Sex and Gender Roles in Relation to Mental Health and Allostatic Load

Robert-Paul Juster, PhD, Jens C. Pruessner, PhD, Alexandra Bisson Desrochers, BSc, Olivier Bourdon, BSc, Nadia Durand, DEC, Nathalie Wan, MA, Valérie Tourjman, MD, Edouard Kouassi, PhD, Alain Lesage, MD, and Sonia J. Lupien, PhD

ABSTRACT

Objectives: Beyond male/female binaries, gender roles represent masculine and feminine traits that we assimilate and enact throughout life span development. Bem proposed that “androgynous” individuals adeptly adapt to different contexts by alternating from a strong repertoire of both masculine and feminine gender roles. By contrast, “undifferentiated” individuals may not adapt as well to social norms because of weak self-endorsed masculinity and femininity.

Methods: Among 204 adults (mean [standard error] age = 40.4 [0.9] years; 70% women) working in a psychiatric hospital, we hypothesized that androgynous individuals would present better mental health and less physiological dysregulations known as allostatic load (AL) than undifferentiated individuals. AL was indexed using 20 biomarkers using the conventional “all-inclusive” formulation that ascribes cutoffs without regard for sex or an alternative “sex-specific” formulation with cutoffs tailored for each sex separately while controlling for sex hormones (testosterone, estradiol, progesterone). Well-validated questionnaires were used.

Results: Independent of sex, androgynous individuals experienced higher self-esteem and well-being and lower depressive symptoms than did undifferentiated individuals. Men manifested higher AL than did women using the all-inclusive AL index ($p = .044$, $\eta_p^2 = 0.025$). By contrast, the sex-specific AL algorithm unmasked a sex by gender roles interaction for AL ($p = .043$, $\eta_p^2 = 0.048$): with the highest AL levels in undifferentiated men. Analysis using a gender index based on seven gendered constructs revealed that a greater propensity toward feminine characteristics correlated only with elevated sex-specific AL ($r = 0.163$, $p = .025$).

Conclusions: Beyond providing psychobiological evidence for Bem's theory, this study highlights how sex-specific AL formulations detect the effects of sociocultural gender.

Key words: gender roles, androgyny, mental health, cortisol, allostatic load, gender schema theory.

INTRODUCTION

The sexes are not equal in their unique susceptibilities to specific stress-related diseases. For example, women experience more psychosocial stressors and protracted sick leave due to mental health problems, whereas men are more prone to physical stressors and ultimately earlier mortality (1,2). Trends in North America reveal that women are increasingly dying of cardiovascular disease (3), whereas rates among men are declining or remaining stable (4). Although heart health was once viewed predominantly as a men's health issue, incidences of

cardiovascular disease are increasing for women worldwide (1,5). To understand how “sex-specific” diseases like cardiovascular disease, mood disorders, somatic complaints, and muscular-skeletal disorders can change throughout history (6), scientists must identify the sociocultural factors that modulate disease susceptibilities. Beyond one's sex, within-sex variations related to gender

AL = allostatic load, ANCOVA = analysis of covariance, AUC_g = area under the curve with respect to ground, CI = confidence intervals, PCA = principal component analysis

From the Centre for Studies on Human Stress (Juster, Desrochers, Bourdon, Durand, Wan, Lupien), Montreal, Quebec, Canada; Research Centre of the Institut universitaire en santé mentale de Montréal (Juster, Durand, Wan, Tourjman, Kouassi, Lesage, Lupien), Montreal, Quebec, Canada; Integrated Program in Neuroscience (Juster, Pruessner) and Departments of Psychiatry and Psychology (Pruessner), McGill University, Montreal, Quebec, Canada; Research Centre of the Douglas Mental Health University Institute (Pruessner, Desrochers), Montreal, Quebec, Canada; Department of Psychology (Desrochers), Université du Québec à Montréal, Montreal, Quebec, Canada; Departments of Medicine and Medical Specialties (Kouassi) and Psychiatry (Tourjman, Lesage, Lupien), Université de Montréal, Montreal, Quebec, Canada.

Address correspondence and reprint requests to Sonia J. Lupien, PhD, Centre for Studies on Human Stress, 7401 Hochelaga, Pavillon Louis Riel, Unit 226, Montreal, Quebec, Canada H1N 3M5. E-mail: sonia.lupien@umontreal.ca

could further explain why these disease trajectories are inconsistently sex-specific (7).

Gender represents a spectrum of sociocultural roles, identities, and orientations that are distinct from one's biological sex determined by genes, anatomy, gonads, and hormones (8,9). Gender socialization leads to the development of gender roles or personality stereotypes we assimilate and endorse via the enactment of masculine or feminine behaviors (10). In the 1970s, Bem proposed that masculine and feminine gender roles are two independent yet complementary constructs. Masculinity was viewed as an instrumental orientation and cognitive focus on action, whereas femininity was viewed as an expressive orientation and affective concern for the welfare of others (11). The process whereby society defines and transforms gender roles is referred to as *gender typing* that modulates cognitive-behavioral processes (12).

In evolving our gender schema throughout life span development, we encode and process gender-based information and systematically appropriate sex-specific behaviors consistent with our *gender identity* representing the sex we see ourselves as (12). Bem argued that “gender-typed” individuals are those that rigidly conform exclusively to their masculine or feminine schemas at the detriment of their psychological well-being. For example, self-identification as a hypermasculine heterosexual man might lead to distress when enacting cross-gender behaviors associated with femininity (e.g., nurturing, sensitive). The paragons of mental health according to Bem were “androgynous” individuals who could effectively navigate both masculine and feminine behaviors in different contexts. By contrast, “undifferentiated” individuals would show below-average propensities of either masculinity or femininity that she proposed was the most maladaptive profile (13). Although immensely influential in the psychosocial literature, Bem's *gender schema theory* (12) and proposal of androgyny versus undifferentiation has yet to be supported using comprehensive biological approaches to measure stress.

In theory, biological sex and psychosocial gender influence threat processing and therefore stress physiology (2). For both sexes, stressful circumstances lead to the malfunctioning of the stress hormone cortisol that contributes over time to disease by recalibrating interconnected neuroendocrine, immune, metabolic, and cardiovascular functions (14). *Allostatic load* (AL) embodies this multisystemic “wear and tear” that chronic stress exerts in synergy with unhealthy behaviors (15). AL algorithms index the number of dysregulated biomarkers reaching subclinical cutoffs based on a given sample's distribution that are predictive of disease (16). Indeed, elevated AL indices are linked to numerous *antecedents* (e.g., poor social networks, workplace stress, life-style behaviors) and *consequences* (e.g., mortality, cardiovascular disease, psychiatric symptoms) (17). Although aging is the strongest correlate of increased AL, sex

differences are inconsistent (18). Critically, few studies ascribe sex-specific cutoffs when calculating AL (19) or none have taken consideration of sex hormones (e.g., testosterone, estrogen) fundamental to stress physiology (20). To further understand sex differences in AL, researchers must endeavor to demarcate gender-based modulation further while accounting for one's sex and vast within-sex differences in basal sex hormone functioning (8).

In the current study, we propose that biological sex and sociocultural gender interactively influence AL (8,21). Research shows that extremely traditional or hegemonic masculine gender-typed individuals (e.g., hostile, dictatorial) exhibit coronary-prone behaviors (e.g., inhibited emotional expression, lack of empathy, homophobia) that overlap with hostility that is linked to elevated AL (22,23). To date, however, little is known of the impact of gender-based factors on AL despite significant overlap with interconnected factors. For example, several of the salubrious effects thus far identified in the AL literature differ according to sex specifically for sociocultural constructs such as spousal support, social networks, and religiosity/spirituality (24–27) that are related to gender.

Applying a *sex- and gender-based analysis* (28) of AL could illuminate how variations in biological sex and psychosocial gender relate to health outcomes. In a pilot study of 30 workers, we found that increased masculine gender roles relative to feminine gender roles were associated with high AL for both sexes and with psychosomatic complaints among masculine women (29). In separate reports of gendered sexual orientation, we found that gay/bisexual men had lower stress reactive cortisol in response to a psychosocial stressor, lower AL, and lower depressive symptoms than did heterosexual men, whereas lesbian/bisexual women had higher reactive cortisol and trending depressive symptoms than did heterosexual women (30,31). These effects were influenced by within-group differences whereby sexual minorities who had completely disclosed or “come out” manifested lower morning cortisol concentrations and psychiatric symptoms than did those who were not fully disclosed. Collectively, these studies demonstrate that measuring sex, gender roles, and sexual orientation reveal within-sex associations that are impossible to discern by assessing sex solely as a binary classification (32).

Measuring gender is complex (33). Recently, in an effort to pin-point the influence of sex and/or gender factors vis-à-vis acute coronary syndrome, Pelletier et al. (34) innovated a composite measure of gender comprising various psychosocial variables such as primary earner status, personal income, number of hours per week doing housework, level of stress at home, and both Bem masculinity and femininity scores. They found that feminine gender was associated with the heaviest burden of cardiovascular disease risk factors (e.g., hypertension, depressive symptoms, smoking) independent of sex (34). Sex and

gender therefore represent overlapping yet distinct phenomena that must be delineated to better predict diseases more prevalent in one sex or the other at a given historical period. Our aim in this study is to assesses sex and gender categorically and continuously vis-à-vis mental health and AL.

Research Question and Hypothesis

Building upon our previous sex/gender research (29–31), the goal of the current study was to apply a comprehensive sex- by gender-based analysis of AL and mental health in a large-sample of healthy workers. In accordance with theoretical work on gender roles (11–13) and the rich biological literature on measuring stress (35), we hypothesized that androgynous individuals would self-report higher self-esteem, well-being, and social support as well as lower symptoms of depression and AL levels than undifferentiated individuals, whereas masculine and feminine gender types were expected to fall in between androgynous and undifferentiated individuals. AL was indexed using 20 neuroendocrine, immune, metabolic, and cardiovascular biomarkers according to the (1) conventional “all-inclusive” formulation that ascribes cutoffs without regard for sex or an alternative (2) “sex-specific” formulation with cutoffs tailored to men and women separately. In accordance, we expected AL to only differ between men and women when using the all-inclusive formulation or to differ according to gender roles when using a sex-specific formulation. Lastly, we followed the approach by Pelletier et al. (34) and calculated a gender index using principal component analysis (PCA) and logistic regression to respectively reduce seven gender factors into components that we expected would correlate only to the sex-specific AL index.

METHODS

Participants

Participants included 60 men and 144 women (cycling: $n = 55$; contraceptive: $n = 47$; postmenopausal: $n = 42$) recruited from the employees of the *Institut universitaire en santé mentale de Montréal* as part of a larger study (36). This is the largest psychiatric hospital in Québec with 1546 employees that are predominantly women (65%) from diverse professions. To achieve a medium to large effect size, an *a priori* power analysis suggested that we achieve a total sample size of 159 participants.

From a pool of 295 prospects, the final distribution of participants is $n = 204$. Note that those that did not participate were not lost due to exclusion criteria. Of those that did participate, occupations are as follows: clinical services ($n = 61$; 29.9%), administration ($n = 35$; 17.2%), research ($n = 28$; 13.7%), social integration ($n = 23$; 11.3%), professional services ($n = 20$; 9.8%), maintenance ($n = 22$; 10.8%), general direction ($n = 9$; 4.4%), and human resources ($n = 6$; 3.0%). Occupational status was coded based on professions using the Nam-Powers-Boyd method (37) previously used by our group (19). Table 1 lists the sample's descriptive statistics for demographics, interpersonal, health behaviors, and physical/mental health domains that were assessed for group differences according to sex and gender roles separately.

General Protocol

This study was approved by the local research ethics board of the *Institut universitaire en santé mentale de Montréal* and adheres to the Declaration of Helsinki. Testing was conducted by two women (N. D. and A. B. D.) and two men (R. P. J. and O. B.) between October 2011 and December 2012 at the Centre for Studies on Human Stress (Montreal, Quebec, Canada). Access to the participant pool was enabled by hospital administrators who allowed workers to participate during their working hours without prejudice and in full confidentiality. In partnership with our communications department, recruitment was promoted via conferences, intranet advertisements, large banners, face-to-face visits to units, and word of mouth. Prospects were instructed to contact our laboratory for a 15-minute screening interview before scheduling appointments.

During their first laboratory visit between 1300 and 1800 hours (arrival time: mean = 1335 hours, standard error [SE] = 0:03) that lasted approximately 90 minutes, participants were a) requested to read and sign the consent form; b) instructed to provide saliva samples every 10 minutes at seven occasions throughout the visit to assess stress reactive cortisol; c) completed a cognitive task (data not reported here); d) exposed to the Trier Social Stress Test (38), a popular laboratory-based psychosocial stressor; e) instructed in the use of our Internet-based questionnaire system; f) provided instructions for diurnal salivary collection to assess day-to-day variations in cortisol; and finally g) debriefed. Between visits, participants collected saliva samples at home and completed electronic questionnaires that took approximately 45 minutes to complete.

During their second visit scheduled between 0800 and 1100 hours (arrival time: mean = 0854 hours, SE = 0:05) that lasted approximately 15 minutes, participants returned materials and partook in a blood draw by a licensed nurse. Upon blood draw and physical examination, a Continental breakfast was provided to break a 12-hour fast. Participants received 50\$ CAD as compensation.

Questionnaires

Questionnaires were completed electronically via a secured Web-based questionnaire interface called the Studies Web Automation Tool that meets all the requirements for conducting ethical and secure encryption of Web-based questionnaires.

Gender Roles

The 60-item Bem Sex Role Inventory (11,39) was used to measure gender roles. This instrument uses a 7-point Likert scale ranging from 1 (*never or almost never true*) to 7 (*always or almost always true*) for masculine (e.g., *ambitious, dominant, self-reliant*) and feminine (e.g., *affectionate, gentle, understanding*) items. Original psychometrics demonstrated acceptable internal consistency for the masculinity subscale ($\alpha = .81$) and femininity subscale ($\alpha = .86$) as well as strong retest reliability ranging from r values = 0.89 to 0.93. In the present study, reliability analyses revealed acceptable internal consistency for the masculinity subscale (women: $\alpha = .78$; men: $\alpha = .79$), albeit weaker reliability for the femininity subscale (women: $\alpha = .61$; men: $\alpha = .60$). Men scored higher on masculinity ($p < .001$, $\eta_p^2 = 0.113$), whereas women scored higher on femininity ($p < .001$, $\eta_p^2 = 0.139$).

In a revised coding system aimed at assessing psychological androgyny (13), Bem recommended dichotomizing masculinity and femininity subscales to create four gender-role classifications: a) *masculine* if only the masculinity score ranked above the median, b) *feminine* if only the femininity score ranked above the median, c) *androgynous* if both the masculinity and femininity scores ranked above the respective medians, and d) *undifferentiated* if both the masculinity and femininity scores ranked below the respective medians. We adopted this approach using sex-specific medians for the masculinity subscale (4.6 for women and 4.8 for men) and femininity subscale (6.1 for women and 5.9 for men). In accordance, sex-specific gender-role groups are distributed as follows: *masculine-typed* (43 women

TABLE 1. Sample Descriptive Statistics Grouped According to Sex and Gender Roles

Information	All	Men	Women	<i>p</i>	Masculine	Feminine	Androgynous	Undifferentiated	<i>p</i>
<i>n</i> (%)	204 ^a	60 (29.4)	144 (70.6)	—	59 (30)	46 (23)	38 (20)	56 (28)	—
Psychosocial gender									
Gender roles									
Bem masculine gender roles, <i>M</i> (SE)	4.63 (0.044)	4.96 (0.078)	4.50 (0.049)	<.001	4.89 (0.065)	4.54 (0.105)	4.87 (0.089)	4.27 (0.068)	<.001
Bem feminine gender roles, <i>M</i> (SE)	5.31 (0.031)	5.05 (0.054)	5.41 (0.034)	<.001	5.27 (0.055)	5.37 (0.068)	5.53 (0.055)	5.15 (0.056)	<.001
Sexuality									
Klein sexual attractions, <i>M</i> (SE)	1.43 (0.082)	1.81 (0.243)	1.29 (0.061)	.004	1.48 (0.171)	1.45 (0.209)	1.29 (0.133)	1.46 (0.121)	.862
Klein sexual behaviors, <i>M</i> (SE)	1.39 (0.090)	1.62 (0.235)	1.29 (0.085)	.102	1.38 (0.177)	1.32 (0.192)	1.14 (0.083)	1.62 (0.196)	.355
Klein sexual fantasies, <i>M</i> (SE)	1.77 (0.102)	1.83 (0.244)	1.75 (0.106)	.719	1.79 (0.207)	1.73 (0.214)	1.56 (0.180)	1.92 (0.194)	.697
Klein sexual orientation, <i>M</i> (SE)	1.53 (0.084)	1.76 (0.236)	1.44 (0.070)	.092	1.55 (0.174)	1.50 (0.196)	1.32 (0.111)	1.67 (0.148)	.593
Kinsey sexual orientation, <i>M</i> (SE)	0.32 (0.083)	0.67 (0.247)	0.18 (0.063)	.008	0.37 (0.180)	0.39 (0.196)	0.14 (0.060)	0.33 (0.152)	.780
Demographics									
Age, <i>M</i> (SE), <i>y</i>	40.4 (0.85)	39.0 (1.62)	41.0 (1.0)	.278	40.8 (1.7)	40.2 (1.8)	39.84 (2.0)	40.73 (1.6)	.982
Socioeconomics									
Working hours per week, <i>M</i> (SE)	34.7 (0.58)	36.2 (1.23)	34.1 (0.63)	.093	34.8 (1.23)	35.9 (1.35)	32.9 (1.1)	34.6 (1.0)	.447
Education, years schooling, <i>M</i> (SE)	16.5 (0.23)	16.4 (0.50)	16.5 (0.25)	.785	17.4 (0.43)	16.3 (0.45)	16.3 (0.43)	15.8 (0.51)	.064
Occupational status, <i>M</i> (SE) ^b	69.4 (1.19)	65.9 (2.85)	70.8 (1.2)	.060	70.53 (2.3)	70.5 (2.73)	67.2 (2.2)	68.3 (2.3)	.729
Civil status				.229					.575
Single, %	33.8	43.3	39.9	—	35.6	23.9	31.6	42.9	—
In a relationship or common law, %	32.8	31.7	33.3	—	33.9	39.1	34.2	25.0	—
Married, %	22.5	18.3	24.3	—	16.9	28.3	21.1	25.0	—
Separated or divorced, %	10.8	6.7	12.5	—	13.6	8.7	13.2	7.1	—
Coupled, binary coded, %	55.4	50.0	57.6	.317	50.8	67.4	55.3	50.0	.280

(Continued on next page)

TABLE 1. (Continued)

Information	All	Men	Women	<i>p</i>	Masculine	Feminine	Androgynous	Undifferentiated	<i>p</i>
Parenthood, No. children, M (SE)	0.98 (0.08)	0.93 (0.16)	0.99 (0.09)		0.83 (0.13)	1.26 (0.20)	1.24 (0.18)	0.68 (0.13)	.034
None, %	50.0	55.0	47.9	—	54.2	45.7	36.8	58.9	—
1, %	13.7	11.7	14.6	—	11.9	8.7	15.8	19.6	—
2, %	27.0	21.7	29.2	—	30.5	26.1	34.2	17.9	—
≥3, %	9.3	11.7	8.3	—	3.4	19.6	13.2	3.6	—
Parents with children living at home, %	64.3	65.7	63.7	.840	57.6	72.0	66.7	62.1	.704
Health behaviors									
Caffeinated beverages per day				.627					.612
None, %	29.9	31.7	29.2	—	23.7	32.6	31.6	35.7	—
1–2, %	45.6	48.3	44.4	—	44.1	50.0	42.1	42.9	—
≥3, %	24.5	20.0	26.4	—	32.2	17.4	26.3	21.4	—
Alcohol consumption per week				.061					.374
None, %	31.4	28.3	32.6	—	20.3	37.0	31.6	39.3	—
1–5, %	52.9	46.7	55.6	—	59.3	52.2	52.6	44.6	—
≥6, %	15.7	25.0	11.8	—	20.3	10.9	15.8	16.1	—
Tobacco smoking				.477					.873
Nonsmokers, %	83.8	86.7	82.6	—	81.4	87.0	81.6	83.9	—
Smoker, %	16.2	13.3	17.4	—	18.6	13.0	18.4	16.1	—
Elicit drug use				<.001					.483
No, %	92.6	83.3	96.5	—	93.2	95.7	86.8	92.9	—
Yes, %	7.4	16.7	3.5	—	6.8	4.3	13.2	7.1	—
Physical and mental health									
Physical health condition(s)				.200					.586
None, %	56.4	65.0	52.8	—	59.3	54.3	68.4	48.2	—
1, %	29.9	26.7	31.3	—	30.5	30.4	21.1	33.9	—
≥2, %	13.7	8.3	16.0	—	10.2	15.2	10.5	17.9	—
Medication(s) prescribed				.404					.797
None, %	54.4	61.7	51.4	—	57.6	45.7	57.9	57.1	—
1, %	31.4	26.7	33.3	—	27.1	39.1	26.3	32.1	—
≥2, %	14.2	11.7	15.3	—	15.3	15.2	15.8	10.7	—

representing chronic stress that showed high internal consistency in our sample ($\alpha = .90$). One-third of Canadians report chronic stress (47) that is moreover theoretically interlinked to AL. We therefore adjust for chronic stress in AL analysis to ensure that any effects of gender-role classification on AL are over and above those attributable to psychosocial factors at home and at work.

Biomarkers

Allostatic Load

AL was calculated using 20 biomarkers reported in Tables 2 and 3. Repeated measures of the stress hormone cortisol allowed us to assess diurnal variation and stress reactive variation that was summarized using time-dependent formula and then incorporated into our AL indices. For diurnal cortisol, we calculated two values representing the *diurnal cortisol awakening response* (difference between +30 minutes after awakening and awakening) and *diurnal cortisol bedtime* based on the average for two non-consecutive workdays reported in detail elsewhere (36,48). For reactive cortisol, six measurements of cortisol collected throughout the Trier Social Stress Test (38) were transformed using the area under the curve with respect to ground (AUCg) formula derived from the trapezoid formula to summarize hypothalamic-pituitary-adrenal axis systemic output (49). Likewise, *heart rate AUCg*, *systolic blood pressure AUCg*, and *diastolic blood pressure AUCg* were used based on four cardiovascular recordings obtained throughout the Trier Social Stress Test.

As part of a 12-hour fasting blood draw on their second visit, a licensed nurse extracted 44 ml of blood from participants. The local hospital laboratory assayed serum *dehydroepiandrosterone-sulphate*, *C-reactive protein*, *albumin*, *creatinine*, *glycosylated hemoglobin*, *total cholesterol*, *high-density lipoprotein*, and *triglycerides*. Serum *insulin* and *fibrinogen* levels were shipped off and assayed at Maisonneuve-Rosemont Hospital (Montreal, Quebec, Canada). Isolated plasma was used to assay *tumor necrosis factor α* and *interleukin-6* at the Centre for Studies on Human Stress.

Two alternative AL formulations were applied to calculate a) an all-inclusive AL index *without* regard for sex differences in biomarker distributions as is generally done in AL studies (16) and b) a sex-specific AL index *with* sex-specific cutoffs (19). In both cases, we used either one-tailed or two-tailed percentiles (50). Specifically, the 75th percentile was used for biomarkers in which high levels are dangerous, the 25th percentile was used for biomarkers in which low levels are dangerous, or both the 12.5th and 87.5th percentiles were used for biomarkers (e.g., cortisol) in which both hypoactive and hyperactive functioning is considered dangerous. Biomarkers attaining these critical cutoffs were ascribed a score of "1," whereas those in a healthy range were ascribed a score of "0". In sum using 20 biomarkers, AL indices represent the number of dysregulated biomarkers for each individual. Any use of medication(s) was automatically added as "1" and none was scored as "0" to AL indices.

Sex Hormones

Sex hormones were used as covariates in main analyses of AL. Our rationale is that accounting for sex hormone variations in both sexes is the strongest way to account for biological sex-based modulation of stress physiology linked to AL (8,51). Although this is critical in stress studies on sex differences (36), few studies adjust accordingly for sex hormone functioning (36).

Two saliva samples were used to assess diurnal testosterone, estradiol, and progesterone concentrations during work and/or home collection in adjunct to diurnal cortisol sampling (36). Reliability analyses revealed acceptable intercorrelations between the two repeated measures of diurnal testosterone (women: $r = 0.62, p < .001$; men: $r = 0.60, p < .001$), estradiol (women: $r = 0.58, p < .001$; men: $r = 0.49, p < .001$), and progesterone (women: $r = 0.83, p < .001$; men: $r = 0.60, p < .001$). No associations were detected vis-à-vis seasonal variation or days since last menstruation among women of reproductive age, even when stratified according to

reproductive status. This emphasizes the importance of accounting for objective biometrics of reproductive functioning instead of subjective reports when assessing sex differences in stress physiology.

Statistical Analysis

Analyses were run using the Statistical Package for the Social Sciences Macintosh Version 22. Power analyses were computed using G*Power 3.1. Missing data for gender roles ($n = 5$) and AL biomarkers ($n = 25$) are reflected by varying degrees of freedom throughout our statistical analyses.

Preliminary analyses assessed psychosocial gender factors, demographics, interpersonal, health behaviors, and physical/mental health domains (Table 1) using analysis of variance or χ^2 when appropriate as a function of sex and of gender roles in turn. A correlation matrix of sex/gender factors and outcomes used Pearson correlations for descriptive purposes (Table 4).

Main analyses used analysis of covariance (ANCOVA) for mental health and AL while adjusting for covariates specified in respective subsections. Significance was set at $\alpha = .05$; however, we report trends at $p < .10$ in the interest of transparency. Post hoc analyses were scrutinized using Tukey analyses with 95% confidence intervals (CIs). Effect sizes are reported throughout and can be interpreted according to the following conventions: $\eta_p^2 \cong 0.01$ represents a small effect, $\eta_p^2 \cong 0.06$ a medium effect, and $\eta_p^2 \cong 0.14$ constitutes a large effect.

Inspired by the method developed by Pelletier et al. (34) to index gender, we used principal axis factoring and logistic regression. Specifically in sequence, a) we identified variables related to psychosocial gender in preliminary analyses of sex differences in sample characteristics (Table 1), b) factor loaded these variables onto components, c) used these components to predict the probability of being a man or a woman, and d) finally used this probability to represent the composite gender index that we correlated with mental health and AL.

RESULTS

Sample Characteristics

Table 1 reports the sample's descriptive statistics. Men had higher Bem masculinity gender-role scores ($p < .001$) than did women who in turn had higher Bem femininity gender-role scores ($p < .001$). Sex and gender roles were also related to sexual orientation in our sample: men showed more nonheterosexual proclivities as evidenced by higher Klein sexual attraction ($p = .004$), higher Kinsey scale scores ($p = .008$), and a trend toward higher Klein sexual orientation averages ($p = .092$) than did women. Despite no group differences according to gender-role classification (p values $> .035$), feminine gender roles were negatively associated with the Klein sexual orientation scale ($r = -0.16, p = .027$) and Kinsey scale ($r = -0.14, p = .050$); however, no associations were found for masculine gender roles (r values > 0.10).

Preliminary analyses of group differences according to sex revealed that women were more likely than men to have a diagnosed mental health condition ($p = .002$), psychotropic use ($p = .010$), past psychiatric sick leave ($p = .031$), and a trend toward higher occupational status ($p = .060$). By contrast, men were more likely than women to use illicit drugs ($p = .001$), while showing trends for consuming more alcohol ($p = .061$) and worked more hours ($p = .093$).

TABLE 2. Biomarker Information by Sex and Gender Roles

Biomarkers (Unit)	All	Men	Women	<i>p</i>	Masculine	Feminine	Androgynous	Undifferentiated	<i>p</i>
Hypothalamic-pituitary-adrenal axis functioning									
Cortisol, diurnal CAR, µg/dl, M (SE)	34 (0.026)	0.33 (0.051)	0.34 (0.03)	.902	0.33 (0.04)	0.43 (0.055)	0.33 (0.06)	0.27 (0.052)	.179
difference									
Cortisol, diurnal bedtime, µg/dl, M (SE)	0.13 (0.007)	0.1295 (0.014)	0.132 (0.010)	.398	0.20 (0.01)	0.22 (0.04)	1.91 (0.02)	0.22 (0.02)	.754
Cortisol, TSST systemic output (AUCg)	8.32 (0.39)	9.8 (0.88)	7.7 (0.40)	.013	8.99 (0.80)	8.32 (0.78)	8.22 (0.77)	7.73 (0.76)	.686
Dehydroepiandrosterone-sulfate, µM	5.43 (0.26)	7.724 (0.62)	4.5 (0.22)	<.001	5.6 (0.52)	6.28 (0.63)	5.13 (0.56)	4.65 (0.41)	.162
Immune/inflammation									
Tumor necrosis factor α, pg/ml	1.32 (0.09)	1.26 (0.08)	1.35 (0.091)	.562	1.24 (0.17)	1.31 (0.09)	1.56 (0.19)	1.20 (0.08)	.318
Interleukin-6, pg/ml	1.19 (0.072)	1.1 (0.11)	1.24 (0.092)	.281	1.08 (0.11)	1.1 (0.092)	1.51 (0.30)	1.12 (0.078)	.177
C-reactive protein, mg/l	3.84 (0.37)	2.523 (0.383)	4.4 (0.50)	.020	3.53 (0.60)	4.30 (0.99)	3.78 (0.92)	3.54 (0.53)	.869
Fibrinogen, g/l	3.23 (0.053)	3.0 (0.10)	3.32 (0.06)	.004	3.23 (0.08)	3.23 (0.15)	3.07 (0.12)	3.31 (0.08)	.532
Lipid metabolism									
Triglycerides, mM	1.16 (0.047)	1.15 (0.087)	1.17 (0.06)	.852	1.1 (0.10)	1.2 (0.097)	1.04 (0.11)	1.27 (0.08)	.337
Low-density lipoprotein cholesterol, mM	3.0 (0.06)	3.035 (0.12)	3.0 (0.07)	.735	2.93 (0.09)	3.05 (0.13)	2.99 (0.16)	2.99 (0.12)	.912
High-density lipoprotein cholesterol, mM	1.393 (0.027)	1.23 (0.05)	1.5 (0.032)	<.001	1.41 (0.05)	1.31 (0.05)	1.44 (0.08)	1.43 (0.05)	.338
Glucose metabolism									
Insulin, pM	62.94 (2.64)	63.221 (5.22)	62.83 (3.1)	.946	59.55 (5.0)	71.64 (5.96)	54.43 (5.16)	64.07 (5.0)	.178
Glycosylated hemoglobin, %	.0562 (0.002)	.062 (0.01)	.0538 (0.0003)	.116	.053 (0.000)	.055 (0.001)	.066 (0.013)	.054 (0.000)	.279
Renal and liver functioning									
Creatinine, µM	65.56 (1.02)	80.24 (1.6)	59.38 (0.84)	<.001	66.0 (1.7)	66.13 (1.83)	63.54 (2.43)	65.17 (2.26)	.840
Albumin, g/l	42.28 (0.234)	44.2 (0.40)	41.47 (0.26)	<.001	42.47 (0.45)	42.28 (0.49)	42.18 (0.59)	42.19 (0.42)	.968
Cardiovascular functioning									
Heart rate, TSST systemic output (AUCg)	3853.2 (141.8)	4121.3 (47.2)	3741.8 (42.3)	.224	3712.8 (66.62)	3728.44 (76.69)	3704.46 (94.49)	3713.93 (63.74)	.997
Systolic blood pressure, TSST systemic output (AUCg)	6176.2 (48.2)	6398.6 (84.0)	3916.2 (37.8)	.003	6162.03 (99.13)	6240.78 (84.93)	6043.11 (104.7)	6224.02 (98.04)	.561
Diastolic blood pressure, TSST systemic output (AUCg)	3923 (32.3)	3929.3 (62.2)	6083.8 (57.0)	.745	3881.36 (59.78)	4009.67 (64.70)	3839.46 (81.97)	3944.02 (61.02)	.336
Anthropometrics									
Waist-hip ratio	.86 (0.01)	.91 (0.02)	.83 (0.01)	<.001	.84 (0.012)	.85 (0.012)	.84 (0.02)	.87 (0.01)	.096
Body mass index, kg/m ²	27.1 (0.4)	27.32 (0.64)	26.93 (0.50)	.666	25.90 (0.81)	28.77 (0.75)	25.68 (0.85)	27.63 (0.76)	.025

CAR = cortisol awakening response; M = mean; SE = standard error; AUCg = area under the curve with respect to ground; TSST = Trier Social Stress Test.

TABLE 3. Biomarker Information Used to Calculate “All-Inclusive” and “Sex-Specific” Allostatic Load Indices

Biomarkers (Unit)	“All-Inclusive” and “Sex-Specific” Allostatic Load Cutoff(s)
Hypothalamic-pituitary-adrenal axis functioning	
Cortisol, diurnal CAR, µg/dl, M (SE) difference	All: <-0.0195 or >0.72; ♀: <-0.02 or >0.71; ♂: <-0.0154 or >0.776
Cortisol, diurnal bedtime, µg/dl, M (SE)	All: <0.047 or >0.233; ♀: <0.05 or >0.24; ♂: <0.042 or >0.30
Cortisol, TSST systemic output (AUCg)	All: <3.85 or >13.83; ♀: <3.6 or >12.6; ♂: <4.513 or >16.86
Dehydroepiandrosterone-sulphate, µM	All: <2.9 ♀: <3.9; ♂: <2.6
Immune/inflammation	
Tumor necrosis factor α, pg/ml	All: >1.51; ♀: >1.4660; ♂: >1.5453
Interleukin-6, pg/ml	All: >1.435; ♀: 1.4818; ♂: 1.2645
C-reactive protein, mg/l	All: >4.35; ♀: >5.1; ♂: >3.7
Fibrinogen, g/l	All: >3.73; ♀: >3.77; ♂: >3.42
Lipid metabolism	
Triglycerides, mM	All: >1.48 ♀: >1.505; ♂: >1.46
Low-density lipoprotein cholesterol, mM	All: >3.52; ♀: >3.5325; ♂: >3.46
High-density lipoprotein cholesterol, mM	All: <1.11; ♀: <1.1975; ♂: <1.0
Glucose metabolism	
Insulin, pM	All: >79.45 ♀: >79.2; ♂: >83.0
Glycosylated hemoglobin, %	All: >0.055; ♀: >0.055; ♂: >0.055
Renal and liver functioning	
Creatinine, µM	All: >73.75; ♀: >65.0; ♂: >87.0
Albumin, g/l	All: <40.00; ♀: <40.0; ♂: <42.0
Cardiovascular functioning	
Heart rate, TSST systemic output (AUCg)	All: <3147.5 or >4402.5; ♀: <3190.625 or >4340.625; ♂: <3075 or >4492.5
Systolic blood pressure, TSST systemic output (AUCg)	All: <5466.3 or >6962.5; ♀: <5446.875 or >6800; ♂: <5700 or >7140
Diastolic blood pressure, TSST systemic output (AUCg)	All: <3397.5 or >4461.25; ♀: <3415 or >4412.5; ♂: <3342.5 or >4562.5
Anthropometrics	
Waist-hip ratio	All: >91; ♀: >0.8815; ♂: >0.964
Body mass index, kg/m ²	All: >30.34; ♀: >30.197; ♂: >30.6185

CAR = cortisol awakening response; M = mean; SE = standard error; ♀ = women; ♂ = men; AUCg = area under the curve with respect to ground; TSST = Trier Social Stress Test.

Preliminary analyses of group differences according to gender roles revealed a trend for years of education ($p = .064$) and significance for number of children ($p = .017$) that differed between groups. Post hoc analyses revealed that undifferentiated individuals had less education than did masculine individuals ($p = .044$) and less children than did feminine individuals ($p = .041$), while trending toward fewer children than androgynous individuals ($p = .076$). In addition, undifferentiated individuals were most likely to have a psychiatric history, whereas androgynous individuals were most likely to have a family psychiatric history

($\chi^2(9) = 24.21, p = .004$). Conversely, androgynous individuals were least likely to have past sick leave themselves ($\chi^2(3) = 9.94, p = .019$). Main analyses therefore adjusted for mental health condition that correlated with psychotropic use ($r = 0.91, p < .001$) and psychiatric history ($r = 0.568, p < .001$).

Sex and Gender Roles in Relation to Mental Health

Mental health was assessed using two-way (sex by gender roles) ANCOVAs adjusted for mental health condition

TABLE 4. Descriptive Statistics and Correlation Coefficients for Key Sex/Gender Variables and Study Outcomes

Variable	M (SE)	Correlations																
		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.
1. Sex (0 = men, 1 = women)	—	—																
2. Age, y	40.4 (0.85)	0.076	—															
3. Masculinity	4.63 (0.04)	-0.336***	-0.059	—														
4. Femininity	5.31 (0.03)	0.373***	-0.014	-0.171*	—													
5. Sexual orientation	1.53 (0.08)	-0.123 [†]	-0.139	0.104	-0.161*	—												
6. Testosterone	85.36 (3.72)	-0.719***	-0.089	0.252***	-0.213**	0.121 [†]	—											
7. Estradiol	3.74 (0.10)	0.109	0.065	0.040	-0.046	-0.086	0.102	—										
8. Progesterone	98.36	0.136 [†]	-0.013	-0.037	-0.031	-0.055	0.078	0.255***	—									
9. Allostatic load	5.46 (0.22)	-0.057	0.298***	-0.053	-0.084	-0.103	-0.043	0.097	-0.068	—								
index (all-inclusive)																		
10. Allostatic load	5.49 (0.23)	0.035	0.307***	-0.113	-0.046	-0.144*	-0.113	0.095	-0.057	0.947***	—							
index (sex-specific)																		
11. Self-esteem	33.26 (0.37)	-0.108	0.056	0.368***	0.078	0.00	0.020	0.044	-0.054	-0.10	-0.122 [†]	—						
12. Well-being	45.78 (1.0)	-0.097	-0.001	0.30***	0.147*	0.028	-0.008	-0.121	-0.167*	-0.110	-0.142 [†]	0.604***	—					
13. Social support	62.36 (0.69)	0.059	-0.164*	0.086	0.110	0.038	-0.081	0.018	-0.018	-0.041	-0.070	0.373***	0.357***	—				
14. Depressive Sx	7.25 (0.57)	0.110	-0.058	-0.181*	-0.109	0.002	-0.015	0.035	0.095	0.189***	0.219**	-0.530***	-0.577***	-0.274***	—			
15. PCA Component 1	0.00 (0.073)	0.185	0.408***	0.029	0.321***	-0.343***	-0.150*	0.033	0.066	0.144*	0.154*	0.241***	0.192**	0.020	-0.069	—		
16. PCA Component 2	0.00 (0.073)	-0.187**	0.064	0.553***	-0.358***	0.557***	0.166*	0.018	0.012	-0.028	-0.074	0.095	0.065	0.005	0.057	0.000	—	
17. Composite gender index	0.72 (0.009)	0.266***	0.244**	-0.346***	0.467***	-0.678***	-0.228**	0.015	0.036	0.120 [†]	0.163*	0.113	0.086	0.008	-0.090	0.698***	-0.706***	—

M = mean; SE = standard error; PCA = principal component analysis.

* $p < .05$; ** $p < .01$; *** $p < .001$; † $p < .10$.

and sexual orientation. Sex was not related to self-esteem ($p = .172$, $\eta_p^2 = 0.011$), well-being ($p = .300$, $\eta_p^2 = 0.006$), social support ($p = .393$, $\eta_p^2 = 0.004$), or depressive symptoms ($p = .223$, $\eta_p^2 = 0.009$). By contrast, gender roles were significantly related to self-esteem ($F(3,174) = 10.4$, $p < .001$, $\eta_p^2 = 0.152$), well-being ($F(3,178) = 6.5$, $p < .001$, $\eta_p^2 = 0.099$), and depressive symptoms ($F(3,174) = 3.0$, $p = .033$, $\eta_p^2 = 0.049$), but only trending for social support ($p = .076$, $\eta_p^2 = 0.038$).

Figure 1 presents mental health as a function of gender roles irrespective of sex. Tukey post hoc analyses revealed that androgyny was related to higher self-esteem in contrast to undifferentiated ($p < .001$, CI = 3.38–8.52), feminine-typed ($p = .010$, CI = 0.52–5.41), and trending for masculine-typed ($p = .056$, CI = –0.042 to 4.92) individuals. In addition, masculine-typed ($p = .002$, CI = –6.04 to –0.98) and feminine-typed ($p = .012$, CI = –5.48 to –0.49) individuals had higher self-esteem than did undifferentiated individuals. For well-being, androgyny individuals ($p < .001$, CI = –19.19 to –5.08), masculine-typed ($p = .011$, CI = –15.47 to –1.44), and feminine-typed ($p = .020$, CI = –14.75 to –0.91) scored higher than did undifferentiated individuals. Finally, androgyny related

to lower depressive symptoms ($p = .047$, CI = 0.044–8.4) than did undifferentiated individuals, whereas the reverse trend was observed for social support ($p = .077$, CI = –0.35 to 9.86). Although descriptive, the reader will note that depressive symptoms were positively correlated with AL reported in the following section (Table 4). Post hoc power analysis using obtained parameters yielded an achieved power ($1 - \beta$) of 0.97.

Sex and Gender Roles in Relation to AL

AL was assessed using two formulations: a) all-inclusive cutoffs based on the entire sample's biomarker distributions and b) sex-specific cutoffs based on biomarker distributions for each sex separately. In both cases, identical two-way (sex by gender roles) ANCOVAs controlled for age, sex hormones (basal testosterone, estradiol progesterone), sexual orientation, chronic stress, and mental health condition.

First, using an all-inclusive AL index (Fig. 2A), a significant main effect for sex revealed that men had higher AL than did women ($F(1,164) = 4.1$, $p = .044$, $\eta_p^2 = 0.025$) in addition to a covariation effect for age ($F(1,164) = 16.9$, $p < .001$, $\eta_p^2 = 0.093$). Exploring individual biomarkers revealed that half the biomarkers differed between the

FIGURE 1. Estimated means ($\pm$ SE) for mental health outcomes as a function of gender roles while adjusting for sexual orientation and mental health condition. SE = standard error. * $p < .050$; ** $p < .010$; *** $p < .001$; † $p < .10$.

FIGURE 2. Estimated means ($\pm$ SE) for “all-inclusive” allostatic load as a function of sex (A) and “sex-specific” allostatic load as function of sex and gender roles (B) while adjusting for age, sex hormones (testosterone, estradiol, progesterone), sexual orientation, chronic stress, and mental health condition. SE = standard error. ** $p < .010$; $p < .050$; † $p < .10$.

sexes (Table 2): compared with women, men had higher cortisol ($p = .013$), dehydroepiandrosterone-sulphate ($p < .001$), creatinine ($p < .001$), albumin ($p < .001$), systolic blood pressure ($p = .003$), and waist-to-hip ratios ($p < .001$) as well as lower C-reactive protein ($p = .020$), fibrinogen ($p = .004$), and high-density lipoprotein cholesterol ($p < .001$).

Second, using the sex-specific AL index (Fig. 2B), we found a significant sex by gender role interaction effect ($F(3,164) = 2.8$, $p = .043$, $\eta_p^2 = 0.048$), a main effect for gender roles ($F(3,164) = 2.8$, $p = .040$, $\eta_p^2 = 0.049$), and a covariation effect for age ($F(1,164) = 17.8$, $p < .001$, $\eta_p^2 = 0.098$). Sex-specific reanalyses and Tukey post hoc comparisons revealed that undifferentiated men had higher AL than did androgynous men ($p = .015$, CI = 0.56–6.78) and trending compared with masculine men ($p = .066$, CI = –0.13 to 5.63). Splitting analysis by gender roles revealed that undifferentiated men had higher AL than did undifferentiated women ($F(1,39) = 10.3$, $p = .003$, $\eta_p^2 = 0.210$). For the main effect of gender roles, feminine-typed had higher AL than did masculine-typed individuals; however, this did not approach statistical significance in post hoc analysis. The only biomarker that differed according to sex by gender roles was higher interleukin-6 among women than men ($p = .014$). In addition, androgynous women had higher concentrations than did masculine ($p = .011$, CI = 0.15–1.53), undifferentiated ($p = .039$, CI = –1.44 to –0.03), and trending in comparison to feminine ($p = .082$, CI = –0.06 to 1.41) women. Post hoc power analysis using obtained parameters yielded an achieved power ($1 - \beta$) of 0.92.

Gender Index Constructed Using PCA and Logistic Regression

Following an approach developed by Pelletier et al. (34), we used a PCA and logistic regression to generate a

continuous gender index that we assessed in association to mental health and AL. Guided by preliminary analyses (Table 1), seven variables showing the most pronounced sex differences were selected based on their correspondence to psychosocial gender: a) Bem masculine gender roles, b) Bem feminine gender roles, c) Klein sexual attractions, d) occupational status, e) work hours per week, f) civil status, and g) number of children.

Table 5 provides the factor loadings for the PCA. Two components attained eigenvalues greater than 1 (eigenvalues of 1.6 and 1.3 cumulatively representing 41.5% of the explained variance) that were retained for varimax rotation. In accordance with convention, variables with a factor load of 0.40 and above were considered significant. First, Component 1 was positively associated with feminine gender roles, occupational status, being in a relationship, and number of children. Second, Component 2 was positively associated with masculine

TABLE 5. Gender-Related Component Factor Loadings Generated by Principal Component Analysis

	Component 1	Component 2
Masculine gender roles	.100	.592 ^a
Feminine gender roles	.460 ^a	–.280
Sexual attractions	–.367	.584 ^a
Occupational status	.450 ^a	.289
Work hours per week	.141	.678 ^a
Civil status ^b	.673 ^a	.093
No. children	.744 ^a	.026

^a Refers to variables that significantly load onto a component according to a cutoff $\geq .40$.

^b Civil status was binary coded as 0 (single/separated/divorced) or 1 (relationship/common-law/married).

gender roles, nonheterosexual attractions, and work hours per week. The two PCA components were calculated for every individual and used in a logistic regression predicting sex to create a composite gender index.

A binary logistic regression with the two PCA components entered as predictors of one's probability of being a man (0) or a woman (1) was significant ($\chi^2(2) = 12.7$, $p = .002$, Cox & Snell $R^2 = 0.07$; Nagelkerke $R^2 = 0.09$). The probability of being a woman was positively associated with PCA Component 1 ($\beta = 0.42$, $SE = 0.17$, $p = .015$, $OR = 1.52$) and negatively associated with PCA Component 2 ($\beta = -0.43$, $SE = 0.18$, $p = .018$, $OR = 0.66$). This probability score generated by the regression is therefore the composite gender index representing the likelihood of one's sex as a man (more negative scores) or a woman (more positive scores) as initially predicted by the PCA components based on seven psychosocial gender-related factors.

Table 4 reveals that our composite gender index is correlated with key sex/gender variables. Despite no significant associations with mental health or the all-inclusive AL index, the gender index was positively correlated with the sex-specific AL index ($r = 0.16$, $p = .025$). To ensure that this association was independent of sex, partial correlations controlling for sex confirmed that the gender index was significantly correlated only with the sex-specific AL index ($r = 0.16$, $p = .029$) and as a trend with the all-inclusive AL index ($r = 0.14$, $p = .053$).

DISCUSSION

The goal of this study was to apply a sex by gender-based analysis of mental health and AL among healthy working adults recruited from a psychiatric hospital. To reiterate, Bem (11–13) postulated that androgynous individuals are psychologically adaptive because they aptly alternate between masculine and feminine gender roles. By contrast, undifferentiated individuals show low propensities for these gender roles and are thought to be at greater risk for psychopathology due to an inability to conform to societal expectations (13). Our hypotheses partially support Bem's theory in that androgyny seems to be biologically adaptive only among men while being psychologically adaptive for both sexes. In addition, a composite gender index representing a greater probability of multidimensional “femininity” was associated with AL based on sex-specific biomarker cutoffs independent of sex. Whether gender is measured categorically or continuously, our results suggest that a gendered lens allows us to understand stress-related disease trajectories that differ between the sexes.

Our sex *by* gender roles interaction for AL provides powerful evidence for Bem's proposal (11–13) that androgyny is advantageous in comparison to undifferentiation. But why would androgyny be protective against AL in men but not women? It is possible that high appropriation of both

masculine and feminine gender roles is not as beneficial for women as it is for men. This androgynous combination is not new to modern women who have needed to appropriate masculine gender roles to break the “glass ceiling” of workplaces worldwide. Indeed, women have increased their participation in the workforce by 126% over the last 30 years (52) and represent 42% of the entire global labor force (1,53,54). However, women around the world suffer more from competitive socioeconomic pressures, job insecurity, limited opportunities, and inadequate social benefits such as insurance and sick leave (5).

Pioneering research from the 1980s onward by Frankenhaeuser and Lundberg revealed that Scandinavian women in nontraditional or “masculine” occupations (e.g., managerial versus clerical) reported more distress, hostility, inability to recover after work, physical complaints when older, and manifested pathophysiological functioning (55,56). The appropriation of multiple gender roles among working women has clearly not been matched by appropriate social equity. In general, women still retain more “feminine” roles and responsibilities than the average man in both work and home life (1,34,57,58). This may explain why the AL burden is heaviest for feminine and androgynous women, but does not seem so for men. Indeed, masculine gender roles were associated with marginally lower AL than feminine gender roles. This is inconsistent with our past finding linking greater masculinity relative to femininity and elevated AL in a much smaller sample of varied workers (29). Undifferentiated gender identity also seems here to be worst among men than women for AL. Future studies assessing factors inside and outside the workplace are warranted to help tease apart these mixed gender effects. In particular, age cohort effects and cross-cultural contrasts should be explored more broadly.

Methodologically, different AL formulations may capture distinct sex- versus gender-based effects. In our study, men had higher AL than women when using the traditional all-inclusive formulation. These sex differences were driven by nine biomarkers, suggesting that using this approach is most appropriate when endeavoring to understand biological sex differences. This was not the case when using the less common sex-specific formulation that instead captured the influence of sociocultural gender-role diversity. These alternative AL indices are not trivial because they could be helpful in nuancing main effects or interactions among gender-based factors (e.g., social networks, family responsibilities) versus biological factors like sex or age. Indeed, this distinction between all-inclusive versus sex-specific AL formulations should be considered in future studies aimed at respectively demarcating between-sex differences or within-sex diversity in AL and stress-related disease pathways.

Beyond our objectives focusing on Bem's gender schema theory, we expand upon recent endeavors to

quantify psychosocial gender. Using a composite gender index constructed using a method developed by Pelletier et al. (34), we found that a greater propensity toward femininity was positively associated to the sex-specific AL index. This gender index was constructed using first our PCA resulting in the reduced seven gendered factors into two components: a) Component 1 derived from feminine gender roles, occupational status, civil status, and number of children and b) Component 2 derived from masculine gender roles, non-heterosexual attractions, and work hours. Our finding therefore compliments the work of Pelletier et al., (34) who showed in a large cohort that a feminine gender index based on seven components corresponded with a greater propensity toward premature cardiovascular disease and recurring complications for 1 year (59). Similarly using the same cohort, those with more feminine gender show poorer access to care than do men and women with more masculine gender-related characteristics (60). Although our result is of weak statistical magnitude, using innovative methods to similarly construct gender indices provides a unique way to delineate psychosocial contexts related to but still distinct from one's sex that are related to AL (61).

Previous research has shown that gender roles are predictive of health outcomes, but not necessarily independently from one's biological sex (62–64). Our findings carefully adjusted for sex hormones and sexual orientation among other covariates, which provides insights specifically due to sex and gender. This is critical when endeavoring to understand health problems, especially when we consider that objective and subjective measures of stress may manifest themselves differently according to sex/gender. Indeed, women self-report more perceived stressors, psychosocial distress, and physical complaints than do men (65–67). By contrast, men are generally more biologically stress responsive at younger ages (68–71). It is possible that changing gender roles might also influence men in ways that have yet to be studied fully using sex- and gender-based approaches. For example, a Swedish study found that paternity leave—“a departure from traditional masculinity”—was associated with decreased mortality among fathers (33). Gendered coping strategies interact with numerous intersecting factors to influence how individuals deal with major stressful events in their lives that should be explored.

Independent of sex, androgynous individuals self-reported higher self-esteem, better well-being, and less depressive symptoms than did undifferentiated individuals. In addition, undifferentiated individuals had lower self-esteem and well-being than did masculine-typed and feminine-typed individuals. In terms of descriptive characteristics, undifferentiated individuals were less educated than masculine gender types, had fewer children than did feminine gender types, and were the most likely to have had a past psychiatric episode. By contrast, androgynous

individuals were more likely to have a family psychiatric history, yet less likely to have had past sick leave. Taken together, these profiles concord with a large body of theoretical and empirical literature on the advantages of androgyny and the disadvantages of undifferentiation (10–13,72). They do, however, beg the following question: who are undifferentiated individuals and what can be done to help them? Although beyond the scope of the current study, answering these questions represents vital empirical and clinical future directions.

Critically, interventions attempting to reverse deleterious pathways may be complicated by the fact that sex is so often totally disregarded in clinical research (73). For example, a critique of 38 Cochrane systemic reviews on cardiovascular disease identified that sex and gender were rarely considered despite mounting evidence that sex influences heart health (74). Given that Cochrane guidelines are considered to use rigorous standards for inclusion, this is particularly troublesome. More broadly in preclinical and clinical research (e.g., randomized controlled trials), the disaggregation of findings on the basis of sex is fundamental (33,75,76). Disregard for sex/gender is especially problematic for the prevalence of numerous stress-related diseases that we argue is crucial for the advancement of the AL model. Measuring sex as a binary classification is clearly not sufficient when attempting to understand differences between men, women, and gender diverse individuals.

Limitations

Several considerations limit our findings. Despite a respectable sample size, men were underrepresented. A systematic review of workplace AL studies reports that men characterize 46% of participants that are predominantly European professionals working in different industries (77). Despite fewer men, our study is unique in assessing diverse professionals of both sexes working in the same hospital environment, but which also constrains generalizability. Relatedly, because the focus of this study was on the role of sex- and gender-based factors, the role of work and home life was not scrutinized beyond inclusion within our gender index. Our sample was moreover heterogeneous and individuals reported varying degrees of distress and mental health. Although all participants were healthy enough to be working, it is difficult to compare our study to those using stricter exclusion criteria or who study samples suffering from specific diseases.

The Bem Sex Role Inventory has psychometric issues (72) and using median splits are controversial. The internal consistency of the femininity subscale was also significantly weaker than that of the masculinity subscale, suggesting that masculine gender roles are preferentially self-endorsed among this sample of psychiatric hospital workers. Most importantly, gender roles have changed considerably since the mid-1970s and may render this

measure of gender roles outdated and in need of an update. Nevertheless, men did score higher on masculinity and women higher on femininity, suggesting that Bem's gendered stereotypes remain applicable today. Although descriptive (Table 4), it is also noteworthy that testosterone concentrations were positively correlated with masculine gender roles and negatively correlated with feminine gender roles, providing unique triangulation of methods that should be explored further. Correcting for multiple comparisons would, however, render these results nonsignificant, emphasizing the preliminary nature of these correlations.

At a theoretical level, our findings force us to conceptually move beyond bipolar views not only of men and women but also of masculinity and femininity. In developing her theory that androgynous individuals are more adaptively "flexible," Bem (11) proposed that masculine and feminine gender roles are independent entities and therefore not binary. However, when interpreting gender roles along continuums, it is easy to resort to simplifications that can make masculine and feminine gender roles seem like oppositional constructs when they are two independent dimensions that are in fact context-specific. Hammarström (78) has noted that gender-role theories assume an opposite coexistence (e.g., "active" versus "passive," men are from Mars and women are from Venus). Researchers must be cautious with such dichotomizations (79). In accordance, we did not find marked differences when contrasting masculine and feminine individuals, but instead when comparing androgynous and undifferentiated individuals or when assessing gender multidimensionally. Although our results provide psychobiological support for Bem's hypothesis, continuous gender indices provided an additional layer of complexity transcending outdated definitions of gender roles. This is consistent with recent approaches that emphasize sex/gender "mosaics" along continuums rather than categories (80).

CONCLUSIONS

To the best of our knowledge, this is the first study to comprehensively assess the influence of biological sex and sociocultural gender roles in relation to AL and mental health. We show that androgyny, especially among men, seems to be psychobiologically adaptive. In addition using various gendered factors, we showed that a tendency toward greater femininity was associated with elevated AL. Our results contribute to a sequestered literature where biomedical perspectives of sex and psychosocial perspectives of gender are rarely studied together (32). Applying a sex- and gender-based analysis recognizes this plurality of individuality and can be viewed as an iterative process that evolves by refining research methodologies that promote equity in science, policy, and practice (28,81). At a methodological level, our AL findings show that different formulations may capture sex effects (all-inclusive) versus gender effects (sex-specific).

In conclusion, our findings suggest that stress researchers should abstain from studying sex as a dichotomy and focus further on gender diversity when endeavoring to understand disease trajectories that are not always sex-specific.

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