the ongoing economic crisis),

overworked and underfunded health care providers regularly prescribed high dosages of antidepressants to their patients in order to avoid repeat patient visits and to make room for more patients, and thus more insurance payments (Lerman 2017). This research has strong implications for medication adherence, due to the pre-existing stigma surrounding both depression and obesity; individuals with depression may not wish to add to their suffering by taking a medication that would cause them to develop another stigmatized condition. However, the exact cause-and-effect biological relationship of obesity and depression has yet to be explained in full; it is uncertain whether depression as an illness is present prior to the individual developing obesity or vice versa (Heo et al. 2006). But whether or not depression has a direct effect on obesity or vice versa, both depression and obesity as diseases are serious health concerns, and obesity leads to a plethora of other diseases, and as such the relationship of depression and obesity should be further examined.

Obesity researchers (Bellisari 2016; Brewis 2011; Greenhalgh 2015; Lerman 2017; Moffat 2010; Trainer et al. 2017) place obesity within sociocultural milieus that make unhealthy food choices easier. Elsewhere, I have argued that obesity and depression ‘do not exist in a social vacuum’ (Lerman 2017, 53); instead they draw on their social environments when interacting syndemically. Specifically, poverty and limited access to education and healthy foods are major contributors to obesity incidence and prevalence, furthermore emphasizing the association between obesity and depression (Aguirre 2009; Bellisari 2016; Brewis 2011; Greenhalgh 2015; Lerman 2017; McCullough and Hardin 2013). For example, in Puerto Rico people are charged exorbitant prices for fresh fruits and vegetables due to the 1920 Jones Act, which enforces a US monopoly on the shipment of fresh produce to the island and which makes it difficult for Puerto Ricans to consistently afford healthy foods (Lerman 2017). Trauma, neglect, and stress, particularly from living in poverty or violent neighborhoods, are precursors to developing obesity later in life (Moore and Pi-Sunyer 2012; Schavey, Puhl, and Brownell 2014). As discussed earlier, depression acts upon the HPA axis by increasing the production of the hormone cortisol, which in addition to increasing insulin resistance also favors storage of abdominal fat (Gluck et al. 2004; Moore and Pi-Sunyer 2012). However, the mechanisms by which cortisol acts on abdominal fat are not yet fully understood (Dockray, Susman, and Dorn 2009; Gluck et al. 2004; Moore and Pi-Sunyer 2012).

As discussed earlier, the long-term consumption of fatty, sugary foods – particularly when spurred both by the desire to return to a happier mental state (i.e., due to depression) and the lack of access to healthier foods (i.e., due to poverty and food deserts) – contributes to the increased risk of obesity and consequent diseases like type 2 diabetes (Bellisari 2016; Greenhalgh 2015; Lerman 2017). When socioeconomic survival is paramount, exercise and healthier, more expensive foods are not highly prioritized (Flores et al. 2008; Greenhalgh 2015). Parents of families living below the poverty line frequently work more than one job in

order to pay the bills, and do not have time or money to cook healthy meals (Brewis 2011; Greenhalgh 2015). Children in low-income households often remain by themselves for long periods of time, watching television instead of playing outside, and are exposed to advertisements for unhealthy foods that are cheaper than healthier fruits and vegetables (Greenhalgh 2015). Blair and colleagues (2013) found that children living in poverty had higher levels of cortisol than children not living in poverty, due to both the depression related to lack of essential resources and the jealousy associated with having less resources than other children. Furthermore, consistently high levels of cortisol in childhood had adverse effects on brain development into adulthood, negatively affecting memory, attention, and emotion control, which in turn made it more difficult to find and keep a job (Blair et al. 2013; Evans and Schamberg 2009; Kopala-Sibley et al. 2017). Obesity is also a heavy economic burden due to the direct costs of treatment and prevention, the indirect costs of morbidity and mortality, and inconsistent insurance coverage of obesity (Finkelstein et al. 2009; Woolford et al. 2013). Indeed, Finkelstein and colleagues estimated in 2009 that the economic burden of obesity in the United States was roughly \$147 billion in 2008, with individuals with obesity spending 46 percent more on inpatient costs than did individuals without obesity. This additional economic drain of obesity on people already living in poverty both increases the risk of depression and contributes to the cycle from which individuals with obesity and depression are unable to disengage. The exorbitant costs associated with obesity both exacerbate depression among individuals with obesity who live in poverty (Greenhalgh 2015; Heo et al. 2006) and highlight the syndemogenic relationship between the two health conditions.

Depression and substance abuse

Following Singer's original syndemic model (1994, 2009), scholars have shown that depression has a syndemic relationship with substance abuse (González-Guarda et al. 2011; Illangasekare et al. 2014; Senn, Carey, and Venable 2010). Among individuals with major depressive disorder, the prevalence of substance abuse ranges from 8.5 to 21.4 percent in the United States alone (Davis et al. 2008), while the Substance Abuse and Mental Health Services Administration found that in 2014, 28.4 percent of adolescents aged twelve to seventeen with a substance abuse disorder suffered concurrently from depression, compared to 10.5 percent of adolescents who suffered from depression but did not have a substance abuse disorder (Hedden et al. 2015). Substance abuse increases the likelihood of participating in risky sexual behaviors, such as trading sex for drugs or having unprotected sex with a potentially infected partner (National Institute on Drug Abuse 2012). Cocaine, alcohol, and methamphetamine (meth) also affect brain viral loads (Arnsten et al. 2002; Baum et al. 2009; Marcondes et al. 2010): levels of dopamine receptors among cocaine, alcohol, and methamphetamines abusers are lower than in the general population, and decreased

dopamine levels are a risk factor for depression (Gideonse 2017; Marcondes et al. 2010; Volkow et al. 2003). Whether decreased levels of dopamine receptors are a pre-existing condition or a result of drug abuse is unclear, but this finding suggests a biological foundation for both the increased prevalence of depression and for the worsening of HIV and AIDS among substance abusers. Gideonse (2017) found that substance abuse can also be indicative of other conditions, such as attention-deficit/hyperactivity disorder (ADHD), for which meth is a rare treatment, as the drug increases dopamine receptivity and reuptake in the brain. Relying too much on meth to treat ADHD can both cause an addiction to meth and increase the likelihood of developing depression. Depression frequently serves as both as a stimulus for and a result of substance abuse (Illangasekare et al. 2014; Nunes and Levin 2008; Senn, Carey, and Venable 2010). For example, alcohol is both a common source of temporary relief for people suffering from depression, and a depressant that causes symptoms similar to depression, such as fatigue (Senn, Carey, and Venable 2010). Overconsumption of alcohol also increases the likelihood of broken relationships, impulsive decision making, and unemployment, all of which contribute to the development of depression (Senn, Carey, and Venable 2010).

Some research suggests that substance abuse and depression are both correlated with the genotype and allele frequencies of the receptor h5-HT_{1B} G861C (Huang et al. 2003), which underlines a genetic relationship between the two that could lead to new therapies, although more research needs to be conducted to explore this relationship. Other studies suggest that low levels of 5-hydroxyindoleacetic acid (5-HIAA), a major metabolite of serotonin, are related to both increased depression and early-onset alcoholism (Atigari et al. 2013; Kreek et al. 2005). This finding has strong implications for the use of SSRIs both as antidepressants and to combat early-onset alcoholism; again, more research is needed.

In keeping with syndemogenic theory, depression and substance abuse also interact on the structural level, particularly through violence (Gideonse 2017; González-Guarda et al. 2011; Marcus and Singer 2017; Singer 2000; Singer and Ziegler 2017). Lawrence and colleagues (2004) found that among women receiving social welfare benefits, those who report domestic abuse also experience elevated rates of depression and substance abuse compared to women who do not report domestic abuse. In their extensive meta-analysis of studies on substance abuse, violence, and HIV, Meyer and colleagues (2011) found that substance abuse accounted for 20–25 percent of variance in high-risk sex, especially among single women, and that women who had experienced physical or sexual abuse were more likely to be substance abusers. Meyer and colleagues (2011) also found that depression was a frequently concurrent issue for individuals who had experienced domestic violence, and that depression was often the underlying motivation to self-medicate with alcohol and drugs among individuals who had been abused. Parsons and colleagues (2012) corroborated this result in their research on gay and bisexual men, finding that study participants often turned

to illicit drugs to alleviate their depression and stigmatization as sexual minorities. Illangasekare and colleagues (2014) also corroborated this, finding that their study participants reported experiencing depression whenever they came down from a drug-induced high, and would then take more drugs to avoid experiencing depression again.

Socioeconomic disadvantages also increase the risk for syndemogenic depression and substance abuse (González-Guarda et al. 2011; Illangasekare et al. 2014; Singer and Ziegler 2017). There is a strong association between poverty and substance abuse (Gideonse 2017; Marcus and Singer 2017; Valdez, Kaplan, and Curtis 2007). Poverty is a major contributor to depression (Mendenhall 2015; Singer 2000; Weaver and Mendenhall 2014), which is associated with elevated rates of violence, crime, and unemployment, which in turn perpetuate poverty and substance abuse rates (Singer and Ziegler 2017; Valdez, Kaplan, and Curtis 2007). Mojtabai and colleagues (2014) found that among their study participants, the cost of care was the single biggest barrier to treatment for both individuals with depression and with substance abuse.

Depression, HIV, and AIDS

Depression also has a syndemogenic relationship with HIV and AIDS (Pfeiffer et al. 2017). Estimates for the prevalence of depression among individuals with HIV and AIDS in the United States range from 20–45 percent (Arseniou, Arvaniti, and Samakouri 2014; Nanni et al. 2015). Depression is underdiagnosed and undertreated among individuals with HIV and AIDS, which may account for the wide prevalence range (Arseniou, Arvaniti, and Samakouri 2014; Zhang et al. 2012). The high prevalence of depression among individuals with HIV and AIDS is due to several reasons, including stigma, deteriorating cognitive and physical function, and social isolation (Pfeiffer et al. 2017; Herek 2014; Simbayi et al. 2007).

Depression is correlated with impaired immunological response and increased mortality among individuals with HIV and AIDS (Marcus and Singer 2017; Illangasekare et al. 2014; Simbayi et al. 2007) and is associated with nonadherence to pharmaceutical therapies and with high-risk sexual behaviors, such as inconsistent condom use, which are, in turn, associated with increased risk of HIV transmission and worsening symptomology (Cook et al. 2004; Wouters et al. 2012). Depression and HIV share several symptoms, such as lack of energy and weight loss (National Institute of Mental Health 2015), which can make it difficult to detect depression in individuals with HIV. Injection drug use and sharing ‘equipment’ propagates the transmission of HIV and AIDS (Singer 1994), with injection drug use accounting for approximately 10 percent of the annual HIV incidence rate in the United States (HIV.gov 2015).

Stigma surrounds HIV and AIDS, which increases the risk of depression and leaves HIV and AIDS untreated (Emard 2017; Pfeiffer et al. 2017). Stigma-induced structural factors underlying HIV and AIDS, such as blocked funding for needle-exchange programs and classes on safe sex, criminalizing consensual sex between two adults of the same sex, and denied housing, also contribute to increased incidence and prevalence rates of depression among individuals with HIV and AIDS among people living on the streets (Gurung et al. 2004; Herek 2014; Marcus and Singer 2017). HIV and AIDS stigma are also common in schools and the workplace, leading to diminished intellectual and practical skills which, in turn, lead to difficulties obtaining permanent employment, which worsens depression outcomes (Gideonse 2017). González-Guarda and colleagues (2016) found that depression mediated the structural factors, particularly homophobia and HIV stigma, that were associated with increased risky sexual behavior and decreased medication adherence among gay Hispanic men in Miami, a conclusion that Emard (2017) corroborated in his research on men who have sex with men (MSM) in Boston.

Depression, HIV, and AIDS are inversely related to education and positively related to economic inequality (González-Guarda et al. 2011; Illangasekare et al. 2014; Lund et al. 2010). Individuals living in poverty report higher levels of depression, HIV, and AIDS, as well as decreased health care access, than the general population (Armstrong and Costello 2002; Khan, Murray, and Barnes 2002; Senn, Carey, and Vanable 2010). For example, African Americans carry the greatest burden of HIV among all ethnic groups in the United States, accounting for 44 percent of new adult infections (Centers for Disease Control and Prevention 2015a). Gay, bisexual, and other MSM are also at elevated risk of contracting HIV and AIDS, accounting for 63 percent of all new HIV infections in 2010 (CDC 2018b). Thirty-six percent of African American gay, bisexual, and other MSM contracted HIV and AIDS in 2010, making them the highest-infected ethnic group in the United States (CDC 2018b). With 27.4 percent of the African American community living in poverty (CDC 2018a; The State of Working America 2015), they are less likely to have and/or be able to afford health care and are more likely to have worse health outcomes. HIV and AIDS are highly stigmatized and embedded in discrimination rooted in homophobia, racism, classism, and ignorance about disease transmission, which both increases the incidence and prevalence of depression and makes it exceedingly difficult for affected individuals to access care (Bogart and Thorburn 2005; Stall et al. 2003; White and Carr 2005). For example, scholars show that systematic racism and homophobia among health care providers prevent gay, bisexual, and MSM African Americans from seeking treatment for HIV and AIDS, which contributes to elevated depression rates and increases their morbidity and mortality rates (Benkert et al. 2006; Nicolaidis et al. 2010; Smedley and Smedley 2005). Experiencing racism also contributes to poorer depression outcomes and sexually risky behavior (Illangasakere et al. 2014; Singer and Ziegler 2017; Wilson et al. 2014). Wilson and colleagues (2014) also

found that Black and Hispanic men were more likely than White men to be imprisoned for minor drug offenses and to be at increased risk for engaging in unprotected sex.

Discussion

In this article, I review several ways that depression interacts on both the structural and biological levels with other health conditions, indicating the presence of several syndemics and that depression is indeed an example of syndemogenesis. My own work (Lerman 2017, 2015) and that of Mendenhall (2016), Trainer and colleagues (2017), Illangasekare and colleagues (2014), and González-Guarda and colleagues (2011), among others, highlight the necessity of taking a multifaceted look at structural and biological underpinnings of depression, particularly as they relate to other diseases. In particular, they build on the work of Ostrach and colleagues (2017) and Singer (and Ziegler 2017; and Bulled 2016, 2015, 2009, 1994) in taking a critical look at the complex ways that depression serves as a case study of syndemogenesis. This theoretical focus draws attention to the need for multilevel actions to address not only the separate structural and biological bases of depression, but also the intersections. For example, the work by Mendenhall (2016) and myself (Lerman 2015) on depression and diabetes emphasizes the need to look at the effects of broader political changes and oppressions on health outcomes, while Trainer and colleagues (2017) show how social stigma (itself state-perpetuated) of the obese body affect depression and vice versa. González-Guarda and colleagues (2016) and Emard (2017) separately found that depression mediated interactions of risky sexual behavior, homophobia, and HIV stigma; these results call for a closer look at depression as not only a health outcome of structural factors or other diseases, but as an active mediator in other structural-biological interactions. These studies highlight the need not only for more research to better understand the syndemogenic pathways of depression but also that more thorough interventions are needed to combat depression along the biological-structural pathways.

The structural factors contributing to these conditions require intensive changes that will take time to fix: funding drug rehabilitation centers' needle exchange programs to reduce the transmission of HIV and AIDS, providing affordable housing and training for jobs to mitigate poverty, and working to build trust with minority populations. However, some changes, such as training health care providers to identify and address depression in their patients, are smaller and are a viable first step in preventing the incidence and prevalence of depression. By doing so, syndemics that include depression may be mitigated or even eradicated. Furthermore, the structural factors, such as poverty, social and domestic violence, and chronic unemployment, that contribute to both depression etiology and to depression's syndemic interactions with the other diseases outlined here highlight the need for large-scale social change. Given the role of structural and biological interactions in the

perpetuation of syndemogenesis, the treatment of biological factors without social change is insufficient to prevent syndemogenic interactions. Such changes might include the creation of new jobs and safe spaces, the development of social networks for vulnerable individuals to protect against domestic violence, the introduction of more paid sick and vacation days, as well as longer maternity leave, investments in early education, and reforms to the criminal justice system to reduce mass incarceration (CDC 2017; Vallas and Botteach 2014).

This research has implications for potential prevention and treatment. Given the syndemogenic nature of depression, it would behoove providers to check patients for depressive symptomology during both regular checkups and specialty (e.g., endocrinologist) visits in order to identify and treat depressive symptoms. Treatment plans for diabetes, obesity, HIV and AIDS, and substance abuse should include plans for treating depression as a matter of course. Family members should be involved in treatment plans in order to offer support for loved ones with depression (Illangasekare et al. 2014; Nawaz, Mane, and Ramakrishna 2015; Park, Katon, and Wolf 2013).

Broader preventive measures are also needed at the structural level. The stigma surrounding depression, obesity, substance abuse, and HIV and AIDS is still pervasive. Safe spaces should be provided for people with these conditions to come together and share their experiences with people without the conditions to offset assumptions and misconceptions of these conditions. The language used to discuss these conditions should be neutral and not derogatory, and fines should be imposed on institutions that fire or refuse to hire individuals with these conditions based on their disease status. In the United States, the federal government should invest in creating jobs by rebuilding infrastructures, renovating abandoned housing, providing subsidized housing, and passing the Paycheck Fairness Act to ensure that all individuals receive the same paycheck for the same work performed regardless of gender, ethnicity, gender or sexual identification, or religion. More money should be dedicated to childcare and education, such as enforcing The Strong Start for America's Children Act, which invests in preschools, childcare, and home visits for pregnant women. Furthermore, the incarceration system should be overhauled. The 'one strike and you're out' policy for government-funded housing should be changed to allow people more opportunities to get back on their feet after being released from prison, and more programs should be instituted that help recently released individuals find employment and affordable housing. People with low-level offenses, such as possession or sales of small quantities of controlled substances, should receive counseling and rehabilitation instead of incarceration; this would also reduce the number of Blacks and Hispanics in the prison system, as many are jailed for minor offenses (Stevens-Watkins et al. 2014; Wilson et al. 2014).

Given the syndemogenic interconnectedness of depression both with other health conditions and injurious social conditions such as poverty, policies should focus on

improving access to mental health care for vulnerable groups, such as people with HIV and AIDS. As individuals living in poverty often lack the financial resources and employment opportunities to move out of poverty, which contributes to the deterioration of depressive symptoms (Lund et al. 2010; Weaver and Mendenhall 2014), programs that offer job training, early-stage housing support, and affordable mental health counseling should be available to individuals living in poverty. Individuals with depression should be included in decision-making processes regarding federal regulations for mental health treatments.

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