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RESEARCH ARTICLE

Relational and individual stress pathways linking discrimination and ageing cardiometabolic health

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Abstract

Perceived discrimination is a significant risk factor for worse ageing health outcomes. Yet, the specific individual and relational stress pathways linking discrimination to disease are less understood, especially in the context of cardiometabolic health. We tested family stress and psychophysiological distress (negative affect and high-risk lipid/fat metabolism) as mediators linking perceived discrimination to cardiometabolic morbidity and health appraisal over 20 years for midlife adults. Using data from participants who completed the Biomarker Project (2004–2009) of the Midlife in the U.S. project, and examining data over the study's three waves (1995–1996, 2004–2006, and 2013–2014), we used structural equation modelling to test pathways for participants who reported zero cardiometabolic conditions at baseline ($n = 799$). Greater Time 1 discrimination was associated with greater Time 2 family strain, which was in turn associated with worse negative affect; worse Time 2 negative affect was associated with worse Time 3 health appraisal; metabolic lipids risk did not serve as an indirect pathway to Time 3 cardiometabolic morbidity ($\chi^2 = 147.74$, $p < 0.001$; RMSEA = 0.056; CFI = 0.902; SRMR = 0.047). The inclusion of family in interventions to mitigate the impact of discrimination may be indicated for promoting cardiometabolic wellness.

KEYWORDS
ageing, family relations, glucose metabolism disorders, heart diseases, lipid metabolism, social discrimination, stress

1 | INTRODUCTION

Perceived discrimination is a chronic stressor and thus a significant risk factor for worse physical health. Recent prevalence estimates suggest that 1 in 4 Americans, across racial/ethnic groups, report experiences of discrimination (Boutwell et al., 2017). Prior evidence supports psychophysiological mechanisms whereby discrimination impacts health, including individuals' heightened stress responses (Pascoe & Smart Richman, 2009), which deteriorate the functioning of physiological systems (Berger & Sarnyai, 2015). A biopsychosocial conceptualisation of distress resulting from discrimination includes impaired psychological wellbeing, relationship stress, as well as worse markers of physiological functioning (Berger & Sarnyai, 2015; Doyle & Molix, 2014; Paradies et al., 2015). Recent research has found evidence for each, including associations between discrimination and affective reactions (Gerrard et al., 2018), family conflict (Kwon, 2020), strain (Priest et al., 2020), and biomarker indicators of stress (Doyle & Molix, 2014). Further, these pathways have begun to

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be substantiated for a broad collection of structurally disadvantaged populations, including sexual minorities (Doyle & Molix, 2015), women (Saban et al., 2018), and racial/ethnic minorities (Priest et al., 2020). However, to date, research has yet to test both individual and relational distress mechanisms linking perceived discrimination and health over the course of ageing.

Comprehensively modelling links between discrimination and disease may be especially relevant in the context of cardiometabolic conditions, including cardiovascular disease and diabetes (Dolezsar et al., 2014). These conditions are stress-related, impacted by social adversity (Friedman et al., 2015) and poor psychological health (Boylan & Ryff, 2015); they are also more prevalent among populations at the greatest risk of experiencing discrimination (e.g., minoritised populations such as Black/African American and Hispanic adults; Centers for Disease Control and Prevention, 2020). New research has established an association between greater perceived stress and greater cardiometabolic risk, including blood glucose, lipids, and blood pressure, across racial/ethnic groups (Lehrer et al., 2020). However, tests of mediating stress pathways linking psychosocial factors to increased cardiometabolic health risk have been few, and often cross-sectional (Farhangi & Jahangiry, 2020), made worse by the infrequent use of large, epidemiological datasets (Lewis et al., 2014). Indeed, research linking discrimination and health has long pointed to the need to specify longitudinal mechanisms which may serve as precursors to emerging health risks (Paradies et al., 2015; Williams et al., 2019). These mediating pathways may also serve as targets for intervention to reduce the impact of discrimination-related stress, essential for addressing health inequities tied to marginalisation and bias (Cedillo et al., 2020). Thus, the purpose of the present study is to test specific stress pathways – individual (i.e., psychophysiological stress) and relational (i.e., family stress) – by which discrimination impacts long-term cardiometabolic health in a large, national sample of ageing adults.

Theoretically, we position family relationships as a mediator, partially explaining the effects of discrimination on individuals' psychological and biological distress (Figure 1). This is informed by prior approaches theorising discrimination as a stressful life experience adversely impacting psychophysiological stress reactivity via negative effects on close family relationship quality (Priest et al., 2020). Broadly, theory frequently links social support to health and disease via psychological and physiological pathways (Uchino et al., 2018). We have expanded this lens to include consideration of a powerful contextual stressor for many. In other words, we theorise that as the stress of discrimination spills over to affect important social connections, the greater family strain that may be experienced serves as a conveyor of stress that is reflected downstream in worse mental health, worse physiological markers of distress, and eventual disease. Though we review research utilising varied definitions and measurement of discrimination, to elucidate the state of the literature in this area, we acknowledge that the variation is an area of growth for discrimination-based stress research, and that our study will specifically examine the impact of self-reported discrimination that is particularly postulated to produce psychophysiological stress cascades to health, given the assessment of individuals' perceptions and awareness of inequitable treatment (Williams et al., 2019).

1.1 | Perceived discrimination and family stress

The literature has established stress-spillover effects of discrimination, into the family environment, whereby family members who report experiencing the chronic stressor of discrimination in their daily lives are more likely to act with hostility towards family, or to be less able to form and maintain positive connections to loved ones (Priest et al., 2020). Family members experiencing discrimination may

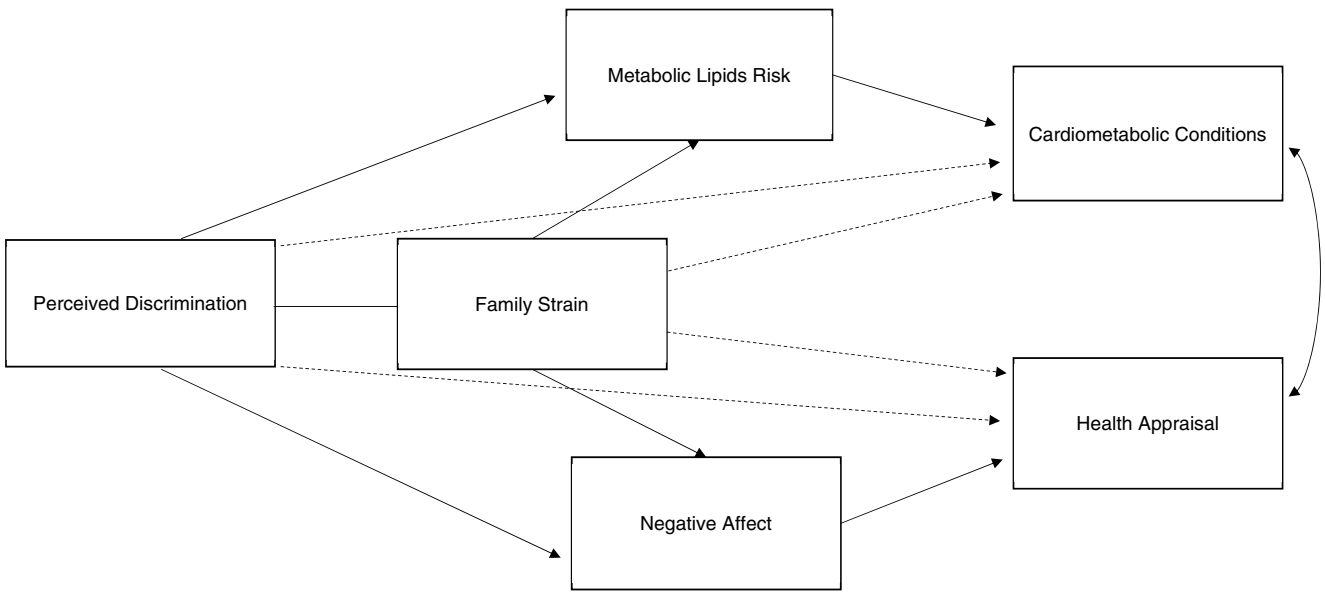

FIGURE 1 Hypothetical model of mediational pathways linking baseline discrimination to cardiometabolic health 20 years later

also be less able to parent with warmth and understanding, or have less energy to contribute to usual family roles, for example, Broadly, associations between discrimination and worse family relationship quality have been found in multiple populations and for many different types of discrimination. For example, prior research investigating racism-based health disparities has found links between perceived discrimination, close relationship strain, and stress biomarkers (Doyle & Molix, 2014) as well as cardiovascular outcomes (Doyle et al., 2018) among African Americans. Similarly, Priest et al. (2020) found associations between discrimination and family strain, which was subsequently linked to disease activity via individuals' stress reactivity for African American participants in the Midlife in the U.S. (MIDUS) study (Ryff, Almeida, Ayanian, et al., 2017). In other prior research, Mexican American men with greater exposure to ethnic discrimination at work demonstrated less warmth to their wives when compared to men in secure, and positive work environments (Hengstebeck et al., 2018), and among Asian Americans, 40% of the association between perceived discrimination and psychological distress was conveyed by family strain, especially family conflict, in this group (Kwon, 2020).

Connections between discrimination and relationship functioning have also been found for sexual minorities. Homophobia can contribute to family strain in the face of greater orientation visibility (Ocobock, 2013). Likewise, Doyle and Molix (2015) found meta-analytic evidence for positive associations between social stigma (including discrimination) and relationship strain among sexual minorities. Links between discrimination and increased family relationship distress also occur in the context of weight-related stigma, where being treated poorly may occur in the family environment, negatively impacting overweight persons' mental health (Carr & Jaffe, 2012). Weight-related discrimination is significantly more likely for women (Puhl et al., 2008), and gender is, in and of itself, frequently a basis for discrimination. However, though discrimination and family relationship quality have each separately been supported as risk factors for cardiovascular disease among women, studies have not consistently linked the two via a stress pathway (Stewart et al., 2018).

Though there is preliminary research on varied types of discrimination and its impacts on relational distress, the bulk of these studies have focussed on racial/ethnic discrimination and, increasingly, sexual orientation. Additionally, despite the influence of chronic experiences of discrimination on stress reactivity, the literature is limited in how it has conceptualised biopsychosocial pathways linking discrimination to cardiometabolic health (Lewis et al., 2014). To better understand how perceived discrimination is linked to cardiometabolic health outcomes, we should understand whether family relationship quality serves as a mechanism of effect.

1.2 | Perceived discrimination and individual stress reactivity

In conceptualising individual stress reactivity as a pathway to cardiometabolic health, research has substantiated both psychological and physiological measures of distress. Our hypothesised model includes both mechanisms (i.e., individual stress pathways including psychological and physiological stress reactivity), reflective of the discrimination-health literature to date (Cuevas & Williams, 2018; Figure 1).

1.2.1 | Psychological stress mechanisms

Research has long connected experiences of discrimination to poor mental health. In fact, though discrimination is more prevalent for adults of lower socioeconomic status, Kessler et al. (2012) suggested that perceived discrimination explains only a small portion of the link between economic disadvantage and mental health because it is so strongly associated with mental health across *all* socioeconomic groups. Increasingly emotional distress – psychological stress, negative affect, depression, and anxiety – is considered a conveyor of the effects of discrimination on physical health (Paradies et al., 2015). For example, Bastos et al. (2015) found that the association between discrimination and self-rated poor health was entirely mediated by anxiety/depression. More recently, Stokes and Moorman (2020) discovered cross-sectional links between discrimination (age, and other), worse self-rated health, and activities of daily living via declines in positive affect and worse psychological well-being; worse positive affect also served as a significant mediator of a discrimination-morbidity link.

Theoretically, psychological stress reactivity (i.e., emotional responses to stress) serves as an important mediator linking contextual and social stress and disease (Cuevas et al., 2013; Uchino et al., 2018). The effects of discrimination on affect may be direct, but may also occur indirectly, via weakened social support and strained close relationships. Greater psychological distress, in conjunction with physiological distress (described below) may serve to wear on the body, promoting worse health outcomes. Prior models examining these links have been supported, including when testing perceived discrimination (Priest et al., 2020). However, these tests, too, have been limited by a reliance on cross-sectional analyses and broad, subjective measures of health.

1.2.2 | Physiological stress mechanisms

Perceived discrimination is also an antecedent for physiological stress, which negatively impacts physical health (Pascoe & Smart Richman, 2009). Cardiovascular markers of physiological distress may include prehypertension, prediabetes, and dyslipidemia, which are often poorly controlled (Kones & Rumana, 2017) and occur among at least 70% of adults in the U.S (Mozaffarian et al., 2016). A growing body of research suggests that discrimination is an important factor in understanding patients' cardiometabolic risks, with a growing need to determine how the effects of discrimination occur throughout the life course to increase cardiometabolic morbidity (Albert & Williams, 2011).

While multiple biological mechanisms have been suggested as linking discrimination and health, there has been a specific focus on lipid/fat metabolism in the context of cardiometabolic disease. Evidence has supported the impact of perceived discrimination on this physiological system as one of a collection of systems reflecting allostatic load (Schwartz, 2017), as well as an independent system via measures of body mass index (BMI), waist-to-hip ratio (WHR), high-density lipoprotein cholesterol (HDL), triglycerides, and low-density lipoprotein cholesterol (LDL) (Van Dyke et al., 2020). Support has also been found for lipid/fat metabolism as a mediator across multiple types of discrimination, including weight- (Tsenkova et al., 2011) and race-related perceived discrimination (Cedillo et al., 2020). The specific mechanisms by which self-reported discrimination affects physiological stress response systems, including lipid/fat metabolism, are less understood (Cedillo et al., 2020; Van Dyke et al., 2020). However, the link is theorised to be stress-specific, whereby the body's many physiological systems mount a coordinated response to stress which, in the face of frequent, chronic, or unrelenting stress, serves to wear and tear on the body to produce worse health and promote ageing. In the context of lipid/fat metabolism, stress reactivity may include concomitant changes in inflammatory markers that affect cholesterol production, or cardiovascular changes including increased blood pressure. Studies support the stress impact on energy production which impacts liver secretion of LDL, and dampens the ability to metabolise lipids out of the body, promoting fat accumulation reflected in increased BMI/WHR, as well as sugar accumulation that converts to triglycerides when unused to respond to environmental stressors (Cedillo et al., 2020).

Though the specific ways in which the stress of discrimination influence individual risk biomarkers remain unknown, it is likely a confluence of psychophysiological stress reactivity. In total, the discrimination-health literature has given less attention to the physiological mechanisms whereby discrimination is linked to disease, and the research that has broached testing these mechanisms has often relied on cross-sectional data (Cuevas & Williams, 2018).

1.3 | Present study

Prior research demonstrates discrimination plays an important role in the development of cardiometabolic illness, cutting across demographic factors. The purpose of the present study is to test relational and individual stress pathways by which perceived discrimination impacts long-term cardiometabolic health for ageing adults. We propose a longitudinal mediation model, and hypothesise greater daily discrimination is associated with worse cardiometabolic health over 20 years via greater family strain, greater negative affect, and greater metabolic lipids risk (Figure 1). We further hypothesise that part of the effect of discrimination on psychophysiological stress is via effects on family strain. Specifically, family strain and psychophysiological distress operate in sequence, such that greater family strain is significantly associated with greater psychophysiological stress in order to partially convey the effects of discrimination on health. Lastly, we hypothesise metabolic lipids risk is associated with objective physical health (i.e., operationalised as aetiology of cardiometabolic conditions), while negative affect is associated with subjective physical health (i.e., operationalised as health appraisal).

2 | METHOD

2.1 | Sample

The sample for the present study includes participants in the longitudinal MIDUS study, who completed the MIDUS 2 Biomarker Project and who reported zero metabolic conditions at baseline (Brim et al., 2011; Ryff, Almeida, Ayanian, et al., 2017; Ryff, Almeida, Binkley, et al., 2017; Ryff et al., 2019). MIDUS is a national project examining biopsychosocial factors impacting ageing health. Data collection was initiated in 1995 with a representative survey of 7108 English-speaking adults age 25–74 years (M age = 46.38, SD = 13.0, 51% female) recruited using random-digit-dialing as well as oversampling across five metropolitan areas (Brim et al., 2011). MIDUS 1 participants completed a telephone interview and self-administered questionnaires by mail. This initial wave was followed by MIDUS 2 (2004–2006), which captured 4963 of MIDUS 1 participants (M age = 55.43, SD = 12.45, 53.3% female). MIDUS 2 participants similarly completed a telephone interview and questionnaire. Additionally, a subset of MIDUS core participants (n = 1054) subsequently participated in the MIDUS 2 Biomarker Project (Ryff et al., 2019), which involved the addition of biomarker assessments and a lab-based health protocol. Data collection for the Biomarker Project occurred at three research centres during 2004–2009, and included a 2-day clinic visit during which participants provided blood, urine, and saliva samples, and completed a physical exam and health assessments (Dienberg Love et al., 2010). The Biomarker subsample of MIDUS is demographically comparable to the larger MIDUS sample (i.e., in regard to age, sex, income, marital status, physical health), though Biomarker participants have somewhat greater education (Dienberg Love et al., 2010; Radler et al., 2018). A third MIDUS wave was conducted in 2013–2014, and included 3294 (M age = 63.64, SD = 11.35; 54.9% female) of the initial MIDUS 1 participants (66.4% of MIDUS 2) (Ryff et al., 2019).

The present study uses the longitudinal subsample of MIDUS participants who completed the MIDUS 2 Biomarker Project and who, for the purposes of testing the present hypotheses (i.e., including aetiology of new cardiometabolic conditions over time), *denied* having cardiometabolic conditions at MIDUS 1, resulting in a final sample of 799 participants for the present analyses (75.8% of MIDUS 2 Biomarker Project participants). Given the long period of time over which the MIDUS study has been ongoing, and the age range of participants (with a focus on midlife), attrition in the MIDUS project is observed (Stokes & Moorman, 2020). MIDUS 1 to MIDUS 2 retention is calculated at 75%, when adjusted for mortality, as documented by Radler and Ryff (2010). We examine missingness in the present sample as a function of baseline age, sex, discrimination,

and MIDUS 2 family strain, negative affect, and metabolic lipids risk. Results of those analyses are presented below.

2.2 | Measures

2.2.1 | Independent variable

We used a continuous measure of daily discrimination completed via the MIDUS 1 self-administered questionnaire and which aggregated the frequency of daily occurrences of nine types of discrimination (Kessler et al., 1999). Items asked, 'How often on a day-to-day basis do you experience each of the following types of discrimination?' and included, 'You are treated with less courtesy than other people,' and, 'People act as if they are afraid of you,' as examples. Response options ranged from 1 (*often*) to 4 (*never*) and were reverse coded such that higher scores indicated more frequent experiences of discrimination. Participant responses were summed; scale scores ranged from 0 to 27. Eight participants were missing responses on this scale (7 missing all nine items); a discrimination score was not computed for these participants.

2.2.2 | Mediating variables

We utilised three measures to capture our hypothesised indirect effects at Time 2, and in order to operationalise family strain and psychophysiological distress. For both the family strain and negative affect measures, captured via the MIDUS 2 self-administered questionnaire, MIDUS researchers used mean imputation to handle missing data: scale scores were calculated for each participant that provided responses for at least one item (Ryff et al., 2019).

Family Strain. The family strain measure included four items, each preceded by the prompt, 'Not including your spouse or partner…' (Walen & Lachman, 2000). Example items include, 'How often do members of your family make too many demands on you?,' and 'How often do they criticise you?' Respondents answered using a scale of 1 (*often*) to 4 (*never*) and participants' responses were recoded (i.e., higher scores indicate greater family strain). Averaged item responses were used as the family strain scale score, totals thus ranging from 1 to 4.

Negative Affect. We operationalised psychological distress as negative affect, which included six items assessing the frequency of negative affect indicators (Mroczek & Kolarz, 1998). Items were preceded by, 'During the past 30 days, how much of the time did you feel…' and included, 'so sad nothing could cheer you up,' 'nervous,' 'restless or fidgety,' 'hopeless,' 'that everything was an effort,' and 'worthless.' Participants responded using a scale of 1 (*all of the time*) to 5 (*none of the time*), and items were recoded such that higher scores indicated higher levels of negative affect. Scale scores were calculated by averaging item responses.

Metabolic Lipids Risk. Physiological distress was operationalised using markers of lipid/fat metabolism obtained by health care professionals in the MIDUS 2 Biomarker Project, that were then assessed for level of risk. Specific lipid/fat metabolism indicators included BMI, waist-to-hip ratio (WHR), triglycerides, high-density lipoprotein cholesterol (HDL), and low-density lipoprotein cholesterol (LDL). BMI was calculated using measurements of weight and height, whereas WHR was calculated using measurements of waist and hip circumferences. Triglycerides, HDL, and LDL (tissue markers of cardiovascular metabolic functioning, each mg/dL) were measured using a fasting blood draw completed during a 2-day clinic visit to one of the three participating project sites (Dienberg Love et al., 2010).

Participants' values on these five indicators were analysed for risk in order to calculate an overall metabolic lipids risk score. We calculated risk scores using high-risk cutoffs established (Gruenewald et al., 2012) and supported in prior research using MIDUS data (Brooks et al., 2014; Priest et al., 2020). Participants' values on the five indicators were designated as either no-risk or high-risk depending on whether the score fell below or at/above the indicator's high-risk cutoff score. For example, participants' triglycerides measurements were categorised as high-risk (assigned a score of 1) if they were at or above 160 mg/dl, or no-risk if they were below 160 (assigned a score of 0). The BMI high-risk cut-point was 32.31, WHR at 0.97, HDL at 41.37 mg/dl, and LDL at 128 mg/dl. These high-risk cutoffs are distinctly different from clinical cutoffs, in that they represent values in upper/lower quartile ranges conveying the greatest risk for problematic lipid/fat metabolism (Gruenewald et al., 2012). An average metabolic lipids risk score was then calculated: participants' scale scores were continuous and ranged from 0 to 1, with higher scores indicating greater metabolic lipids risk (a score of 1 indicating 100% of the indicators were in the high-risk range for that specific participant).

2.2.3 | Dependent variables

We operationalise ageing cardiometabolic health using two measures: number of metabolic conditions and health appraisal.

Cardiometabolic Conditions. The number of cardiometabolic conditions included a summation of the presence or absence of heart conditions suspected or confirmed by a physician, high blood pressure/hypertension (occurring in the past 12 months), and diabetes/high blood sugar (occurring in the past 12 months), for a range of 0–3 total cardiometabolic conditions. While the latter two indicators were assessed at MIDUS 3 via the self-administered questionnaire, diagnosed heart conditions were assessed via the project's telephone interview. The number of cardiometabolic conditions for the present sample averaged 0.53 (SD = 0.73, n = 660 reporting), with 40% of these participants reporting the presence of at least one of these three indicators of cardiometabolic disease.

Health Appraisal. Participants were asked in the MIDUS 3 telephone interview to rate their overall health on a scale of 1 (*excellent*) to 5 (*poor*); as such, higher scores reflect worse self-rated health. This one-item measure of self-rated health is widely used in research estimating physical health outcomes due to its significance

as a predictor of morbidity and mortality (DeSalvo et al., 2006). This variable was entered as a continuous variable given evidence of its robustness when treated thusly in prior MIDUS research (Woods et al., 2020). In the present sample, 11.6% reported poor/fair health at MIDUS 3.

2.2.4 | Control variables

We control for the effects of baseline reports of age, sex, income (dichotomised at the median, 0 = *below the median*, 1 = *at/above the median*), and family history of heart attack (i.e., number of biological family members who had previously experienced a heart attack), on Time 3 ageing health. We also control for the impact of three baseline health behaviours on Time 2 metabolic risk, including: smoking (1 = *smoking cigarettes regularly now*, 0 = *not currently smoking cigarettes*), problematic alcohol use (i.e., using a four-item version of the Michigan Alcohol Screening Test [Selzer, 1971] to assess the presence of alcohol-related problems in the past 12 months, a measure validated by MIDUS investigators [Grzywacz & Marks, 1999] where 1 = *problematic alcohol use*, 0 = *no problematic alcohol use*), and exercise (assessed as a measure of whether participants engaged in moderate or vigorous physical activity at least several times a week during summer and/or winter, either while at their paid job, while performing chores, or during their free time; examples of moderative activity included light tennis and brisk walking, while vigorous activity included running and lifting heavy objects; Brim et al., 2011). Exercise was also coded dichotomously, whereby participants who reported moderate or vigorous physical activity in any area during either season, several times per week, were coded as '1' (*achieved aerobic activity levels*), while participants who reported no moderate/vigorous activity at any time, or that occurred less frequently, were coded as '0,' an approach validated in prior research investigating cardiovascular health outcomes (Author). Finally, we regress each of our mediators and the observed ageing health variables on their corresponding MIDUS 1 reports, at baseline (e.g., MIDUS 2 family strain on MIDUS 1 family strain; MIDUS 2 metabolic lipid risk on MIDUS 1 BMI and WHR; see Supplementary Table1 for descriptive statistics).

2.3 | Analyses

We used Mplus (Muthén & Muthén, 2017) (Version 8.2) to conduct structural equation modelling (SEM) and employed full information maximum likelihood (FIML) with robust standard errors. FIML accommodates missing data by using all available data (assuming data missing at random, discussed below) and calculates parameter estimates with standard errors robust to non-normality (see Table 1 for skewness/kurtosis statistics). We evaluated model fit using root mean square error of approximation (RMSEA), comparative fit index (CFI), and standardised root mean square residual (SRMR). We determined good model fit using recommended cutoffs (Kenny et al., 2015; Kline, 2015): RMSEA <0.08 (and closer to 0.05), CFI >0.90 (closer to 1.00), and SRMR <0.10 (closer to 0.05), indicating close alignment between the hypothesised model and the data. Table 1 includes a correlation matrix; coefficients do not preclude the use of SEM (i.e., multicollinearity is not indicated).

3 | RESULTS

3.1 | Sample demographics

The present sample of 799 MIDUS participants was, at baseline, an average 44.94 years old (*SD* = 11.43), 55.3% female, and 93.1% White (2.8% Black/African American, 2.0% other, <1% Native American/Alaska Native, Asian/Pacific Islander, and multiracial,

TABLE 1 Discrimination, mediating variables, and cardiometabolic health dependent variables: Correlations (*N* = 799)

Variables	1	2	3	4	5	6
1. Daily discrimination T1						
2. Family strain T2	303***					
3. Negative affect T2	0.213***	0.281***				
4. Metabolic lipids risk T2	0.036	−0.009	0.054			
5. Health appraisal T3	0.155***	0.156***	0.272***	0.123**		
6. Metabolic conditions T3	−0.022	0.045	0.001	0.230***	0.267***	-
M	3.34	2.02	1.46	0.24	2.36	0.53
SD	4.11	0.56	0.51	0.25	0.98	0.73
α	0.91	0.76	0.83	-	-	-
Skewness	1.30	0.42	1.92	0.97	0.55	1.20
Kurtosis	1.67	0.16	5.16	0.30	−0.07	0.65

Note: T1 = Time 1/MIDUS 1, T2 = Time 2/MIDUS 2, T3 = Time 3/MIDUS 3.
$^{**}p < 0.01$, $^{***}p < 0.001$.

each). While the majority of the sample was married at baseline (72.1%), an additional 12% reported never having married, 11.9% were divorced, 2.3% were widowed, and 1.8% reported a marital status of separated. Additionally, 45.3% reported having obtained a college degree or higher level of education, while 28.2% reported some college, 22.7% reported having graduated high school, and 3.9% reported having earned a GED or having less than a high school education. Lastly, the median household income for the present sample equalled $65,500 ($M$ = $82,550.83, SD = $61,187.27), and included wages, pension, and social security or other governmental assistance.

Though our sample included participants who denied having a cardiometabolic condition at baseline, we are able to characterise additional health indicators at baseline. Specifically, the average BMI was greater than 25.0 (i.e., overweight). Additionally, the mean WHR was 0.94 (median = 0.94, SD = 0.06) for men and 0.81 (median = 0.80, SD = 0.08) for women indicating, on average, participants were approximately at recommended cutoffs (Lear et al., 2010). Reflective of the health status of this specific sample, solely 2 participants reported using a prescription medication for hypertension, and 22 for high cholesterol (2.8% of the sample); zero participants reported current prescription medications used for diabetes.

Regarding our variables of interest, 61.6% (n = 492) of the sample reported at least some experience of daily discrimination at baseline. Of these, 70.7% (n = 348) indicated reasons why they experienced discrimination, including gender (46.3%; n = 161, 144 of which were women); age (21%; these respondents ranged from 25 to 74 years, with an average age of 44.84 years, SD = 13.27); race (18.1%; n = 63, 39 of whom were White, 14 were Black/African American, 2 were Native American/Alaska Native, 2 were Asian/Pacific Islander, 3 identified as multiracial, and 3 identified as other); and height or weight (15.8%; with BMI ranging from 16.95 to 42.98, M BMI = 29.94, SD = 6.56). Additional reasons for discrimination experienced included appearance (11.5%); religion (6.0%; n = 21, 17 of whom identified their religious preference as a Christian religion); ethnicity/nationality (5.5%; n = 19, 16 of whom were U.S. citizens and 4 of whom identified as Hispanic/Latino); sexual orientation (5.2%; n = 18, 8 of whom identified as gay/lesbian, and 2 as bisexual, while the remainder identified as heterosexual, the sole response options); physical disability (2.0%); and, other (19.3%). Of note, participants were allowed to indicate more than one main reason for the discrimination, thus, percent estimates total greater than 100%.

3.2 | Missingness

Participants missing at Time 3 (n = 62) were significantly older (M = 51.81 years) than those who completed the third MIDUS wave (M = 44.36 years; F = 25.01, p < 0.000), though there was no significant difference between the groups in regard to sex (χ^2 = 0.006, p = 0.937). Participants missing at Time 3 also scored higher on MIDUS 2 negative affect (M = 1.44, SD = 0.49) than those who completed MIDUS 3 (M = 1.44, SD = 0.49; F = 8.68, p = 0.003). Participants missing at Time 3 did not significantly differ in baseline discrimination (M = 3.69, SD = 4.01) from participants who completed MIDUS 3 (M = 3.31, SD = 4.12; F = 0.47, p = 0.493). Participants who did not complete MIDUS 3 also did not significantly differ in levels of MIDUS 2 family strain (M = 2.02, SD = 0.61) from those who completed MIDUS 3 (M = 2.02, SD = 0.57; F = 0.01, p = 0.943), nor did they differ in metabolic lipids risk scores (M = 0.27, SD = 0.26 vs. M = 0.23, SD = 0.25; F = 1.77, p = 0.184). Thus, we determined data are missing at random and the selected control variables are necessary to include. Specifically, we include age, given its likely influence on health at MIDUS 3, as well as sex, and income. We also control for baseline BMI, WHR, family strain, negative affect, and health appraisal to maximise our ability to understand the influence of our hypothesised variables on cardiometabolic health while controlling for autocorrelations (additional control variables are described above). Missing data are addressed in model-testing using FIML.

3.3 | Model testing

Results demonstrated a good fit to the hypothesised model (χ^2 = 147.74, p < 0.001; RMSEA = 0.056 [90% CI = 0.046, 0.066]; CFI = 0.902; SRMR = 0.047; Figure 2). As hypothesised, a greater frequency of daily discrimination experiences reported at baseline was associated with greater family strain 10 years later (controlling for baseline family strain; Supplementary Table2). However, Time 1 discrimination was not significantly associated with Time 2 negative affect nor Time 2 metabolic lipids risk.

Regarding our proposed mediating variables, greater family strain was associated with worse Time 2 negative affect. In turn, worse negative affect at Time 2 was associated with worse health appraisal at Time 3. Contrary to our expectations, however, family strain at Time 2 was not significantly associated with Time 2 metabolic lipids risk. Time 2 metabolic lipids risk was significantly associated with later metabolic conditions, such that greater lipids risk was associated with a greater number of metabolic conditions 10 years later, as expected. Instead, family strain was directly associated with later metabolic conditions: greater family strain was associated with a greater number of metabolic conditions at Time 3. As hypothesised, neither Time 3 health appraisal nor number of metabolic conditions were significantly associated with baseline daily discrimination.

In sum, Time 1 daily discrimination was associated with Time 2 family strain, which was associated with negative affect and directly with Time 3 metabolic conditions; worse Time 2 negative affect was associated with worse Time 3 health appraisal. Metabolic lipids risk operated as an independent predictor of later metabolic conditions in this model, and was associated neither with daily discrimination nor family strain. The strength of associations between baseline WHR and BMI with mid-point metabolic lipids risk, and these risk scores with later number of diagnosed conditions, suggests that though the full sample denied the presence of heart conditions, hypertension, or

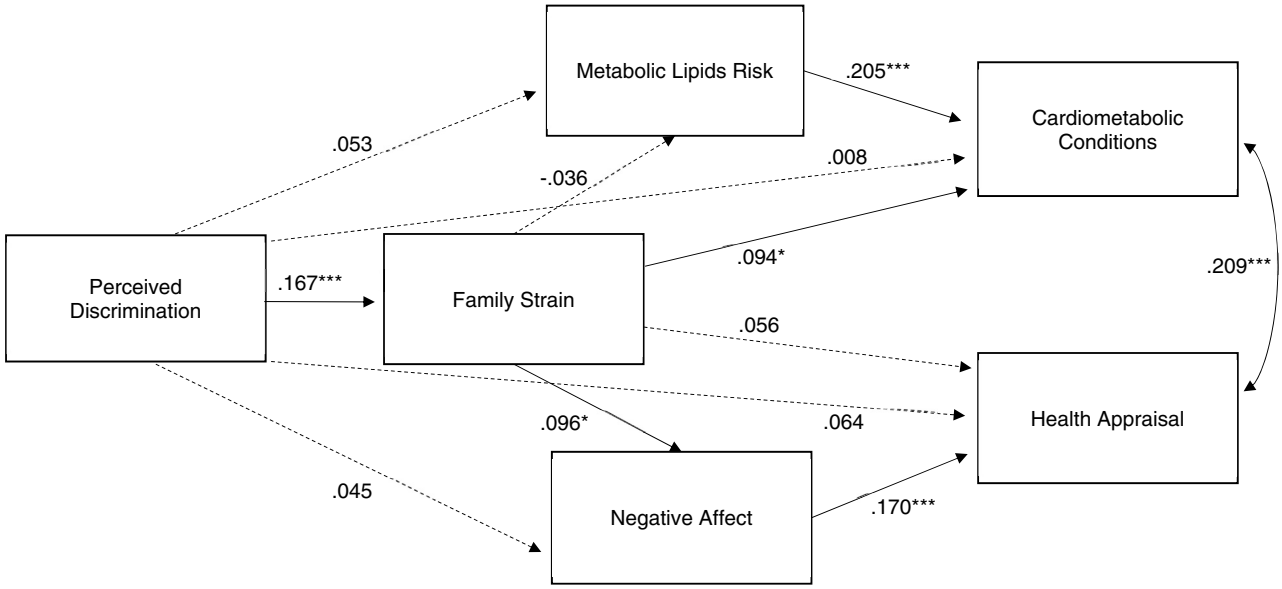

FIGURE 2 Standardised path coefficients for indirect effects of perceived daily discrimination on health ($n = 711$); $\chi^2 = 147.74$, $p < 0.001$; RMSEA = 0.056; CFI = 0.902; SRMR = 0.047. $^*p < 0.05$, $^{***}p < 0.001$. Variance explained for family strain ($R^2 = 0.322$, $p < 0.001$), negative affect ($R^2 = 0.318$, $p < 0.001$), metabolic lipids risk ($R^2 = 0.315$, $p < 0.001$), health appraisal ($R^2 = 0.206$, $p < 0.001$), and metabolic conditions ($R^2 = 0.138$, $p < 0.000$) were each significant

diabetes, those that went on to be diagnosed likely had strong premorbid indicators of their eventual health present at baseline (Supplementary Table2). The model accounts for a significant amount of variance family strain ($R^2 = 0.322$, $p < 0.001$), negative affect ($R^2 = 0.318$, $p < 0.001$), metabolic lipids risk ($R^2 = 0.315$, $p < 0.001$), health appraisal ($R^2 = 0.206$, $p < 0.001$), and metabolic conditions ($R^2 = 0.138$, $p < 0.000$).

3.3.1 | Indirect effects

As hypothesised, family strain significantly mediated the effects of daily discrimination on Time 2 negative affect, whereby the direct association was nonsignificant (Table 2). Further, negative affect significantly mediated the effects of family strain on later health appraisal. In other words, any direct effect of greater family strain on worse health appraisal was rendered nonsignificant, and was instead an effect conveyed via worse negative affect. Conversely, as indicated in results above, discrimination was not associated with metabolic lipids risk directly, nor indirectly via family strain. In addition, metabolic lipids risk did not serve as a link between family strain and metabolic conditions, which were directly linked.

Our tests of full model mediation – specifically, whether the effects of discrimination on physical health over 20 years were conveyed via interim family strain and psychophysiological distress – demonstrated partial support for our hypotheses. Specifically, a significant total and total indirect effect of Time 1 daily discrimination on Time 3 health appraisal was observed (Table 2). Results demonstrated the effect of daily discrimination on this measure of ageing health over 20 years was conveyed via greater family strain and worse negative affect. Conversely, while the direct effect of discrimination on Time 3 metabolic conditions was nonsignificant, the total indirect effect was significant, and the specific indirect effect of discrimination via family strain (though not hypothesised) was significant, indicating more stressful family relationships at Time 2 conveyed the effects of Time 1 discrimination experiences on the number of cardiometabolic conditions developed over 20 years of adulthood. In total, the present indirect pathways demonstrated weak effects.

4 | DISCUSSION

Overall, the tests of indirect effects produced mixed results. We found support for our hypotheses regarding the serial effects of discrimination on family strain, strain on negative affect, and negative affect on health appraisal. This is an important mechanism of effect over 20 years of ageing, indicating that greater daily discrimination is linked to later declines in self-rated health via relational and psychological distress pathways. However, though individual parameter estimates were significant, we found a weak mediation effect, indicating a need for study replication. With this limitation in mind, it is possible our model results extend the literature: whereas prior tests have found associations between perceived discrimination, family strain, stress reactivity, and physical health, these studies have been limited by cross-sectional data and narrowly defined samples (Doyle & Molix, 2014; Priest et al., 2020).

Further, we hypothesised family strain as a partial mediator of the link between discrimination and negative affect, given prior research demonstrating direct discrimination-affect associations.

TABLE 2 Standardised point estimates and significance levels for indirect effects (Standard errors in parentheses; $N = 711$)

Indirect Pathway	Standardised	*p*	95% CI
Daily discrimination T1→ family strain T2 → negative affect T2			
Total	0.061 (0.045)	0.175	[−0.027, 0.149]
Direct	0.045 (0.046)	0.325	[−0.045, 0.135]
Indirect	**0.016 (0.007)**	**0.024**	[0.002, 0.030]
Daily discrimination T1 → family strain T2 → metabolic risk T2			
Total	0.047 (0.034)	0.166	[−0.019, 0.112]
Direct	0.053 (0.035)	0.135	[−0.016, 0.122]
Indirect	−0.006 (0.006)	0.343	[−0.019, 0.006]
Family strain T2 → negative affect T2 → health appraisal T3			
Total	0.072 (0.039)	0.064	[−0.004, 0.148]
Direct	0.056 (0.039)	0.157	[−0.021, 0.133]
Indirect	**0.016 (0.008)**	**0.048**	[0.000, 0.032]
Family strain T2 → metabolic risk T2 → metabolic conditions T3			
Total	**0.086 (0.044)**	**0.047**	[0.001, 0.158]
Direct	**0.094 (0.042)**	**0.027**	[0.011, 0.177]
Indirect	−0.007 (0.008)	0.329	[−0.023, 0.005]
Daily discrimination T1 → health appraisal T3			
Total	**0.083 (0.038)**	**0.029**	[0.009, 0.158]
Direct	0.064 (0.039)	0.105	[−0.013, 0.141]
Indirect via family strain T2	0.009 (0.007)	0.177	[−0.004, 0.023]
Indirect via negative affect T2	0.008 (0.008)	0.335	[−0.008, 0.023]
Indirect via family strain T2, negative affect T2	0.003 (0.001)	0.059	[0.000, 0.006]
Total indirect	**0.020 (0.010)**	**0.039**	[0.001, 0.038]
Daily discrimination T1 → metabolic conditions T3			
Total	0.33 (0.039)	0.395	[−0.043, 0.110]
Direct	0.008 (0.041)	0.844	[−0.073, 0.089]
Indirect via family strain T2	**0.016 (0.007)**	**0.034**	[0.001, 0.030]
Indirect via metabolic risk T2	0.011 (0.007)	0.147	[−0.004, 0.025]
Indirect via family strain T2, metabolic risk T2	−0.001 (0.001)	0.345	[−0.004, 0.001]
Total indirect	**0.025 (0.010)**	**0.011**	[0.006, 0.045]

Note: T1 = Time 1/MIDUS 1; T2 = Time 2/MIDUS 2; T3 = Time 3/MIDUS 3; CI = confidence interval. Significant pathways ($p < 0.05$) indicated in bold.

However, we found support for family strain as an indirect pathway, failing to find a significant direct association between discrimination and negative affect 10 years later. Prior research supports the ordering of these effects from broader social and contextual factors, and larger systems, to individual well-being via the smaller social systems of family units and dyadic relationships (McNeil Smith et al., 2019; Priest et al., 2020). Moreover, whereas the present study focussed on the distressing impacts of discrimination on family relationship quality, prior research has also explored family support as a buffer against the adverse effects of discrimination (Gerrard et al., 2018; McNeil Smith et al., 2019). Thus, the dynamic interplay between discriminatory experiences, family processes, and mental health should be explored more, and include a focus on both family strain and support. This line of research will also benefit from additional waves of data: our model was advantageous in testing pathways over a 20-year span, but was limited by the three MIDUS waves. Thus, our ability to test temporal links between our mediating variables is hampered; this project does not establish causality.

We did not find support for links between discrimination and lipid metabolism nor cardiometabolic morbidity. This likely indicates there are additional, yet unknown, mediators conveying the effects of discrimination on ageing cardiometabolic health. It is notable that the

present sample includes almost 800 adults without a cardiometabolic condition at baseline. It is possible that, in a sample already diagnosed with a cardiometabolic disease, discriminatory experiences accelerate ageing via the distress mediators tested here. Further, previous research on the topic has noted that a latency period may exist between the experience of discrimination and its impact on health (Pavalko et al., 2003). The current study examined the effects of discrimination after 10 and 20 years, but it is possible that the effects of discrimination are more impactful over a longer period of time or at an earlier stage of life.

Further, discrimination has been associated with a variety of health behaviours, including diet (Brodish et al., 2011) and seeking medical care (Blanchard & Lurie, 2004). Though we included several health behaviours as control variables in the present model, it may be that health behaviours in fact serve as an additional mediator, linking discrimination and cardiometabolic health via worse self-care. Health behaviours may also serve as a moderator in the proposed model, amplifying the effects of negative affect on later health appraisal via impaired health care access, decreased exercise, or worse sleep, for example, In sum, while discrimination may not be significantly associated with predictors of cardiometabolic disease, they may affect the degree of success in the management of these diseases.

4.1 | Clinical implications

The results of this study may implicate important areas of prevention and intervention aimed at mitigating the effects of discrimination on health. Primary care, for example, is primed for detecting risk factors through preventative screening before they become disabling. Screening in primary care for depression is recommended (Siu et al., 2016) and would capture symptoms of negative affect, such as sadness, hopelessness, or worthlessness. However, the results of the present model-testing indicate that primary care clinicians may be able to intervene earlier in this stress-disease process if discrimination was also assessed regularly. Initiating regular assessment of discrimination in primary care would necessitate physician training specific to addressing this social determinant of health, and building safety among disadvantaged patient populations.

Family relationship quality may serve as an additional area amenable to targeted interventions in order to disrupt the discrimination-disease pathway. As found in prior research (Doyle & Molix, 2014; Kwon, 2020; Priest et al., 2020) and supported in the present study, greater experiences of discrimination are associated with greater family strain. This is a direct effect, but also means that discrimination's effects cascade into the support systems that surround healthcare. Thus, family-based interventions aimed at reducing conflict and enhancing cohesion may serve to interrupt the longitudinal effects of discrimination on negative affect and worse health. It may be possible to leverage the involvement of family members in primary care while addressing discrimination, as described above, or to expand individually-oriented treatment aimed at modulating discrimination-related distress to include family (Anderson et al., 2019).

While intervention is proposed in our discussion of the current results, this is an area of the literature that needs more focus (McNeil Smith et al., 2019; Priest et al., 2020). This study invites us to ask the question about whether discrimination also impacts how families support cardiometabolic disease self-management. Finding effective methods of intervening in the quality of our closest relationships, and those impacted daily by experiences of discrimination, is critical given the pathways outlined in this project. Further, as the populations at greatest risk of experiencing discrimination are likely at greatest risk of cardiometabolic health disparities, testing interventions to moderate the negative impacts of mistreatment on health is crucial.

4.2 | Limitations & future research

Though this project reflects a meaningful advance in the study of stress pathways linking discrimination and ageing cardiometabolic health over 20 years, there are limitations of note for interpreting the findings. First, though discrimination is a widely reported experience (Boutwell et al., 2017), the focus of the current analysis on discrimination, broadly, without considering specific types of discrimination limits our ability to understand whether the present pathways differ among racial/ethnic groups or for sexual minorities, for example, Lastly, the present measure of discrimination did not assess for the chronicity of discrimination, a variable that may attenuate the impacts of discrimination on health. It may be that participants' consistent experiences of high levels of oppression over several years has a greater effect on the quality of their relationships and their mood, as compared to time-limited discrimination. Each of these areas reflects next steps that discrimination-health research should take.

Related, the present sample was mostly White and highly educated. Though MIDUS is cutting-edge, it is notably limited in its core samples' diversity. The present use of the Biomarker Project limits the sample's diversity further, as these participants undoubtedly had greater economic resources tied to their ability to participate in this intensive lab-based data collection (Woods et al., 2020). While we have tested age, sex, and income as control variables, there are undoubtedly additional confounding variables that may be important to consider in future theoretical iterations of the present model. These sample limitations may impact our ability to generalise implications – research or clinical – to samples that are more racially or socioeconomically diverse, and who may be more adversely impacted by discrimination. Further, there may be unique mechanisms by which discrimination impacts the health of racial minority groups (recently examined using cross-sectional MIDUS data of African American participants (Priest et al., 2020)), including variations in the pathways tested in the present project.

Additionally, the present sample was used to test, in part, pathways to cardiometabolic disease aetiology over 2 decades of adulthood. As such, the sample reported zero cardiometabolic conditions at baseline. However, as a result, we may have included a healthiest

subsample of MIDUS participants who were not experiencing heart conditions, hypertension, nor diabetes by midlife. Thus, our nonsignificant discrimination-metabolic lipids pathway may reflect a type II error, in that persons at greatest risk of health disparities tied to discrimination were excluded. Additionally, as we included participants who completed three MIDUS waves, we may, have excluded participants whose cardiometabolic health fared worse, and thus were unable to complete the project due to severe disability or death. Incorporating cardiometabolic morbidity, disease timing, as well as mortality in future model-testing will be key.

We are also aware of limitations tied to measures used in the present study, including self-reported cardiometabolic diagnoses. First, relying on participant self-report may obscure those who otherwise meet diagnostic criteria but lack access to healthcare. Second, we are unable to assess condition severity. It will be important for future research to include medical record data. Further, the present analyses utilise high-risk cutoff scores for lipid/fat metabolism established by Gruenewald et al. (2012). Though this decision was intentional, in order to model the greatest risk for problematic lipid metabolism, these cutoffs represent a higher threshold for determining risk than had we used established clinical cutoffs. As such, this may reflect an alternate test for future research in exploring discrimination-disease pathways, as well as meaningful clinical outcomes amenable to intervention.

4.3 | Conclusion

Perceived discrimination is a significant contextual stressor, with important implications for worse ageing health outcomes. The present study is among the first to test specific biopsychosocial stress pathways linking daily experiences of discrimination to health over the course of 20 years. As such, it provides important evidence of the relational and affective mechanisms tying daily discrimination to disease. Increased stress in families and greater negative affect reflect specific distress reactions in the face of denigration. Ultimately, the goal would be to mitigate these pathways through addressing the effects of discrimination early on, intervening in the quality of family relationships, and minimising stress reactivity. Further research is needed to substantiate the present pathways, and inform intervention efforts for ageing adults, though the present project takes a meaningful step in that direction.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to report.

DATA AVAILABILITY STATEMENT

MIDUS data are available via the Inter-university Consortium for Political and Social Research at ICPSR.umich.edu.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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