Impact of Stress and Strain on Current LGBT Health Disparities

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Robert-Paul Juster, Jennifer A. Vencill, and Philip Jai Johnson

Introduction

Health inequalities are experienced by sexual and gender minority populations as a consequence of stigma and represent a national public health priority [1]. Despite social progress in North America, perceived discrimination attributable to sexual orientation is reported by 29–78% of lesbian, gay, bisexual, and transgender (LGBT) Canadians [2] and 42% of LGBT Americans [3]. Further, violence against gender nonconforming lesbian, gay, bisexual, and transgender people remains "alarmingly high," with approximately 20–25% of lesbian and gay people reporting some form of violence within their lifetimes. Indeed, these figures likely underestimate the experience of violence and discrimination against LGBT people, as the US federal survey on violence does not commonly contain questions on sexual orientation or gender identity [4].

These distinct experiences of violence and discrimination, which create cumulative stress and strain for LGBT individuals, are referred to as *minority stress* [5, 6]. Minority stress can be defined as the enduring stress that sexual minority individuals experience as a result of their minority status within a pervasively stigmatizing social climate [5, 7]. Meyer [8] identified three overarching characteristics: (1) minority stress is *uniquely* experienced by LGBT individuals and is different from mundane stressors encountered by people from majority or nonstigmatized backgrounds; (2) minority stress is *chronic*, ranging from mundane offenses to extreme instances of harassment and violence; and finally (3) minority stress is *socially based* and caused by other people, groups, institutions, and political processes. It should be noted that much of the data in support of this model is derived from research on gay men or men who have sex with men (MSM), although recent research has supported the applicability of the model for lesbian/bisexual women's experiences [9–14] as well as those of transgender individuals [15, 16]. Moreover for transgender individuals, "sexual minority" does not necessarily apply as stigma is related to gender identity.

The experience of minority stress can be thought of as the consequence of experiencing a combination of specific processes: (i) enacted, (ii) felt, and (iii) internalized stigma. Specifically, (i) *enacted stigma* comprises the objective or external events of discrimination and stigma people experience; (ii) *felt stigma* is the expectation of rejection and vigilance that arises in response to such events; and (iii) *internalized stigma* is the internalization of negative attitudes, feelings, and internal representations of a sexual minority identity [6, 12]. As defined by Stuenkel and Wong [17], enacted stigma refers to the hostile behaviors and perceptions, also known as bias and discrimination, of majority group individuals toward an individual stigmatized or seen as different [18]. However, the experience of stigma can occur in the absence of overt discrimination. For example, felt stigma represents the internalization of perceived stigma that leads people to engage in concealment to avoid rejection, bias, and discrimination.

Similarly, LGBT individuals will often engage in identity concealment behaviors so as to avoid being "outed" and potentially becoming the target of prejudicial reactions. Unlike heterosexual individuals for whom stigma tends to be salient when sexual orientation becomes personally relevant [19], among LGBT individuals for whom sexual orientation forms an inextricable component of identity, stigma becomes an ever-present phenomenon, with concealment, expectations of rejection, and hypervigilance being understandable (but not always inevitable) consequences.

R.-P. Juster, PhD (✉) • P.J. Johnson, PhD
Program for the Study of LGBT Health, Division of Gender, Sexuality, and Health, Columbia University, 722 W 168th Street, Room 337, New York, NY 10031, USA
e-mail: robertpauljuster@gmail.com

J.A. Vencill, PhD
Program in Human Sexuality, Department of Family Medicine & Community Health, University of Minnesota Medical School, Minneapolis, MN, USA

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For bisexual and transgender individuals, the experience of stigma comes from both heterosexual individuals and within the LGBT community [19–21]. Although little research has examined attitudes toward bisexual and transgender individuals within the commonly and perhaps erroneously perceived "monolithic" LGBT community, lesbians and gay men often see the issues experienced by bisexual and transgender people as completely separate from their own [20], and transphobic attitudes have been shown to be particularly prevalent among gay men [22]. Thus, divisions within the LGBT community can generate unique forms of minority stress.

Minority stress processes affect the psychological, physical, and behavioral health of LGBT individuals [1]. Many of the health consequences, such as anxiety and mood disorders, physical complaints, maladaptive substance use, and cardiovascular disease, are catalyzed and/or exacerbated by psychosocial stress (Fig. 4.1). However, additional research is urgently needed to elucidate the biological mechanisms that explain how minority stress "gets under the skin" to affect the health and well-being of LGBT individuals [23].

This chapter will outline the neurobiology linking chronic stress to health outcomes, as well as recent research developments applying biological approaches to describe LGBT health disparities as they relate to minority stress and trauma. Our focus will be on stress physiology and the development of the allostatic load model used to describe "wear and tear" on the brain and body caused by chronic stress and unhealthy behaviors. We will also discuss how healthcare providers can incorporate this knowledge to deliver LGBT healthcare in a competent and sensitive manner. The next section will begin with a brief introduction to stress physiology and explain how initial adaptive mechanisms can become maladaptive when chronically activated under stressful circumstances.

Biological Stress

Stress is broadly defined as a real or interpreted threat to an individual that results in biological and behavioral responses. The stress-disease literature includes three broad perspectives with regard to measurement of stress and subsequent coping: environmental, psychological, and biological. As a multidimensional construct, stress involves interactions among *inputs* (environmental stressors), *processes* (subjective psychological distress), and *outputs* (objective biological stress responses). Though often investigated separately [24, 25], these elements of stress and coping are best studied in conjunction with one another, as each dimension can impact the others. For example, the release of stress hormones as part of the biological response to environmental and psychological stress mobilizes energy to promote adaptation (e.g., behaviors that function to distance a person from an environmental stressor or damp down maladaptive psychological processes).

Absolute stressors (e.g., natural disasters, sexual assault) that threaten survival lead invariably to acute stress responses and, potentially, to posttraumatic distress. By comparison, *relative stressors* (e.g., negotiating traffic, public speaking) threaten one's well-being only if the person deems them stressful. As is principally the case for relative stressors, situations that are novel, unpredictable, threaten self-preservation, and/or diminish one's sense of control contribute additively to biological stress responses [26, 27]. Cumulative exposure to multiple relative stressors can render an individual more susceptible to traumatic symptoms (e.g., hypervigilance) in the face of an absolute stressor or accumulated relative stressors that "break the camel's back," so to speak. Based on the minority stress model outlined earlier, chronic internal and external stressors – and subsequent stress responses – may be more pernicious and emotionally salient among LGBT individuals.

Biological stress responses are activated whenever real or interpreted threats are detected via neural systems. The interpretation of a "threat" triggers the *sympathetic-adrenal-medullary* (SAM) axis to release *catecholamines* (e.g., adrenalin) within seconds from the adrenal medulla. This response system is fast-acting and reflexive, preparing the body to respond almost immediately to threat. Similarly, the neural interpretation of "threat" activates the paraventricular nucleus of the hypothalamus to release *corticotropin-releasing factor* (CRF), which in turn activates the *hypothalamic-pituitary-adrenal* (HPA) axis. Specifically, CRF travels through a portal system linking the hypothalamus to the pituitary gland, where it signals the secretion of *adrenocorticotropic hormone* (ACTH) from the capillary-rich environment of the anterior pituitary. Systemic ACTH then travels to the adrenal glands, where it precipitates cellular activities in the zona fasciculata region of the adrenal cortex to produce the glucocorticoid *cortisol*, which in turn is responsible for transforming fat into sugar to fuel biobehavioral responses [28]. Compared to the SAM axis, the HPA cascade is slower, occurring within minutes after the perception of a threat. Thus, the SAM and HPA axes synergistically mobilize energy necessary for adaptation; however, this comes at the cost of acute and/or chronic recalibration of many biological functions that ensure health of the whole organism [29] (Fig. 4.2).

The brain's ultimate role during stress is to detect threat and promote adaptation. In addition to the pituitary and hypothalamic control of the HPA axis, there are three major brain structures involved in the regulation of stress responses: (i) the *hippocampus*, which is linked to memory and cognition, in addition to being implicated in negative

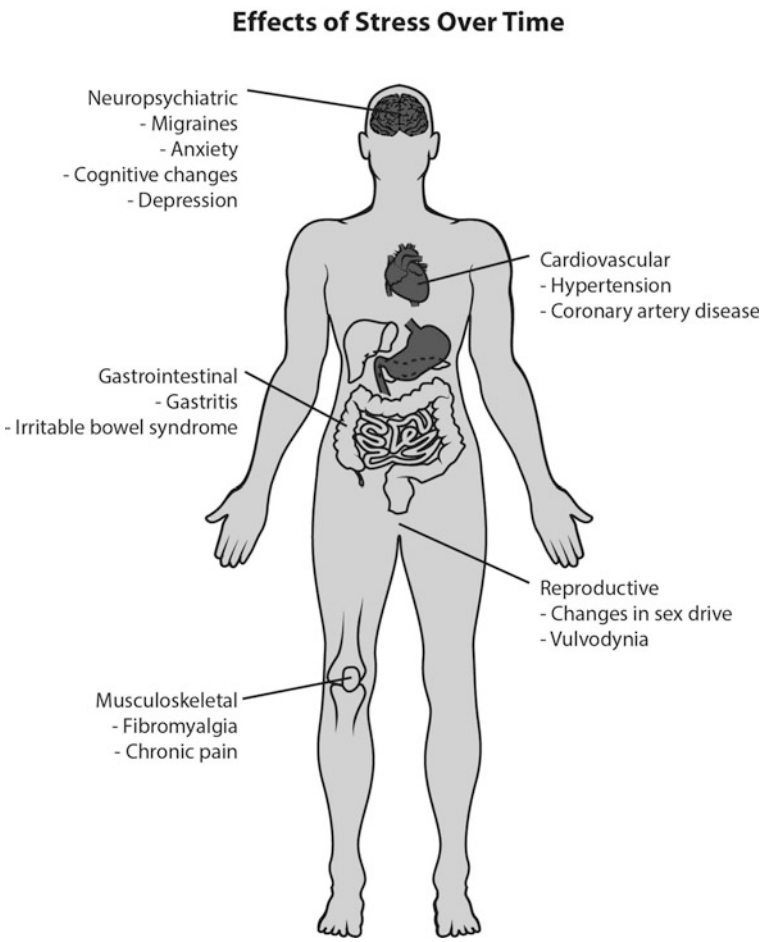

Fig. 4.1 Biological effects of stress on the brain and body

feedback regulation that shuts down the HPA axis; (ii) the *amygdala*, which is responsible for threat perception and emotional processing with outputs to SAM axis and neuroendocrine regulatory systems; and (iii) the *frontal cortex*, which is involved in cognition and exerting top-down control over subcortical structures and the development of coping responses [30–35]. With regard to HPA-axis regulation, the hippocampus is inhibitory, the amygdala is excitatory, and the frontal cortex can be both. Neural regulation of allostatic mechanisms is further shaped by individual differences in constitutional (genetics, development, experience), behavioral (coping and health habits), and historical (trauma/abuse, major life events, stressful environments) factors that ultimately determine one's vulnerability and/or resilience to stress.

Life Cycle Model of Stress

Lupien et al. [36] proposed that the consequences of chronic stress and/or trauma depend on age of exposure and, accordingly, brain development of specific regions regulating the HPA axis. Environmental stress in the prenatal period affects the development of the hippocampus, prefrontal cortex, and amygdala, and shapes the neural development of these regions. After birth, the effects of postnatal stress vary according to environmental exposures: for instance, maternal separation during childhood generally leads to *increased* secretion of cortisol, whereas exposure to severe abuse is associated with *decreased* levels of cortisol. It is important to note that, from the prenatal period onward, all developing brain areas are sensitive to the effects of stress hormones; however, some areas undergo rapid growth during key critical windows. From birth to 2 years old, for example, the developing hippocampus is most vulnerable to the effects of stress. By contrast, exposure to stress that persists over a longer duration between birth through late childhood can lead to changes in volume of the amygdala, which continues to develop until the late 20s.

During adolescence, the rapid development of the hippocampus slows down but continues to show marked plasticity as evidenced by perpetual neurogenesis of the dentate gyrus [37]. Other stress-regulatory regions, including the frontal cortex, continue to mature into adulthood. Consequently, stress exposure during the transition into emerging adulthood can have major effects on the frontal cortex. Studies

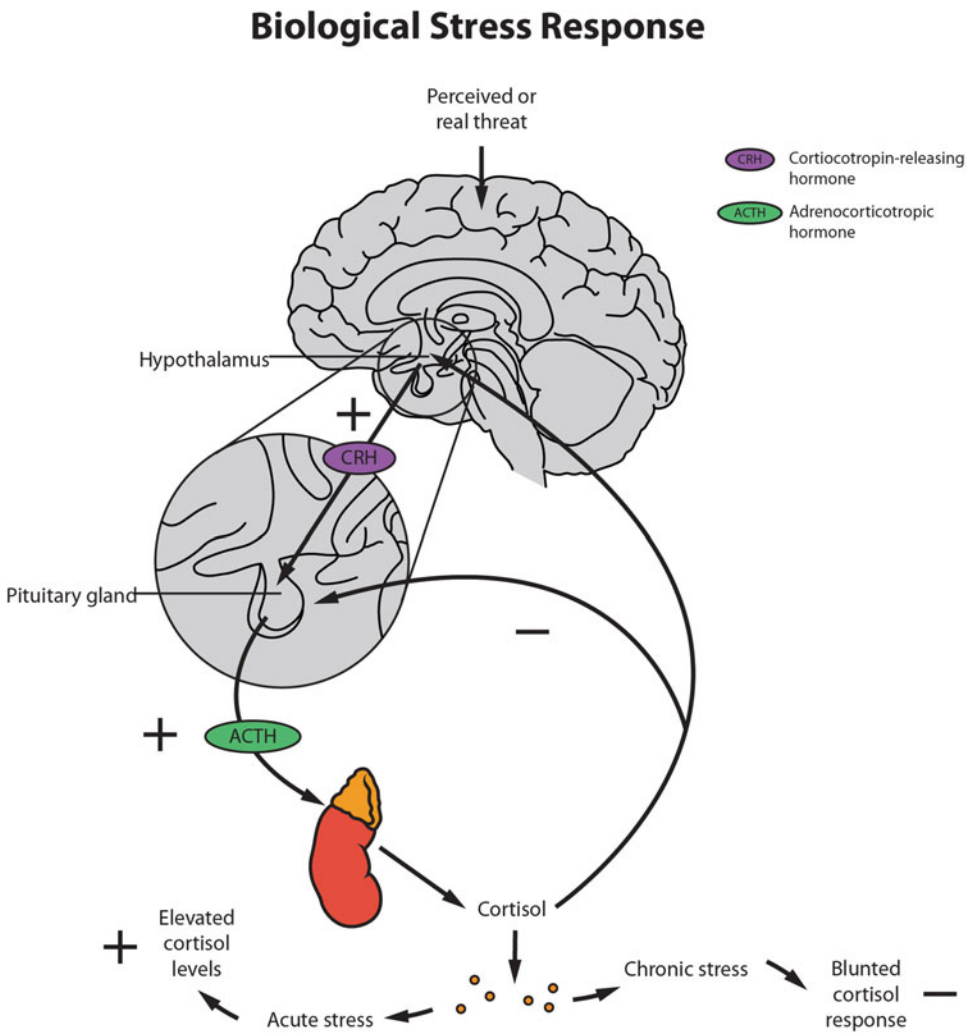

Fig. 4.2 Hypothalamic-pituitary-adrenal (HPA) axis contributing to biological stress response

show that adolescents are highly vulnerable to stress because of pubertal changes in gonadal hormones and sensitivities of the HPA axis that can persist into adulthood. In adulthood and into older age, the brain regions that undergo the most rapid decline as a result of aging are once again highly vulnerable to the effects of stress hormones, including the manifestation of effects from earlier life [36].

Lifelong brain changes ultimately diminish a person's ability to adapt, leading to subtle recalibrations in stress responsivity that could be used to detect disease trajectories [38]. According to the life cycle model of stress and a growing body of preclinical research, regional volumes of these neurological structures in conjunction with biological signatures (e.g., hypercortisolism vs. hypocortisolism) can be used to predict differential risk profiles for specific psychopathologies (e.g., depression vs. PTSD) in adulthood as well as predict that traumatic experiences might have occurred in early life [36]. From a clinical perspective, however, direct measurement of central nervous system substrates is costly and potentially invasive, while indirect assessment using peripheral biomarkers routinely collected in blood draws (e.g., glucose, cholesterol) could be compiled with stress biomarkers (e.g., adrenalin, cortisol).

Life stressors resulting from stigma are believed to render LGBT individuals more vulnerable to a variety of mental health conditions [39]. We believe that specific psychopathological trajectories can be demarcated by distinct biological signatures related to stress hormones and stress-related biomarkers. While extant literature on LGBT health has focused on psychosocial questionnaires and population surveys, few interdisciplinary studies have assessed physiological measures of biopsychosocial stress among LGBT individuals [40]. Moreover, with the exception of research focused on the HIV/AIDS pandemic among sexual minority men [41–46], biological stress mechanisms have not been extensively investigated among healthy LGBT populations. The following sections will provide the reader with emerging literature that applies measures of stress biology to understanding health inequities experienced by LGBT individuals.

Reactive Cortisol

Stress responses are adaptive in the short term, while long-term activations can result in physiological dysregulation. The *reactivity hypothesis* [47] proposes that exaggerated physiological and behavioral reactivity to stressors is a risk factor for stress-related diseases such as cardiovascular disease, among others [48, 49]. Such pathophysiological reactivity is potentially discernable by examining the magnitude of physiological stress responses in controlled laboratory settings.

Stress reactivity has traditionally been defined according to increases in stress biomarkers from baseline and upon stressor exposure; however, the prolongation and total duration of stress responses persisting after the stressor ceases are also critical to consider [50, 51]. Indeed, the reactivity hypothesis has been criticized for often ignoring or dismissing physiological *recovery*, a period after exposure that is characterized by much individual variability [52–54] and that may have significant clinical implications for LGBT individuals. For instance, rumination is associated with delayed cortisol recovery [55] and evidence suggests that sexual minorities may experience more ruminative processes than heterosexuals [23, 56, 57].

Stress reactivity and recovery could also extend clinically to treatments aimed at addressing psychological, emotional, and physiological responses to minority stress (e.g., systematic desensitization, biofeedback, and ecological momentary assessment (EMA)). EMA refers to methods that ask participants to repeatedly self-report their affective, behavioral, and cognitive states in naturalistic setting and has been used, for example, to demonstrate a relationship between a lifetime history of discrimination and current smoking status among Black and Latino men living in the USA [58]. We believe that using such tools to examine dynamic changes in stress reactivity and related phenomena that occur in response to gender and sexual minority stress processes would significantly expand our understanding of the factors that contribute to resilience and health among LGBT individuals.

A body of emerging research is assessing stress reactivity in LGBT populations. The first study on this topic was conducted by Hatzenbuehler and McLaughlin [59], who reported that LGB individuals growing up in less socially tolerant states evidenced blunted cortisol reactivity and hypothesized that this dampened HPA-axis pattern might indicate a pathophysiological profile associated with trauma and fatigue [60]. A novel study comparing LGB men and women to heterosexual individuals of both sexes demonstrated that sexual orientation modulates endocrine stress reactivity [61]. Eighty-seven participants were exposed to a psychosocial stressor involving public speech and mental arithmetic. Results revealed that lesbian/bisexual women demonstrated higher cortisol levels 40-min poststressor than heterosexual women, while gay/bisexual men demonstrated lower cortisol levels throughout testing compared to heterosexual men who peaked 20-min poststressor, as is usually observed [26].

The latter study showed that gay/bisexual men demonstrate stress reactivity profiles more closely aligned with those of heterosexual women, while lesbian/bisexual women show patterns more akin to those of heterosexual men. Although speculative, the delayed peak observed among lesbian/bisexual women could be indicative of ruminative processes. This would be consistent with reports by Hatzenbuehler and colleagues [23, 56, 57] who showed that lesbians and gay men are more ruminative than heterosexuals in response to stigma-related stressors. Importantly, rumination is associated with delayed poststressor cortisol recovery [55]. While ruminative cognitive-behavioral processes were not assessed, this approach represents a promising avenue for future inquiry, especially in the context of further understanding mental health.

In contrast to findings among women and consistent with a gender-based reversal in male-typical HPA-axis hyperreactivity [62], lower overall cortisol concentrations were observed throughout testing among gay/bisexual men relative to heterosexual men. From a sexual minority stress perspective and in light of Hatzenbuehler and McLaughlin's [63] findings showing a blunted cortisol response among young sexual minority adults exposed to high-structural stigma environments as adolescents, this suggests that gay/bisexual men may be displaying HPA-axis downregulation. Indeed, an expanding literature is examining the relationship between hypocortisolism and severe stressors early in development [64, 65] or in the face of traumatic experiences [66], both of which are ubiquitous among sexual minority men [1]. The functional significance of this blunted cortisol stress reactivity to a psychosocial stressor must be further delineated since it is not clear whether this hormonal profile represents an adaptive or maladaptive process among sexual minority men. As will become evident in the following section, assessing circadian variations in stress hormone levels may prove to be a valuable technique that can be used to discern an individual's level of vulnerability and/or resilience.

Diurnal Cortisol

Stress hormones can be measured diurnally to capture naturalistic variation. For instance upon awakening, the *cortisol awakening response* (CAR) represents a normal surge in cortisol levels reaching maximal concentrations approximately

30 min after awakening [67]. This surge is followed by gradually declining cortisol concentrations throughout the day as pulsatile secretion decreases in amplitude and frequency [68]. The nadir usually occurs around midnight, after which cortisol levels start to rise again during the early morning hours [69]. These dynamics are normal mechanisms that help ensure adaptive functioning of metabolism, cognition, and so on. Measuring diurnal cortisol can be complemented by ecological momentary assessment of emotional and social processes occurring throughout the day that can have clinical applications.

Like stress reactivity, diurnal HPA-axis functioning can be used to identify disease vulnerabilities. A meta-analysis of 62 studies concluded that while the CAR is positively associated with workplace stress and general life stress, it is negatively associated with symptoms of burnout, fatigue, and exhaustion [70]. Hypocortisolism is a phenomenon that occurs in approximately 20–25% of patients suffering from stress-related diseases like chronic fatigue syndrome, fibromyalgia, PTSD, burnout [71, 72], and atypical depression, to name a few [60]. By contrast, increased HPA-axis functioning during the afternoon and evening has been strongly associated with depressive symptoms [73, 74]. Figure 4.3 illustrates how psychopathological conditions can be hypothetically conceptualized to differ in terms of distinct biological signatures that we believe can one day be applied in clinical practice to differentiate conditions with otherwise overlapping symptomatologies.

Diurnal cortisol is beginning to be applied in LGBT research with particular regard to stigma and "coming out." Benibgui [75] found that LGB emerging adults (ages 17–27) from Montréal with low social support experienced increased psychosocial stress that corresponded to increased depressive symptoms and decreased self-esteem. While the majority of the sample (77–88%) had disclosed their sexual orientation to family members, LGB youth with increased internalized homophobia had flatter cortisol profiles that corresponded to an increased vulnerability to adverse mental health conditions [75]. Another study showed that compared to those who did not disclose their sexual orientation at work, disclosure was unexpectedly associated with higher cortisol levels and negative affect among LGB individuals [40]. Shedding light on this nonintuitive finding is another study showing that gay men who disclosed their sexual orientation to supervisors reported significantly higher hostility in their work environments, significantly lower perceived promotion opportunities, and significantly higher turnover intentions as evidenced by their desires to quit [76].

In contrast, disclosure of sexual orientation can also have positive effects on diurnal cortisol and mental health [77]. Using the same sample described earlier vis-à-vis stress reactivity [61], LGB individuals who had completely disclosed their sexual orientation to family and friends demonstrated lower morning cortisol levels and fewer symptoms of depression, anxiety, and burnout than those who had not completed the disclosure process. Future research would do well to also correlate disclosure to persons outside one's immediate interpersonal network.

Allostasis and Allostatic Load

Thus far, we have focused on stress hormones in a cortisol-centric manner that does not consider other related biological systems. As an inherently adaptive mechanism in reactive and diurnal contexts, physiological dynamics in

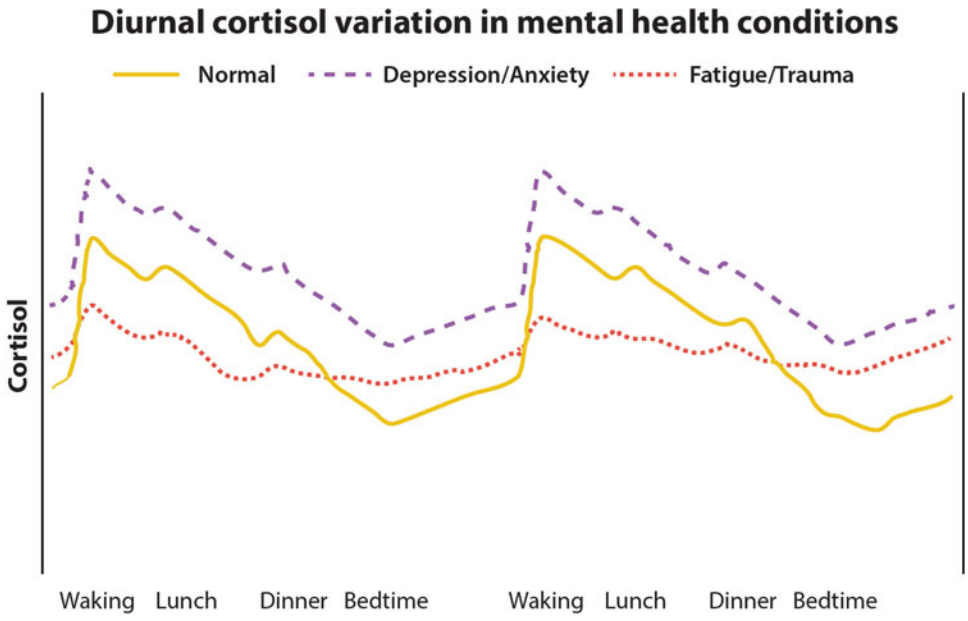

Fig. 4.3 Hypothetical diurnal cortisol profiles in normal and psychopathological conditions

stress hormone functions are examples of *allostasis*, defined as adaptive biological processes that preserve "stability through change" [78]. The neurobiologist Sterling and the epidemiologist Eyer coined the term *allostasis* to describe dynamic, multifaceted biological processes that maintain physiological stability by recalibrating homeostatic parameters and matching them appropriately to meet environmental demands [78]. Analogous to our understanding of resilient systems that have the capacity to dynamically adjust and stabilize when faced with perturbations [79], allostatic processes likewise alter metabolic functioning via compensatory and anticipatory mechanisms in both reactive and diurnal contexts. Compensatory alterations during acute stress include, for example, decreased digestive and bodily growth/repair processes that are adjusted to accommodate increased neurological, cardiovascular, respiratory, and immunological activities that are metabolically taxing. Under these circumstances, allostasis becomes taxing and differs from normal responsivity as an allostatic state.

Four potential pathophysiological profiles representing allostatic states have been outlined [39]. First, *repeatedly activated responses* refer to simply too much stress in the form of repeated, novel events that cause cumulative elevations of stress mediators over sustained periods of time. Second, *nonhabituating responses* refer to failure to habituate or adapt to the same stressor that leads to the overuse of stress mediators because of the failure of the body to dampen or eliminate the hormonal stress response to a repeated event. Third, *prolonged responses* represent a failure to shut off either the hormonal stress response or to display the normal trough of the circadian patterns. Fourth, *inadequate responses* represent hypoactive stress responses that may involuntarily allow other systems, such as inflammation, to become hyperactive. In essence, allostatic states reflect response patterns in which physiological systems become over or underactive, leading to multisystemic physiological dysregulations.

The multisystemic strain attributable to chronic stress, adversity, and trauma is referred to as allostatic load [80]. *Allostatic load* (AL) is defined as the multisystemic "wear and tear" the brain and the rest of the body experience when repeated allostatic responses exact their noxious toll when exposed to chronic stress. Under such conditions, stress hormones like adrenaline and cortisol first become misbalanced and induce an interconnected cascade of interdependent biological processes that sequentially collapse as individual biomarkers become dysregulated and lead to disease outcomes [81]. AL can be indexed using combinations of stress-related biomarkers to represent physiological dysregulation [82].

Validation using longitudinal data from the MacArthur Studies of Successful Aging cohort led to a *count-based AL index* representing the following ten biomarkers [82]: 12-h urinary cortisol, adrenaline, and noradrenaline output; serum dehydroepiandrosterone-sulfate (DHEA-S), high-density lipoprotein (HDL), and HDL-to-total cholesterol ratio; plasma glycosylated hemoglobin; aggregate systolic and diastolic blood pressures; and waist-to-hip-ratio. Participants' values falling within high-risk quartiles (clinical and preclinical ranges based on percentiles) with respect to the sample's biomarker distributions are dichotomized as "1" and those within normal ranges as "0." Once tabulated, these are summed to yield an AL index ranging from a possible 0 to 10 which can then be used to predict health outcomes.

The thematic advantages of applying an elevated-risk-zone system when scoring AL are fivefold as they represent (1) *early warning signals*, since cutoffs are anchored at subclinical thresholds; (2) *multi-finality*, in that similar AL algorithms predict different tertiary outcomes; (3) *flexibility*, since calculations are based on different biomarker combinations; (4) *synergism* that captures the cumulative interaction of numerous biomarkers; and finally (5) *antecedents* that powerfully predict individual variation in AL [83]. In sum, AL algorithms are objective reflections of biological functioning that are intricately interconnected with genetic, neurological, developmental, behavioral, cognitive, and social factors.

Clinical Allostatic Load Index

The AL index is thus far a research measure that may become useful as a clinical tool in the future; however, it is not yet ready for prime time, as clinical norms have yet to be established. In cases where medical professionals currently measure other stress-related biomarkers in standard blood tests (e.g., fibrinogen, cytokines, cortisol), attention is typically placed on values reaching clinically significant levels based on population norms if these exist for any given novel biomarker. For readers interested in knowing how to determine an AL index for clinical and research investigative purposes, a simple formulation can be used to calculate the index based on clinical reference ranges used in current practice for diagnostic purposes. For each biomarker value included, a subclinical cutoff can be easily calculated based on normative clinical ranges. Note that for some emerging biomarkers, like cortisol, clinical norms have yet to be established.

For example, consider total cholesterol, with a normal range between 3.3 and 5.2 nmol/L. First, to determine the *range*, subtract the lower limit from the upper limit ($5.2 - 3.3 = 1.9$). Second, to determine the *quartile*, divide the range by four ($1.9/4 = 0.475$). Finally, to determine the *cutoff*, either subtract the quartile from the upper limit for the upper cutoff ($5.2 - 0.475 = 4.725$) or add the quartile

to the lower limit for the lower cutoff (3.3 + 0.475 = 3.775) in the case of biomarkers such as HDL cholesterol, DHEA-S, and albumin where lower levels may be associated with health risk. Based on this example, a patient with total cholesterol of 4.725 nmol/L or higher would receive a score of "1," while values below this cutoff would be scored as "0." A clinical AL index is therefore the sum of subclinically dysregulated biomarkers for a given individual. Previous work demonstrated that a clinical AL index was associated with increased subjective reports of chronic stress, frequency of burnout symptoms, and hypocortisolemic profiles characteristic of fatigue states [72].

A review by Juster et al. [84] of nearly 60 empirical studies suggests that AL indices incorporating subclinical ranges for numerous biomarkers (mean = 10; range = 4–17) predict clinical outcomes better than traditional biomedical methods that address only clinical thresholds for single biomarkers. Importantly, AL inclusion of neuroendocrine and/or immune biomarkers is stronger than metabolic syndrome parameters or systemic clusters in the prediction of stress-related conditions like cardiovascular disease and psychopathology. The most consistent causes of AL are increased age, low socioeconomic status, non-white race/ethnicity, workplace stress, and involvement in emotionally taxing activities such as caregiving. In the context of LGBT health, Fig. 4.4 illustrates how sexual minority stress relates to stress physiology and AL that are in turn predictive of both physical and mental health conditions.

Sexual orientation and developmental aspects related to sexual identity formation are related to AL. In the same study that assessed diurnal cortisol described above [77], analyses examined 21 biomarkers related to neuroendocrine, immune, metabolic, and cardiovascular functioning and teased apart between-group (sexual orientation) and within-group (disclosure processes) differences. Results showed no between-group differences as a function of sexual orientation except that gay/bisexual men evidenced fewer depressive symptoms and AL driven by lower triglycerides, BMI, and cytokine levels than heterosexual men. While no overall AL differences were found as a function of full disclosure, a follow-up analysis found that retrospective coping strategies during sexual identity formation were critical. Specifically, retrospective avoidance coping strategies (e.g., trying to forget everything, keeping one's emotions to one's self, using medication to feel better) during sexual identity formation and disclosure were associated with current elevations in perceived stress, daily hassles, and AL [85]. By contrast, seeking social support was associated with less perceived stress. Taken together, these preliminary findings suggest that the coping strategies enacted during key developmental periods unique to LGBT individuals could help protect against AL and poorer mental health.

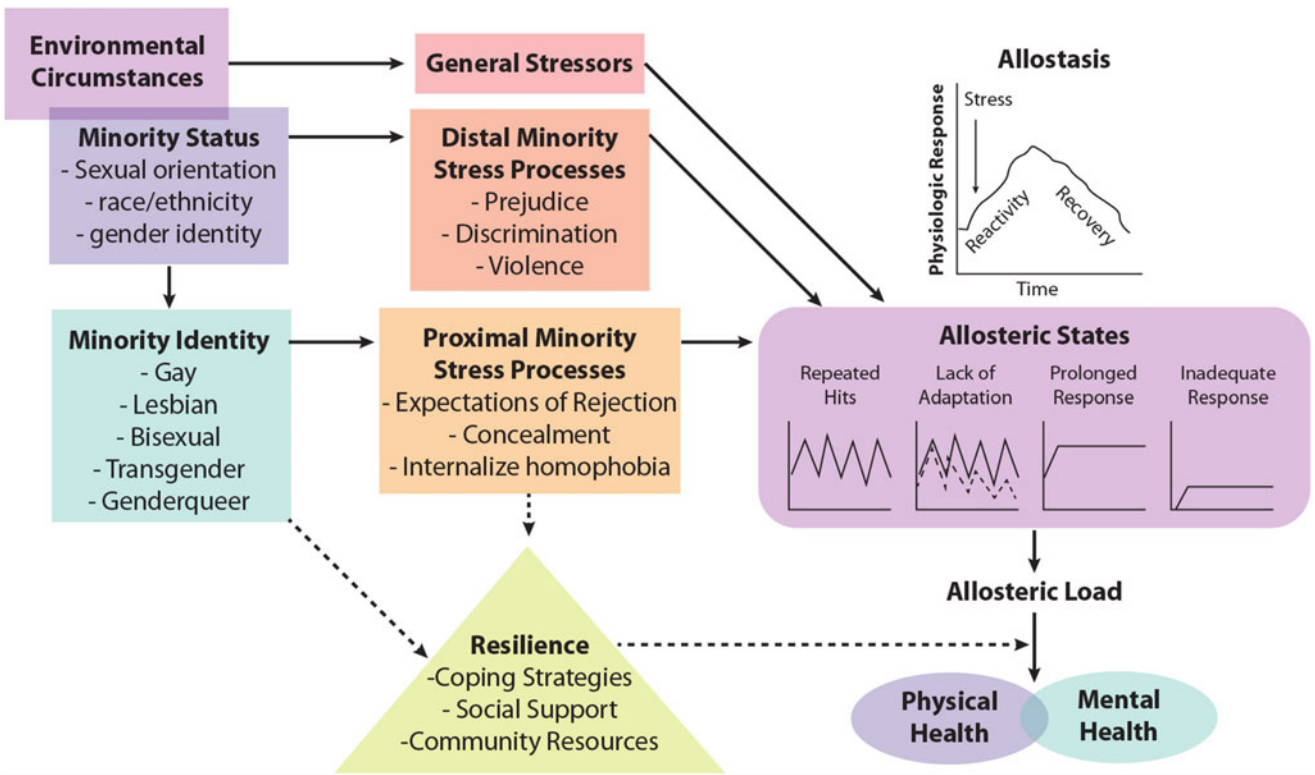

Fig. 4.4 Conceptual model of sexual minority stress and allostatic load (Adapted from Refs. [6, 36])

Psychosocial Implications and Clinical Perspectives

While further research is needed to explore and delineate the potential pathways to resilience for LGBT individuals, a growing body of research points to the benefits of establishing identity-related social support for LGBT individuals [7, 86–87]. DiFulvio and colleagues [86] specifically note the value of *social connectedness* for LGBT youth, which refers to the perception of individuals that they belong, are cared for, and can feel empowered within a given context. In a qualitative study utilizing in-depth interviews with 15 sexual minority youth, these authors outlined a process of negotiating an identity seen as different and defective with family and friends and, in the face of rejection, developing the ability to reclaim this identity and derive empowerment through connections with similar peers.

The importance of having accepting others in one's support network and the benefits conferred by eliminating individuals who might be critical or unsupportive of one's sexual identity is also critical for older LGBT adults [88]. Surprisingly, Snapp and colleagues did not find any impact of community support on development of self-esteem among LGBT young adults, whereas both family and peer support were found to be strong predictors [87]. Perhaps more worrisome, exposure to similar others increases sexual risk-taking among LGBT young adults [89]. Thus, healthcare providers must be sensitive to the fact that mere exposure to similar others and including them in one's support network does not guarantee positive effects on mental health and health behaviors. The key feature is that support network members be included because they are aware of the individual's sexual identity and can provide acceptance and affirmation.

One vital way in which healthcare providers can provide affirmation and acceptance for an LGBT person's sexual and/or gender identities is by identifying and normalizing the impact of minority stress and trauma on the individual's lived experience. This normalization process involves providing psychoeducation to LGBT individuals regarding the multifaceted effects of family, societal, and peer rejection and violence, as well as assurance that it is common and understandable to develop a plethora of thoughts, feelings, physiological reactions (e.g., heart racing, sweating, etc.), and behaviors in response to threats to one's identity and safety. Indeed, the hypervigilance that some individuals develop in response to stigma-related stressors can in fact be adaptive in encouraging avoidance of situations in which safety might be compromised.

Normalizing these experiences can help provide LGBT individuals with avenues to form more supportive connections (particularly in a group-based context), and derive empowerment by attributing their distress to stigma rather than personal failings and recognizing that their emotional, cognitive, and physiological responses make sense in light of the hostility of their social environments [90]. Psychoeducation can also include providing information on the psychobiology of stress reactivity in the context of stigma, as has been discussed by Fisher [91] in the context of responding to past trauma. Specifically, Fisher [91] suggests that providing psychoeducation on the psychobiology of self-injurious behaviors in the midst of a traumatic experience might help decrease an individual's experience of shame and encourage exploration of more adaptive coping strategies [91]. Unfortunately, no published research has examined the effect of psychoeducation on the psychobiology of response to identity-based stigma and trauma or the psychological well-being and coping strategies of LGBT individuals. While psychoeducation may be a therapeutically useful tool, providers must utilize this strategy with caution and only when the individual has developed a sense of security and trust in the relationship with the provider.

It should be noted that we have, for the most part, discussed responses to stress and treatment approaches as uniform among LGBT individuals, perhaps inadvertently reiterating the perceived monolithic nature of the LGBT community. In actuality, it is important for researchers and practitioners to note that experiences of sexual minority stress can differ widely on the basis of an individual's gender, gender identity, and sexual orientation. These experiences can also differ vastly when the intersections of race, physical ability, age, and other identity categories are taken into account. For example, the prejudicial experiences directed at *genderqueer* individuals (i.e., those who do not identify with the gender binary as male nor female or who may view their identity as beyond gender or in-between) may be significantly different from those directed toward male-to-female or female-to-male transgender individuals [21]. Therefore, responses demonstrated by individuals in response to minority stressors must be examined within the diversity of their identities and lived experiences.

In order to maximize the ability of an intervention to foster resilience, psychoeducation may be most effective when it includes opportunities to build supportive networks and a strong emphasis on identifying an individual's strengths and positive coping strategies in response to both past and ongoing stigma- and trauma-based stressors [92]. Healthcare providers can demonstrate affirmation of their LGBT individuals' lived experiences by pointing out that the very resources utilized to adapt to minority stress can be the very same resources used during recovery from the effects of enacted and felt stigma.

Social Policy Implications

Knowledge generated in the stress-disease literature expands our understanding of health inequalities that carry critical conceptual implications for social policy. Social justice focuses on the philosophy of equality of opportunity. For example, *gender relations* refers to expectations related to etiquette and understanding how we relate to each other, while *institutionalized gender* refers to the ways that gender is constructed within large social systems that dictate value systems, social class, and hierarchies of privilege [93]. Institutionalized stigma and heterosexism include, for example, the denial of marriage rights, disadvantaged treatment in schools and workplaces, and disenfranchisement of sociocultural resources like religion and spirituality that often dehumanize LGBT individuals and contribute to further distress [1, 94]. These macro-level factors have important conceptual implications for scientist-practitioners. For example, the use of stress biomarkers could be used to discern the existence of LGBT healthcare inequalities before and after social policy changes trickle down into systems more proximate to the individual.

Social inequalities have health consequences [95]. Compelling research shows that LGBT Americans living in states without policies that protect against hate crimes and employment discrimination experience significantly higher rates of mental distress than those living in states with protective policies [96]. Likewise, LGBT individuals living in states with constitutional amendments banning same-sex marriage experience increased rates of generalized anxiety disorder, depressive disorders, and alcohol abuse. Geopolitical strata with antigay prejudice are associated with increased rates of all-cause mortality among sexual minorities [97]. By contrast, those living in states that recognize same-sex marriages show no increased development of these conditions [98].

Social policy changes can affect the health of sexual minorities. A pioneering study documented significant decreases in general medical and mental healthcare visits and costs among gay men 12 months after Massachusetts legalized same-sex marriage [99]. This study demonstrates how changes in distal policies can progressively eliminate institutionalized stigma and promote public health benefits [99]. Given ongoing debate in the United States and worldwide concerning, for example, same-sex marriage, a fascinating social experiment would be to assess biological stress indices as a function of American states with and without protective policies over time to further understand the relation between social policy and biological processes. In theory, LGBT Americans exposed to less structural stigma should evidence different biological signatures than LGBT individuals from less progressive geo-political strata.

Structural stigma experienced by sexual and gender diverse minorities is modifiable. North America is undergoing geo-political changes that necessitate research evidence to help inform, for instance, the remaining American states without protective legislations and many nations worldwide that still criminalize homosexuality. This makes the comprehensive measurement of stress biomarkers a crucial endeavor, providing us with an objective biometric of macro-level effects that can inform policy makers of the pernicious effects of institutionalized gender and how to improve the health of marginalized groups. The health and well-being of sexual minorities is not a matter of political debate but a matter of public health.

Practitioners can help LGBT clients identify their internal strengths and foster resilience by creating support networks and engaging in advocacy efforts for public policy change and social reform. Thus, practitioners must become informed about local resources in order to refer their clients to advocacy groups, activist events, panel discussions, and pride marches where it is possible to speak out against the experiences of stigma and violence and receive community support [86]. Commitment to and participation in such community engagement can, as DiFulvio eloquently states, "serve as a way for [LGBT individuals] to make meaning of an identity that has been silenced and allows them to regain a sense of power over their lives" (p. 616). At the same time, in light of the identity-based violence that has, through pervasive societal stigma, become an inextricable part of the social fabric of LGBT lived experience, recommendations to engage in community advocacy efforts must be made collaboratively with LGBT clients and following a thorough assessment of the extent of their support network and safety.

Case Scenario

Ashlee is a 19-year-old White female who grew up in the rural Midwest and is just completing her first year of college (Fig. 4.5). She presents to the campus health clinic complaining of significant fatigue, irritability, and recent pain with urination. Ashlee reports that she has been oversleeping, missing her classes, and generally feeling "kinda blah." During the visit, she indicates to you that this is her first time at the campus health clinic and inquires whether her parents might find out about the appointment or have access to her records. She seems nervous about being at the clinic and you notice her tendency to keep an eye on the exit. During the intake, Ashlee looks surprised when you ask her about recent sexual partners as part of the routine intake process. She thanks you for not assuming her partners' genders and shares that during a recent visit to

Fig. 4.5 Ashlee is a 19-year-old white female who grew up in the rural Midwest and is just completing her first year of college

her family physician, she was asked "how many men" she had been sexual with over the past year. Ashlee disclosed that she has recently come out to herself and her college friends that she is a lesbian. She is tearful in describing her expectation that her family will "disown" her if they learn about her sexual orientation. She also fears that her parents may become physically violent towards her, as they have hit her in the past when they didn't like decisions she'd made.

Discussion Questions

1. How can healthcare agencies and institutions ensure inclusivity of sexual and gender minorities when it comes to forms/paperwork, screening questions, and routine intake procedures?
2. What additional information might you want or need to gather from this patient? Why or how might that information be useful in your assessment and treatment of Ashlee?
3. How might the trauma of being rejected by one's family impact biomarkers and AL?
4. How can healthcare agencies and institutions communicate that they are safe and affirming environments for LGBT individuals?
5. What are a healthcare provider's legal and ethical responsibilities when a patient discloses feeling at risk of violence?

Summary Practice Points

- Patients like Ashlee who have trauma histories commonly present with "garden variety," stress-related symptoms and concerns of somatic health. Arriving at an accurate diagnosis can be challenging and necessitates an integrated approach with diverse professionals.
- It is important to screen all patients for trauma, and particularly those who, like Ashlee, appear to be triggered when asked about sexual contact. Screening should include specific questions about sexual assault, abuse, coercion or harassment, and intimate partner violence.
- In addition to considering the contribution of Ashlee's sexual identity to her lived experience, it is important to inquire about other, overlapping stigmatized identities (e.g., rural background, history of sexual abuse, etc.), as research suggests that multiple stressors can produce additive effects.

Key issues to explore with patients like Ashlee include sexual health, self-acceptance of sexual minority identity, and disclosure, including a consideration of the differential mental health impact of nondisclosure versus active concealment of identity. Additionally, it is important to learn more about Ashlee's current coping mechanisms (positive and negative), particularly the presence or absence of supportive interpersonal connections. As noted throughout this chapter, enhancing one's engagement with affirming and accepting social networks is an important predictor of health and well-being among LGBT populations.

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