REGULAR ARTICLE

Physical Activity Buffers the Adverse Impacts of Racial Discrimination on Allostatic Load Among Indigenous Adults

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Abstract

Background Racial discrimination has been associated with biological dysfunction among ethnic minorities. The extent to which regular physical activity (PA) may buffer this association is unknown.

Purpose To examine the association between past-year racial discrimination and allostatic load (AL) stratified by PA within a sample of Indigenous adults.

Methods Data were collected from Indigenous adults attending university in a city in western Canada between 2015 and 2017. The Experiences of Discrimination Scale was used to assess discrimination and the Godin–Shephard Leisure-Time Physical Activity Questionnaire assessed PA. A composite of seven biomarkers assessing neuroendocrine, cardiovascular, metabolic, and immune system function measured AL. Linear regression models examined associations adjusted for confounders (N = 150).

Results In the insufficiently active group, every 1 point increase in racial discrimination (up to a maximum of 9) resulted in approximately one third of a point increase in AL score. In the sufficiently active group, the association between racial discrimination and AL score was not statistically significant.

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Conclusions A growing body of research suggests racial discrimination is associated with multisystem biological dysregulation and health risks. Increased action to address racism in society is a priority. As that work unfolds, there is a need to identify effective tools that racialized groups can use to buffer the effects of racism on their health. The present findings suggest that engagement in regular PA may attenuate the pernicious effects of discrimination on biological dysfunction.

Keywords: Racial discrimination · Racism · Health · Stress · Indigenous · Physical activity · Allostatic load

Introduction

Racism has significant effects on health, and surveys indicate Indigenous people across many countries experience high levels of racial discrimination across life domains [1, 2]. Racial discrimination has wide-ranging impacts that go beyond mental distress to include alterations in stress biomarkers across multiple domains [3–8]. The cumulative impacts of discrimination on biological health may play a role in the health inequities observed across many ethnic minority populations, including Indigenous populations [9]. Addressing the overarching problem of systemic racism is of paramount importance to equity and population health [10]. While that work unfolds, there is a need to identify potential strategies that minority populations can use to buffer the effects of racism on their health.

The present study is part of a larger project that used a *Two-Eyed Seeing* perspective to understand how racial discrimination may be impacting the health and well-being of Indigenous adults, as well as potential strategies that individuals can use to buffer these impacts. Two-Eyed Seeing refers to learning to see from one eye with the strengths of Indigenous knowledge systems and ways of knowing and the other eye with the strengths of Western knowledge systems and ways of knowing and, importantly, learning to examine problems using the best knowledge available from both systems [11]. Using the same data set examined in the present study, we found that the association between racial discrimination and multisystem biological dysregulation may be buffered by Indigenous cultural continuity [12, 13]. The present analysis sought to examine the role that physical activity (PA), as a Western concept of promoting health, may play in attenuating the impact of racial discrimination on multisystem biological dysregulation, operationalized using allostatic load (AL).

Allostasis is the process by which the body responds and adapts to stressors by changing the defended levels of a regulated physiological variable, sometimes referred to as "stability through change" [14, 15]. Allostasis requires the release of chemical signals that act as primary mediators, including the activation of the hypothalamic-pituitary-adrenal (HPA) axis. In the face of unremitting stress, the continuous secretion of primary mediators leads to physiological dysregulation and the development of secondary outcomes, including risk factors for cardiovascular and metabolic disease. Eventually, AL leads to morbidity as a tertiary outcome [15]. The model of AL was developed to quantify the cumulative physiological "wear and tear" that results from repeated cycles of allostasis by measuring a combination of primary mediators and secondary outcomes [16, 17]. Higher AL is associated with greater risk of premature mortality, with a cumulative measure of AL serving as a better predictor of mortality than individual biomarkers analyzed separately [18–20]. Further, Seeman et al. found that a summary measure of AL provides important information about differential mortality risk that cannot be determined from measuring individual biological markers [21].

While AL is known to increase with age as a result of the cumulative effects of allostasis across the lifespan, at any given age, there can be a range of AL experienced by people, resulting from differential life experiences and exposures to social stressors [21]. For example, among African Americans, lifetime discrimination has been associated with increased AL in middle adulthood [4]. Another study found that a measure of chronic everyday discrimination experienced between 16 and 18 years of age was positively associated with AL at age 20 [22]. Allen et al. documented a nonlinear association between discrimination and AL that was moderated by educational attainment among African American women [7].

In the present analysis, we sought to examine whether PA served as a resilience factor that could buffer the impacts of racial discrimination on AL. Resilience represents the ability to sustain relatively normal functioning despite significant adversity or risks [23]. Within a buffering hypothesis, resilience is conceptualized as a separate dimension to risk, acting to moderate the impact of risk on an outcome [24]. Thus, rather than being investigated as a correlate of an outcome, a resilience variable is examined as a factor that may attenuate the association between a correlate and an outcome, typically in a stratified analysis [24].

While PA is associated with lower AL among ethnic minorities [25, 26], to our knowledge, no studies have explicitly examined the role of PA in buffering the impacts of racial discrimination on AL. Yet, such an impact is plausible given research suggests that regular PA might confer resilience by optimizing neuroendocrine and physiological responses to physical and psychosocial stressors [27]. Indeed, habitual activity has been shown to moderate HPA axis reactivity to acute stress in a lab setting [28]. Thus, the objective of this study was to examine the role that PA may play in buffering the impact of racial discrimination on AL. We hypothesized that regular PA would attenuate the association between racial discrimination and AL among Indigenous adults.

Methods

Study Design

Study procedures were approved by the Institutional Human Participant Research Committee. Participants were recruited using posters and ads placed in e-newsletters on campus. Participant recruitment and data collection began in September 2015 and continued over four academic terms, ending in April 2017. The final sample size was 150 Indigenous adults. Informed consent was obtained from all individual participants included in the study.

Participatory Action Research

This study was conceptualized using a participatory action research framework [29]. An Indigenous Advisory Committee made up of key elders and members of the Indigenous community in our territory was assembled to discuss pertinent research questions. Working together, we set study priorities and made data collection decisions. The Committee suggested Indigenous university students as a population focus given many have overcome significant hardships to attend university. Thus, it was suggested that this population would be a particularly good test of the hypothesis that racial discrimination and other forms of social adversity could impact AL as this group may be more resilient in the face of stressors relative to other Indigenous adults. More than half of all Indigenous Canadians aged 25-64 years have a postsecondary qualification, including 34% who have completed university or college [30]. The number of Indigenous adults who are attending and completing university or college is growing with each Census cycle [30]. Similar to Indigenous populations in other countries, Indigenous Canadians are also urbanizing rapidly, with the majority of the population now living in cities rather than Indigenous communities [31]. Thus, while the population focus for this study (Indigenous university students aged 18-60 years) does not represent all Indigenous peoples in Canada, it does represent both a large and growing segment of this population and is in keeping with larger urbanization and educational trends across many Indigenous populations worldwide [32].

Working with the Indigenous Advisory Committee, we determined that saliva rather than blood samples would be taken. As saliva is a substance that comes from the body, a system was put in place in consultation with Indigenous elders to ensure that the wishes of participants were honored. The consent form provided participants the option of having their saliva samples returned to them upon analysis or to have their saliva samples included in an Indigenous ceremony led by an elder that returned the samples to the Earth.

Procedures

Respondents were asked to confirm eligibility by email/ phone (i.e., identified as Indigenous, were current postsecondary students, and were 18 years or older). Participants then attended an on-campus study office to complete consent procedures, paper-and-pencil surveys, and the physical assessments needed to calculate AL score (mean completion time = 90 min) during standard office hours (9:00 AM to 4:00 PM). Saliva samples were collected at three time points during this visit using the passive drool technique. Participants rinsed their mouth with water upon arrival and the first sample was collected after completing a portion of the questionnaire. The remaining samples were taken 30 and 60 min later. Whole saliva samples were collected in a 2 mL microcentrifuge tube using a Saliva Collection Aid (Salimetrics, State College, PA). During data collection, salivary samples were stored in a small in-office freezer and then transferred to a -80°C freezer until analysis.

For the assessment of cortisol awakening response (CAR), participants were instructed to collect saliva samples at home, over two consecutive days, at three time points: immediately upon wake-up, 30 min after wake-up, and before bed. Participants were instructed to place a swab (Salimetrics, State College, PA) under the tongue for 3 min and then place it in a prelabeled tube

and put it in their freezer. When all six samples were collected, the participant contacted the research assistant to coordinate sample return. We used CAR expert consensus guidelines to increase adherence, including clearly explaining the importance of strict adherence to sampling times, emphasizing the importance of collecting Sample 1 immediately upon awakening, encouraging participants to ask questions via text message/email/ phone, providing take-home instructions, having participants record data collection time points in a diary log, advising participants to place kits beside the bed for morning collection, and text messaging the evening before sampling to highlight instructions [33]. Participants returned the samples in an insulated lunch kit with a freezer pack given to them during the in-office visit and samples were then transferred to a -80°C freezer until analysis. Participants were given an honorarium of \$50 for in-office measures and \$50 for at-home measures.

Data Collection and Measurements

Allostatic load

A 2017 systematic review by Johnson et al. called for a more critical approach in the calculation of AL indices to ensure that biomarkers used captured the *biological effects of psychosocial stress* rather than markers of physiologic dysfunction more generally [34]. Taking this into consideration, we operationalized AL using markers from the three biological domains that framed the original AL index (i.e., neuroendocrine, cardiovascular, and metabolic) [16]. We also added an immune marker given the general agreement that immune function should also be included in AL calculations [35].

- Cardiovascular markers: Resting systolic and diastolic blood pressures were measured using a Life Source automated sphygmomanometer (Auto Control Medical, Mississauga, ON). Three readings were taken. The first was taken approximately 15 min after the participant arrived, once they had completed the consent process and answered the first part of the survey package in a seated position. This reading was discarded. Two additional readings were taken approximately 15 min and 30 min after the first, while the participant was seated and completing the survey package. These two measures were averaged.
- Neuroendocrine markers included dehydroepiandrosterone-sulfate (DHEA-S) and CAR. DHEA-S concentrations in various body fluids, including saliva, have been shown to decrease with aging, chronic stress, and inflammation [36]. Petros et al. suggested that salivary DHEA-S could be a biomarker of resilience to adversity [37]. All samples were analyzed in duplicate. As per the manufacturer's

suggestion for DHEA-S, the three in-office samples were pooled and mixed for analysis. Alterations in the CAR have been associated with stress and both amplified and blunted CAR has been associated with poor health outcomes [38, 39]. To examine CAR, the wake-up (S1) and 30 min postwake-up (S2) samples taken at home on the second day were analyzed, and the percentage change in cortisol between S1 and S2 was calculated. Day 1 at-home samples were not used because there was more missing data for Day 1. CAR represents the sharp rise in cortisol levels across the first 30–45 min following morning awakening; in healthy adults, the magnitude of CAR ranges between 50% and 156% [40].

- 3. *Metabolic markers* included body mass index (BMI) and waist circumference (WC). To calculate BMI, height and weight were measured to the nearest 0.5 cm and 0.1 kg, respectively, using a Health-O-Meter mechanical beam scale and stadiometer. WC was measured at the top of the iliac crest to the nearest 0.5 cm. Although correlated (Pearson's r = .87 in this sample), both measures were included in the AL score as each is associated with health risk.
- 4. *Immune markers*: C-reactive protein (CRP) is an acute-phase protein produced in the liver that provides a biomarker of systemic inflammation. Previous research has confirmed the validity of salivary CRP measurement [41]. We measured CRP using the third in-office saliva sample.

AL risk assessment was based on the distribution of the study sample for salivary CRP and DHEA-S by dividing the sample into sex-specific quartiles with high risk defined by the highest quartile for CRP and the lowest quartile for DHEA-S. For the remaining biomarkers, we used standard cut points to define higher risk [42, 43]. Table 1 shows the range and the high-risk cut points used for all biomarkers. Consistent with prior studies, 1 point was assigned if the variable was high risk and 0 if not [44]. Scores were summed to create a total score for AL.

Racial discrimination

The Experiences of Discrimination Scale is a valid and reliable measure of self-reported racial discrimination that has been used across many ethnic groups [45]. The situation score is derived by counting the number of situations (one to nine) in which racial discrimination was experienced. Previous research suggests that Indigenous Canadians experience high levels of racism and that a scale adjusted to measure experiences in the past 12 months is recommended to ensure sufficient variability [1, 46]. Consistent with previous research, each question in the present study was worded as follows, with information in brackets reflecting words added, and X reflecting the situation tested: (In the past 12 months) have you experienced discrimination, been prevented from doing something, or been hassled or made to feel inferior at X because of your (Aboriginal) race, ethnicity or color? Internal consistency of the measure in this study was good ($\alpha = .82$).

Physical activity

The Godin–Shephard Leisure-Time Physical Activity Questionnaire is a four-item valid and reliable measure of self-reported PA completed during leisure time for at least 15 min over a typical week [47–50]. The scale examines the number of times an individual engages in leisuretime PA (LTPA). The score derived represents total weekly moderate to strenuous activity, called a Leisure

Table 1. Mean, range, and cut points used for allostatic load (AL) biomarkers (N = 150)

Biomarker	Range	Mean	SD	Cut-point female	Cut-point male
1. Cardiovascular					
Resting SBP (mm Hg)	90, 150	119.3	13.0	>140	>140
Resting DBP (mm Hg)	59, 111	78.3	10.3	>90	>90
2. Neuroendocrine					
DHEA-S (pg/mL)	188.5, 16,055.6	4,284.0	3,743.0	<1,419.5	<2,865.1
CAR	-98.8, 771.7	70.6	152.6	<50.0 or >156.0	<50.0 or >156.0
3. Metabolic					
BMI (kg/m ²)	18.8, 48.5	29.2	6.8	>30.0	>30.0
WC (cm)	68.9, 166.4	98.7	18.5	>88.0	>102.0
4. Immune					
CRP (pg/mL)	55.1, 3,150.0	459.5	686.9	>397.8	>711.8
Total AL Score	0–6	2.5	1.3		

BMI body mass index; *CAR* cortisol awakening response; *CRP* C-reactive protein; *DBP* diastolic blood pressure; *DHEA-S* dehydroepiandrosterone-sulfate; *SBP* systolic blood pressure; *WC* waist circumference.

Score Index (LSI), which was calculated by multiplying the number of bouts of moderate and strenuous LTPA by 5 and 9, respectively, and then summing these numbers. The LSI is in arbitrary units and can rank people according to their habitual activity, with a higher score indicating a greater volume of leisure-time PA. The LSI was also used to classify participants into sufficiently active (LSI >24) and insufficiently active (LSI \leq 24); Amireault and Godin provide evidence of the validity of this classification system [47].

Covariates

Age, sex, and perceived income group were measured so that they could be tested as potential confounders. Perceived income was assessed by a question that asked participants which income group they currently identified with. The five response options were: upper income, upper-middle income, middle income, lower-middle income, and lower income. Income group was assessed in this manner to improve validity and reduce missing data, given that university students may not know their household income if they are living with their parents. Previous Indigenous research has documented low missing values when income was measured with Indigenous participants in our territory using these categories as compared to categories that use dollar amounts (25% missing data vs. 2% missing data) [51–53].

Missing Data

Data were collected from 150 participants, 35 of whom chose to not complete and/or return at-home samples. For an additional eight, we were unable to calculate valid CAR because their at-home salivary sample was collected more than 45 min after their recorded wake time or their recorded wake time on two consecutive days was 2 standard deviations (SDs) higher than the average wake time for the sample [33]. Also, two participants did not complete questions about discrimination in the past 12 months and one did not report their age. We conducted separate variance *t*-tests for the mean values of valid cases and missing cases across continuous variables. The p-values were not significant across all comparisons, indicating that there were no group differences between the variance of valid cases and missing cases. Cases with missing values were not systematically different from cases without missing values across variables (Little's MCAR test chi-square = 45.7, degrees of freedom [df] = 67, p = .98). This means, for example, that participants who followed the at-home protocol instructions and returned their salivary samples did not systematically differ from participants who did not across the variables examined. With these criteria met, we used the Missing Values Analysis extension with SPSS 26.0 to conduct multiple imputation. Values were drawn from regression-predicted posterior distributions using over 50 iterations. All items from the six variables examined in this paper were included in the imputation (i.e., seven AL items used to create the AL outcome score, nine racial discrimination items used to create the racial discrimination score, four PA items, and three sociodemographic covariate items). Each imputed variable item was constrained to the minimum and maximum range for that item. After imputation, AL score, racial discrimination score, and PA score were computed using their respective items for each imputed data set and analyzed jointly as pooled results per Rubin's formula [54].

Analysis Strategy

Measures were summarized with means \pm SDs for continuous variables and frequencies for dichotomous variables. Linear regression models were used to quantify the change in AL per unit change in the number of situations in which discrimination had been experienced in the past year, with AL examined as a continuous variable. To examine the role PA might play in buffering the impacts of past-year racial discrimination on AL, an interaction between racial discrimination and PA was modeled by forming a product term between the two continuous variables and calculating two R^2 values, one for the main-effects-only model and another with the product term added, with the interaction deemed present if the difference between the two R^2 values was statistically significant as determined by an F-statistic. A series of confounders were individually tested before entry into the main model (age, sex, and current income group). Those associated with AL at p < .20 were retained. Confounders were tested as potential interaction variables using LOWESS curves and multiplicative interaction terms before entry into the final model; none were found. Data were analyzed using IBM SPSS 26. A sensitivity analysis was conducted to examine associations using listwise deletion for missing cells (n = 104; analysis not shown) compared to the imputed data set (n = 150; Table 3). There was no change in the statistical significance of findings across study objectives using either method.

Results

Sample Characteristics

Sample characteristics are shown in Table 2. All participants were adult postsecondary students and most (98.1%) attended school full time. The mean age was 28.1 years (SD = 8.9, range = 18–57 years). Almost three

Table 2.	Characteristics of the sampl	e

Characteristics	Total n (%)	
Total sample		
Sex		
Female	109 (72.6)	
Male	41 (27.3)	
Age		
18–24 years	67 (44.7)	
25–34 years	48 (32.0)	
35–44 years	27 (18.0)	
45+ years	8 (5.3)	
Income group as adult		
Upper middle/upper income	9 (6.7)	
Middle income	35 (23.1)	
Lower middle	70 (50.0)	
Low income	36 (20.2)	
Physical activity		
Sufficiently active	89 (59.3)	
Insufficiently active	61 (40.7)	
Physical activity mean (SD)	32.9 (21.7)	
Racial discrimination mean (SD)	2.4 (2.1)	

SD standard deviation.

quarters were female in keeping with the gender balance for Indigenous students in most Canadian universities [55]. Most identified as low-middle or low income. The mean LSI for PA was 32.9 ± 21.7 with a range from 0 to 97. Approximately 75% of the sample (75.3%) had experienced discrimination in the past year. On average, participants had experienced racial discrimination across 2.4 of the 9 situations examined (SD = 2.1, range = 0-7 situations), most frequently in stores and restaurants, in a public setting, and at school. The mean AL score for this sample was 2.5 out of a possible 7 (SD = 1.3, range = 0-6). The AL median and mode were both 2.0. The AL skewness value was 0.49, suggesting that the distribution of AL scores was approximately symmetric for this sample. The AL kurtosis value was -0.2 indicating low kurtosis and low outliers (i.e., the extreme values are less than that of the normal distribution).

Past-Year Racial Discrimination, AL, and PA

As shown in Table 3 (Model 1), every additional situation in which racial discrimination had been experienced in the past year resulted in a 0.16 point increase in AL score, adjusting for PA (the LSI), age, sex, and income. Every 1 point increase in the LSI was associated with a 0.01 point decrease in AL score, adjusting for racial discrimination, age, sex, and income. A score of 25 or greater on the LSI can be used to classify an individual as sufficiently active [47]. Thus, data from Table 3 (Model 1) suggest adults who met minimum criteria for sufficient PA, with an LSI score of 25, had an AL score that was one quarter of a point lower than adults with an LSI score of 0 (i.e., unweighted beta coefficient = $-.01 \times$ an LSI score of 25).

Taken together, Model 1 explained 25% of the variance in AL score. In Model 2, a multiplicative interaction term for racial discrimination and PA was added to the model. The product term was statistically significant. A hierarchical *F-statistic* comparing the R^2 values for the main-effects-only model and Model 2 with the product term added was statistically significant (*F*-change statistic = 4.04, df = 140, p = .049). Model 2 explained 27% of the variance in AL score.

To examine the role that PA might play in buffering the impact of racial discrimination on AL, the sample was stratified into sufficiently physically active (n = 89)and insufficiently physically active (n = 61) groups. Approximately, 60% of the sample was sufficiently active. On average, insufficiently active adults reported racial discrimination across 2.2 out of a potential 9 situations in the past year (median = 2.0, range = 0-7) compared to 2.5 situations among sufficiently active adults (median = 2.0, range = 0-7); this difference was not statistically significant (independent samples *t*-test = -0.99, df = 145, p = .34). Associations between past-year discrimination and AL stratified by PA group were modeled in linear regression models. As shown in Table 3 (Model 3), every additional situation in which racial discrimination had been experienced in the past year resulted in approximately a third of a point increase in AL score, adjusting for age, sex, and income. Taken together, Model 3 explained 32% of the variance in AL score. Within the sufficiently active group (Model 4), the association between racial discrimination and AL was weaker and no longer statistically significant.

Discussion

Over the past decade, the adverse impacts of racial discrimination on health have become increasingly clear [3–5, 9, 22]. This growing body of research is important and creates a sense of urgency for more action to address racial discrimination in society. Yet, at the same time, these findings can create a sense of concern among racialized groups about the consequences of discrimination on their health, given that few studies have examined strategies that individuals can use to buffer these health impacts. The purpose of this study was to examine the role that PA might play in buffering the negative impact of racial discrimination on AL, given that PA is associated with reduced AL more generally [25, 26]. We found that the association between racial

	N	$Adj R^2$	SE	<i>B</i> (95% CI)	р
Model 1: Full sample	150	0.25			
Past-year discrimination			0.05	0.16 (0.07, 0.25)	.001
Physical activity			0.01	-0.01 (-0.02, -0.01)	.03
Age			0.01	0.05 (0.03, 0.07)	.001
Sex			0.21	0.10 (-0.51, 0.31)	.64
Income			0.12	-0.04 (-0.28, 0.20)	.75
Model 2: Full sample	150	0.27			
Past-year discrimination			0.09	0.31 (0.14, 0.49)	.001
Physical activity (PA)			0.01	0.08 (-0.01, 0.64)	.97
Past-year discrimination × PA			0.01	-0.01 (-0.01, 0.00)	.049
Age			0.01	0.05 (0.02, 0.07)	.001
Sex			0.21	0.10 (-0.51, 0.31)	.62
Income			0.12	-0.06 (-0.30, 0.18)	.61
Model 3: Insufficiently active group	61	0.32			
Past-year discrimination			0.08	0.35 (0.18, 0.51)	.001
Age			0.02	0.02 (-0.01, 0.06)	.25
Sex			0.29	0.29 (-0.96, 0.38)	.40
Income			0.19	-0.24 (-0.62, 0.13)	.20
Model 4: Sufficiently active group	89	0.22			
Past-year discrimination			0.05	0.08 (-0.02, 0.19)	.13
Age			0.02	0.06 (0.03, 0.09)	.001
Sex			0.30	0.05 (-0.46, 0.57)	.84
Income			0.17	0.07 (-0.24, 0.37)	.68

Table 3. Linear regression models for the direct effects of past-year discrimination score on AL score, with and without stratification by physical activity^a (N = 150)

B unstandardized beta weight; CI confidence interval; SE standard error.

^aStatistically significant variables presented in bold.

discrimination and AL was attenuated among adults who reported engaging in regular PA. Among adults who were insufficiently active, AL score increased by more than a third of a point (0.35) for each additional situation in which racial discrimination had been experienced in the past year. On average, this subgroup had experienced discrimination across 2.2 situations over the past year, resulting in an AL score that was three quarters of a point (0.77) higher than their peers who had not experienced discrimination, after adjustment for age, sex, and income. This is significant, given that there is a dose-response association between higher cumulative AL and premature mortality [18-20]. Specifically, in a 2017 prospective cohort of 4,500 adults, a 1 unit increase in AL (i.e., one biomarker being scored in the higher risk quartile) was associated with an 8% increased risk of dying from all causes in 10 years after adjustment for age, sex, and income [20]. Adults in the present study had experienced racial discrimination in zero to seven situations in the past year, resulting in a potential AL increase that ranged up to 2.5 points for the insufficiently active group.

AL is typically operationalized using markers of physiologic dysfunction across cardiovascular, metabolic, neuroendocrine, and immune system domains [16, 35], all of which are affected by PA. It is well documented that cardiovascular and metabolic systems are positively impacted by regular PA [56, 57]. In terms of neuroendocrine function, regular PA has been shown to blunt the acute reactivity of the HPA axis to stress across several studies [27, 28, 58]. For example, Gerber et al. found that postsecondary students with high perceived stress and low vigorous PA had greater cortisol reactivity to a standardized psychosocial stressor than students who met minimum PA guidelines for vigorous activity. Students with high perceived stress and high PA demonstrated adrenocortical stress reactivity that was comparable to students who reported low perceived stress [59]. A recent review similarly concluded that regular PA reduces the overall cortisol response to stress [60]. Given that the HPA axis is a primary mediator of AL, the effects of PA on HPA activity may reduce or delay the cumulative effects of repeated cycles of allostasis [14].

Regular PA has also been shown to improve immune system function and reduce inflammation [61, 62]. Systemic inflammation has been identified as a potential link between psychological stress and the risk of chronic disease, and a number of studies have identified associations between experiences of discrimination and markers of inflammation [63, 64]. Gay et al. found that meeting PA guidelines was associated with both lower AL and lower inflammation in Mexican American men and women [26]. Reduced inflammation also positively impacts cardiovascular and metabolic health and, therefore, may have both a direct and indirect impact on AL score [65]. Thus, the key finding of this study-that regular PA attenuates the association between experiences of racial discrimination and AL-is biologically plausible. Additional research is needed to repeat these novel findings across samples and populations, preferably using longitudinal study designs so that the temporal sequence can be examined with certainty.

Strengths and Limitations

Strengths of this study include guidance by an Indigenous Advisory Committee, the use of a validated measure of racial discrimination and PA, and the use of a cumulative AL approach to examine the impact of discrimination on the body, after controlling for age, sex, and income. Limitations include the use of a cross-sectional design that precludes inferences about causation and the temporal sequence of racial discrimination and AL, more female than male participants, and a relatively small sample of university students, which may not be generalizable to the general population. Approximately 30% of the sample had missing data for CAR as this measure required at-home data collection and sample return. Response bias due to self-report measures is a concern for both racial discrimination and PA. Research suggests racial discrimination, given that it is often implicit in nature and pervasive in society, may not always be perceived and reported [45]. Thus, self-reported discrimination may result in underreporting, revealing only a small portion of the actual effect of racial discrimination on the individual [66]. For salivary biomarkers, we needed to accommodate student course schedules and, thus, could not standardize the time of the day for data collection across participants, which may have been useful for some biomarkers (e.g., DHEA-S, CRP). We also note that the analysis of salivary biomarkers did not adjust for flow rate, which is a limitation for the measurement of DHEA-S. Finally, a sample size calculation could not be estimated given that the associations examined were novel for the population under study, and there was a dearth of studies within other populations that could be used to estimate sample size when this study began in 2015.

Conclusions

The findings of this study combine with others to highlight the negative consequences of racial discrimination and underline the importance of increased action to address this pervasive adverse determinant of health. As this work unfolds, research can identify tools that racialized groups may use to buffer the impacts of racism on their personal health. The findings of this study suggest that regular PA could help offset the physiologic impacts of racial discrimination on their bodies. This study also underlines the importance of ensuring that all people have access to opportunities for PA, especially those who may face a disproportionate burden of stress in their daily lives.

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Compliance with Ethical Standards

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards All authors report no conflicts of interest.

Authors' Contributions J.L.C. and C.L.C. performed the analysis and interpretation of the data, and drafted the manuscript. All authors contributed to conceptualizing and designing the study and collecting the data. All authors read and approved the final manuscript.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This study was reviewed and approved by the Human Participant Research Committee in the Office of Research and Innovation Services at the University of Lethbridge (Protocol #2014–046).

Informed Consent All participants gave written informed consent.

References

- Currie C, Wild TC, Schopflocher D, Laing L. Racial discrimination, post-traumatic stress and prescription drug problems among Aboriginal Canadians. *Can J Public Health*. 2015;106:e382–e387.
- 2. Shepherd, CCJ, Li J, Cooper MN, Hopkins KD, Farrant BM. The impact of racial discrimination on the health of Australian

Indigenous children aged 5–10 years: Analysis of national longitudinal data. *Int J Equity Health*. 2017;16:116.

- 3. Paradies Y, Ben J, Denson N, et al. Racism as a determinant of health: A systematic review and meta-analysis. *PLoS One*. 2015;10:e0138511.
- Ong AD, Williams DR, Nwizu U, Gruenewald TL. Everyday unfair treatment and multisystem biological dysregulation in African American adults. *Cultur Divers Ethnic Minor Psychol.* 2017;23:27–35.
- Lewis TT, Cogburn CD, Williams DR. Self-reported experiences of discrimination and health: Scientific advances, ongoing controversies, and emerging issues. *Annu Rev Clin Psychol.* 2015;11:407–440.
- Brody GH, Yu T, Miller GE, Chen E. Discrimination, racial identity, and cytokine levels among African-American adolescents. *J Adolesc Health.* 2015;56:496–501.
- Allen AM, Thomas MD, Michaels EK, et al. Racial discrimination, educational attainment, and biological dysregulation among midlife African American women. *Psychoneuroendocrinology*. 2019:99:225–235.
- Chae DH, Nuru-Jeter AM, Adler NE, et al. Discrimination, racial bias, and telomere length in African-American men. *Am J Prev Med.* 2014;46:103–111.
- Siddiqi A, Shahidi FV, Ramraj C, Williams DR. Associations between race, discrimination and risk for chronic disease in a population-based sample from Canada. *Soc Sci Med.* 2017;194:135–141.
- Browne AJ. Moving beyond description: Closing the health equity gap by redressing racism impacting Indigenous populations. Soc Sci Med. 2017;184:23–26.
- Marshall M, Marshall A, Bartlett C. Two-eyed seeing in medicine. In: Greenwood M, De Leeuw S, Lindsay NM, eds. *Determinants of Indigenous Peoples' Health: Beyond the Social*. 2nd ed. Toronto, ON: CSP Books Inc.; 2018.
- Currie CL, Copeland JL, Metz GA, Chief Moon-Riley K, Davies CM. Past-year racial discrimination and allostatic load among indigenous adults in Canada: The role of cultural continuity. *Psychosom Med.* 2020;82:99–107.
- Currie CL, Copeland JL, Metz GA. Childhood racial discrimination and adult allostatic load: The role of Indigenous cultural continuity in allostatic resiliency. *Soc Sci Med.* 2019;241:112564.
- McEwen BS. Stress, adaptation, and disease. Allostasis and allostatic load. Ann N Y Acad Sci. 1998;840:33–44.
- 15. Sterling P, Eyer J. Allostasis: A new paradigm to explain arousal pathology. In: Fisher, S., Reason J, ed. *Handbook of Life Stress, Cognition and Health.* New York: John Wiley & Sons; 1988:629–649.
- 16. McEwen BS, Stellar E. Stress and the individual. Mechanisms leading to disease. *Arch Intern Med.* 1993;153:2093–2101.
- Seeman TE, Singer BH, Rowe JW, Horwitz RI, McEwen BS. Price of adaptation–allostatic load and its health consequences. MacArthur studies of successful aging. *Arch Intern Med.* 1997;157(19):2259–2268.
- Castagné R, Garès V, Karimi M, et al.; Lifepath Consortium. Allostatic load and subsequent all-cause mortality: Which biological markers drive the relationship? Findings from a UK birth cohort. *Eur J Epidemiol.* 2018;33:441–458.
- Borrell LN, Dallo FJ, Nguyen N. Racial/ethnic disparities in all-cause mortality in U.S. adults: The effect of allostatic load. *Public Health Rep.* 2010;125:810–816.
- Robertson T, Beveridge G, Bromley C. Allostatic load as a predictor of all-cause and cause-specific mortality in the general population: Evidence from the Scottish Health Survey. *PLoS One.* 2017;12:e0183297.

- Seeman TE, Crimmins E, Huang MH, et al. Cumulative biological risk and socio-economic differences in mortality: MacArthur studies of successful aging. *Soc Sci Med.* 2004;58:1985–1997.
- Brody GH, Lei MK, Chae DH, Yu T, Kogan SM, Beach SRH. Perceived discrimination among African American adolescents and allostatic load: A longitudinal analysis with buffering effects. *Child Dev.* 2014;85:989–1002.
- Fletcher D, Sarkar M. Psychological resilience. *Eur Psychol.* 2013;18(1):12–23.
- Johnson J, Wood AM, Gooding P, Taylor PJ, Tarrier N. Resilience to suicidality: The buffering hypothesis. *Clin Psychol Rev.* 2011;31:563–591.
- 25. Upchurch DM, Rainisch BW, Chyu L. Greater leisure time physical activity is associated with lower allostatic load in white, black, and Mexican American Midlife Women: Findings from the national health and nutrition examination survey, 1999 through 2004. Womens Health Issues. 2015;25:680–687.
- Gay JL, Salinas JJ, Buchner DM, et al. Meeting physical activity guidelines is associated with lower allostatic load and inflammation in Mexican Americans. *J Immigr Minor Health*. 2015;17:574–581.
- 27. Silverman MN, Deuster PA. Biological mechanisms underlying the role of physical fitness in health and resilience. *Interface Focus.* 2014;4:20140040.
- Puterman E, O'Donovan A, Adler NE, et al. Physical activity moderates effects of stressor-induced rumination on cortisol reactivity. *Psychosom Med.* 2011;73:604–611.
- McTaggert R. Principles for participatory action research. *Adult Educ Q.* 1991;41(3):168–187.
- 30. Government of Canada. *Education in Canada: Key Results* From the 2016 Census. 2017.
- Statistics Canada. Aboriginal peoples in Canada: Key results from the 2016 Census. Ottawa, ON: Statistics Canada, Catalogue no. 11-001-X; 2017.
- Anderson I, Robson B, Connolly M, et al. Indigenous and tribal peoples' health (The Lancet-Lowitja Institute Global Collaboration): A population study. *Lancet.* 2016;388: 131–157.
- Stalder T, Kirschbaum C, Kudielka BM, et al. Assessment of the cortisol awakening response: Expert consensus guidelines. *Psychoneuroendocrinology*. 2016;63:414–432.
- Johnson SC, Cavallaro FL, Leon DA. A systematic review of allostatic load in relation to socioeconomic position: Poor fidelity and major inconsistencies in biomarkers employed. *Soc Sci Med.* 2017;192:66–73.
- 35. Segerstrom SC, Miller GE. Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychol Bull.* 2004;130:601–630.
- Maninger N, Wolkowitz OM, Reus VI, Epel ES, Mellon SH. Neurobiological and neuropsychiatric effects of dehydroepiandrosterone (DHEA) and DHEA sulfate (DHEAS). *Front Neuroendocrinol.* 2009;30:65–91.
- Petros N, Opacka-Juffry J, Huber JH. Psychometric and neurobiological assessment of resilience in a non-clinical sample of adults. *Psychoneuroendocrinology*. 2013;38(10):2099–2108.
- Berger M, Leicht A, Slatcher A, et al. Cortisol awakening response and acute stress reactivity in first nations people. *Sci Rep.* 2017;7:41760.
- Adam EK, Kumari M. Assessing salivary cortisol in large-scale, epidemiological research. *Psychoneuroendocrinology*. 2009;34:1423–1436.
- Clow A, Thorn L, Evans P, Hucklebridge F. The awakening cortisol response: Methodological issues and significance. *Stress.* 2004;7:29–37.

- Labat C, Temmar M, Nagy E, et al. Inflammatory mediators in saliva associated with arterial stiffness and subclinical atherosclerosis. J Hypertens. 2013;31:2251–2258; discussion 2258.
- 42. Juster RP, Moskowitz DS, Lavoie J, D'Antono B. Sex-specific interaction effects of age, occupational status, and workplace stress on psychiatric symptoms and allostatic load among healthy Montreal workers. *Stress.* 2013;16:616–629.
- 43. Gustafsson PE, San Sebastian M, Janlert U, Theorell T, Westerlund H, Hammarström A. Life-course accumulation of neighborhood disadvantage and allostatic load: Empirical integration of three social determinants of health frameworks. *Am J Public Health.* 2014;104:904–910.
- 44. Beckie TM. A systematic review of allostatic load, health, and health disparities. *Biol Res Nurs.* 2012;14:311–346.
- 45. Krieger N, Smith K, Naishadham D, Hartman C, Barbeau EM. Experiences of discrimination: Validity and reliability of a self-report measure for population health research on racism and health. Soc Sci Med. 2005;61:1576–1596.
- Currie CL, Wild TC, Schopflocher DP, Laing L, Veugelers P. Racial discrimination experienced by aboriginal university students in Canada. *Can J Psychiatry*. 2012;57:617–625.
- 47. Amireault S, Godin G. The Godin-Shephard leisure-time physical activity questionnaire: Validity evidence supporting its use for classifying healthy adults into active and insufficiently active categories. *Percept Mot Skills*. 2015;120:604–622.
- Godin, R.; Shephard RJ. Godin leisure-time exercise questionnaire. *Med Sci Sports Exerc.* 1997;29:S36–8.
- Godin G, Shephard RJ. A simple method to assess exercise behavior in the community. *Can J Appl Sport Sci.* 1985;10:141–146.
- Sallis JF, Saelens BE. Assessment of physical activity by selfreport: Status, limitations, and future directions. *Res Q Exerc Sport.* 2000;71(suppl 2):1–14.
- Currie CL, Wild TC, Schopflocher DP, Laing L, Veugelers P. Illicit and prescription drug problems among urban Aboriginal adults in Canada: The role of traditional culture in protection and resilience. *Soc Sci Med.* 2013;88:1–9.
- Currie CL, Wild TC, Schopflocher DP, et al. Enculturation and alcohol use problems among aboriginal university students. *Can J Psychiatry*. 2011;56:735–742.
- Smith, G, Currie, CL, Battle J. Exploring gambling impacts in two Alberta Cree communities: A participatory action study. In: Belanger Y, ed. *First Nations Gaming in Canada*. Winnipeg, MB: University of Manitoba Press; 2011;118–139.

- 54. Rubin D. *Multiple Imputation for Nonresponse in Surveys.* Hoboken, NJ: Wiley Interscience; 2004.
- 55. Ferrao V. Women in Canada. A Gender Based Statistical Report. 89-503-X. Ottawa, ON: Statistics Canada; 2010.
- Myers J, Kokkinos P, Nyelin E. Physical activity, cardiorespiratory fitness, and the metabolic syndrome. *Nutrients*. 2019;11:1652.
- Zhang D, Liu X, Liu Y, et al. Leisure-time physical activity and incident metabolic syndrome: A systematic review and dose-response meta-analysis of cohort studies. *Metabolism.* 2017;75:36–44.
- Gerber M, Pühse U. Review article: Do exercise and fitness protect against stress-induced health complaints? A review of the literature. *Scand J Public Health.* 2009;37: 801–819.
- 59. Gerber M, Ludyga S, Mücke M, Colledge F, Brand S, Pühse U. Low vigorous physical activity is associated with increased adrenocortical reactivity to psychosocial stress in students with high stress perceptions. *Psychoneuroendocrinology*. 2017;80:104–113.
- 60. Chen C, Nakagawa S, An Y, Ito K, Kitaichi Y, Kusumi I. The exercise-glucocorticoid paradox: How exercise is beneficial to cognition, mood, and the brain while increasing glucocorticoid levels. *Front Neuroendocrinol.* 2017;44:83–102.
- Nieman DC, Wentz LM. The compelling link between physical activity and the body's defense system. J Sport Health Sci. 2019;8:201–217.
- 62. Truba TN, Doan J, Currie CL, Copeland JL. Short-term changes in daily movement behaviour influence salivary C-reactive protein in healthy women. *Appl Physiol Nutr Metab.* 2018;43:854–856.
- Panza GA, Puhl RM, Taylor BA, Zaleski AL, Livingston J, Pescatello LS. Links between discrimination and cardiovascular health among socially stigmatized groups: A systematic review. *PLoS One.* 2019;14:e0217623.
- Cohen S, Janicki-Deverts D, Miller GE. Psychological stress and disease. JAMA. 2007;298:1685–1687.
- Pinckard K, Baskin KK, Stanford KI. Effects of exercise to improve cardiovascular health. *Front Cardiovasc Med.* 2019;6:69.
- Berger M, Sarnyai Z. "More than skin deep": Stress neurobiology and mental health consequences of racial discrimination. *Stress.* 2015;18:1–10.