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# Affect regulation and allostatic load over time<sup> $\star$ </sup>

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#### ARTICLE INFO ABSTRACT Keywords: Objective: Emerging work suggests that affect regulation strategies (e.g., active coping, anger expression) predict Affect regulation disease and mortality risk, with sometimes divergent estimates by sex or education levels. However, few studies Allostatic load have examined potential underlying biological mechanisms. This study assessed the longitudinal association of Coping affect regulation with future allostatic load. Coping variability Method: In 2004–2006, 574 participants from the Midlife in the United States study completed validated scales Anger assessing use of nine general and emotion-specific regulatory strategies (e.g., denial, anger expression). As a Emotion regulation proxy for how flexibly participants regulate their affect, variability in the use of regulatory strategies was operationalized using a standard deviation-based algorithm and considered categorically (i.e., lower, moderate, greater variability) to assess non-linear effects. Participants also provided data on relevant covariates and 24 allostatic load biomarkers (e.g., cortisol, blood pressure). In 2017-2021, these biomarkers were again collected. Linear regressions modeled betas ( $\beta$ ) and 95 % confidence intervals (CI) examining associations of affect regulatory constructs with future allostatic load. Results: In fully-adjusted models including initial allostatic load, general regulatory strategies were unrelated to future allostatic load. Yet, greater versus moderate affect regulation variability levels predicted lower allostatic load ( $\beta$ =-0.14; 95 %CI: -0.27, -0.01). Only among more educated participants, greater use of anger expression predicted lower allostatic load, while the reverse was noted with anger control ( $\beta_{expression} = -0.12$ ; 95 %CI: $-0.20, -0.05; \beta_{control}=0.14; 95$ %CI: 0.05, 0.24). Conclusions: While general regulatory strategies appeared unrelated to allostatic load, greater variability in their use and anger-related strategies showed predictive value. Subsequent studies should examine these associations in larger, more diverse samples.

## 1. Introduction

In the face of perceived stress, adaptive fluctuations in our biological systems (e.g., increase in cortisol or blood pressure) facilitate the

stability of organism function, a phenomenon called allostasis. However, prolonged stress may lead to chronic shifts in biological system function, resulting in elevations or deficits of system biomarkers, or altered patterns of function, thus leading to allostatic load (McEwen, 2002).

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Allostatic load can be indexed using biomarkers representing the autonomic, neuroendocrine, metabolic, and immune systems (Mauss and Jarczok, 2021; Wiley et al., 2016, 2017). Over time, higher allostatic load predicts detrimental health outcomes, including an increased risk of all-cause and cardiovascular disease (CVD)-related mortality, (Parker et al., 2022) and CVD incidence (Gillespie et al., 2019), suggesting this physiological wear and tear is especially detrimental for long-term health.

Prior research has sought to identify modifiable factors, including psychological markers, that may alter allostatic load levels. Previous longitudinal work has highlighted relationships between greater psychological distress (e.g., depressive symptoms (McClain et al., 2022), perceived stress (Upchurch et al., 2015)) and higher allostatic load, as well as between greater psychological well-being (e.g., purpose in life (Lewis and Hill, 2023), emotional vitality (Deen et al., 2020)) and lower allostatic load. Relatedly, theoretical models in health psychology and social epidemiology have theorized that upstream psychological regulatory processes, including stress-related coping and emotion regulation, may contribute to disease outcomes, partly via biological processes (Trudel-Fitzgerald et al., 2017, 2024a; Epel et al., 2018). To our knowledge, however, no longitudinal study has explored the association of such regulatory processes with allostatic load.

# 2. Stress-related coping, emotion regulation, and physical health

Coping has been defined as "cognitive and behavioral efforts to master, reduce, or tolerate the internal and/or external demands" (Folkman, 1984). These strategies are frequently conceptualized as adaptive (e.g., actively coping with stressors) or maladaptive (e.g., denying the presence of stressors), given the direction of their associations with mental and physical health outcomes (Hoyt et al., 2024; Kato, 2015). Accordingly, previous longitudinal studies have shown that greater use of adaptive coping strategies was associated respectively with lower CVD and mortality risk, whereas the reverse was noted with greater use of maladaptive coping strategies, beyond statistical control for sociodemographic, behavioral, and health covariates (Trudel-Fitzgerald et al., 2022; Ng et al., 2024; Roohafza et al., 2021; Svensson et al., 2016). Some data further suggest sociodemographic factors may modify these associations, whereby coping exposures predicted CVD outcomes among women and more educated individuals only (Ng et al., 2024).

In parallel to coping, emotion regulation describes the way individuals experience and express their emotions (Gross, 1998). These strategies are also typically divided into adaptive and maladaptive coping strategies. Most longitudinal health research has focused on the regulation of anger specifically. Evidence suggests that greater anger aggressive/disruptive expression, typically deemed a maladaptive strategy, was related to higher CVD risk (Davidson and Mostofsky, 2010) and cancer mortality (Trudel-Fitzgerald et al., 2021), beyond relevant covariates. By contrast, a more adaptive form of anger regulation, namely constructive anger expression, predicted lower CVD risk but only among men (Davidson and Mostofsky, 2010). Given these linkages of coping and emotion regulation, separately, with long-term health outcomes, unresolved questions include the comparability of coping and emotion regulation as health predictors, and the identification of underlying biological pathways.

#### 2.1. Affect regulation and its association with physical health

Research on coping and emotion regulation have often been considered in parallel, rather than jointly, because of key conceptual differences (e.g., focus on responses to stressors *vs.* emotions) (Trudel-Fitzgerald et al., 2023; Troy et al., 2023). However, because they share more similarities than initially acknowledged, scholars have recently encouraged their joint consideration under the broader affect

regulation framework (Troy et al., 2023). Besides the adaptive vs. maladaptive nature of coping and emotion regulation strategies, researchers from both fields also theorize that the connotation of any regulatory strategy may be judged as adaptive or maladaptive depending on the context (Cheng et al., 2014; Bonanno and Burton, 2013). Thus, optimal use of regulatory strategies may be instead represented by flexibility, or an individual's ability to select the appropriate regulatory strategy for the present situation. Previous studies have conceptualized regulatory flexibility as the amount of the numerical variability observed in the frequency use of distinct strategies (Cheng et al., 2014; Blanke et al., 2020). For instance, using a standard deviation-based algorithm (Trudel-Fitzgerald et al., 2022, 2024b; Blanke et al., 2020), lower variability (displaying high evenness in scores across strategies) suggests all strategies are used at a fairly equal frequency across situations, greater variability (displaying high unevenness in scores across strategies) suggests only a few strategies are frequently used, and moderate variability (displaying moderate unevenness in scores across strategies) suggests several strategies are used with varied frequencies and possibly reflects an effort to find the best strategy for each situation. Emerging research shows the predictive value of such variability in long-term health outcomes, such as longevity (Trudel-Fitzgerald et al., 2022, 2024b).

Despite evidence linking psychological processes to health outcomes, and allostatic load to health outcomes, few studies have examined the association between psychological regulatory processes and allostatic load specifically. For example, in the Jackson Heart Study, women who used more disengagement strategies like avoidance had higher allostatic load beyond sociodemographic and behavioral covariates; associations for men were null (Fernandez et al., 2015). In the Midlife in the United States Study (MIDUS), greater use of anger control, but not anger expression, was associated with lower allostatic load, above sociodemographic and chronic disease characteristics (Zilioli et al., 2017). While informative, these studies were cross-sectional, which limits interpretation about directionality of effects. Moreover, MIDUS and other studies adopted either the coping or the emotion regulation framework. As advised in the recent affect regulation framework (Troy et al., 2023), consideration of regulatory strategies from both frameworks allows a more comprehensive assessment of how individuals cope with stressors and regulate their emotions; it also permits the comparison of their predictive value in relation to allostatic load. Lastly, to our knowledge, none of these studies considered variability, which likely represents a more nuanced view of how individuals regulate their affect relative to the (mal)adaptive dichotomization predominantly used in previous research.

#### 2.2. The present study

We examined associations of affect regulation indicators (i.e., general and emotion-specific strategies, and variability in their use) and allostatic load using 17 years of follow-up data available from the MIDUS. Based on prior work examining these strategies with health outcomes (Trudel-Fitzgerald et al., 2022, 2024b; Ng et al., 2024), we hypothesized that strategies typically deemed adaptive relate to lower allostatic load, while strategies usually seen as maladaptive relate to higher allostatic load. Given limited work on regulatory variability and health (Trudel-Fitzgerald et al., 2022; Ng et al., 2024; Cheng et al., 2014), we examined this association without a priori hypotheses. Because previous research also suggests that psychological regulatory processes and allostatic load may vary by sex (Cheng et al., 2014; Juster et al., 2019), age (Piazza et al., 2019), socioeconomic status (Upchurch et al., 2015), and psychosocial stress levels (McEwen, 2002; Upchurch et al., 2015), we also considered these sociodemographic and psychosocial indicators as potential effect modifiers of study associations.

#### 2.3. Method

#### 2.3.1. Study Sample

The MIDUS is a national cohort study of noninstitutionalized English-speaking adults between the ages of 25–74 at baseline, recruited through random-digit-dialing. Once selected, participants were interviewed at three separate time points: MIDUSI (N=7108; 1995–1996), MIDUSII (N=4963; 2004–2006; 70 % response rate from MIDUSI), and MIDUSIII (N=3294; 2013–2015; 66 % response rate from MIDUSI). For each time assessment, data was collected via a phone interview and self-administered questionnaire (SAQ). The SAQs were mailed to the participant's residence. Comprehensive biological assessments, medical histories, and an additional SAQ were collected as part of MIDUSII (2004–2009) and MIDUSIII (2017–2021). The current study sample includes all respondents with data on all affect regulation variables at MIDUSII (2004–2006) and allostatic load at MIDUSII (2004–2009) as well as MIDUSIII (2017–2021), with all covariates from MIDUSII imputed using multiple imputation (N=574; Supplemental Figure 1).

## 2.4. Measures

#### 2.4.1. Affect Regulation

As mentioned previously, scholars have recently encouraged the joint consideration of coping and emotion regulation constructs under the broader affect regulation framework, because they are two key mechanisms that influence affect regulation and, in turn, promote psychological resilience (Troy et al., 2023). Despite their distinct targets, whereby coping focuses on stressors whereas emotion regulation focuses on emotion, recent evidence further demonstrates a substantial conceptual and measurement overlap between coping and emotion regulation strategies (Trudel-Fitzgerald et al., 2023). Consequently, it has been proposed to consider them jointly in future research to widen the repertoire of available measures and compare their correlates (Trudel-Fitzgerald et al., 2023). How one generally copes with stressful events and regulates emotions, as a disposition, was assessed through two validated self-reported measures at MIDUSII.

To measure general regulatory strategies, participants completed a modified version of the 60-item Coping Orientation to Problems Experienced (COPE) Inventory (Carver et al., 1989), which describes how someone typically manages stressful events. This version includes 24 items categorized into 6 subscales that represent distinct strategies. Positive Reinterpretation & Growth (e.g. "I look for something good in what is happening"), Active Coping (e.g. "I take direct action to get around the problem"), and Planning (e.g. "I try to come up with a strategy about what to do") are typically deemed as adaptive, while Focus on & Venting of Emotions (e.g. "I feel a lot of emotional distress and find myself expressing those feelings a lot"), Denial (e.g. "I pretend that it hasn't really happened", and Behavioral Disengagement (e.g. "I give up trying to reach my goal") are usually judged as maladaptive given the direction of their association with psychological distress and well-being in prior work (Cheng et al., 2014; Penley et al., 2002).

Across subscales, item scores were rated on a scale from (1) *A lot* to (4) *Not at all*, which were summed and reverse-coded to create a total score for each subscale ranging from 4 to 16, with higher scores indicating more frequent use of the strategy. These strategies had acceptable-to-high internal consistency at the current study baseline (MIDUSII; Cronbach alpha,  $\alpha$ =0.72–0.83; Supplemental Table 1). Of note, scores were very stable over 10 years, when compared with those obtained at MIDUSIII (most Pearson correlations, *r*=0.51–0.67; Supplemental Table 1), reinforcing the dispositional nature of this measure. Following MIDUS recommendations, subscale scores were computed for cases with valid data on at least half of the items. For items with remaining missing values, the mean value of completed items was then used. Scores from each strategy were standardized using z-scores to facilitate comparison with results obtained from other affect regulation exposures in the current study and those observed in previous studies.

We implemented the following standard deviation-based algorithm based on prior research (Trudel-Fitzgerald et al., 2022, 2024b; Ng et al., 2024; Blanke et al., 2020) to create a dispositional Between-Strategy Index, or the affect regulation variability score, using information from the COPE subscales:

$$SD_{(\text{between})i} = \sqrt{\frac{1}{L-1} \times \sum_{s=1}^{L} (\mathbf{x}_{si} - \mathbf{M}_{(\text{between})i})^2}$$

where  $x_{si}$  corresponds to the value of strategy *s* of individual *i* for the total number of strategies *L*.

As discussed, affect regulation variability describes the extent to which participants may use different regulatory strategies across different situations. Thus, participants with lower variability are more likely to use their set of strategies at an equal frequency across situations (displaying high evenness in scores across strategies) while those with greater variability are more likely to use some strategies and rarely use others (displaying high unevenness in scores across strategies). Participants with moderate variability are likely to engage in several strategies with varied frequencies (displaying moderate unevenness in scores across strategies), possibly reflecting an effort to find the best strategy for each situation. An example with fictitious data that illustrates the computation and the interpretation of the Between-Strategy Index score is provided in Table 1. The affect regulation variability score was divided into tertiles (lower, moderate, and greater) (Cheng et al., 2014; Blanke et al., 2020), to examine possible non-linear associations with allostatic load, as done previously in MIDUS and other studies (Trudel-Fitzgerald et al., 2022, 2024b; Ng et al., 2024). Mean strategy use score was also adjusted for in all models with affect regulation variability to reflect that participants with consistently low or high mean strategy scores are unable to show high levels of variability due to floor or ceiling effects (Trudel-Fitzgerald et al., 2022, 2024b; Ng et al., 2024).

Emotion-specific regulatory strategies were measured with the validated Spielberger Anger Expression Inventory (Spielberger, 1988), which included 8 items that capture subscales on anger expression and suppression, respectively, and 4 items on anger control. Participants responded to items (e.g., Anger expression: "In general, when I feel angry or furious I lose my temper"; Anger suppression: "I keep things in"; Anger Control: "I control my temper") on a scale from (1) Almost never to (4) Almost always. These strategies had moderate-to-high internal consistency at the current study baseline (MIDUSII;  $\alpha$ =0.53–0.80; Supplemental Table 2). Scores were also fairly stable over 10 years, when compared with those obtained at MIDUSIII (r=0.45-0.58; Supplemental Table 2), supporting the dispositional nature of these strategies. Following MIDUS guidelines (University of Wisconsin Madison, 2020), a composite score for each subscale was computed by summing across all items for which there were no, or only one missing value. Mean substitution was used in cases with only one missing value. Higher scores indicated greater frequency of use. Here again, scores from each anger subscale were standardized using z-scores to facilitate comparison with results obtained from other affect regulation exposures in the current study and those observed in previous studies.

#### 2.4.2. Allostatic Load

Allostatic load was computed using 24 biomarkers representing 7 physiological domains collected at MIDUSII and MIDUSIII, respectively: *Sympathetic Nervous System* (epinephrine, norepinephrine), *Parasympathetic Nervous System* (low frequency spectral power, high frequency spectral power, SDRR, RMSSD), *Hypothalamic Pituitary Adrenal Axis* (DHEAS, Cortisol), *Inflammation* (CRP, IL-6, fibrinogen, sE-Selectin, sICAM-1), *Cardiovascular* (resting SBP, resting DBP, resting heart rate), *Glucose Metabolism* (HbA1c, fasting glucose, insulin resistance), and *Lipid Metabolism* (BMI, WHR, triglycerides, HDL cholesterol, LDL cholesterol). For each biomarker, participants were separated into high/low categories using sex-specific quartile cut-offs (Juster et al., 2016). As done previously (Podber and Gruenewald, 2023), the proportion of biomarkers that were categorized as high was calculated for each domain for participants that had valid data on at least half of the

Table 1

Example of the dispositional	Between-Strategy Index to	capture an individual's	general level of affect reg	gulation variability	with fictitious data.

Participant number	Strategy 1	Strategy 2	Strategy 3	Strategy 4	Strategy 5	Strategy 6	6 Dispositional Between-Strategy Index	
1	4	4	4	3	3	3	0.55	
2	1	1	1	2	2	2	0.55	
3	6	1	3	1	1	1	2.04	
4	0	0	3	3	3	0	1.64	
5	6	6	6	0	0	0	3.29	
6	0	0	6	6	0	0	3.10	

*Notes.* The Dispositional Between-Strategy Index and this example table are adapted from Blanke and colleagues' study on the Between-Strategy Index (Blanke et al., 2020) and is based on a prior similar investigation led in MIDUS examining the association of regulatory variability with lifespan (Trudel-Fitzgerald et al., 2022). Data are from six fictitious participants and their rating of the frequency with which they used six regulatory strategies on a scale from 0 (not at all) to 6 (all the time). Individuals displaying lower variability scores (e.g., participants 1 and 2) would generally use all strategies to a similar extent (displaying high evenness in their scores across strategies) across circumstances, whereas those with greater variability scores (e.g., participants 5 and 6) would be more likely to select only a few strategies from their repertory and rely heavily on them without using other strategies (displaying high unevenness in their scores). By contrast, individuals exhibiting moderate variability scores (e.g., participants 3 and 4) might use several strategies but each to a different extent or use a few strategies to a modest extent, possibly reflecting an attempt to find the best strategy in a given context (displaying moderate unevenness in their scores) (Trudel-Fitzgerald et al., 2022).

biomarkers in the domain; the proportion of the 7 domain scores were subsequently summed to create a continuous total allostatic load score ranging from 0 to 7 for participants with a score on at least 6 of the 7 domains.

#### 2.4.3. Covariates

Information on various covariates was collected at baseline (MID-USII). Factors that could potentially confound the affect regulationallostatic load associations included age (continuous), biological sex (male, female), racial minority status (white, non-white [Black and/or African American, Native American or Aleutian Islander, Asian or Pacific Islander, Other, Multiracial]), marital status (married/living with partner, separated/widowed/divorced, never married), income (\$0–24,999, \$25,000–44,999, \$45,000–74,999, \$75,000–200,000), highest level of education (less than high school/general educational development (GED), high school diploma/GED, some college/Bachelor's degree or higher), prevalent/history of heart disease (yes, no), and prevalent/history of cancer (yes, no).

Health behaviors, which could either confound or lie on the pathway relating affect regulation to allostatic load, encompassed physical activity, smoking status, and alcohol consumption. Physical activity was measured with many items capturing moderate-to-vigorous physical activity separately by season and situation (employment, leisure, and chores). Participants were categorized as active if they indicated they were moderately or vigorously active at least once per week in both summer and winter, from any situation. Smoking status was ascertained via two items that asked the participant whether they (1) had ever smoked or (2) currently smoke. If a participant responded "no" to the first item, they were classified as a "never smoker", if they responded "yes" to the first and "no" to the second were classified as a "former smoker", and if they responded "yes" to both items were classified as "current smokers". Alcohol consumption was assessed with one item that asked participants how many drinks they would typically drink per day during the time they drank the most. Following public health guidelines on chronic disease prevention and other empirical evidence (Dietary Guidelines for Americans, 2020; Koga et al., 2022), females who reported more than zero but less than or equal to one drink per day, and males who reported more than zero but less than or equal to two drinks per day were classified as moderate drinkers, which represents a more favorable level of alcohol consumption. Females who reported more than two drinks and males who reported more than three drinks per day were classified as heavy drinkers, and were combined with participants who reported not drinking into a second category representing less favorable alcohol consumption level (Dietary Guidelines for Americans, 2020; Koga et al., 2022).

Moreover, to account for baseline MIDUSII levels of allostatic load, a sex-specific allostatic load score, constructed in parallel to the outcome

at MIDUSIII detailed above, was considered as a covariate (continuous). Lastly, a summary stress score (continuous), also available at MIDUSII, was considered as a potential effect modifier in secondary analyses, aside from age (median-split), sex (male vs. female), and education (high school diploma/GED and less vs. some college or more). Following previous research (Slopen et al., 2018), this comprehensive MIDUS summary stress score aggregates ten stress domains across the life course, including psychological work stress, physical work stress, work-family spillover, perceived inequality (in family, home and work opportunities), relationship stress, neighborhood stress, discrimination, current financial stress, past year problems in immediate family, stressful life experiences, and early life stress. Standardized scores from each domain are combined to create the summary score, which is then standardized using z-scores for analysis.

#### 2.5. Statistical analyses

All analyses were conducted with SAS, version 9.4. Descriptive statistics of the study sample were calculated, with percentages presented for categorical variables, and means and standard deviations (SD) presented for continuous variables.

#### 2.5.1. Primary models

Linear regression models were constructed, examining associations between each continuous standardized affect regulation variable and continuous allostatic load, with baseline covariates added sequentially. More specifically, Model 1 was adjusted for age only, and Model 2 additionally controlled for other sociodemographic characteristics (sex, racial minority status, marital status, education). Model 3, our core model, further included health conditions (heart disease, cancer). Because health behaviors could be either confounders, intermediate pathways, or affect regulation strategies themselves (i.e., someone can drink wine to manage anger) (Trudel-Fitzgerald et al., 2024a; Park and Iacocca, 2014), physical activity, smoking, and alcohol consumption were then included in an exploratory Model 4. Model 5 additionally adjusted for allostatic load at baseline. These same nested models were repeated using categorical affect regulation variability levels as the exposure, evaluating all possible contrasts (i.e., moderate vs. lower, greater vs. lower, greater vs. moderate).

#### 2.5.2. Secondary models

To assess effect modification by age, sex, education levels, and psychosocial stress levels, interaction terms between each affect regulation variable and potential effect modifier (e.g., continuous denial\*categorical sex) were included to core Model 3 described above. For interaction terms that were statistically significant at the p<0.05 level, stratified models were then constructed.

## 3. Results

#### 3.1. Descriptive statistics

Table 2 presents descriptive characteristics of the study sample. On average, participants were middle-aged (M=52.23 years; SD=9.93), with a similar representation of males and females. Most were White (95.50 %) and married/living with a partner (75.26 %), while approximately half attended at least some college (50.96 %) and had incomes greater than \$45,000 (40.71 %). The vast majority did not have a history/prevalent heart disease (93.06 %) or cancer (90.72 %), and about half to two-thirds of the sample were physically active (54.01 %), never smokers (43.81 %), and moderate alcohol drinkers (67.60 %).

#### 3.2. Primary analyses

Table 3 first presents results from linear regression models examining associations between general regulatory strategies and allostatic load. In age-adjusted models (Model 1), there were no clear associations between any of these strategies and allostatic load. However, estimates trended in the expected directions, with adaptive strategies related to lower allostatic load, and maladaptive strategies related to higher allostatic load. Associations between these general regulatory strategies and allostatic load remained non-significant following covariate adjustment.

Associations between affect regulation variability and allostatic load are also reported in Table 3. In an age-adjusted model, greater variability was related to lower allostatic load (Model 1,  $\beta$ = -0.17; 95 %CI: -0.32, -0.02) compared to moderate variability levels. Further adjustment for sociodemographic, health, and behavioral covariates did not alter this association (e.g., Model 4,  $\beta$ = -0.17; 95 %CI: -0.32, -0.02). Additional adjustment for baseline allostatic load only slightly attenuated the estimate (Model 5,  $\beta$ = -0.14; 95 %CI: -0.27, -0.01). Greater or moderate variability compared to the lower variability levels were also related to lower allostatic load score across models, but estimates did not reach statistical significance.

Lastly, Table 3 shows results of the associations between emotionspecific regulatory strategies and allostatic load. Estimates from ageadjusted models were all not statistically significant, although they hint to lower allostatic load with greater use of anger expression and to higher allostatic load with greater use of anger control and suppression, respectively. Estimates remained non-significant with further adjustment for sociodemographic, health, and behavioral covariates. However, when adjusting for baseline allostatic load (Model 5), anger expression became marginally related to a lower allostatic load score (per 1-SD increase,  $\beta = -0.06$ ; 95 %CI: -0.12, 0.0001), while anger control became significantly associated with a higher allostatic load score (per 1-SD increase,  $\beta = 0.07$ ; 95 %CI: 0.004, 0.14).

#### 3.3. Secondary analyses

Tests for interaction assessing effect modification by age, sex, education, or stress levels were virtually all not statistically significant (p>.05), except for two interaction terms between anger-specific regulation strategies and education levels. Findings from related stratified analyses are presented in Supplemental Table 3 and Fig. 1. Specifically, among adults with some college or more, greater use of anger expression was related to lower allostatic load (e.g., per 1-SD increase in fullyadjusted Model 5,  $\beta$ = -0.12; 95 %CI: -0.20, -0.05), whereas greater use of anger control was associated with higher allostatic load (e.g., per 1-SD increase in fully-adjusted Model 5,  $\beta$ = 0.14; 95 %CI: 0.05, 0.24). Among adults with a high school diploma or less, associations of anger expression and control, respectively, with allostatic load, were nonsignificant.

#### Table 2

Descriptive statistics of sociodemographic and health-related covariates, affect regulation exposures and allostatic load outcomes (N=574).

	M (SD)	N (%)
Sociodemographic and health covariates (MIDUS		
Age	52.23 (9.93)	
Sex Male		308 (53.66
Female		266 (46.34
Racial minority status (N=555)		25 (4.50)
Marital status		
Married/Living with Partner		432 (75.26
Separated/Widowed/Divorced		95 (16.55)
Never Married		47 (8.19)
Education (N=573)		
Less Than High School		109 (19.02
High School Diploma/GED		172 (30.02
Some College or More		292 (50.96
income (N=533) \$0-24,999		214 (40.15
\$25,000-44,999		102 (19.14
\$45,000–74,999		120 (22.51
\$75,000–200,000		97 (18.20)
Prevalent or history of heart disease (N=562)		39 (6.94)
Prevalent or history of cancer (N=571)		53 (9.28)
Physical activity <sup>a</sup>		
Active		310 (54.01
Inactive		264 (45.99
Smoking (N=404)		100 ( 10 6 1
Never Smoked		177 (43.81)
Past Smoker Current Smoker		172 (42.57 55 (13.61)
Alcohol consumption <sup>b</sup>		55 (15.01)
None or Heavy		186 (32.40
Moderate		388 (67.60
Affect regulation exposures (MIDUSII)		
General regulatory strategies (Range 4–16)		
Positive Reinterpretation & Growth	12.46 (2.42)	
Active Coping	12.60 (2.11)	
Planning	13.05 (2.34)	
Focusing on & Venting of Emotion Denial	9.37 (2.82)	
Behavioral Disengagement	5.67 (1.91) 6.57 (2.12)	
Emotion-specific regulatory strategies	0.07 (2.12)	
Anger Expression (Range 4–32)	12.92 (3.13)	
Anger Control (Range 4–16)	10.20 (2.17)	
Anger Suppression (Range 4–32)	14.86 (4.10)	
Sex-specific allostatic load outcomes (MIDUSIII)		
Fotal Allostatic Load Score (0–7)	1.35 (0.89)	
Sympathetic Nervous System (SNS)	0.07 (0.17)	
Epinephrine Score		134 (23.34
Norepinephrine	0.00 (0.04)	143 (24.91)
Parasympathetic Nervous System (PNS)	0.20 (0.34)	115 (00.02
Low Frequency Spectral Power High Frequency Spectral Power		115 (20.03
SDRR		115 (20.03 115 (20.03
RMSSD		115 (20.03
HPA	0.09 (0.19)	110 (20100)
DHEAS		144 (25.09
Cortisol		143 (24.91
Inflammation	0.25 (0.28)	
CRP		143 (24.91
L-6		144 (25.09
Fibrinogen		141 (24.56
-		143 (24.91
E-Selectin		
SE-Selectin SICAM–1	0.24 (0.28)	
E-Selectin SICAM–1 CVD	0.24 (0.28)	143 (24.91
SE-Selectin SICAM–1 CVD Resting Pulse	0.24 (0.28)	143 (24.91 22 (3.83)
E-Selectin MCAM–1 CVD Resting Pulse Resting Systolic Blood Pressure	0.24 (0.28)	143 (24.91 22 (3.83) 126 (21.95
sE-Selectin sICAM–1 CVD Resting Pulse Resting Systolic Blood Pressure Resting Diastolic Blood Pressure		143 (24.91 22 (3.83)
E-Selectin SICAM–1 CVD Resting Pulse Resting Systolic Blood Pressure Resting Diastolic Blood Pressure Glucose Metabolism	0.24 (0.28) 0.25 (0.34)	143 (24.91 22 (3.83) 126 (21.95 49 (8.54)
E-Selectin SICAM-1 CVD Resting Pulse Resting Systolic Blood Pressure Resting Diastolic Blood Pressure Glucose Metabolism Homair Hba1c (N=572)		143 (24.91 22 (3.83) 126 (21.95
SE-Selectin SICAM–1 CVD Resting Pulse Resting Systolic Blood Pressure Resting Diastolic Blood Pressure Glucose Metabolism Homair		143 (24.91 22 (3.83) 126 (21.95 49 (8.54) 143 (24.91
E-Selectin ICAM-1 VD Resting Systolic Blood Pressure Resting Diastolic Blood Pressure Jlucose Metabolism Jomair Haalc (N=572)		143 (24.91 22 (3.83) 126 (21.95 49 (8.54) 143 (24.91 48 (8.39)

(continued on next page)

#### Table 2 (continued)

	M (SD)	N (%)
Waist Hip Ratio (N=573)		127 (22.16)
Triglyceride		49 (8.54)
HDL Cholesterol		114 (19.86)
Total Cholesterol		377 (65.68)

*Notes.* These statistic descriptives were conducted before multiple imputation was implemented.

<sup>a</sup> Active=participants indicated they were moderately or vigorously active at least once per week in both summer and winter, from any context (i.e., employment, leisure, and chores); Inactive=participants not classified as Active.

<sup>b</sup> Moderate drinkers=females who reported more than zero but less than or equal to one drink per day, and males who reported more than zero but less than or equal to two drinks per day; Heavy drinkers=females who reported more than two drinks and males who reported more than three drinks per day.

### 4. Discussion

This study examined longitudinal associations between affect regulation and allostatic load. Results revealed specificity in the associations among various indicators of affect regulation and allostatic load up to 17 years later. Specifically, the use of general strategies (e.g., planning, denial) was not predictive of allostatic load. Rather, greater affect regulation variability in the use of these strategies were related to lower allostatic load later on. In addition, the role of emotion-specific regulatory strategies in allostatic load varied by education levels. Stratified analyses indeed showed that, among more educated participants only, greater use of anger expression was related to lower allostatic load while greater use of anger control was associated with higher allostatic load over time. However, age, sex, and stress levels did not modify the associations of all affect regulation exposures with future allostatic load. Besides, estimates were only barely attenuated across models progressively adjusting for sociodemographic, health, and behavioral covariates, as well as baseline allostatic load.

#### 4.1. General regulatory strategies and allostatic load

Since non-significant estimates were obtained with general regulatory strategies, one could argue that allostatic load is unlikely to be a plausible pathway connecting such regulatory strategies with long-term health outcomes (e.g., CVD incidence, mortality risk), even if this association has been reported in several previous studies (Roohafza et al., 2021; Svensson et al., 2016; Davidson and Mostofsky, 2010; Trudel-Fitzgerald et al., 2021, 2024b). Notwithstanding, results trended in the expected direction, with strategies typically deemed adaptive being related to lower allostatic load and strategies usually viewed as maladaptive related to higher allostatic load. It is also worth noting that magnitude and direction of estimates were only slightly smaller than significant results obtained in prior longitudinal research. For example, among 985 MIDUS participants, greater purpose in life is related to lower allostatic load 10 years later (per 1-SD increase,  $\beta = -0.09$ ; 95 %CI: -0.15, -0.02) (Zilioli et al., 2015). Lastly, the relatively small sample size in our study (N=574) and related limited statistical power may have restraint the capacity to detect small but true associations.

## 4.2. Affect regulatory variability levels and allostatic load

Our findings also suggest that participants with greater variability in the use of these general strategies, relative to those displaying moderate variability levels, have lower allostatic load. Albeit not statistically significant, both moderate and greater variability levels were also associated with lower allostatic load when compared with lower variability. These results appear at first somewhat unexpected. As discussed previously, individuals with greater variability, hence displaying high unevenness in scores across strategies, may rely on the same few strategies regardless of the situation. In contrast, those with moderate variability, hence displaying moderate unevenness in scores across strategies, may alter their strategies depending on the situation. In turn, individuals categorized as having moderate variability would be more flexible/less rigid than their counterparts with greater variability in how they regulate their affect across contexts. Based on this premise, greater variability should be associated with *poorer* rather than better health

## Table 3

Linear regressions modeling the associations between affect regulation exposures (MIDUSII) and a sex-specific continuous allostatic load score (MIDUSIII), N=574.

	Model 1		Model 2 C		Core Mo	Core Model 3		Exploratory Model 4		Model 5	
	β	95 % CI	β	95 % CI	β	95 % CI	β	95 % CI	β	95 % CI	
General regulatory strategies (p	er 1-SD inc	rease)									
Positive Reinterpretation &	-0.02	(-0.09, 0.04)	-0.03	(-0.10, 0.04)	-0.03	(-0.10, 0.04)	-0.02	(-0.09, 0.04)	-0.03	(-0.09, 0.03)	
Growth											
Active Coping	-0.03	(-0.11, 0.04)	-0.02	(-0.09, 0.06)	-0.02	(-0.09, 0.06)	-0.01	(-0.08, 0.07)	0.01	(-0.06, 0.07)	
Planning	-0.02	(-0.09, 0.05)	-0.01	(-0.08, 0.06)	-0.01	(-0.08, 0.06)	0.001	(-0.07, 0.07)	0.02	(-0.04, 0.08)	
Focusing on & Venting of	0.03	(-0.05, 0.11)	0.03	(-0.05, 0.11)	0.03	(-0.05, 0.11)	0.03	(-0.05, 0.11)	0.05	(-0.02, 0.12)	
Emotion											
Denial	0.06	(-0.02, 0.14)	0.04	(-0.04, 0.12)	0.03	(-0.05, 0.11)	0.04	(-0.05, 0.12)	0.03	(-0.03, 0.10)	
Behavioral Disengagement	0.06	(-0.02, 0.14)	0.05	(-0.03, 0.13)	0.04	(-0.03, 0.12)	0.04	(-0.04, 0.12)	0.03	(-0.03, 0.10)	
Affect regulation variability lev	'els†										
Moderate vs. Lower	-0.13	(-0.31, 0.06)	-0.14	(-0.32, 0.05)	-0.14	(-0.32, 0.05)	-0.14	(-0.33, 0.04)	-0.10	(-0.25, 0.06)	
Greater vs. Lower	-0.11	(-0.26, 0.03)	-0.11	(-0.26, 0.04)	-0.11	(-0.26, 0.04)	-0.11	(-0.25, 0.04)	-0.10	(-0.22, 0.02)	
Greater vs. Moderate	-0.17	(-0.32, -0.02)	-0.17	(-0.33, -0.02)	-0.17	(-0.33, -0.02)	-0.17	(-0.32, -0.02)	-0.14	(-0.27, -0.01)	
		*		*		*		*		*	
Emotion-specific regulatory stra	ategies (per	1-SD increase)									
Anger Expression	-0.06	(-0.13, 0.02)	-0.05	(-0.12, 0.02)	-0.05	(-0.12, 0.03)	-0.05	(-0.12, 0.03)	-0.06	(-0.12, 0.0001)§	
Anger Control	0.05	(-0.03, 0.13)	0.06	(-0.02, 0.13)	0.06	(-0.02, 0.13)	0.06	(-0.01, 0.14)	0.07	(0.004, 0.14)*	
Anger Suppression	0.05	(-0.03, 0.14)	0.06	(-0.03, 0.14)	0.06	(-0.03, 0.14)	0.05	(-0.04, 0.13)	0.05	(-0.02, 0.12)	

§  $p \leq .10$ ; \*  $p \leq .05$ ;  $\beta$ =beta estimate, CI=confidence interval.

 $^\dagger$  All affect regulation variability analyses are additionally adjusted for the mean strategy score.

Model 1: age-adjusted

Model 2: Model 1 + sex, race, marital status, income, education at baseline (MIDUSII)

Model 3: Model 2 + prevalent/history of heart disease and cancer at baseline (MIDUSII)

Model 4: Model 3 + physical activity, smoking, and alcohol consumption at baseline (MIDUSII)

Model 5: Model 4 + allostatic load at baseline (MIDUSII)

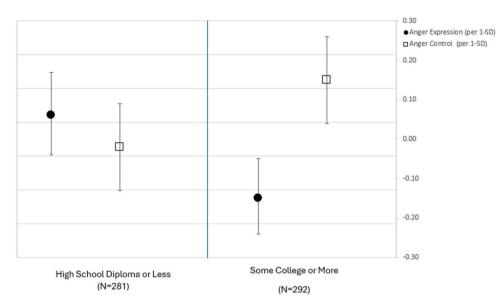


Fig. 1. Associations of emotion-specific regulatory strategies and allostatic load, stratified by highest level of education. *Notes*. Models are adjusted for sociodemographic characteristics and health status (core Model 3). Stratified sample sizes do not add up to the total N=574 because one participant only had missing data on education level. Nonetheless, these analyses were computed after multiple imputation, on N=574.

outcomes compared to moderate variability. Accordingly, prior MIDUS research indicated that greater relative to moderate variability levels were associated with a *shorter* lifespan (Trudel-Fitzgerald et al., 2022), which is inconsistent with the current results. Yet, the analytic sample of this prior MIDUS study was less healthy than the current one (e.g., 28 % with a major chronic disease vs. 7–9 % with heart disease or cancer, 51 % vs. 46 % physically inactive, 16 % vs. 14 % current smokers, 80 % vs. 32 % unfavorable alcohol consumption).

The current variability findings, however, are aligned with other longitudinal results obtained in the Nurses' Health Study (Trudel-Fitzgerald et al., 2024b), which showed that both moderate and greater variability levels are associated with a longer lifespan, among a sample that also encompasses fairly healthy individuals (e.g., 0 % with major chronic disease, 8 % current smokers, 42 % unfavorable alcohol consumption). An alternative hypothesis raised by the authors of the Nurses' Health Study investigation is that participants in their greater variability level showed less contrast/unevenness in the scores of strategies used, compared to the contrast observed among participants with a greater variability level in the analogous MIDUS study. In other words, greater variability levels might be beneficial for health, but only to a certain level. Altogether, it is possible that the role of affect regulation variability in long-term health outcomes varies according to health status and affect regulation characteristics of the sample under study.

#### 4.3. Emotion-specific regulatory strategies and allostatic load

When considering emotion-specific strategies, the present analyses showed significant associations between greater anger expression and lower allostatic load, and between greater anger control and higher allostatic load, which were marginal in the overall sample and clearly significant among more educated adults. Unlike our findings, in a crosssectional MIDUS study, greater anger control was marginally associated with *lower* levels of inflammation markers that are IL-6 and CRP (but not fibrinogen) when assessed in the overall sample and not clearly associated with these two inflammatory markers based on education levels (Boylan and Ryff, 2013). However, consistent with our results, greater anger expression was marginally related to lower IL-6 and fibrinogen (but not CRP) levels among more educated participants (Boylan and Ryff, 2013).

These partially conflicting findings may be explained by differences in sample sizes and study designs used in our longitudinal study (N=574) compared to the cross-sectional one (N=1054). In fact, it is possible that attempts to keep one's cool or to calm down fast, which are anger control strategies measured in MIDUS, are related to physical health benefits when considered concurrently with allostatic load; yet, engaging in these control strategies may yield some wear and tear in the longer-term because the implementation of such strategies may be effort-intensive, for instance among individuals who often experience strong feelings of anger.

#### 4.4. Potential pathways and effect modifiers

These observed associations of affect regulation variability and anger regulatory strategies, respectively, with future allostatic load may be explained by several potential pathways and effect modifiers. First, scarce but promising findings suggest that the ways individuals cope with stressors and regulate their emotions are associated with health behaviors (Park and Iacocca, 2014; Holt et al., 2014), which are known to modulate future allostatic load (McEwen, 1998). Yet, when controlling for physical activity, smoking, and alcohol consumption in the current models, estimates were remarkably robust, which reduces such possibility. Other potential pathways, including intermediate biological processes like mitochondrial functioning (Picard et al., 2014) and cellular aging more broadly should be investigated in future work.

Second, affect regulation may be particularly potent for allostatic load among specific subgroups only, including individuals of racial minority status (Boylan et al., 2015) and those experiencing elevated objective or subjective stressors (Christensen et al., 2019). In the current sample, there were too few non-White participants (<5 %) to accurately examine this research question. However, we were able to consider interaction terms between affect regulation exposures and a comprehensive stress score that encompass both objective and subjective stressors (e.g., financial stress, perceived inequality). No effect modification was observed, suggesting that affect regulation may contribute to allostatic load over time regardless of the stress experienced or perceived.

#### 4.5. Limitations and strengths

The present study is not without its limitations. As noted previously, the sample was relatively healthy and homogeneous in terms of race/ ethnicity (<5% were of minority status), which limits the results

generalization. However, given the challenges that exist in maintaining a cohort over several decades, and repeatedly collecting biomarkers from participants, this relatively healthy and homogeneous sample is still able to make an important, though preliminary, contribution to the literature. Moreover, the experience of psychosocial stressors over the 17-year period was not documented in MIDUS. Yet, the current results showed that the affect regulation-allostatic load relationship did not differ by stress levels reported at baseline, and affect regulation exposures were fairly stable between MIDUSII and MIDUSIII, hence reducing concerns that subsequent stressful events would impact substantially our conclusions. Lastly, our emotion-specific regulatory measure focused on angry feeling and captured three strategies only. Thus, future research should assess a greater number of anger-specific strategies in order to calculate variability in the use of these strategies, as well as consider regulatory measures specific to other emotions (e.g., sadness) (Zaid et al., 2021).

A notable strength of the present study is its use of longitudinal, prospective data with 17 years of follow-up, boasting a robust research design with a greater ability to support accurate temporality and directionality in contrast with other study designs like cross-sectional and retrospective ones. In respect to measurement, the use of objective biomarkers to assess allostatic load at both baseline and follow-up, and the use of validated scales to ascertain affect regulation strategies also support the robustness of the study. Compared to other studies that focus on the inherent (mal)adaptive value of individual regulatory strategies, the use of variability levels to represent the extent to which individuals may use different strategies in different situations allows further understanding of affect regulation as a complex phenomenon rather than a binary characteristic.

#### 5. Conclusions

Although general affect regulation strategies like planning and denial do not seem to predict future allostatic load levels over a 17-year period in this sample of 574 relatively healthy midlife and older adults, greater affect variability levels appeared to have a predictive protective value. Moreover, emotion-specific regulatory strategies, namely anger expression and anger control, were each associated with allostatic load in opposite directions, especially among more educated individuals. Additional research with larger, more diverse samples, is needed to further understand how the way individuals cope with stressors and regulate their emotions may affect biological processes that contribute to long-term health.

#### CRediT authorship contribution statement

**Claudia Trudel-Fitzgerald:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Funding acquisition, Conceptualization. **Robert-Paul Juster:** Writing – review & editing, Methodology. **Tara Gruenewald:** Writing – review & editing, Methodology. **Amanda E. Ng:** Writing – review & editing, Writing – original draft, Visualization, Methodology, Formal analysis, Data curation.

#### **Declaration of Competing Interest**

The authors do not have any declarations of interest to disclose.

#### Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.psyneuen.2024.107163.

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