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Mechanisms Linking Childhood Harsh Fathering to Adult Sons' Health

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Objective: Fathering is now recognized as influencing children's health, yet the physiological and behavioral pathways linking harsh fathering to men's health—particularly in the context of intergenerational fathering—remain less understood. This study examined the long-term effects of harsh parenting by Generation 1 (G1) fathers during Generation 2 (G2) men's childhood on physical and mental health problems in G2's adulthood, focusing on three potential processes: (a) G2 men's physiological stress regulation reflected in diurnal cortisol patterns, (b) the quality of the G2 men–G3 child relationship, and (c) the sequential mediation of these two processes. ***Method:*** Data from three waves of the Midlife Development in the U.S. survey (MIDUS) of 265 G2 men were analyzed using latent growth modeling and structural equation modeling. ***Results:*** Results revealed that G1's harsh fathering in childhood significantly increased the risk of G2 men's physical health problems in middle and old adulthood through dysregulated diurnal cortisol patterns, specifically flattened diurnal slopes in early to mid-adulthood, even while controlling for covariates. Neither the direct impact of harsh fathering nor the mediating mechanisms through the G2–G3 relationship were significant. ***Conclusions:*** The present findings suggest that the effects of harsh fathering during childhood on adult sons' health are long-lasting, and the stress regulation system may serve as a key underlying mechanism in this relationship. This study highlights the need for evidence-based fatherhood programs aimed at preventing harsh parenting practices.

Public Significance Statement
Childhood harsh fathering increases sons' physical health problems in middle and old adulthood by disrupting daily cortisol rhythms during early to mid-adulthood. These findings highlight the lasting impact of harsh fathering, with disrupted stress regulation as a key mechanism.

Keywords: harsh fathering, stress regulation, diurnal cortisol patterns, father–child relationship, men's health

Studies suggest that fathering may have long-term effects on their offspring's health in adulthood (Diggs et al., 2017; Schoppe-Sullivan & Fagan, 2020), as well as their experiences as parents (Jessee & Adamsons, 2018). Understanding how childhood experiences with fathers influence adult health is critical for identifying long-term developmental consequences of harsh parenting and for providing important insights into how childhood adversity contributes to intergenerational patterns of risk. However, despite growing evidence linking harsh fathering to negative health outcomes in adulthood, the mechanisms underlying this association remain poorly understood. The present study aimed to examine potential mechanisms underlying the long-term effects of exposure to Generation 1 (G1) fathers' harsh fathering in childhood to Generation 2 (G2) men's health outcomes in middle and old adulthood

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The authors declare no conflicts of interest.

The study data from the Midlife Development in the U.S. (MIDUS) project and related documentation are available for download at https://www.icpsr.umich.edu/. The analysis code used in this study is not publicly available.

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(aged 43–70 years). Specifically, three processes were examined: (a) through physiological stress regulation during G2 men's early to mid-adulthood (aged 34–60 years), (b) through the relationship quality between G2 men and their children (Generation 3 [G3]) during G2 men's middle and old adulthood, and (c) through a sequential pathway involving G2 men's poor stress regulation followed by lower relationship quality with their G3 children.

Exposure to Harsh Fathering in Childhood and Adult Men's Health

A large body of research suggests that negative parenting experiences are one of the most significant determinants of adult health (Chen et al., 2017). In particular, harsh parenting—defined as "hostile, coercive, and inappropriately controlling" (Rueger et al., 2011, p. 8) behaviors involving "emotional and physical maltreatment" (Naranjo, 2009, p. iii), has been linked to enduring negative effects on physical and psychological health. Both prospective and retrospective studies have consistently demonstrated that individuals exposed to harsh parenting during childhood report a higher number of chronic conditions and poorer self-rated health (Schafer et al., 2014); higher body mass index (Lei et al., 2021); and increased somatic complaints, anxiety, depression, and borderline personality disorder (Allen, 2008) in adulthood. Children who experienced paternal aggression during childhood (e.g., hostility, angry coerciveness) were also found to exhibit increased alcohol use during emerging adulthood (Diggs et al., 2017).

Notably, harsh parenting is not uncommon, with parents responsible for 76% of child maltreatment cases, making them by far the primary perpetrators, and fathers involved in 44% of cases (U.S. Department of Health and Human Services et al., 2025). Data from the Midlife Development in the U.S. (MIDUS) study indicated that about 25% of adults reported emotional maltreatment from their mothers and 28% from their fathers during childhood, as well as 7% and 9% reported physical maltreatment from their mothers and fathers, respectively (Schafer et al., 2014). Studies that include parenting by both mothers and fathers have shown that harsh fathering exerts unique or additive detrimental effects on offspring's outcomes (Simmons et al., 2018), highlighting the distinct and significant role of fathering. Moreover, sons are more frequently reported as victims of harsh parenting by fathers (Wang et al., 2018). Growing evidence indicates that the effects of parenting on offspring may be stronger in same-sex dyads (i.e., father–son or mother–daughter) than in opposite-sex dyads (i.e., father–daughter or mother–son; Xu et al., 2018), suggesting that harsh fathering is likely to have a more pronounced negative impact on sons than on daughters. For instance, a study of Chinese children showed that fathers' harsh parenting had a stronger effect on sons' aggression than on daughters' (Chang et al., 2003). Similarly, another study showed that father–son conflict was associated with diminished social skills and increased behavioral problems in boys, whereas mother–daughter closeness was linked to enhanced social competence and reduced behavioral problems in girls (Xu et al., 2018). This may be because of the similarity effect, where children are more likely to identify with and model the behaviors, attitudes, and emotional responses of a same-gender parent (Matetovici et al., 2025; Xu et al., 2018). Although these findings highlight the importance of considering gender dynamics in parenting research, the long-term consequences of harsh fathering, particularly its enduring effects on sons' adjustment, remain insufficiently explored. In particular, little is known about how childhood exposure to harsh fathering shapes men's health in middle and old adulthood, including chronic health conditions.

Processes Linking Harsh Fathering in Childhood to Adult Men's Health

One of the key processes linking early caregiving experiences to long-term health is the stress regulation system. Several theoretical models, such as the biological embedding of childhood adversity model (Miller et al., 2011) and the risky family model (Repetti et al., 2002), propose that early adverse caregiving disrupts the development of the hypothalamic–pituitary–adrenal (HPA) axis, leading to dysregulated stress responses that persist into adulthood. It has been well established that the HPA axis is especially vulnerable to early caregiving environments and has long-term implications for individuals' physical and mental health (Stroud et al., 2019). Stressors activate the HPA axis, triggering the release of hypothalamic corticotropin-releasing hormone (CRH) and initiating the endocrine response to stress (e.g., the release of adrenocorticotropic hormone [ACTH] and subsequent release of glucocorticoids, such as cortisol in humans; Gershon et al., 2013).

Beyond its role in acute stress responses, the HPA axis is also involved in regulating stress response by controlling diurnal patterns of cortisol secretion. A well-regulated HPA axis is characterized by a robust circadian rhythm of cortisol secretion—high upon waking, peaking approximately 30 min after waking, and declining throughout the day and reaching its lowest point around bedtime (Adam et al., 2017). In contrast, a dysregulated stress system is reflected in flatter or blunted cortisol patterns with lower morning and/or elevated evening levels, which are commonly observed among individuals who have experienced chronic stress in early years, such as adverse caregiving environments (Adam et al., 2017). Although the short-term activation of the HPA axis can be adaptive and facilitates coping with immediate challenges, chronic stress and prolonged release of cortisol can lead to dysregulation of the HPA axis function (Gershon et al., 2013). Limited evidence has shown that fathering experienced in childhood exerts long-term effects on the HPA axis, as seen in both cortisol reactivity to acute stress (Mills-Koonce et al., 2011) and diurnal patterns of cortisol (Choi et al., 2021). For example, a long-term prospective multigenerational study in the United States showed that higher levels of positive father involvement during childhood led to a well-regulated diurnal cortisol pattern in young adulthood (i.e., a steeper slope characterized by increased intercept and greater reduction throughout the day; Choi et al., 2021).

The dysregulated HPA axis can contribute to various health problems, given its broad regulatory functions. Cortisol, for instance, modulates immune activity by signaling immune cells (e.g., monocytes and macrophages) to suppress inflammation (Chen et al., 2017). Individuals exposed to repeated stress often show diminished responses to these anti-inflammatory signals, along with elevated levels of inflammatory markers (Miller et al., 2011). Cortisol also influences other biological systems, including cardiovascular, metabolic, neurobiological, and reproductive systems, all of which have significant implications for long-term

health (Sapolsky et al., 2000). Indeed, growing evidence suggests that a flatter diurnal cortisol slope is associated with a range of adverse health outcomes (e.g., poor immune and inflammatory outcomes, cancer, depressive symptoms or diagnosis of depression, and obesity; for a review, see Adam et al., 2017). However, no study has fully addressed the indirect pathways linking harsh fathering in childhood to adult men's health through diurnal cortisol slopes.

G1's negative parenting and aggressive behaviors during G2's childhood have also been linked to G2's interpersonal difficulties (Cui et al., 2010), which may, in turn, contribute to G2's poor health outcomes across the lifespan (Cohen, 2004). Chronic childhood stress caused by harsh parenting can alter the response tendencies of the brain's corticolimbic circuitry toward emotional stimuli, leading to heightened threat vigilance, negative beliefs about others, interpersonal conflict, and subsequent difficulties in forming and maintaining social relationships (Miller et al., 2011). The risky family model also explains that harsh family climates undermine children's social competence and emotion processing, potentially increasing the risk of later interpersonal difficulties (Repetti et al., 2002).

Among the G2 men's close relationships, those with their own children may serve as a particularly important link in understanding the developmental processes from G1's harsh fathering to G2 men's health outcomes. A large body of research has shown that G1's negative parenting leads to G2's similarly negative parenting practices and poor parent–child relationships when G2 becomes a parent (Belsky et al., 2005). Multiple theoretical models, including social learning theory (Capaldi et al., 2003; Cui et al., 2010), social information processing models, and attachment theory (Finzi-Dottan & Harel, 2014), have suggested behavioral and cognitive mechanisms through which exposure to aggressive and harsh parents contributes to the adoption of ineffective parenting practices and poor relationships with children in the next generation. Using prospective data on three generations, Capaldi et al. (2003) found that G1's poor parenting, assessed when G2 men were aged 9–12 years (e.g., harsh and inconsistent discipline in both parents), predicted G2 men's poor fathering (e.g., harsh discipline, poor discipline) when their G3 children were 16–30 months old. This finding suggests that G1's parenting during G2 men's childhood can lay an important foundation for their ability to establish positive and healthy relationships with their own children. As social relationships are among the most robust predictors of health and mortality (Cohen, 2004), fatherhood, in particular, is increasingly recognized as having significant implications for men's physical and mental health, beyond its impact on child outcomes (Palkovitz, 2002; Schoppe-Sullivan & Fagan, 2020). Fathers tend to view emotional and cognitive bonds with their children as central to fatherhood (Palkovitz, 2002), and the quality of these relationships has emerged as key in understanding the health impacts of parenthood (Schoppe-Sullivan & Fagan, 2020).

Most research on intergenerational continuities in parenting has focused on socioemotional and cognitive factors (e.g., G2 parents' social isolation, social information processing patterns, mental illness; Berlin et al., 2011; Capaldi et al., 2003), resulting in a limited understanding of physiological mechanisms (for a review, see Alink et al., 2019). Several animal studies suggest that the effects of fathering on sons' stress regulation may contribute to multigenerational continuity in fathering (for a review, see Feldman et al., 2019). In humans, the neurophysiological model of intergenerational continuities in harsh parenting posits that early exposure to child abuse or parental aggression may disrupt stress regulation systems, which increases the likelihood of negative or harsh parenting practices in the next generation (Alink et al., 2019). Similarly, Margolin et al.'s (2016) work suggests how childhood exposure to family aggression alters physiological regulatory processes, which are linked to current stress response during interpersonal conflict and aggressive behavior toward intimate partners or children. Taken together, these studies suggest a potential double mediation pathway: G1's harsh fathering may lead to dysregulated cortisol functioning in G2 men, which in turn affects the quality of G2 men's relationship with their G3 child and ultimately impacts G2 men's health.

The Present Study

The present study examined the long-term effects of G1's harsh fathering during G2 men's childhood on their health in middle and old adulthood, using data from the Midlife Development in the U.S. (MIDUS), a large national survey of adults. The G2 men's health in middle and old adulthood was represented by physical and mental health problems. We specifically examined the following mediating processes through (a) G2 men's stress regulation, represented by diurnal cortisol patterns during G2 men's early to mid-adulthood (paths *b* and *c* in Figure 1), (b) the relationship quality between G2 men and their G3 children during G2 men's middle and old adulthood (paths *d* and *e* in Figure 1), and (c) a sequential mediating pathway in which the negative effects of G1's harsh fathering on G2 men's health are transmitted through G2 men's poor physiological stress regulation and subsequently low-quality relationship with their G3 children (paths *b*, *f*, and *e* in Figure 1). This effort can inform effective prevention and intervention strategies to reduce harsh fathering, promote positive father–child relationships, and ultimately improve men's health and adjustment.

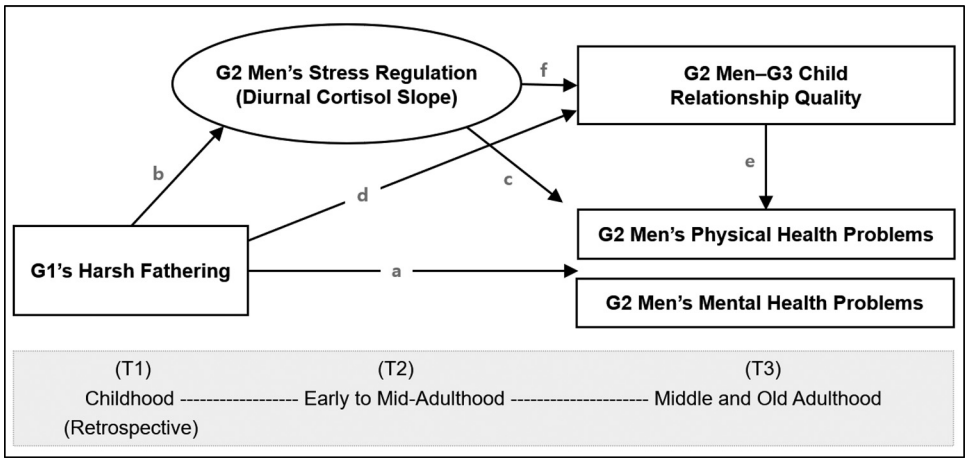
Method

Data and Participants

Data were drawn from the MIDUS, a national multidisciplinary survey of adults, designed to investigate factors that may contribute to age-related variations in individuals' health and well-being (https://www.midus.wisc.edu). The participants were recruited from a random-digit-dialing approach and were English-speaking noninstitutionalized adults aged 25–74. The first assessment (Time 1 [T1]) was completed by 7,108 adults between 1995 and 1996, and the participants were followed for the second assessment from 2004 to 2006 (Time 2 [T2], $N = 4{,}963$) and the third assessment from 2013 to 2014 (Time 3 [T3], $N = 3{,}294$). A randomly selected subsample of T2 ($n = 2{,}022$) participated in the National Study of Daily Experiences, a daily diary study of daily stress for 8 days, and provided saliva 4 times a day on 4 of the 8 assessment days (Ryff & Almeida, 2017). At T1, participants were on average 46.38 years old ($SD = 12.98$, range = 20–75), 65.69% of them were married, and 90.67% identified as White. MIDUS data and related documents can be downloaded from https://www.icpsr.umich.edu/.

Figure 1

Effects of G1's Harsh Fathering on G2 Men's Health Problems in Adulthood Through G2 Men's Stress Regulation and Relationship With G3 Children.



Note. G1 = Generation 1; G2 = Generation 2; G3 = Generation 3.

Of the full MIDUS sample (N = 7,288, including newly recruited at T2), the analytic sample was restricted to male participants (n = 3,440) who met the following inclusion criteria: (a) aged 60 or younger at T2 (N = 1,543), given that HPA axis functioning and its association with health may vary with age (Ice et al., 2004); (b) reported having at least one biological, adopted, step-, or foster children at any wave (T1–T3; N = 1,329); and (c) provided at least one saliva sample at T2 (n = 371). In addition, to ensure the independence of the respondents in the analytic sample, siblings of the main respondents and one of each twin pair were excluded, resulting in a study sample of 265 men.

At T2, on average, the men were 49.42 years old (SD = 7.06) and had 2.54 children (SD = 1.51), 85.28% were married, and the men's youngest child was, on average, 17.45 years old (SD = 9.14). At T3, the men were 58.68 years old on average (SD = 7.09), and 91.06% were White. The median level of education for men was completion of a 2-year college or vocational program (associate degree). Ethical approval was exempted by the Institutional Review Board at Yonsei University, as this study involved secondary data analysis (7001988-202210-HR-1729-01E).

Measures

G2 Men's Exposure to Harsh Fathering by G1

As noted earlier, the present study defines harsh parenting as "hostile, coercive, and inappropriately controlling" (Rueger et al., 2011, p. 8) parenting behavior and "emotional and physical maltreatment of children" (Naranjo, 2009, p. iii). G1's harsh fathering during G2 men's childhood was measured using six retrospective items assessed at T1. G2 men reported on G1 fathers' overly strict discipline behaviors during their childhood (three items; e.g., How much did he stop you from doing things that other kids at your age were allowed to do?) on a 4-point Likert scale, ranging from 1 (*not at all*) to 4 (*a lot*). G2 men also reported on the frequency of three types of abusive behaviors by their fathers (i.e., emotional, physical, and severe physical abuse; three items). For each type of abusive behavior, several examples were provided: for emotional abuse, "Insulting you or swearing at you," for physical abuse, "Throwing something at you," and for severe physical abuse, "Kicking, biting, or hitting you with a fist." The items were rated on a 4-point Likert scale, ranging from 1 (*never*) to 4 (*often*). The six items showed adequate reliability (Cronbach's α = .77) and were thus averaged to create the G1 harsh fathering score.

G2 Men's Health Problems During Middle and Old Adulthood

G2 men's physical and mental health problems were assessed at T3 using measures of subjective health and the presence of chronic conditions in the past 12 months. Physical health was evaluated using two indicators. First, a single item assessed men's subjective perception of their overall physical health ("In general, would you say your physical health is excellent, very good, good, fair, or poor?"). Responses ranged from 1 (*excellent*) to 5 (*poor*), with high scores indicating greater health problems. Second, chronic physical conditions were assessed by asking whether participants had experienced or received treatment for each of 37 physical conditions (e.g., asthma/bronchitis/emphysema, and stomach trouble). Each condition was coded as 1 if present and 0 if absent. The number of chronic physical conditions was summed, ranging from 0 to 19. These two indicators were standardized and averaged for the analysis. Men's mental health problems were assessed using two indicators. Subjective mental health was assessed using the same single-item format as physical health and rated on a 5-point Likert scale. Chronic mental health conditions were assessed by asking about the presence of two conditions: anxiety/depression and alcohol/drug problems. Scores ranged from 0 to 2. These two indicators were also standardized and averaged for the analysis.

G2 Men's Stress Regulation During Early to Mid-Adulthood: Diurnal Cortisol Slope

G2 men's stress regulation system was represented by diurnal salivary cortisol slopes drawn from the National Study of Daily

Experiences at T2. The men provided four saliva samples daily for four consecutive days (at waking, 30 min after waking, before lunch, and before bed). They received a home saliva collection kit and were instructed not to eat, drink, brush their teeth, or have any caffeinated products before collecting saliva. They also completed short phone interviews about their daily lives across the assessment days. The saliva samples were mailed to the University of Wisconsin's MIDUS Biological Core. The cortisol concentrations were estimated by the Luminescence immunoassay (IBL, Hamburg, Germany). The intra- and interassay coefficients of variation were lower than 5% (see Ryff & Almeida, 2017 and Almeida et al., 2009 for detailed information on saliva collection and the reliability and validity of this procedure).

Consistent with prior studies (Otto et al., 2018; Piazza et al., 2018), a series of data cleaning for the cortisol values was conducted by excluding (a) cortisol values of 60 nmol/L or higher, (b) the first and second collections of each day if the interval between wake-up time and the first collection exceeded 15 min, (c) the second collection of each day if the time interval between the first and second collection exceeded 60 min, and (d) all four collections of the day if the men slept for less than 4 hr or for 12 hr or more on that day. As a result, the present analysis included 858, 810, 1,000, and 999 cortisol samples (the total number of cortisol samples = 3,667) at waking, 30 min after waking, before lunch, and before bed, respectively (the total number of cortisol samples = 3,667). The average was 6.38, 6.97, 12.75, and 22.69 hr for each collection time (SD = 1.34, 1.37, 1.65, and 1.69).

The present study considered both statistical and conceptual factors in selecting an analytic approach for cortisol data with a nested structure (collection-, day-, and individual-level). First, a series of analyses was conducted to examine the existence of significant day-to-day variation. Internal consistency reliabilities of cortisol levels collected at the same timeframe across 4 days ranged from .62 to .78, and repeated measures analyses of variance (ANOVA) showed nonsignificant day-to-day variation. Group size-adjusted intraclass correlation coefficients (ICCs; group mean reliability), calculated using the Spearman-Brown formula (Bliese, 2000), ranged from 0.65 to 0.78 for each collection. In general, intraclass correlation coefficients of 0.7 or higher indicate high within-group similarity and justify the use of aggregated group-level data in analysis (Park & Ko, 2005). Such minimal day-to-day variation suggests that most variance in cortisol levels occurs within a day (across sampling timepoints) or between individuals.

Second, the analytic objective was considered. Studies examining the effects of time-level covariates (e.g., specific contexts during cortisol collection) on cortisol levels often use multilevel modeling (Stoffel et al., 2021), whereas others employ latent growth modeling (LGM), which is particularly effective for modeling intraindividual change and interindividual differences in trajectory parameters (e.g., mean levels and slopes; Choi et al., 2021). The present study aimed to estimate the structural form and interindividual variability of the diurnal cortisol slope, rather than the effects of contextual or day-level predictors. Accordingly, we employed LGM, which also accounts for measurement error by estimating cortisol trajectories as latent variables.

Taken together, cortisol values from the same timeframe were averaged across 4 days and used in the LGM. The day-level covariates were adjusted in the conditional model. Mean cortisol values were 15.61 nmol/L (SD = 6.78), 21.00 nmol/L (SD = 8.97), 7.26 nmol/L (SD = 4.53), and 3.30 nmol/L (SD = 3.47) at waking, 30 min after waking, before lunch, and before bed, respectively, representing a clear circadian rhythm.

Father–Child Relationship Quality Between G2 Men and G3 Children

Father–child relationship quality was measured with a single item reported by the G2 men at T3, "How would you rate your overall relationship with your children these days (including biological, step, or adopted)?" Responses were rated on an 11-point scale, ranging from 0 (*worst possible relationship*) to 10 (*best possible relationship*).

Control Variables

G2 men's age, race, education level, and marital status assessed at T3, as well as G1 mothers' harsh parenting assessed at T1, were included as control variables to adjust for their potential effects on G2 men's health. G2 men's age was represented in years, and their race was coded 1 for White and 0 for non-White. G2 men's education level was included as an ordinal variable (1 [*no school/some grade school*] to 12 [*PhD or other professional degree*]). G2 men's marital status was coded 1 for married and 0 for unmarried. G1 mother's harsh parenting was measured in the same manner as the G1 father's and was controlled for in the analysis, as recommended by many researchers (Fagan et al., 2014). G2 men's reports of their mothers' overly strict discipline and abusive behaviors (six items, Cronbach α = .73) were averaged to create an indicator of G1 mothers' harsh parenting. In addition, as in previous studies (e.g., Choi et al., 2021), three additional T2 variables that may influence diurnal cortisol patterns were included in the model: (a) A dummy variable to indicate whether the cortisol collection day was a weekend (1) or not (0), (b) men's average sleep time across 4 collection days, and (c) men's time spent in vigorous physical activities or exercise averaged across 4 collection days.

Data Analysis

Data preparation and descriptive analyses were conducted in SPSS 26, and the proposed models were tested in Mplus 8 (Muthén & Muthén, 1998–2021). A two-step approach was used: (a) LGM to obtain factor scores for diurnal cortisol slopes as indicators of G2 men's stress regulation system, and (b) structural equation modeling (Figure 1) to test the proposed longitudinal model examining effects of G1's fathering on G2 men's health problems, mediated through G2 men's stress regulation system (represented by the slope factor scores from the first step) and G2 men–G3 children relationship quality.

The model fit was evaluated using chi-square, root-mean-square error of approximation (RMSEA $\leq$ .08), standardized root-mean-square residual (SRMR $\leq$ .08), comparative fit index (CFI $\geq$ .90), and Tucker–Lewis index (TLI $\geq$ .90; Keith, 2015). The significance of the proposed paths was determined using bias-corrected bootstrapping with sample iterations set to 5,000, which is considered a rigorous method for testing the significance of mediating effects when the sampling distribution is asymmetric (Hayes, 2009). The path is considered statistically significant if the bootstrapped 95% confidence interval (CI) does not include zero. The 95% CIs are presented in brackets.

In the first step, LGM was performed to examine diurnal cortisol slopes, with four observed cortisol values from each collection time (i.e., at waking, 30 min after waking, before lunch, and before bed), which were averaged across 4 days. LGM has been widely employed to identify diurnal cortisol slopes (Choi et al., 2021). LGM estimates two latent growth factors: an intercept representing the initial cortisol level upon waking and a slope representing the direction and rate of change throughout the day. These two growth factors are individual-level random parameters, allowing for variation between individuals. Because the typical diurnal cortisol rhythm is not linear (i.e., high levels upon waking, a rise 30–40 min after waking, a rapid decline throughout the day, and the lowest levels at bedtime; Adam et al., 2017), linear spline models have been used (Choi et al., 2021). These models fix the slope factor loadings for the first (i.e., at waking) and the last cortisol sample (i.e., at bedtime) at 0 and 1, respectively, while allowing the loadings for the second and third cortisol samples to be freely estimated. The intercept and slope factors are allowed to covary, as is typically done (Duncan & Duncan, 2009). To account for the non-normal distribution of the cortisol data, maximum likelihood estimation with robust standard errors was employed.

The unconditional linear spline model fit the data well, $\chi^2(3) = 4.467$, $p > .05$, RMSEA = 0.043, SRMR = .056, CFI = 0.984, TLI = 0.969. For the intercept factor, the mean and variance were 15.647 and 24.608, respectively, and both were significant. For the slope factor, the mean and variance were −12.277 and 23.907, respectively, and both were also significant. The negative slope mean indicated a decreasing pattern of cortisol during the day by 12.28 nmol/L. The conditional model that included three covariates (i.e., collection on weekends, the total amount of sleep, and the amount of exercise time) fit the data well, $\chi^2(9) = 10.990$, $p > .05$, RMSEA = 0.029, SRMR = .038, CFI = 0.984, TLI = 0.968. The residual variances of the intercept and slope factors remained significant. The adjusted slope factor scores derived from the conditional model were used in the subsequent analysis.

In the second step, structural equation modeling was conducted to test the proposed model (Figure 1). G1's harsh fathering during G2 men's childhood was included as the independent variable, and G2 men's physical and mental health problems in adulthood were included as the dependent variables. This model included three specific processes: (a) stress regulation, based on factor scores for diurnal cortisol slopes obtained in the first step; (b) the G2 men–G3 child relationship; and (c) a sequential pathway involving both stress regulation and the G2 men–G3 child relationship. Of the control variables, G1's harsh mothering and harsh fathering were allowed to covary because they are likely to be significantly correlated with each other (Diggs et al., 2017). The cortisol slope and the G2 men–G3 child relationship were allowed to covary with the control variables, except those with near zero correlations.

To accommodate missing data, full information maximum likelihood was used, as it is known to provide less biased estimates than other traditional approaches to handling missing data (Enders & Bandalos, 2001). In the present study, missing rates of all study variables were about 10% or lower except for the G2 men–G3 child relationship (19.62%). Although those with data on the G2 men–G3 child relationship reported greater physical and mental health problems than those without ($p < .05$), the two groups did not differ significantly on any other variables. The potential implications of the missing data are discussed further later in the text.

Results

Descriptive Statistics of Study Variables

Table 1 presents the means, standard deviations, and bivariate correlations for all study variables. G1's harsh fathering in G2 men's childhood was positively correlated with G2 men's diurnal cortisol slope, indicating that G1's harsh fathering was associated with G2 men's flatter cortisol slopes. G1's harsh fathering was also positively correlated with G2 men's physical health problems in adulthood. G2 men's diurnal cortisol slope was negatively correlated with the G2 men–G3 child relationship quality and positively correlated with G2 men's physical health problems. That is, better relationship quality between G2 men and G3 child, and fewer physical health problems in G2 men were significantly correlated with steeper diurnal cortisol slopes. Of the control variables, G2 men's race and education were negatively correlated with their physical health problems, indicating that being White and having higher education were linked to fewer physical health problems. Higher education level and being married were also correlated with fewer mental health problems among G2 men.

Table 1
Means, Standard Deviations, and Bivariate Correlations Among the Study Variables (N = 265)

Study variables	1	2	3	4	5	6	7	8	9	*M*	*SD*
1. G1's harsh fathering (1–4, T1)										2.34	0.61
2. G2's stress regulation (diurnal cortisol slope, T2)	.14*									−12.29	3.95
3. G2 men–G3 child relationship quality (0–10, T3)	.00	−.14*								8.65	1.58
4. G2's physical health problems (T3)	.16*	.20**	−.12							0.02	0.88
5. G2's mental health problems (T3)	.12	.04	−.06	.55**						0.02	0.88
6. G2's age (T3)	.12	−.03	.13	.08	−.12					58.68	7.09
7. G2's race (White = 1/non-White = 0, T3)	−.02	−.02	.10	−.13*	.05	.02				0.91	0.29
8. G2's education (1–12, T3)	−.17**	−.15*	.09	−.25**	−.18**	−.02	.01			7.74	2.34
9. G2's marital status (married = 1/unmarried = 0, T3)	.01	.00	.11	−.10	−.15*	.10	.02	.14*		0.83	0.38
10. G1's harsh mothering (1–4, T1)	.39**	.16**	−.01	.06	.07	.01	.04	−.17*	−.01	2.14	0.50

* $p < .05$. ** $p < .01$.

Pathways From the G1's Harsh Fathering to G2 Men's Health Problems in Adulthood

The proposed model fit the data well, $\chi^2(4) = 0.474$, $p > .05$, RMSEA = .000, SRMR = .006, CFI = 1.000, TLI = 1.000. G1's harsh fathering during G2 men's childhood was not directly associated with physical or mental health problems in G2 men's middle and old adulthood, while controlling for the effects of G2 men's age, race, education, marital status, and G1's harsh mothering. However, as shown in Table 2 and Figure 2, G1's harsh fathering during G2 men's childhood was significantly associated with a smaller decline in G2 men's cortisol levels across the day (β = .142 [.021, .261]), suggesting a flatter diurnal slope in their early to mid-adulthood. In addition, a flatter diurnal cortisol slope was significantly associated with greater physical health problems in G2 men's middle and old adulthood (β = .158 [.037, .266]), even after adjusting for the effects of confounders. This mediating path was statistically significant (β = .022 [.001, .048]), indicating that G1 fathers' harsh parenting during G2 men's childhood significantly increased the risk of developing physical health problems decades later, through dysregulated diurnal cortisol patterns. The mediating path via cortisol slope to mental health problems was not significant.

The mediating path via the G2 men–G3 child relationship quality was not significant. Similarly, the sequential mediating path through stress regulation and G2 men–G3 child relationship quality was also not significant. Of the control variables, G2 men's higher education level was significantly associated with fewer physical health problems (β = −.199 [−.313, −.079]), and mental health problems (β = −.142 [−.280, −.010]). G1's harsh mothering was not significantly associated with G2 men's physical and mental health problems in middle and old adulthood.

We conducted a robustness check by including participants aged 60 and older ($N = 463$), and the model fit the data well, $\chi^2(4) = 3.872$, $p > .05$, RMSEA = .000, SRMR = .013, CFI = 1.000, TLI = 1.000. The mediating path from G1's harsh fathering to G2's physical health problems through a flatter diurnal cortisol slope remained significant (β = .019 [.003, .040]).

Conclusions and Discussions

Growing interest in fatherhood has brought greater recognition of the important role of fathers in shaping both children's and their own health and adjustment. However, relatively few studies have examined the underlying mechanisms through which fathering influences long-term outcomes, particularly in adult sons. Drawing on multiple theoretical models, including Miller et al. (2011) biological embedding of childhood adversity model, the present study examined how childhood harsh fathering affects adult men's health through: (a) physiological stress regulation, (b) the G2 men–G3 child relationship, and (c) the sequential mediation. We used three waves of the MIDUS, spanning early to old adulthood in American men.

It was found that G1's harsh fathering during G2 men's childhood did not directly increase G2 men's physical and mental health problems in middle and old adulthood. Instead, the proposed stress regulation process was supported: G2 men's retrospective accounts of G1's harsh fathering significantly increased their risk of physical health problems in middle and old adulthood, through dysregulated diurnal cortisol patterns in early to mid-adulthood (i.e., a flattened cortisol slope or smaller reduction across the day). This association remained significant even after controlling for G1 mothers' harsh parenting and other potential confounders. Our findings highlight the need for rigorous scientific evidence to inform policies and interventions aimed at promoting positive fathering (Schoppe-Sullivan & Fagan, 2020), as preventing harsh fathering is as important as addressing its consequences. Furthermore, systematic screening for adverse childhood experiences, including harsh parenting by each parent, should be embedded within health care and educational systems to enable early identification and support.

It is also important to recognize that effective intervention efforts should address a broad spectrum of harsh parenting

Table 2

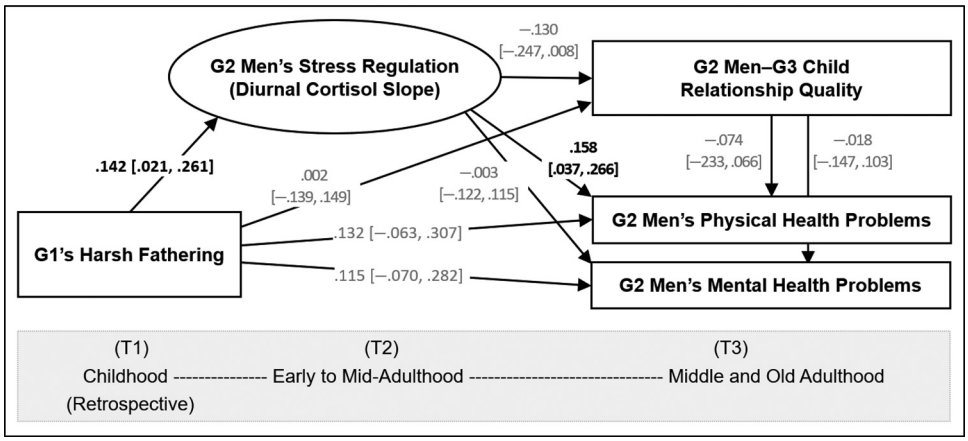
The Effects of the Experience of G1's Harsh Fathering on G2 Men's Health Problems in Adulthood (N = 265)

T1 (retrospective reports on childhood)	T2 (early to mid-adulthood)	T3 (middle and old adulthood)		Standardized estimates	95% bootstrap confidence intervals
G1's HF →			G2's PHPs	.132	[−.063, .307]
G1's HF →			G2's MHPs	.115	[−.070, .282]
	G2's SR →		G2's PHPs	**.158**	**[.037, .266]**
	G2's SR →		G2's MHPs	−.003	[−.122, .115]
		G2–G3 RQ →	G2's PHPs	−.074	[−.233, .066]
		G2–G3 RQ →	G2's MHPs	−.018	[−.147, .103]
G1's HF →		G2–G3 RQ		.002	[−.139, .149]
	G2's SR →	G2–G3 RQ		−.130	[−.247, .008]
G1's HF →	G2's SR			**.142**	**[.021, .261]**
G1's HF →	G2's SR →		G2's PHPs	**.022**	**[.001, .048]**
G1's HF →	G2's SR →		G2's MHPs	.000	[−.020, .019]
G1's HF →		G2–G3 RQ →	G2's PHPs	.000	[−.022, .015]
G1's HF →		G2–G3 RQ →	G2's MHPs	.000	[−.014, .008]
G1's HF →	G2's SR →	G2–G3 RQ →	G2's PHPs	.001	[−.001, .006]
G1's HF →	G2's SR →	G2–G3 RQ →	G2's MHPs	.000	[−.003, .003]

Note. G = generation; HF = harsh fathering; SR = stress regulation (diurnal cortisol slope); RQ = relationship quality; PHP = physical health problem; MHP = mental health problem. Statistically significant estimates are indicated in bold. Results of the control variables are not shown.

Figure 2

Standardized Results of the Effects of G1's Harsh Fathering on G2 Men's Health Problems in Adulthood Through G2 Men's Stress Regulation and Relationship With G3 Children.



Note. G1 = Generation 1; G2 = Generation 2; G3 = Generation 3. Statistically significant estimates are indicated in black.

practices, including both strict disciplinary behaviors and abusive acts. In this study, harsh fathering was assessed using items that captured these two dimensions. Their significant correlation suggests a shared underlying construct of harsh parenting, supporting the importance of considering these two dimensions together. Although overt child abuse is broadly condemned, a persistent belief holds that strict disciplinary behaviors—such as hostile, coercive, or overly controlling behaviors—are acceptable or even necessary for shaping children's behavior (Higgins, 2025; Petani & Jurina, 2022). This may reflect a misunderstanding between authoritarian and authoritative parenting, as well as the relatively limited public awareness of the negative consequences associated with authoritarian parenting (Sarwar, 2016). The present results highlight the conceptual and empirical overlap between overly strict discipline and abusive parenting behaviors, as well as their shared long-term detrimental effects on child health.

Our finding also underscores the role of the dysregulated stress regulation system as both a consequence of early harsh fathering and a robust predictor of long-term health problems, especially physical health problems, in adult sons (Stroud et al., 2019). Notably, the lack of direct effects of G1's harsh fathering implies that not all individuals exposed to harsh parenting will necessarily develop health problems in later life. Rather, the significant mediating path highlights the stress regulation system as a modifiable mechanism. As Miller et al. (2011, p. 980) argue, childhood stress does not "lock" physiological systems into permanent set points, but instead calibrates their operating ranges, setting the upper and lower bounds for how cells respond to future challenges. These responses can then be further refined by later experiences (Miller et al., 2011). Our findings underscore the importance of developing and implementing evidence-based interventions focused on improving stress regulation for individuals who have experienced harsh fathering.

Findings also showed that the process involving G2 men–G3 child relationship quality in G2's middle and old adulthood was not statistically significant. Multiple theories have posited stress regulation and social relationships as key processes linking adverse childhood experiences to long-term health outcomes (Gershon et al., 2013; Miller et al., 2011; Repetti et al., 2002). Chronic exposure to stress during childhood can disrupt both the body's stress regulation system and the ability to form close relationships, increasing vulnerability to chronic inflammation and long-term health risks (Chung et al., 2009; Miller et al., 2011). However, few empirical studies examined these two processes simultaneously. In the present study, only the pathway through dysregulated stress regulation was significant, suggesting that physiological processes may play a more central role in linking childhood harsh fathering to later physical health problems. This does not imply that G2 men–G3 child relationship quality is not important. Rather, it may reflect the broader and more pervasive impact of the stress regulation system, which influences a wide range of individuals' reactivity to daily stressors (Schlotz et al., 2011). It is also important to note that the relationship quality was assessed when G2 men's youngest child was on average 25.55 years, a developmental stage when father–child dynamics may be less salient in shaping fathers' health. Future research should examine G2 men's earlier parenting years to better understand how their relationship with G3 children may shape both their own and their children's long-term adjustment.

The present findings should be considered within the context of several limitations. First, the mediating effects were only significant for physical health problems. This discrepancy between physical and mental health outcomes may partly reflect differences in measurement variability: Of the 39 chronic conditions assessed, only two pertained to mental health problems, yielding a restricted score range (0–2) and limited variability compared with physical health conditions. Future research should incorporate more comprehensive measures of mental health. Second, despite using three waves of data spanning nearly 20 years, this study used adult participants' retrospective reports of their parents' behavior during childhood. Despite limitations such as recall bias, retrospective

studies are effective for capturing long-term developmental histories and individuals' subjective interpretations of early experiences (Giele & Elder, 1998). Retrospective self-reports of adverse childhood experiences have been widely used in the literature and have yielded consistent associations between adverse childhood experiences and later health outcomes (Anda et al., 2010). Nonetheless, future research would benefit from prospective multiinformant studies to further validate the present findings. Third, this study focused on G2 men who had children, as the primary aim of the study was to examine their adjustment as fathers. However, this may limit the generalizability of the findings to the broader population because individuals exposed to harsh parenting are less likely to get married or maintain stable marital and parental relationships (Covey et al., 2013). Future research should include more diverse samples. Fourth, although the hypothesized direction from G2 men–G3 child relationship to G2 men's health outcomes was grounded in growing evidence on the health implications of fatherhood (Schindler, 2010; Schoppe-Sullivan & Fagan, 2020), the possibility of reverse causality cannot be ruled out. Fathers with health problems may be more likely to experience difficulties in their relationships with their children. Future research is needed to verify the directionality of these associations. Lastly, the missingness of some variables appeared to be nonrandom. To address this, full information maximum likelihood was employed, as it is known to be superior to other conventional approaches even in the presence of nonrandom missingness (Enders & Bandalos, 2001). Although the likelihood of selective missingness was minimal, the findings should be interpreted with caution.

Taken together, this study deepens our understanding of how harsh fathering in childhood can shape long-term health outcomes, primarily through the disrupted stress regulation system. Despite robust evidence linking early adverse experiences to later health (Anda et al., 2010), few studies have empirically examined the processes underlying this association, particularly in relation to men's experiences of fathering across generations. Our findings underscore the critical importance of preventing harsh parenting and enhancing the stress regulation system in those exposed to it. Furthermore, research must go beyond identifying risk to also illuminate pathways of resilience. Identifying protective factors that buffer the impact of early adversity or characterizing individuals who maintain positive health outcomes despite histories of harsh fathering will advance both theory and practice. Although the present study used a composite measure of harsh fathering that incorporated two conceptually and empirically overlapping components, future studies may also benefit from investigating the unique and shared contributions of overly strict discipline and abusive parenting. Moreover, there is a pressing need to examine the potential health benefits of positive fathering behaviors, such as warmth, responsibility, and involvement—dimensions that have only recently gained recognition as central to fatherhood. Future research may also benefit from identifying other potential factors —such as neuroticism—that could link early adversity and health outcomes. By integrating insights into developmental timing, physiological processes, and parenting dynamics, future work can better inform intervention strategies aimed at disrupting the intergenerational transmission of risk. Ultimately, bridging research and practice will require investments in evidence-based fatherhood programs, broader screening for adverse childhood experiences, and expanded access to interventions that foster both physical and biological resilience.

Abstracto

Objetivo: Hoy día se reconoce que la paternidad influye en la salud de los hijos, pero los mecanismos fisiológicos y conductuales que vinculan la paternidad autoritaria con la salud de los hombres —particularmente en el contexto de la transmisión intergeneracional de la paternidad— aún no se comprenden completamente. Este estudio examinó los efectos a largo plazo de la crianza autoritaria ejercida por los padres de la Generación 1 (G1) durante la infancia de los hombres de la Generación 2 (G2) en los problemas de salud física y mental de estos últimos en la edad adulta, centrándose en tres posibles procesos: 1) la regulación fisiológica del estrés en los hombres de la G2, reflejada en los patrones diurnos de cortisol; 2) la calidad de la relación entre los hombres de la G2 y sus hijos de la Generación 3 (G3), y 3) la mediación secuencial de estos dos procesos. ***Métodos:*** Se analizaron los datos de tres oleadas de la encuesta sobre el Desarrollo en la Edad Adulta Intermedia en Estados Unidos (MIDUS, por sus siglas en inglés) de 265 hombres de la generación G2 utilizando modelos de crecimiento latente y modelos de ecuaciones estructurales. ***Resultados:*** Los resultados revelaron que la crianza paterna autoritaria en la infancia de la generación G1 aumentó significativamente el riesgo de problemas de salud física en los hombres de la generación G2 durante la edad adulta media y tardía, a través de patrones de cortisol diurno desregulados, específicamente con pendientes diurnas aplanadas en la edad adulta temprana y media, incluso controlando las covariables. Ni el impacto directo de la crianza paterna autoritaria ni los mecanismos mediadores a través de la relación G2-G3 resultaron significativos. ***Conclusiones:*** Los presentes hallazgos sugieren que los efectos de una crianza paterna autoritaria durante la infancia en la salud de los hijos adultos son duraderos, y el sistema de regulación del estrés podría ser un mecanismo subyacente clave en esta relación. Este estudio subraya la necesidad de programas de paternidad basados en la evidencia, dirigidos a prevenir las prácticas parentales autoritarias.

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