# Social Determinants of Chronic Pain in the United States

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## Abstract


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Social aspects of the biopsycho*social* framework of pain have been under theorized and under researched. We proposed a theory-driven conceptual framework to expose multilevel intersectional systems of inequity and pathways through which they may shape pain over the life course and across generations. Using directed acyclic graph-informed regression in a nationally representative sample of US adults, we estimated large relative inequities in high-impact chronic pain (HICP) and number of site-specific pains, especially by family income and education. Inequities in low-impact chronic pain and site-specific pains were smaller. In intersectional decomposition analysis, adults in nearly all doubly marginalized positions of race/ethnicity and socioeconomic position (SEP) were estimated to have higher absolute HICP burden than non-Hispanic White adults with high SEP, and lower absolute HICP burden than expected based on the sum of SEP and race/ethnicity disparities alone. Enhanced conceptualization, measurement, and analytic strategies for social determinants of pain may contribute to research and policy strategies towards population health equity.

**Dedication**

To my family, whose love and support are always with me across any physical distance; to my father, who teaches me to dream big, and does not measure efforts provide me with tools and opportunities to pursue those objectives; to my mother, who raised me to see and care about all people as myself; to my brother, whose treasured friendship and shared memories warm my heart at all times; to my maternal grandmother, who teaches me "jogo de cintura" and grace to enjoy life's journey and overcome whatever barriers may come along; to my paternal grandparents, who inspired me to be curious and do my best given the circumstances. To my partner, whose steady encouragement and understanding push me to grow while cherishing what I have already accomplished. To my friends back home, in Seattle, and around the world, whose connections are a continuous source of strength and joy. To my teachers at Colégio Santa Cruz, who taught me the value of critical thinking and writing. To my dental school mentors at the University of São Paulo, who introduced me to the challenges and rewards of diagnosing and treating pain, as well as the world of scientific research, and international collaboration. To my University of Minnesota mentors, clinic staff, and patients, who nurtured me like family, inspired me to follow an academic career, and opened countless doors to my professional development. Finally, to my University of Washington mentors, instructors, TA and RA supervisors, and Summer Institute team. I am deeply grateful for your time, feedback, and role modelling. I will continue to try mirroring some of the best features I have seen in your work, such as empathy, clarity, critical thinking, passion, humility, flexibility, and persistence. You have inspired me to do well-grounded work and to continue building it towards consequential epidemiology.

**Acknowledgements & positionality**

I acknowledge with gratitude that this work was conducted in the traditional territories of the Coast Salish peoples of the Duwamish, Suquamish, Stillaguamish, Tulalip, and Muckleshoot nations. I recognize them as original and current stewards of this land, air, and water; whose culture and livelihood resists ongoing colonization and erasure.

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I was born in Tupi and Guarani Mbya land, currently called São Paulo, Brazil, with ancestry who were European colonizers and displaced immigrants. I am currently able-bodied, and have benefited from undeserved social, educational, and economic privileges because my social positions. While I have experienced some degree of relative disadvantage based on language, citizenship, and ethnicity as a primarily Portuguese-speaking alien Latina in the United States, my work is limited by a lack of personal insight into the lived experience of chronic pain, and the perspectives of most of the intersectional social groups that I mention in my presentation. This work is primarily aligned with a critical theory lens. I seek to understand how complex power dynamics contribute to the health of populations, so that my work may enable interventions that promote equity and social justice.

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## 1. General introduction

Chronic pain is a major public health problem in the United States (US) with estimated societal costs of $560 to $635 billion annually (Gaskin et al., 2012). Furthermore, there is evidence that the burden of chronic pain is disproportionally carried by women, persons with lower socioeconomic position (SEP), racial/ethnic minorities, and rural residents (Dahlhamer et al., 2018). There are large chronic pain disparities by individual-level SEP, with strong gradients by income, education, employment, and insurance status. For instance, prevalence of high-impact chronic pain (HICP, i.e., pain that interferes with life or work activities on most days or every day for 6 months or longer) of US adults living in poverty is 4.5 times that of those with family income ≥400% the federal poverty level and more than twice the national average (Dahlhamer et al., 2018).

Despite pain being widely accepted as a biopsycho*social* experience and these well-described social disparities, social factors are often disregarded as non-modifiable or included in research as nuisance adjustment variable. Nevertheless, race/ethnicity and SEP constitute some of the most prominent social features that shape population health and health inequities in the US. Racial and socioeconomic pain inequities have been widely reported, however, there is lingering inaccuracy in theory, methods, and interpretation of findings (Meghani and Chittams, 2015). Informed by critical theory-based frameworks of a variety of disciplines (Link and Phelan, 1995; Krieger, 1994; McEwen and Stellar, 1993; Kuh et al., 2003; Crenshaw, 1989; McCartney, 2019; Ford and Airhihenbuwa, 2010), we undertook theoretical, methodological, and empirical work to investigate social determinants of chronic pain.

Specifically, we aimed to: 1) Propose a multilevel conceptual framework outlining social determinants of chronic pain and pathways of embodiment through which they may shape individual biology and psychology; 2) Construct a measure of relational social class for the National Health Interview Survey (2016-2017) and investigate whether there are non-gradient patterns in the distribution of HICP by social class.; 3) Use directed acyclic graph (DAG)-informed regression analysis to estimate the associations of educational attainment, family income, social class, family savings/investments and home tenure status with HICP, low-impact chronic pain (LICP), and site-specific pain outcomes; 4) Use intersectional decomposition analysis to quantify the joint disparity in HICP and its components, at intersections of race/ethnicity and SEP.

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## 2. Multilevel social determinants of pain and pain inequities

### 2.1. Introduction

Social context has been shown to shape the variability in pain responses to injuries for over a hundred years (Beecher, 1956). Moreover, since the advent of the biopsychosocial model of illness (Engel, 1977), most pain science and care has acknowledged that social factors play an integral part in pain experience and expression (Gatchel et al., 2007; Loeser and Melzack, 1999; Waddell et al., 1984). During the last two decades, there has been growing evidence of the importance of social determinants and consequences of pain, as well as awareness of large social inequities in the prevalence and impact of pain. Nevertheless, pain diagnosis, management, research, and education continue to be overwhelmingly directed to intrapersonal aspects of pain, that is, biological and psychological determinants (Blyth et al., 2007).

Part of this disconnect may be attributable to the lack of clarity of current conceptualizations of pain in systematically addressing relationships between social factors at multiple levels of organization and how they integrate with biological and psychological factors to influence pain. (Blyth et al., 2007; Edwards et al., 2016). Shared observations of pain inequities do not seem to have translated into a shared understanding of causal pathways and strategies for intervention among those in the field; theoretical elaboration is needed to advance the field towards equity (Krieger, 2001).

Extant evidence of social aspects of pain has emphasized individual social characteristics (e.g., social roles and identities) and interpersonal dynamics on pain onset, impact, and prognosis (Vervoort et al., 2018). We focus this section primarily on the structural role of societal and group-level social determinants in shaping interpersonal and individual

exposures, vulnerability to and consequences of pain, which may be targeted by social equity and policy strategies for population health. Using a critical theory-driven framework based on ecological, intersectional and life course principles, this work aims to provide a scaffold to expand the understanding of the multifactorial etiology of chronic pain and pain inequities, as well as their impact on individuals and society. Social determinants of pain have been severely underexplored, especially at group and societal levels. Improved clarity concerning multilevel factors that may shape pain and its sequelae may allow for clearer causal hypothesis generation and testing, maximizing the potential range and effectiveness of interventions to reduce the burden of pain and its social consequences. Our conceptual framework seeks to support research and practice to overcome the uncritical depiction of pain inequities by sociodemographic groups as if they were naturally occurring individual or interpersonal differences due to static traits, in disregard of historical and systemic power relations that drive social stratification (Bowleg, 2019).

First, we presented the main theories that guided the development of the conceptual framework (2.1.1.), accompanied by figures that explain each of the corresponding conceptual framework elements (Figures 1-6). These were followed by the explanation of the framework itself (2.2.), including an illustrative causal pathway (2.2.1.), and the figure of the full conceptual framework (Figure 7). We then reviewed examples of determinants of pain from the literature at each of the levels of organization (2.2.2—2.2.4.), with an emphasis on upstream factors, and concluded with a discussion of the main points.

### 2.1.1. Foundational principles

#### 2.1.1.1. Critical stance

Despite a general lack of explicit statements on epistemological stance (i.e., assumptions about the nature, limitations, and justification of human knowledge (Hathcoat et al., 2017) in public health and biomedical research including pain, the predominant approach to knowledge is implicitly aligned with a (post)positivist position. Positivist epistemologies seek to objectively measure, discover, and describe reality; Social phenomena – such as social stratification – are often not central to inquiry, framed as natural or inevitable (Kinchelow and McLaren, 2005). Conversely, interpretivist epistemologies posit that the meaning and construction of knowledge are subjective, placing great emphasis on individual appraisals of and reactions to their social context. These two stances are the most common within pain research, with positivism generally centering pain biology, and interpretivism centering pain psychology.

Critical epistemologies view the positivist claims of science neutrality as complicit with oppression (Bowleg, 2019; Kinchelow and McLaren, 2005; Krieger, 2020). For instance, in a racist society, omission and inaction in the face of need will favor the continuation of racist policies and practices (Jones, 2018; Kendi, 2019; Krieger, 2020). Critical research is not satisfied with the goal of increasing knowledge and embraces a transformative purpose for social justice (Kinchelow and McLaren, 2005), which makes it an ideal platform to expand inquiry, expose and challenge the power dynamics that generate health inequities. The lack of cogent population strategies for pain research and policy despite its enormous societal burden (Blyth et al., 2019) and well described inequities may be partially explained by the nearly exclusive focus on individual-level risk factors and treatment options of positivist and interpretivist conceptualizations of pain, and a dearth of critical approaches to the multilevel social determinants of pain. We embrace a critical stance by framing social

inequities as causal factors in pain etiology and aiming to help enable social change towards the elimination of undue excess burden of pain among marginalized groups.

#### 2.1.1.2. Intersectionality

Our framework is guided by the multidimensional aspects of social systems of privilege and oppression, which intersect the experience of individuals simultaneously and create health inequities (Figure 1). Persons are not singly defined by one social category at a time (e.g., race, gender, class, ableness, immigration status), but their experiences and health are shaped by the combination of their social positions and the relative value attributed to them in a given context and historical moment. It follows that social categories are not static and monolithic. Persons may live at uniquely oppressive or invisible intersections between multiple marginalized positions, relative to those who occupy positions of privilege. Importantly, intersectionality opposes traditional 'risk-factor epidemiology' and does not attribute the health inequities associated with particular identities to the individuals or to their identities. Intersectional identities are proxies for relative social positions in hierarchies of power. The social and material context of intersectional social positions are hypothesized to shape the life conditions and experiences of individuals (Evans et al., 2018).

Across this spectrum, persons may experience oppression from some of their social identities while simultaneously benefiting from privilege due to other aspects of their social positions (Bowleg, 2012; Crenshaw, 1991). Few studies have used an intersectional framework to consider heterogeneity of pain phenomena across multidimensional social positions (Anastas et al., 2020; Befus et al., 2018; Brady et al., 2019; Goodin et al., 2018; Pryma, 2017; Wiklund et al., 2016).

#### 2.1.1.3. Multilevel socio-ecological approach

Intersectional systems of privilege and oppression are established in and operate across multiple levels of organization. The original application of the biopsychosocial framework had multilevel features based on systems science principles. In contrast to biomedical reductionism, it placed the person in a hierarchically arranged continuum of systems contained within each other, and not reducible to the sum of its parts. From molecule and organelle to society-nation and biosphere, the person was represented as the highest unit of the within-organism hierarchy and the lowest unit of the social hierarchy (Engel, 1980). However, current implementations of the biopsychosocial perspective rarely do justice to this broad multilevel structure. We propose to reinstate a conceptualization of pain that considers multiple layers of systems beyond the individual (Figures 2 and 3).

Ecological frameworks have also been widely used in public health to contextualize the study of populations within the many layers of social organization that constrain and facilitate health and disease (Krieger, 2001). To illustrate the multilevel structure, we highlight *pathways of embodiment* through which the social context may affect pain (Figure 4): how societal arrangements of power as well as human history, ecological context and individual experiences come to be literally incorporated to shape our biology (Krieger, 2001).

#### 2.1.1.4. Life course perspective

We incorporate elements of longitudinal dynamics into our framework based on the life course perspective (Figure 5), which emphasizes the role of time and timing in the study of health and disease over the lifespan of individuals and over historical time across generations (Kuh et al., 2003). Life course research seeks to identify dynamic patterns such

as cumulative effects of exposures over time, periods of development in which certain exposures would be more likely to influence health (i.e., sensitive periods), or exclusive periods during which exposures could have an effect on health (i.e., critical periods). This approach, which has been occasionally explored in pain research (Dunn, 2010; Goosby, 2013; Lacey et al., 2013; Muthuri et al., 2018; Zouikr and Karshikoff, 2017) bolsters investigations of etiological processes, as well as the optimal timing for deployment of preventive and treatment measures (Jones et al., 2019) to address pain and its consequences (Figure 6).

### 2.2. A multilevel framework of pain etiology and impact

We present the proposed framework (Figure 7) with illustrating examples, emphasizing connections within and between levels of organizations as well as over time. We define 'social factors' as systems (e.g., institutions, policies, normative beliefs), processes (e.g., relationships, interactions) and positions (e.g., group membership, roles), that emerge from or are modified by the engagement of individuals with each other. Social factors at multiple levels of organization (e.g., societal, groups, interpersonal, individual) interact with each other and shape individual biological and psychological make up over the life course and across generations. Social factors which manifest anywhere along pathways of embodiment of pain – whether protective or harmful – are referred to as *social determinants of pain*. They may shape the risk of exposure to pain, the distribution of risk factors for pain and/or the distribution of resources to prevent, manage and mitigate the consequences of pain.

Socially patterned flexible resources to avoid hazards and protect health (Link and Phelan, 1995) include material conditions (e.g., living in a clean, safe and walkable neighborhood, financial stability, access to nutritious foods), emotional, cognitive and behavioral assets

(e.g., acquired through education and positive social environments), free time, social connections and access to high quality healthcare, all of which may facilitate "healthy lifestyles" to prevent and manage pain, including appropriate sleep, nutrition and physical activity. We endorse the concept of behavioral justice, which posits that "no group should bear a disproportionate share of health problems resulting from inadequate resources for engaging in healthy behaviors" (Adler and Stewart, 2009). Interlocking systems of self-serving domination and privilege, such as racism, sexism, ableism, and classism, trespass all levels of social organization from societal and institutional to interpersonal and internalized presentations (Krieger, 2020). These and other human-made systemic power structures, called *social determinants of equity* (Jones, 2014), jointly shape the distribution of downstream social determinants of health to produce differences in health outcomes between social groups which are avoidable, unnecessary, and unjust; (Whitehead, 1992) that is, health *inequities*. As articulated by Diderichsen and colleagues, the social context produces health inequities through four main mechanisms: 1) social stratification, 2) differential exposure, 3) differential vulnerability, and 4) differential social and economic consequences of ill health (Diderichsen et al., 2001).

The social context is embodied through pathways that generally flow from higher to lower levels of organization. Broadly, norms, policies, and systems operate as societal determinants of pain when they structure the distribution of risks and resources between social groupings, according to power hierarchies and other societal ethnocultural values. Group membership and spaces in which people live, work and play can be sources of strength and belonging, or the basis for social oppression and discrimination (Krieger, 2020). Likewise, interpersonal relationships and interactions are guided by higher-level parameters, and can provide both safety (e.g., empathy, support) and threat (e.g., violence,

exclusion) inputs, which modulate the salience and impact of noxious stimuli (Karos et al., 2020; Krahé et al., 2013). Families and close relationships are important actors in social learning of pain meanings (Craig, 2018) through observation (Craig, 1983), social reinforcement, (Walker et al., 2008), and social comparison processes (Festinger, 1954), which influence cognitive appraisals and affective components of the pain experience, as well as appropriate pain expression and behaviors. In turn, social communication of pain shapes how others perceive and react to it (Craig, 2018). Acute and chronic stress, social threat and deprivation may be ultimately embodied into pain through various mechanisms embedded in biological systems such as neuroendocrine, immune, inflammatory, autonomic, and epigenetic, as well as in the biological substrates of perceptual, affective, cognitive, and behavioral processes.

#### 2.2.1. Example pathway of embodiment: socioeconomic position

Although individual socioeconomic conditions are well document risk factors for pain, multilevel causal pathways through which they operate have not be adequately theorized. Here we illustrate, from upstream to downstream, one socioeconomic pathway that may influence pain. For instance, economic systems (i.e., societal) shape the value of skills, goods, and services, which in turn determine distribution of labor and production, and associated class exploitation and domination (i.e., group-level) (McCartney et al., 2019). These create potential to generate occupational conflicts and employment-related distress, (i.e., interpersonal), and place some workers at higher risk of injury, higher levels of job instability and earning unfairness, as well as to limit autonomy and access to high quality healthcare (i.e., individual) (Peckham et al., 2019; Prins et al., 2015). These factors may affect biological (e.g., risk of exposure to pain, inadequate pain management, neuroimmune stress response) and psychological (e.g., mood, maladaptive cognitive and behavioral

patterns) risk factors for pain, again operating through biological systems. Importantly, intersectional social positions influence personal history, starting potentially from inherited epigenetics and in-utero environment, with bearing on general health status, stress reactivity and adaptation to these insults (Wallack and Thornburg, 2016). Furthermore, although classism and socioeconomic position may be the most obvious drivers of pain inequities in this example, racism, sexism, heterosexism, gender binarism, nationalism and ableism, among other social determinants of equity, may also contribute to making persons who are members of multiple marginalized groups more likely to face more precarious (un)employment conditions while also having less access to salutary resources.

Recognizing the potential for dynamic feedback and reciprocal influence, chronic pain may also have consequences including impact on other aspects of physical and mental health, as well as alterations in social functioning, development, and opportunities, which become the biopsychosocial context for pain later in life (Murray et al., 2020; Rosenbloom et al., 2017), or in the early life environment of offspring (Nelson et al., 2018; Stone and Wilson, 2016). Biological endowments and their maturation interact with life experience over time to determine changes in the human capacity to experience and manage pain. Early life (Goosby, 2013; Zouikr et al., 2016), preadolescence (Denk et al., 2014), and the transition from adolescence to early adulthood (Rosenbloom et al., 2017) are periods of important biopsychosocial development, which are thought to be sensitive to the determination of pain (and health) resilience and vulnerability.

Inequalities may accumulate over the life course (Ferraro and Shippee, 2009); evidence suggests there is an increase in socioeconomic inequities in the prevalence of adult chronic pain up to the age of 65 years (Goosby, 2013), with a contribution of parental education

(often considered a proxy for childhood socioeconomic position) independent of current socioeconomic position. Above 65 years, relative socioeconomic inequities in pain seem to decrease with age, consistent with the 'age-as-a-leveler' hypothesis and robust to possible selective mortality (Celeste and Fritzell, 2018). Potential 'leveling' mechanisms offered to explain this partial convergence included social policies and more homogenous welfare programs for the elderly which reduce the impact of social stratification, as well as a preponderance of biological determinants of health in that age group.

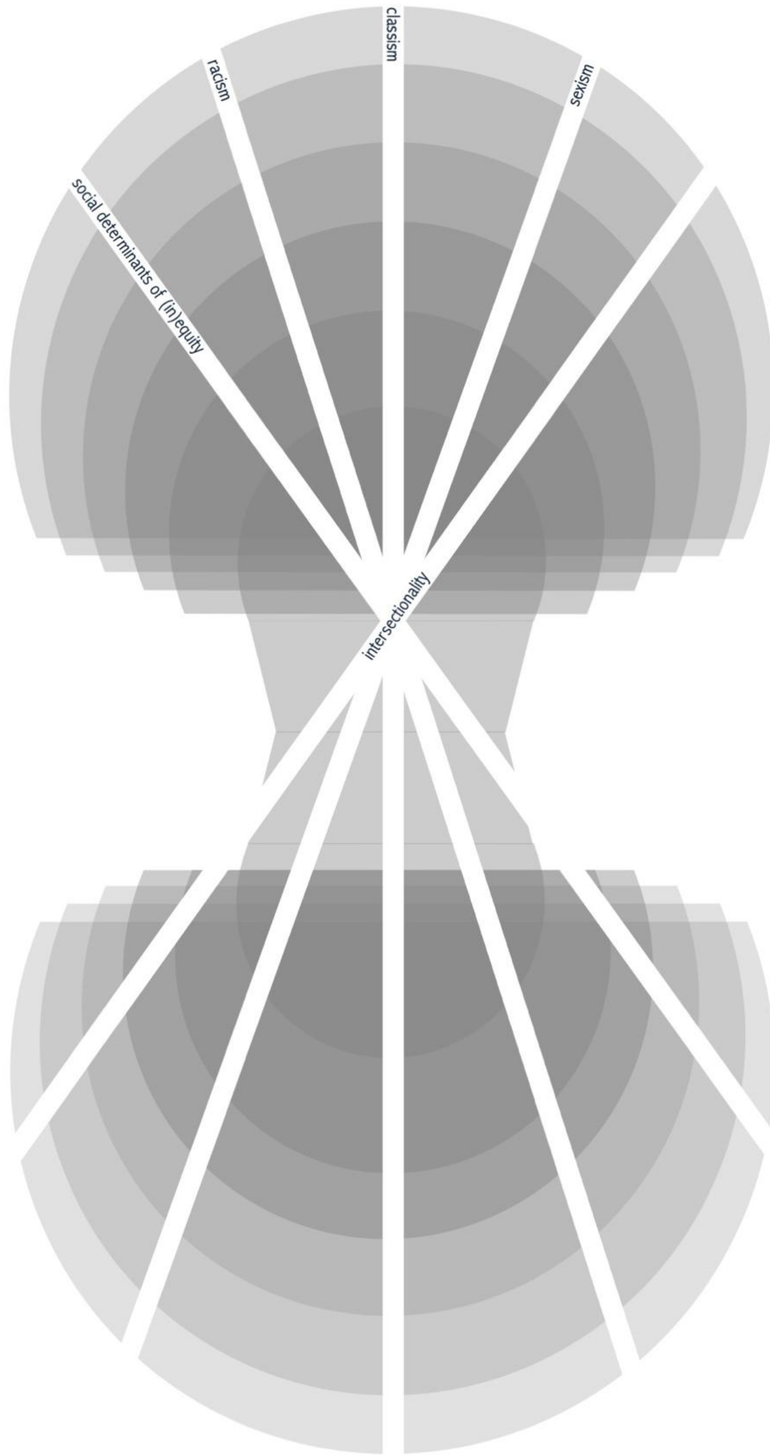


**Figure 1: Social determinants of (in)equity & intersectionality.** *Social determinants of (in)equity are* human-made interrelated systems of power that structure social stratification and relative social hierarchies within and between social groups (e.g., racism, sexism, classism, ableism, ageism, nativism, heterosexism, gender binarism, etc.). They manifest in different forms at all levels of organization (e.g., structural, personally mediated, internalized), and jointly define dynamic, contextual, and subjective *intersectional* social positions.

**Figure 2: Multilevel <u>social</u> determinants of pain** across a spectrum of nested levels (e.g., societal, group, interpersonal and individual) shape the conditions of living in which people are born, develop, relate to each other, work, and age. Risks and resources for health and social thriving are inequitably distributed according to intersectional social positions.

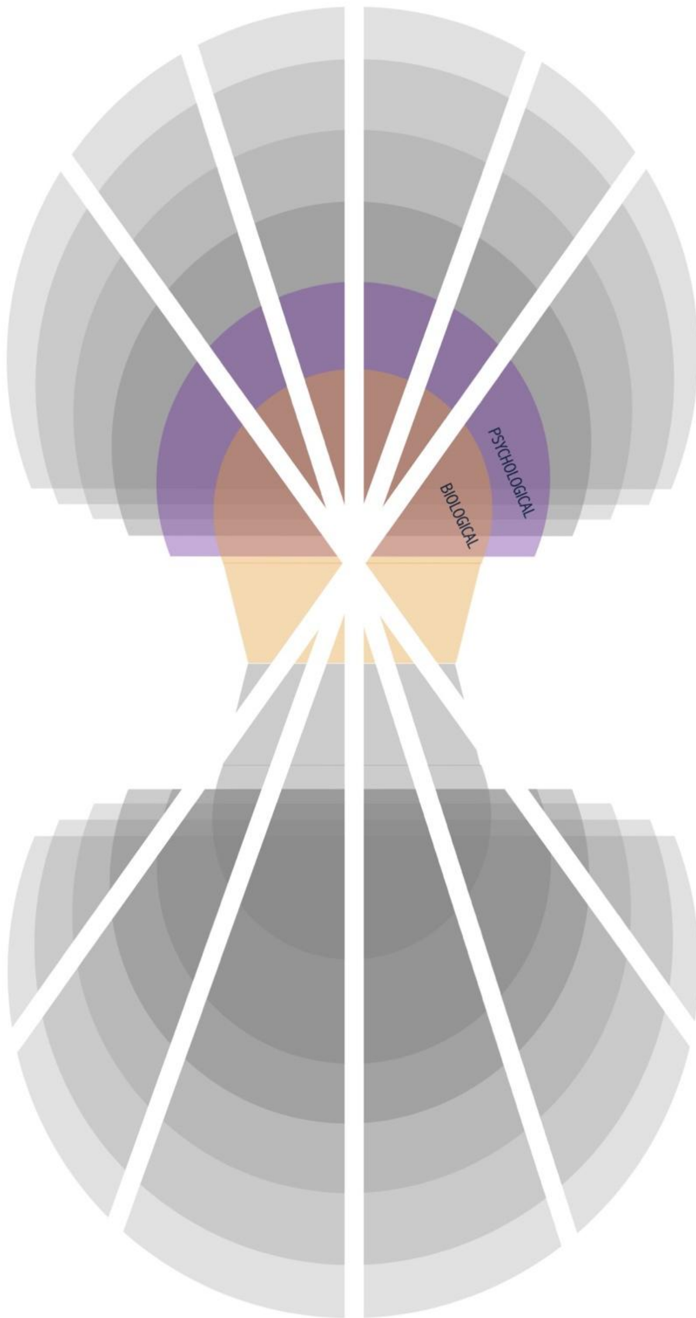


**Figure 3:** **<u>Psychological and biological</u> determinants of pain** are the result of historical and ongoing interactions between inherited (epi)genetics and environmental conditions, which are embedded in and shaped by multilevel social structures. Pain experience and expression are modulated by cognitive, affective, and behavioral processes that have evolved to allow for learning from social cues about threats to integrity. Physiological responses to stress, pain and signals of threat and deprivation (i.e., allostatic load) may contribute to the (dys)function of neurological, immune, endocrine, autonomic systems, resulting in a pro-inflammatory state and sensitization of pain processing systems.

**Figure 4: Pathways of embodiment** through which social factors affect pain are generally oriented from distal to proximal. Higher levels are more likely to drive and constrain lower levels of organization. Social inequities may lead to *differential exposure* to pain (e.g., increased risk of injury, violence, accidents, physical overload, reduced material, and social resources to prevent pain) and *differential vulnerability* to pain (e.g., acute and chronic stress, mental ill health, reduced material and social resources to maintain a healthy lifestyle and manage acute pain).

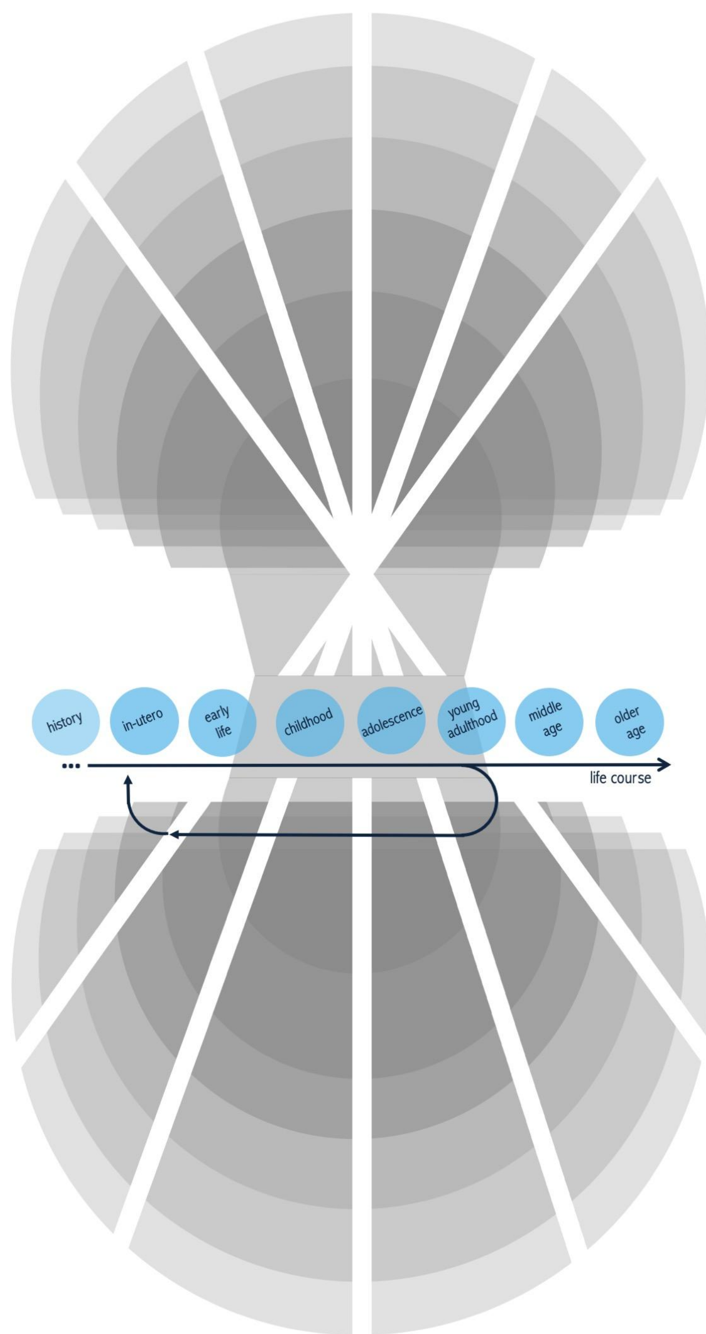


**Figure 5: Life course and intergenerational trajectories of pain.** *Life course* stages are associated with changes in biopsychosocial functions relevant to stress reactivity, as well as pain sensitivity, appraisal, and coping. Critical and/or sensitive periods may present optimal points for intervention. The effects of multilevel determinants of pain may accumulate over the life course (e.g., adverse social conditions earlier life predisposing to later onset or worsening of pain) and play a role in the *intergenerational* transmission of pain (e.g., epigenetic, behavioral, and social environment conditions of parent influencing onset and prognosis of pain in offspring).

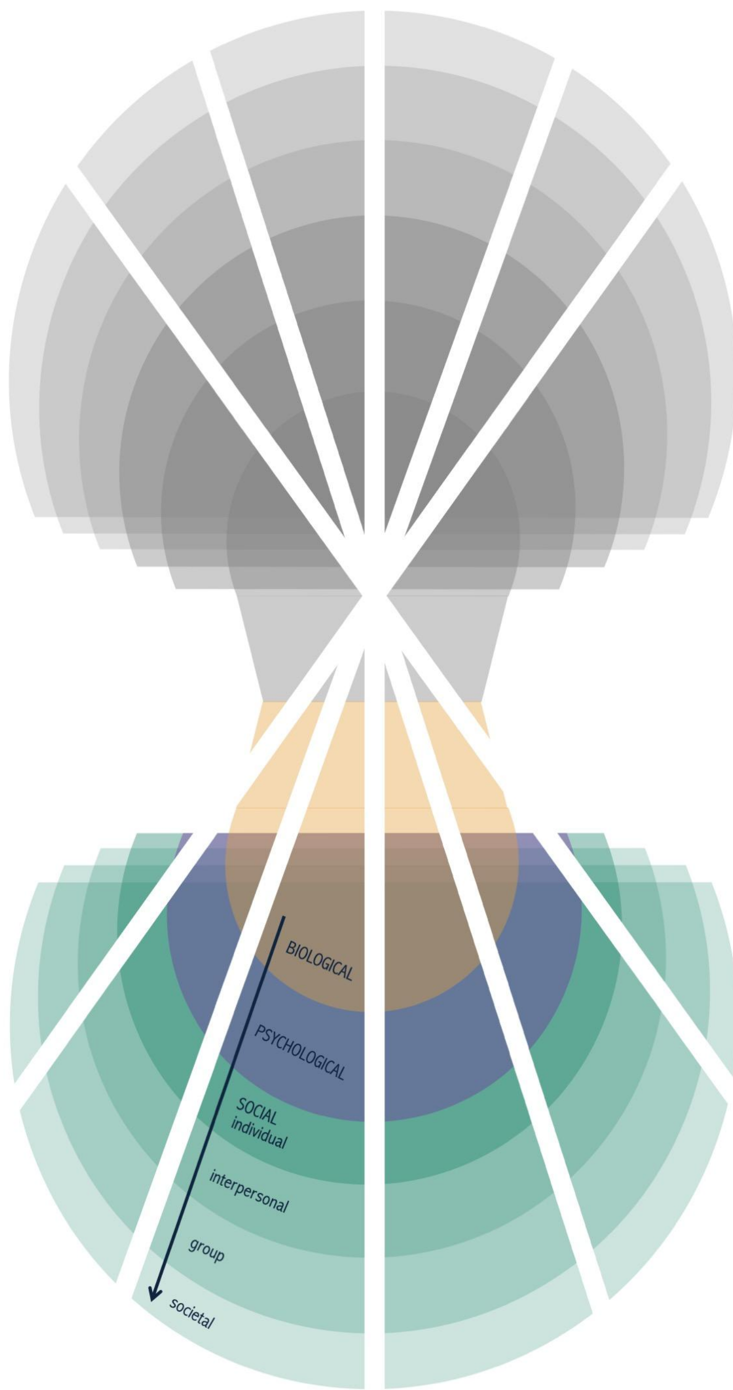


**Figure 6:** ***Multilevel consequences of pain.*** Especially when chronic, pain may be associated with increases in the risk of other physical and mental health conditions, which jointly take a toll on individual self-perception, interpersonal relationships, and participation in valued social roles, with substantial impact on population burden of disability. Social determinants of (in)equity produce *differential consequences* of pain with greater impact on persons at marginalized intersectional social positions, through barriers to material and social resources to manage pain.

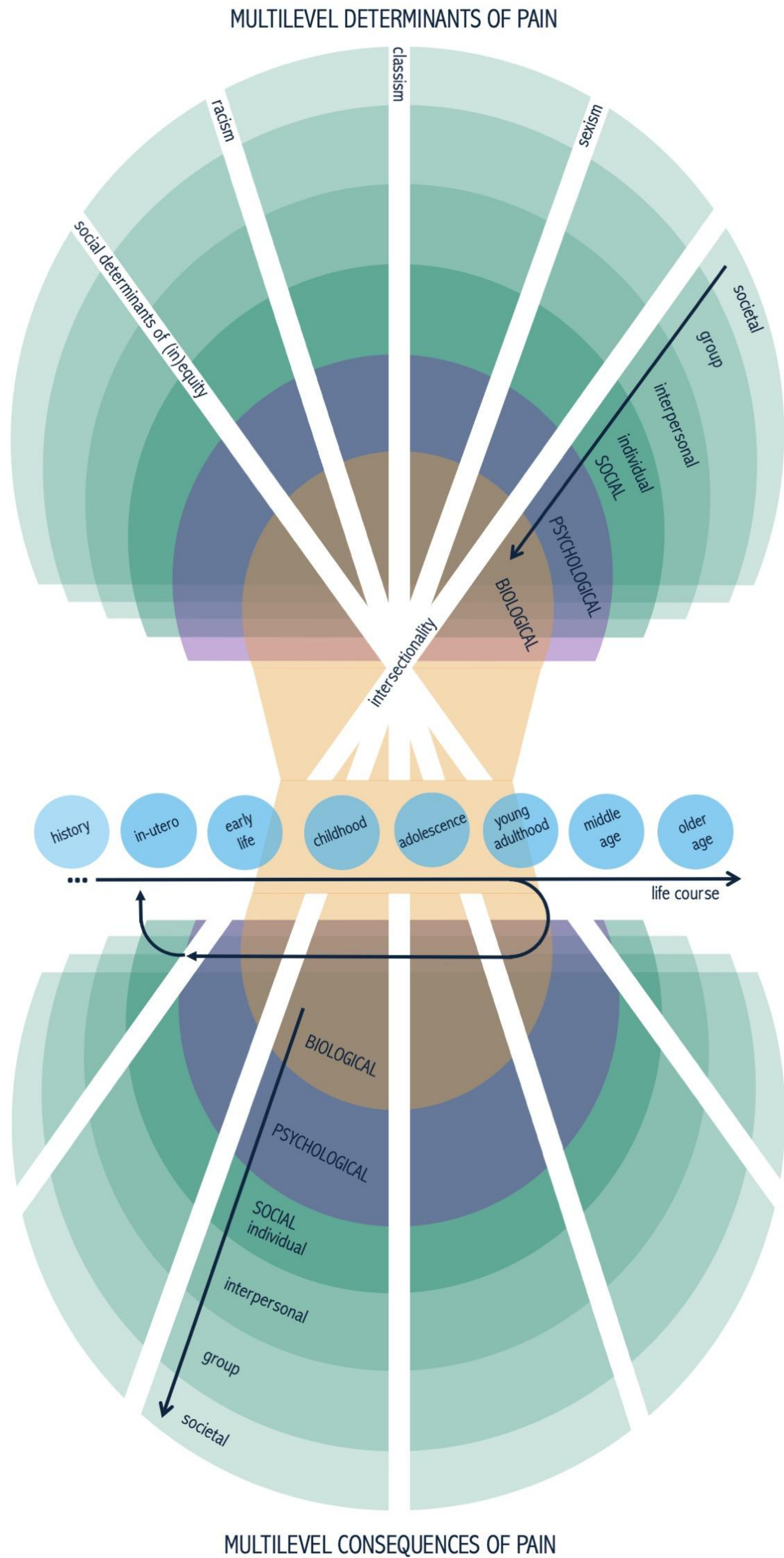


**Figure 7:** ***Multilevel framework of pain etiology and impact***
Social determinants of (in)equity shape the distribution of multilevel social determinants of pain according to intersectional social positions. Social determinants interact with each other through social pathways of embodiment, to shape individual psychological and biological determinants of pain experience and expression over the life course. In turn, pain, especially when chronic, may impact individual biopsychosocial function. Multilevel social consequences of pain are also shaped by intersectional social positions and stage of development, including potential intergenerational transmission of illness

### 2.2.2. Societal and systemic determinants of (in)equity and pain

Sociopolitical, economic, and cultural systems – societal determinants – regulate social relations of institutions and individuals, defining rights and responsibilities, determining the social and international order, and the distribution of power and resources across social groupings within and between countries (Solar and Irwin, 2010). As recognized by the 'Health in All Policies' approach (World Health Organization, 2013), the conditions of living, development and opportunities that influence health of different social groups across place and time are a function not only of healthcare and public health policies, but policies across sectors such as labor, fiscal, electoral, housing, transportation, food and agriculture, trade and industry, land use, education, media, military, environment, criminal justice, and welfare (Solar and Irwin, 2010). Some systems of beliefs and practices are based on unjust ideas of superiority of certain groups, which are used to motivate and justify the control, domination, and exploitation of other groups for their benefit (e.g., racism and white supremacy, sexism and patriarchy, gender binarism, heterosexism, ableism, ageism, nationalism, imperialism, etc.) (Krieger, 2020; Ng and Muntaner, 2014). These systems of oppression and discrimination are manifested at all levels of organization (thus, called systemic), and exert their power to create and maintain social structures and dynamics in favor of their privilege.

In short, societal, and systemic factors are the source of social stratification and may affect pain by trickle down influence in group-level, interpersonal and individual social functioning by shaping life opportunities, autonomy and material living conditions, as well as exposure to acute and chronic stressors such as discrimination, violence, and deprivation. Explicitly outlining societal and systemic determinants reveals inequality-generating mechanisms that explain why we observe unequal distribution of more proximal

risk factors for illness (for pain, e.g., mood and sleep disorders), and social gradients in health (Ng and Muntaner, 2014). Furthermore, awareness of the upstream social determinants also challenges biological essentialism and narratives that normalize health inequities (i.e., claims of inherent superiority and/or normality of dominant groups), to identify paths for accountability, social change, and population health equity (Krieger, 2020).

For instance, lower neighborhood collective efficacy (defined as "social cohesion among neighbors combined with their willingness to intervene on behalf of the common good") has been associated with racial inequities in violent victimization (i.e., a putative interpersonal determinant of racial pain inequities). However, complex systems modeling suggests that interventions to increase collective efficacy might not reduce these inequities without efforts to first eliminate residential racial segregation (Cerdá et al., 2014), which can be traced to societal-level racist policies such as "redlining" and anti-black racist policing (Bailey et al., 2020). Likewise, the acquisition of education, occupation, income and wealth - robustly associated with poorer pain outcomes at different stages of life (Bonathan et al., 2013; Dahlhamer et al., 2018; Dorner et al., 2011; Janevic et al., 2017) – does not occur at random in the population, but it is socially patterned through societal norms, policies and practices designed to preserve the status quo, such as labor exploitation, intergenerational transfer of wealth, restriction of opportunities through educational credentials, racism (e.g., school-to-prison pipeline, residential segregation, etc.), and sexism (e.g., unequal pay for equal work, unequal burden of family care and domestic labor, etc.) (Ng and Muntaner, 2014). The downstream effects of fundamental social determinants are unlikely to be sustainably eliminated by interventions that focus exclusively on specific intermdiary mechanisms, because the causal pathways that produce health inequities are multiple and

dynamic (Link and Phelan, 1995).

Barriers to social, economic, and political fulfillment are not mutually exclusive, but tend to be interconnected through their shared structural mechanisms (Kinchelow and McLaren, 2005), such that people at the intersections of disadvantaged positions can experience multiple limitations in resources and clusters of stressors over the life course and across generations (Pearlin et al., 2005). For example, in the face of generalized economic instability such as that spurred by the COVID-19 pandemic, loss of employment and food insecurity among those who become unemployed may disproportionately burden individuals who are part of racial, ethnic, sexual and gender minorities, as well as single adults living with children (Raifman et al., 2021).

### 2.2.3. Group and community determinants of pain

People are born to or acquire membership in different groups or social categories (e.g., family, friends, work, gender, social class, race/ethnicity, religion, and neighborhood), which play central roles in various aspects of their social lives, with potential implications for chronic pain. Social category membership shapes life experiences of members to the extent that societal and systemic factors distribute risks and resources across different groups, sometimes creating marginalized status. Although many of these social categories have conventionally been conceptualized as individual-level characteristics (e.g., race, ethnicity, socioeconomic status), we maintain that group membership and their socially constructed position in the hierarchies of social stratification are the basis for systematic constrains or optimization of opportunities for health and social thriving, as well as environmental contexts where people live, work and play.

For example, individuals' families are complex, dynamic systems whose characteristics and resources may influence the onset, maintenance, and management of pain (Palermo and Chambers, 2005) during important phases of neurodevelopment (McLaughlin, 2016). Exposure to a family member with chronic pain increases an individual's risk of developing chronic pain (Campbell et al., 2018). Pathways of intergenerational transmission of pain may include social learning (e.g., modelling and reinforcement) (Craig, 1983), adverse childhood experiences (ACEs, e.g., uncertainty associated with chronic parental illness or in extreme cases neglect) and elevated risk of child depression (Brown et al., 2020; Dennis et al., 2019; Higgins et al., 2019). Family social and economic deprivation is strongly associated with both ACEs (Bellis et al., 2013) and anxious or insecure attachment style (Fergusson et al., 2004; Stansfeld et al., 2008), which, in turn, are associated with adult pain outcomes and sleep disturbances (Adams and McWilliams, 2015; McWilliams, 2017; Romeo et al., 2017; Von Korff et al., 2009). The inequities in the prevalence of high-impact chronic pain by socioeconomic position (Dahlhamer et al., 2018) persist into old age, but evidence from developed countries suggest they are most prominent in midlife (Celeste and Fritzell, 2018; Grol-Prokopczyk, 2017).

Social group membership can also be subject to discrimination, which affects the pain experience (Brown et al., 2018). Racism – although not usually explicitly labeled as such – has received much empirical attention in this area. For example, among African Americans, there is a consistent relationship between perceived discrimination and chronic pain, independent of socioeconomic and health-related factors (Burgess et al., 2009; Edwards, 2008; Goodin et al., 2013; Haywood et al., 2014). Similar associations have also been found for Vietnamese Americans, Filipino Americans, Chinese Americans (Gee et al., 2007). Perceived workplace discrimination by age, education, birth region, sex, and employment

status, was also associated with prevalence of low back pain, adjusting for covariates including job physical demands in a large study in South Korea (Lee et al., 2017); Interestingly, these effects were limited to workers at workplaces without a labor union, whereas there was no evidence of associations between these types of discrimination for workers of workplaces with labor unions, regardless of individual union membership (Lee et al., 2017).

Discrimination and bias based on other social identities has also been linked to pain, such as gender (Brown et al., 2018; Klonoff et al., 2000; McDonald et al., 2014; Samulowitz et al., 2018), socioeconomic status (Willems et al., 2005) and other marginalizing personal identities (Craig et al., 2020). Inappropriate pain care and invalidating judgements due to social group membership may also contribute to inequities in pain and suffering. For example, there is experimental evidence that, despite equivalent facial expressions and self-reported pain intensity, the pain of women is systematically underestimated and perceived to be more likely to benefit from psychotherapy compared to that of men, who are perceived to be more likely to benefit from pain medicine (Zhang et al., 2021). Interactions between social categories such as race and SES have also been found to result in different biases in pain care (Anastas et al., 2020). While fraudulent misrepresentations of pain in the interest of escaping from responsibilities or securing financial gain are likely to be rare, there can be suspicion of the credibility of patient claims from health care professionals, caregivers, employers, insurance adjusters, or others (Ziano and Wang, 2019), especially for women (Werner and Malterud, 2003; Zhang et al., 2021), persons of non-conforming gender identity and at intersections of other marginalized social positions such as Black race and low socioeconomic status (Boerner et al., 2018; Pryma, 2017).

Differences in pain by gender are also shaped by the social environment (Bernardes et al., 2008; Boerner et al., 2018). Women typically have greater sensitivity to pain elicited by laboratory experiments and are more affected by painful conditions across the lifespan (Fillingim, 2017; Fillingim et al., 2009). This variation is thought to be explained by both biological and psychosocial factors, including constructs concerning normative standards, such as masculinity and femininity. Stereotypical views of masculinity are characterized by greater stoicism, strength, and independence, whereas femininity is more often associated with greater emotional, caring and sociable characteristics (Wood and Eagly, 2015).

Patterns of gender norms about pain are consistent with the concepts of hegemonic masculinity and andronormativity, where all persons are evaluated against standards of stereotypical masculine attributes and behaviors, considered prevailing and normative (Samulowitz et al., 2018). Given pain and its expression can be considered a signal of vulnerability and need for help, chronic pain, especially primary and idiopathic, is often seen as feminine, of less importance relative to more "objective" ailments and frequently has its legitimacy questioned (Samulowitz et al., 2018). Taken together, evidence suggests that some of the differences in pain between men and women are not fixed or inherent, but partly learned, flexible and dependent on social context, generally serving to reinforce male power dominance.

Group membership is often associated with the quantity of social connections and quality of social networks, for example, through access to educational and family social networks and social closure mechanisms (McCartney et al., 2019; Weeden, 2002). Measures of social group integration and connection have also been found to impact pain. Higher neighborhood social cohesion – or the degree to which neighborhood members feel they can

trust and rely on one another to cooperate and help each other when in need – may be protective against chronic pain and disability, for example, through higher levels of physical activity (Gebauer et al., 2020). Similarly, higher social capital (e.g., participation in local community feeling of trust, neighborhood connections, and friend connections) was associated with lower pain in persons with fibromyalgia, and lower odds of having dental pain, musculoskeletal pain, depression and psychosomatic symptoms in population-based studies (Aslund et al., 2010; Boehm et al., 2011; Santiago et al., 2013). In experimental studies, synchronized group activities (i.e., choir singing, rowing, and dancing) have been shown to increase social bonding and increase pain thresholds, even when group members are not known to each other (Lewis and Sullivan, 2018; Tarr et al., 2016; Weinstein et al., 2016).

Neighborhood built and social environments, which are socially patterned, may also influence pain outcomes through conduciveness to physical activity, and social connectedness, as well as exposure to toxic pollutants (potentially pro-inflammatory), crime (threat to integrity that may promote pain sensitivity), injury, and psychosocial distress,(Brooks Holliday et al., 2018). For example, walkability and proximity to parks have been associated with lower levels of pain, pain-related disability, and better physical functioning in adolescents, net of family socioeconomic characteristics and pain history (Schild et al., 2016). In contrast, neighborhood disadvantage has also been associated with a variety of negative pain outcomes, such as higher pain intensity and interference during the first year after a motor vehicle collision (Ulirsch et al., 2014), higher onset of pain interference in adults aged 50 and over (Jordan et al., 2008), higher pain-disability in adults with chronic pain (Green and Hart-Johnson, 2012) and lower health-related quality of life in children with sickle cell disease pain (Palermo et al., 2008).

### 2.2.4. Interpersonal and individual determinants of pain

Social, political, and economic systems structure the life experiences and resources of different social groups, constraining interpersonal interactions and relationships, as well as individual social roles and identities. In turn, this immediate social context influences pain and adjustment to pain by shaping cognitive-affective processes, including beliefs, appraisals, and pain-related health behaviors. For example, those with long-term painful conditions that may or may not be the result of an accident often implicate others in the onset and trajectory of their pain, including health professionals, family, drivers of vehicles, insurers, or employers for reasons including contributing to injury, inadequate assessment or medical treatment or poor interpersonal treatment (McParland et al., 2011; Scott et al., 2016). Consistent evidence demonstrates perceptions of injustice have an adverse effect on pain, disability, and mental health (Carriere et al., 2020). However, a focus on individual perceptions has the potential to be hijacked by political agendas that seek to normalize injustices (Lynch et al., 2000).

Although many evidence-based clinical treatments for pain seek to modify individual brain states, cognitive content, cognitive coping and behaviors, as well as potential sources of behavioral reinforcement in the patient's immediate environment (Jensen, 2011), it is important to highlight that neurodevelopment and brain function are themselves shaped by the social context, for example through early life conditions (e.g., threat, deprivation, stressors and protective factors), primarily driven by family social circumstances (Lopez et al., 2021; McLaughlin, 2016; Von Korff et al., 2009). Furthermore, persons who embody intersections of marginalized identities are disproportionately burdened by adverse conditions throughout the life course, including adverse childhood experiences (Slopen et al., 2016), physical injury (Padalko et al., 2019; Piatt Jr., 2015; Singh et al., 2017; Wang et

al., 2015), and chronic stress, which can lead to anxiety, depression, sleep disturbances and pain sensitization (Geronimus et al., 2011; Juster et al., 2010). These inequities are compounded by multidimensional systematic constraints in access to and quality of housing, education, and occupation, as well as early parenting, with consequences for income, wealth, recreation, nutrition, physical activity, sleep and healthcare (Durfey et al., 2019; Murray et al., 2020). These circumstances in turn limit cognitive, emotional, behavioral, social, and material resources to stay healthy and manage illness when it occurs (Phelan et al., 2010). Therefore, as emphasized throughout this text, exclusively focusing on individual-level perceptions and behaviors does not address upstream social causes of pain and is likely not sufficient to reduce pain inequities.

### 2.3. Discussion

Chronic pain is the leading cause of morbidity and disability globally (Rice et al., 2016). This major public health challenge is reflected in widespread debilitating prevalence that is often resistant to current treatments, as well as systemic inequities in access to and quality of care (Blyth et al., 2015; Institute of Medicine (IOM), 2011). The broad biopsychosocial conceptual framework of pain (Darnall et al., 2017; Gatchel et al., 2007; Hadjistavropoulos et al., 2011) has provided a useful counterpoint to the biomedical model, bolstering theoretical and treatment advances especially in the psychological domain. However, the social aspects of pain – often conflated with psychological aspects under the term 'psychosocial' – have been under-theorized and under-researched in efforts to understand pain. Pain long has been defined as a sensory and affective experience (Raja et al., 2020; Turner and Arendt-nielsen, 2020), largely omitting explicit recognition of its social features (Williams and Craig, 2016). The emphasis on sensory and affective mechanisms supports established pharmaceutical, biomedical, and psychological interventions. Failure to

recognize social aspects of pain neglects significant complexity at a cost to research priorities, prevention, and management of pain. We provide evidence of numerous isolated studies that have documented the role of social aspects of pain as risk factors, modulators, and contributors to pain-related disability, but a general conceptual framework synthesizing potential causal pathways between multilevel social factors and pain has been absent.

Furthermore, major pain models have conceptualized pain etiology at the individual level (Gatchel, 2004; Maixner et al., 2011; Melzack, 1999) or accounted for the role of the immediate social context at the interpersonal level (Craig, 2009; Hadjistavropoulos et al., 2011; Palermo et al., 2014), limiting the range and complexity of social factors considered (e.g., recent systematic review of social determinants of low back pain (Karran et al., 2020). This close-up focus on the individual with pain is consistent with the predominantly clinical approach of disciplines engaged in the diagnosis and management of pain, generally justified by the concern with investigating determinants that could be addressed through healthcare interventions (e.g., clinically modifiable factors). Yet, the considerable focus on manipulable intervention targets has been deemed socially conservative (Schwartz et al., 2016); the individual approach leaves out numerous opportunities for discovery and interventions on social factors which could greatly influence the onset and severity of chronic pain of populations (Mills et al., 2019). By contextualizing individuals within social structures, we hope to encourage collaborations between disciplines across the multilevel spectrum to expose and seek alternatives to policies, institutions and practices that create and maintain pain inequities. Likewise, strengths, resources and resilience can also be interrogated as societal and community-level targets to prevent and mitigate the impact of pain.

Learning from the trajectory of the broader field of health equity research (Adler and Stewart, 2010), we aim to facilitate pain research to advance past describing social gradients in pain. Building onto influential social determinants of health frameworks (Dahlgren and Whitehead, 1991; Diderichsen et al., 2001; Krieger, 1994; Susser and Susser, 1996), we provide a platform for the study of multilevel causal pathways and mechanisms through which the social context may affect pain at each place and historical moment. Furthermore, we highlight the importance of accounting for intersectional social forces that shape exposures and vulnerability to pain, as well as material and social resources to prevent and treat pain over time. Finally, we incorporate a life course perspective, stimulating future work to consider cumulative or differential effects of social factors on pain across generations and at various life stages. We encourage pain researchers to explicitly acknowledge these principles when formulating scientific questions, selecting analytical strategies, interpreting results, and considering strengths and limitations.

There is ongoing debate about theories and methods for causal inference in social epidemiology with implications for consequential research and the translation of knowledge into transformative social action for health equity (Hernán, 2018; Krieger and Davey Smith, 2016). There are large and persistent social inequities in pain which share fundamental social causes with a plethora of related health conditions (e.g., mental health, sleep and substance use disorders). Collectively, these conditions account for the highest burden years lived with disability globally (GBD 2016 Disease and Injury Incidence and Prevalence Collaborators, 2017), with extensive detrimental effects on social outcomes and structures. Therefore, the impact of scientific evidence and interventions can be maximized by expanding interdisciplinary collaborations between these illness-focused fields and areas such as public health, public policy, social work, and the social sciences.

Reframing pain etiology through a social justice, multilevel, intersectional and life course lens, makes it inevitable to confront fundamental social causes of pain and pain inequities. This stance also invites pain researchers, practitioners, and educators to reflect on ways in which we can best leverage our privilege to promote health equity. A*gency and accountability* are important for revealing and quantifying the role of institutions such as government, businesses, and the public sector in the production of chronic pain and suffering; furthermore, these concepts apply to the responsibility that comes with the knowledge about societal and systemic determinants, and the choice to incorporate (or ignore) an approach that actively seeks to deconstruct social and health inequities (Bowleg, 2019; Krieger, 2001).

For example, research, care, and advocacy can contribute to a culture of pain equity by challenging discourse and practices that prioritize the legitimacy of "physical" pain and interrogating who benefits from the perpetuation of a predominantly biomedical approach; dismantling narratives of onset and persistence of chronic pain as a personal deficit; listening to and amplifying the voices of people with pain (Shapiro, 2020), especially those at intersections of multiple marginalizing social positions; supporting equitable education, hiring and advancement practices within our institutions in favor of diversity and representation in healthcare (Anderson et al., 2020), academia and other spaces of power; demanding accountability from systems responsible for inadequate prevention, care and widening social inequities (Meghani, 2011); advocating for policies that reduce social inequities, and distribute interventions more equitably across social groups (Phelan et al., 2010).

Universally applied population health interventions may help to reduce the overall population burden of illness; however, these approaches tend to provide greater health benefits to more privileged subpopulations, exacerbating inequities (Frohlich and Potvin, 2008). Therefore, policies should directly address inequities by focusing on removing multilevel social barriers to health for vulnerable populations (Frohlich and Potvin, 2008). Opportunities for intervention can be envisioned and investigated at multiple policy 'entry points', which correspond to the main mechanisms that drive health inequities (Diderichsen et al., 2001): 1) reducing social inequities by addressing the mechanisms of stratification and oppression, 2) reducing differential exposures to pain, 3) reducing differential vulnerability to pain, and 4) reducing differential material and social resources to manage pain and mitigate pain-related disability.

Interventions for social, political, and economic justice – fundamental social causes of illness and health (Link and Phelan, 1995) - may target multiple levels of organization (Blyth et al., 2007), with potential effects on multiple health outcomes. For example, strengthening public education systems with a focus on historically marginalized communities and affirmative action may: 1) allow for upward social mobility and associated health gains (Vable et al., 2019), 2) expand occupational options, facilitate demands for healthy working conditions and community safety, 3) enhance social and emotional development, prevent early parenthood and intergenerational transmission of illness, strengthen social networks, healthy lifestyles and well-being (Choi et al., 2021; Mirowsky and Ross, 2003), 4) promote financial stability and paid sick leave so that the ability to engage in evidence-based non-pharmacological treatments for pain such as meditation and yoga (Bushnell et al., 2013) are not a privilege of dominant groups, improve access to high-quality healthcare provided by a diverse workforce. A framework that facilitates the

understanding of pathways of embodiment through which the social context affects pain expands the possibilities of inquiry into the vastly underexplored pain prevention and treatment strategies beyond the individual level.

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## 3. Primary data source and population

### 3.1. The National Health Interview Survey (NHIS)

For analysis in Sections 4-7, we used data from the NHIS, from years 2016 and 2017, as described below. The NHIS uses a multistage area probability design that includes clustering and stratification to obtain a nationally representative sample of approximately 35,000 households and non-institutional group quarters (e.g., college dormitories) per year. Data is obtained through in-person interviews, performed continuously throughout the years, and stratified by states and the District of Columbia (Center for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCHS), 2018). All adult members of selected households ≥17 years are invited to participate, then one civilian adult in each household is randomly selected to complete the Sample Adult survey, with persons aged ≥65 years, who identify as of Black, Hispanic, or Asian race/ethnicity having a higher chance to be selected in order to oversample and improve precision of estimates in these minority groups (Center for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCHS), 2018).

This study included a nationally representative sample of adults aged ≥25 years who had ever worked in civilian occupations. Public data files for each year (i.e., person, family, household, sample adult, functioning and disability, and 5 files for imputed income) were merged using the person, family, and household IDs, then concatenated across the two years, and treated as a pooled cross-sectional data set for 2016-2017 (Centers for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCHS), 2018). Since we were interested in evaluating associations between adult socioeconomic position and pain, we limited the sample to those aged ≥25 years, the age by which the vast majority would have had a chance to work and complete their education. In the weighted sample,

3.8% of adults 25 years and over reported having never worked in civilian occupations (Suppl. Table 1). Among these, only 2.9% reported not having worked in the past week due to being in school (0.1% if all adults 25 and over) (Suppl. Table 2).

Due to indications that missing data in income and earnings was not completely at random (i.e., missingness was associated with observed individual and family-level characteristics), NHIS staff performed multiple imputation accounting for the complex sampling design, individual and family-level variables. The procedures, described in detail in technical documentation (Division of Health Interview Statistics National Center for Health Statistics (NCHS), 2017), produce five imputed data sets. Imputation aims to adjust for bias due to missing data under the assumption that, given observed covariates considered in the procedure, missingness is randomly distributed in the data. Pooled analysis of multiple imputed data sets ensures the precision of the estimates is consistent with the uncertainty in the imputed values (Rubin, 1996). Additionally, imputation performed by NHIS staff also has the advantage of considering the inclusion of restricted variables and other confidential information about reasons for missing data in the procedures (Division of Health Interview Statistics National Center for Health Statistics (NCHS), 2017).

Occupational characteristics were obtained based on the current job of adults who were working during the week prior to the NHIS interview. Adults who were not working (i.e., “looking for work” or “not working and not looking for work”) during the week prior to the interview were asked if they had “ever held a job or worked at a business.” Those who responded affirmatively and were either retired or ≥65 years, were then asked about the job they had held the longest. Those who had ever worked in civilian occupations and were younger than 65 years were asked about their most recently held job. Adults who had never

worked in civilian occupations were excluded from the analysis, since their occupational status was undefined.

### 3.2. Key variables

Measures of socioeconomic position conceptualized as *exposures* of interest were the defined as follows:

- Educational attainment ('bachelor's degree or higher' [reference], 'some college or associate's degree', 'high school graduate', 'GED or equivalent', 'less than high school'). The GED is a high school equivalency diploma available in the US, obtained after passing a General Educational Development test;
- Relational social class, as operationalized in Section 4.2.1. (5-class NS-SEC: 'managerial and professional occupations' [reference], 'intermediate occupations', 'small employers and own account workers', 'lower supervisory and technical occupations', 'semi-routine and routine occupations');
- Family income as a percent of the US Census Bureau federal poverty thresholds (FPT) ('≥400% FPT' [reference], '300-399% FPT', '200-299% FPT', '100-199% FPT', '>100% FPT'). Family income (top-coded to the weighted average of the $95^{th}$ percentile of the five imputed data sets) is divided by the FPT of the survey year, which is adjusted annually, accounts for family size, and number of related children under 18 years, and multiplied by 100 (United States Census Bureau, 2016). For example, the cut off points for inclusion in the reference category of ≥400% was an annual family income of ≥$77,272 for a family of three with one related child under 18, or ≥$48,912 for a single person. Analytic categories were produced from a continuous measure. Preliminary analysis informed by cutoff points from previous

literature (Persmark et al., 2020; Singh et al., 2017) sought to obtain categories with adequate sample size and similar effect size for the association with HICP;

- Family members with savings or investments ('any' [reference], 'none'); and
- Home tenure status ('owned or being bought' [reference], 'rented', 'other arrangement'.

Categories regarded as "higher" or detaining generally more power, prestige, or resources were selected as reference categories.

When asked about pain, participants were instructed: "Please refer to pain that lasted a whole day or more. Do not report aches and pains that are fleeting or minor." The primary study *outcome* was the defined as follows:

- High-impact chronic pain (HICP, 'yes', 'no'), defined as pain that limits life or work activities 'most days' or 'every day' for 6 months or longer, according to previous research and National Pain Strategy recommendations (Dahlhamer et al., 2018; Interagency Pain Research Coordinating Committee, 2016).

Additional secondary outcomes included:

- Low-impact chronic pain (LICP, 'yes', 'no'), defined as pain on 'most days' or 'every day' for 6 months or longer, but limitation due to pain on life or work activities reported as 'never' or 'some days';
- Low back pain ('yes', 'no', 3 months or longer);
- Neck pain ('yes', 'no', 3 months or longer);
- Severe headaches or migraines ('yes', 'no', 3 months or longer);
- Facial pain ('yes', 'no', 3 months or longer); and
- Limiting arthritis ('yes', 'no', any joint pain, aching, stiffness during past 30 days with any current limitation).

- Number of site-specific pains (0 to 5, count of pain sites listed above)

The following sociodemographic characteristics, were conceptualized as key covariates

- Age (‘25-34 years’, ‘35-44 years’, ‘45-54 years’, ‘55-64 years’, ‘65-74 years’, ‘75-84 year’s, ‘≥85 years’);
- Sex (‘female’, ‘male’);
- US citizenship (‘yes’, ‘no’);
- Census region (‘Northeast’, ‘Midwest’, ‘South’, ‘West’);
- Ethnicity (‘Hispanic or Latino’, ‘non-Hispanic or Latino’); and
- Race (self-identified Office of Management and Budget [OMB] categories + multiple races: ‘White only’, ‘Black or African American only’, ‘American Indian or Alaska Native only’, ‘Asian only’, ‘Not releasable’, ‘Multiple races’).

According to the U.S. Census Bureau, these OMB categories “generally reflect a social definition of race recognized in this country and not an attempt to define race biologically, anthropologically, or genetically.” (U.S. Census Bureau, 2020)

- Marital status (CDC standard for legal marital status categories: separated, divorced, married, single/never married, widowed)

### 3.3. Appendix

#### 3.3.1. Supplemental Tables

**Supplemental Table 1.** Work status among adults ≥25 years, NHIS 2016-2017

| | % | 95% CI lower | 95% CI upper |
|---|---|---|---|
| Had job last week | 62.0 | 61.3 | 62.7 |
| No job last week, had job past 12 months | 5.1 | 4.8 | 5.4 |
| No job last week, no job past 12 months | 29.1 | 28.4 | 29.7 |
| Never worked | 3.8 | 3.5 | 4.1 |

Adults who had never worked in civilian occupations were excluded from the analysis since their social class was undefined. N=54,336; pop. est.=216,000,469

**Supplemental Table 2.** Reasons for not working in the past week among adults ≥25 years who have never worked in civilian occupations, NHIS 2016-2017.

| | % | 95% CI lower | 95% CI upper |
|---|---|---|---|
| Temporary non-health leave | 0.4 | 0.0* | 0.9 |
| Looking for work | 5.8 | 4.4 | 7.3 |
| Student | 2.9 | 1.6 | 4.1 |
| Taking care of house/family | 37.0 | 34.0 | 40.1 |
| Retired | 25.3 | 22.7 | 27.9 |
| Disabled or temporary health leave | 24.9 | 22.2 | 27.6 |
| Not working for other reasons | 3.6 | 2.4 | 4.9 |

These adults were excluded from the analysis since they had never worked in civilian occupations and had an undefined social class. N=1,925; population estimate=8,245,412.
*Model estimates produced negative value for lower bound of 95% confidence interval, which was manually edited to zero.

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# 4. Relational social class and high-impact chronic pain

## 4.1. Introduction

Assumptions about the distribution and generating mechanisms of social factors affect how they are measured, interpreted, and acted or not acted upon. Since at least the 1800's, there's good documentation of the effects of poor living and working conditions on health (Engels, 1845; Virchow, 2006). Initially, the effects of differing levels of social standing on morbidity and mortality were conceptualized as dichotomous, such that falling below a meaningful threshold of poverty, unemployment and political disenfranchisement was thought to be a cause of poor health (Adler and Stewart, 2010).

### 4.1.1. The social gradient in health

Around the 1980's there was a shift to a gradient, or social stratification paradigm, still predominant to this day in biomedical and other fields of research (Singh et al., 2016). The Whitehall studies of British civil servants provided important evidence that differences in health outcomes extended across the spectrum of occupational ranks (Marmot et al., 1991, 1984). The social stratification approach predicts a monotonic relationship between socioeconomic status (SES) and health, where higher SES is associated with better health outcomes. This type of hypothesis has received extensive empirical validation, to the point that the absence of socioeconomic gradients in health outcomes are considered exceptional cases worthy of study (Phelan et al., 2010).

Stratification approaches are usually based on empiricist, positivist epistemology, although theoretical underpinnings may not be explicitly presented (Muntaner and Gunn, 2019). Under these tenets, SES is typically conceptualized as a unidimensional continuous parameter, based on individual attributes such as income, educational attainment, and

occupational classifications (Muntaner et al., 2010), or as a composite index of such items (Ganzeboom et al., 1992; Muntaner, 2019). Inequality is thought to operate on a continuous spectrum, with an "unlimited number of graded distinctions between occupational groups" (Ganzeboom et al., 1992), which generally implies there is social mobility across these levels (Muntaner et al., 2010). *Health* inequalities are thought to be generated due to individuals at lower socioeconomic positions having greater health-damaging exposures and lesser health-protecting resources (Adler and Rehkopf, 2008). Explanatory mechanisms may include material and psychosocial pathways (Marmot and Wilkinson, 2001), such as living conditions, nutrition, environmental toxins, work safety, job control, discrimination, and stress (Adler and Rehkopf, 2008; Marmot et al., 1997).

#### 4.1.2. Relational social class

In parallel to the social gradient in health, relational social class theories, rooted primarily in Neo-Weberian and Neo-Marxian sociological traditions, have been used to explain the mechanisms underlying the distribution of social and economic resources as the root causes of subsequent health inequities (Muntaner and Gunn, 2019). These theories posit that political-economic power relations places people into discrete *social classes* or socioeconomic positions (SEP) and consider how different socioeconomic inequity-generating mechanisms (e.g., exploitation, domination, credentialism) shape material and psychosocial exposures that are relevant to health (McCartney et al., 2019; Muntaner et al., 2010). As opposed to the term SES, generally used in the context of gradient approaches to conceptualization of socioeconomic characteristics, the term SEP denotes the relational nature of socioeconomic classes relative to each other (Glymour et al., 2014). Although there may be some hierarchical ordering between these groups, the categorical nature of this conceptualization allows relational social class analysis to explain non-monotonic patterns in health, breaks

in the social gradient of health. Relational measures of SEP are generally operationalized as categorical, based on the relative power of economic positions held such as by level of control over productive assets (e.g., owner, employer vs. worker; manager, supervisor, non-supervisor; expert, credentialled, non-credentialed) (Krieger et al., 2005; McCartney et al., 2019; Muntaner et al., 2010; Wright, 1984).

Neo-Weberian class theories have focused on the inequality-generating mechanism of credentialism (also called social closure and opportunity hoarding, i.e., uneven allocation of skills and knowledge that are labelled as socially valuable, restricting access to labor markets, privileged positions, prestige, and social networks). Bourdieusian class theories have also described how early life circumstances are thought to produce markers - habitus and distinction - used as basis for class discrimination through social closure (McCartney et al., 2019). On the other hand, Neo-Marxist class theories emphasize the conflict over production through ownership and management relations as the main inequality-generating mechanism (Muntaner et al., 2010). Individuals are thought to use their agency to make rational choices to maximize their welfare given the constraints of their class position (e.g., employers hire, and workers are hired to maximize their welfare), resulting in exploitation and domination (Muntaner et al., 2010). Credentialism, and control over productive assets then determine factors such as job content, income, physical and psychosocial workplace exposures, social protection, and social networks, which consequently affect health (Eisenberg-Guyot and Prins, 2020; Krieger et al., 1997; Prins et al., 2021, 2015).

Although Neo-Weberian (interpretivist) and Marxian (critical theory) theories are based on different epistemologies, both oppose post-positivist stances that seeks to produce objective,

value-neutral knowledge (Hathcoat et al., 2017; Muntaner, 2019). Additionally, Neo-Marxian and Neo-Weberian relational social class theories have been integrated in the work of scholars such as Erik Olin Wright, which considers socioeconomic gradients to be the empirical *result* of underlying complex class interactions, which can also have non-gradient consequences (Muntaner et al., 2010; Wright, 1984). For instance, the concept of contradictory class locations predicts poorer health for workers in the "middle" of class hierarchies, contrary to the social gradient framework (Wright, 1984). The contradiction of their class positions, which can present differently according to place and historical context, is that they embody aspects of both ownership and labor (e.g., are subordinated by owners and higher-level workers, while also subordinating lower-level workers) (Wright, 1984). For examples, present-day lower-level supervisors in capitalist societies may experience increased psychosocial distress due to workplace demands without substantial control over decision-making, which is not offset by the presumed material benefits of slightly higher occupational ranks as compared to front line workers (Prins et al., 2015).Workers in these class positions with contradictory features have been found to have higher levels of depression and anxiety than those above and below them (Muntaner et al., 2003; Prins et al., 2015).

In summary, gradient and relational approaches are thought to capture different aspects of social inequality. For instance, most occupations are compatible with various class positions (Muntaner, 2019). Technical characteristics and prestige rankings of various occupations (e.g., job title, industry, manual workers vs. professionals, white collar vs. blue collar), have been considered by gradient approaches, while relational approaches emphasize labor relations, relative autonomy, and control over resources (e.g., owner, manager, supervisor, worker, self-employed). Despite fundamental theoretical differences, some authors advocate

for including both types of measures as they complement each other for a more complete picture of socioeconomic position as a determinant of health (Muntaner et al., 2010).

#### 4.1.3. Gaps in the pain literature

Although extensive literature has demonstrated a social gradient in pain (e.g., Dahlhamer et al., 2018; Todd et al., 2019), to our knowledge, none have investigated relational social class. A few studies have highlighted 'anomalies' in the social gradient of pain (Zajacova et al., 2020, 2012). For example, using a large nationally representative sample from the NHIS 2010-2017 (including some of the data used in the present study), adults with some college but no post-secondary degree have been found to have a higher prevalence of pain than adults with a high school diploma who did not attend college, adjusting for sociodemographic characteristics (Zajacova et al., 2020). The authors hypothesized that aspects of occupation and socioeconomic position not considered in their study warranted further investigation as potential explanations for the observed distribution of pain by education which followed non-monotonic patterns. Although they used a continuous scale of occupational prestige (Nam-Powers-Boyd) – aligned with a gradient approach - and an indicator of current employment (i.e., employed, unemployed, not in the labor force), relational social class was not investigated.

Additionally, definitions of pain that fail to account for pain severity or pain interference with life or work activities are thought to lead to an underestimation of socioeconomic inequities (Grol-Prokopczyk, 2017). For this reason, we elected to use a primary outcome measure that captures chronic pain impact (i.e., high-impact chronic pain [HICP]). HICP, defined as being associated with substantial restriction of participation in work, social, and

self- care activities for six months or more, has been for population research by the US National Pain Strategy (Interagency Pain Research Coordinating Committee, 2016).

Theory-driven SEP measures such as relational social class can reveal group differences that would be obscured or considered anomalous under the dominant social gradient paradigm. Few studies in the United States have explored the role of relational social class and health (Barbeau et al., 2004; Eisenberg-Guyot and Hajat, 2020; Eisenberg-Guyot and Prins, 2020; Krieger et al., 1997; Krieger and Fee, 1994; Prins et al., 2015). We operationalized a Neo-Weberian relational social class measure which can be used for US National Health Interview Survey (NHIS) data 2010-2018, with methods adapted from Krieger et al., 2005. We then tested for the presence of "contradictory class locations" (Wright, 2005) in US adults, which predict non-linear associations in the distribution of high-impact chronic pain by social class.

We aimed to: a) present a novel the operationalization of the five-class United Kingdom (UK) National Statistics Socio-Economic Classification (NS-SEC) for NHIS data; and b) investigate whether the association of relational social class with HICP was consistent with relational theories predicting non-gradient patterns.

## 4.2. Methods

### 4.2.1. Operationalizing the NS-SEC for NHIS 2016-2017 data

The NS-SEC is a Neo-Weberian measure of relational social class influenced by the Erikson-Goldthorpe class scheme, which has been included in the UK census in 2001, substituting two previously used UK measures, the Registrar's General Social Class and

the Socio-economic Groups (Muntaner et al., 2010; United Kingdom Office for National Statistics, 2010a). The NS-SEC conceptual development considered types of labor contract (salaried, hourly wage work, mixed), employment position (employer, self-employed, supervisory, or non-supervisory employee), and size of firm (<25, ≥25 employees).

We operationalized five-class NS-SEC to be used with data from the US NHIS 2016-2017. We used the 'self-coding method' to derive the following relational social classes: 1- Managerial and professional occupations, 2- Intermediate occupations, 3- Small employers and own account workers, 4- Lower supervisory and technical occupations, 5- Semi-routine and routine occupations (Krieger et al., 2005; United Kingdom Office for National Statistics, 2010b). According to theoretical and measurement principles of the NS-SEC, these classes should not be regarded as ordinal scales (United Kingdom Office for National Statistics, 2010a).

Methods to build the NS-SEC from US NHIS have been described by Krieger et al., 2005. However, variable availability precludes their direct application in more recent data. Specifically, methods used by Krieger et al. 2015 were derived for NHIS 2000 data, which contained civilian occupational codes based on 1995 Standard Occupational Classification (SOC), recoded as 41 categories nested within 13 broad occupational groups. In contrast, NHIS 2010-2018 used occupational codes based on 2010 SOC, recoded as 93 categories nested within 22 broad occupational groups.

To overcome the discrepancy in number of NHIS occupational groups, we used a harmonized NHIS occupational category variable from IPUMS NHIS (Blewett et al., 2019), which is available for both NHIS 2000 (year used by Krieger et al. 2005) and 2016-2017

(years used for the present study). IPUMS staff constructed the harmonized variable based on a series of technical papers and crosswalks aimed to enhance comparability across different occupational coding schemes by accounting for the proportion of each occupation that was split or aggregated into different occupational groups (Blewett et al., 2019).

First, we loaded the 2000 NHIS data obtained from IPUMS, and cross-tabulated the harmonized occupational variable and the original occupational variable from NHIS 2000 to produce a crosswalk between the two. Then, we used the harmonized variable to merge the crosswalk with the NHIS 2016-2017 data. This allowed each sample adult from the 2016-2017 in the present study who had ever worked in civilian occupations to have a value for occupational categories from NHIS 2000, used by Krieger et al 2005. Therefore, each respondent had the necessary variables to build the NS-SEC:

- NHIS occupational categories (with NHIS 2000 correspondence);
- Employment position (employee, self-employed, employer)
- Firm size (<25, ≥25 employees), and;
- Employee supervisory status (whether one supervised employees as part of work)

The five-class NS-SEC variable was then built in two steps: 1) translating NHIS occupational categories into the eight NS-SEC occupational groups; and 2) cross-referencing NS-SEC occupational group with employment position, firm size, and employee supervisory status. For the first step, we implemented the same correspondence between the 41 NHIS occupational categories and the eight NS-SEC occupational groups "self-coded" according to Krieger et al. 2005. The NHIS occupational code 25 (food service) appeared twice in their table, as 'Technical and craft occupations' and 'Routine manual and service occupations'. We opted to assign code 25 to 'Routine manual and service occupations', since workers in

food service fitting this type of category (e.g., line cook, server) would be more numerous than those possibly considered to be in 'Technical or craft occupations' (e.g., chef, restaurant manager). For the second step, the information about eight NS-SEC occupational groups was then combined with employment position, size of company, and supervisory status to derive the final five-category relational social class measure (Table 1), adapted from Krieger et al., 2005 and United Kingdom Office for National Statistics, 2010b.

Per the NS-SEC derivation matrix, relational social class 1- 'Managerial and professional occupations' was composed of all persons who were in 'Modern professional' (e.g., teacher, physiotherapist, artist, software designer) and 'Traditional professional occupations' (e.g., accountant, physician, scientist, engineer) NS-SEC occupational groups, all employers or managers in firms with ≥25 employees regardless of occupational group, supervisory employees in 'Clerical and intermediate occupations' (e.g., supervisors: secretary, personal assistant, office clerk, nursing assistant), as well as supervisory and non-supervisory employees in 'Senior manager or administrators' (e.g., finance manager, chief executive) and 'Middle and junior managers' (e.g., office, retail, bank, or warehouse manager).

Relational social class 2- 'Intermediate occupations' was composed of non-supervisory employees in 'Clerical and intermediate occupations'. Relational social class 3- 'Small employers and own account workers' included all employers or self-employed workers with <25 employees, except those in 'Modern professional' and 'Traditional professional occupations'. Relational social class 4- 'Lower supervisory and technical occupations' was composed of supervisory employees in 'Semi-routine' (e.g., postal worker, machine operator, security guard, farm worker, receptionist, sales assistant) and 'Routine manual and service occupations' (e.g., van driver, cleaner, packer, waiter/waitress), as well as supervisory and

non-supervisory employees in 'Technical and craft occupations' (e.g., motor mechanic, fitter, plumber, train driver). Finally, relational social class 5- 'Semi-routine and routine occupations' was composed of non-supervisory employees in 'Semi-routine' and 'Routine manual and service occupations'.

**Table 1. NS-SEC five-class self-coded derivation method derivation matrix**

| NS-SEC occupational groups | Large employers or managers | Small employers or self-employed | Supervisory employees | Non-supervisory employees |
|---|---|---|---|---|
| Modern professional occupations | 1 | 1 | 1 | 1 |
| Clerical and intermediate occupations | 1 | 3 | 1 | 2 |
| Senior managers or administrators | 1 | 3 | 1 | 1 |
| Technical and craft occupations | 1 | 3 | 4 | 4 |
| Semi-routine manual and service occupations | 1 | 3 | 4 | 5 |
| Routine manual and service occupations | 1 | 3 | 4 | 5 |
| Middle and junior managers | 1 | 3 | 1 | 1 |
| Traditional professional occupations | 1 | 1 | 1 | 1 |

Large employers have ≥25 employees, small employers have <25 employees, self-employed have no employees. Numbers correspond to relational social classes: 1- Managerial and professional occupations, 2- Intermediate occupations, 3- Small employers and own account workers, 4- Lower supervisory and technical occupations, 5- Semi-routine and routine occupations.

#### 4.2.2. Statistical analysis

To investigate the association between relational social class and HICP for the presence of non-gradient patterns, we used survey weighted Poisson regression to obtain adjusted prevalence ratios (aPR) and 95% confidence intervals (CI), adjusting for education, age, sex, race, ethnicity, US citizenship, marital status, and Census region.

### 4.3. Results

The burden of HICP by relational social class was similar for the reference category ‘managerial and professional occupations’ and ‘intermediate occupations’ (aPR=1.00, 95% CI 0.88, 1.13), adjusted for education and demographic characteristics; both lower than all other classes.

Although ‘semi-routine and routine workers’ often constitute lower ranks in occupational hierarchies, their adjusted burden of HICP relative to ‘managerial and professional occupations’ was not the highest among relational social classes, as would have been predicted by a hypothesis of social gradient in health. Lower supervisory and technical occupations had the highest burden of HICP relative to the reference class (aPR=1.37, 95% CI 1.22, 1.53), followed by small employers and own account workers (aPR=1.26, 95% CI 1.09, 1.45) and semi-routine and routine workers (aPR=1.25, 95%CI: 1.13, 1.39).

### 4.4. Discussion

Three relational social classes were identified as having higher burden of HICP compared to managerial and professional occupations. Relational class measures can provide insight into the complexity of socioeconomic position that may be obscured by strictly gradient-wise

measures and conceptualizations (i.e., modeling SEP as a continuous variable). Moreover, inequity-generating mechanisms that are supported by relational theories such as exploitation and domination may guide the development of interventions for population-level prevention or mitigation of inequities (e.g., strengthening labor movements, fair compensation policies, alternative ownership, and management structures) (Prins et al., 2021). There were modest non-gradient patterns in the distribution of HICP by social class that may be suggestive of the relational concept of contradictory class locations. Alternatively, we could interpret these three groups as having similar effect sizes.

Given the novel character of this analysis, further evaluation of additional measures of relational social class are needed to test whether our findings are sensitive to the operationalization of the concept. Measures developed for or validated for the current US occupational and social context would be particularly useful.

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# 5. Socioeconomic pain inequities in US adults: High-impact chronic pain

## 5.1. Introduction

Three main goals of measuring SEP in health research have been identified (Galobardes et al., 2007): 1) to describe and monitor the social distribution of health and disease; 2) to explain causal mechanisms of socioeconomic health inequities; and 3) to statistically adjust for confounding by socioeconomic circumstances. Research in the pain field has reflected primarily goals one and three. Socioeconomic variables have often been presented as non-modifiable (van Hecke et al., 2013), and few studies have explored etiological pathways of socioeconomic pain inequities. We aimed to use social theories and directed acyclic graphs (DAGs), a causal inference tool, to expand the conceptualization of SEP in relation to pain for the benefit of all three goals.

Socioeconomic group differences can be explored by approaches that formally consider causal *pathways of embodiment* (Krieger, 2001) between the social context and health to inform potential health equity interventions. Additionally, clear and complete conceptualizations of the roles of SEP measures as potential confounders, colliders and mediators through DAGs, can enhance study validity by informing SEP measure selection, statistical model-building and interpretation, even when SEP is not central to the study in question (Akinkugbe et al., 2016; Fleischer and Roux, 2008).

Regression models in pain research often include variables that potentially lie on the causal pathway between SEP and pain. These adjustments may lead to an underestimation of total socioeconomic pain inequities – unless used as part of procedures to estimate controlled direct effects, with consideration to assumptions of no mediator-outcome confounding and no exposure-mediator interactions (VanderWeele and Vansteelandt, 2010).

For example, a recent study using a large nationally representative sample from the NHIS 2010-2017 (including some of the data used in the present study), assessed the association between education and pain. In fully adjusted models (including employment status, occupational prestige, income, home tenure status, smoking, alcohol use, body mass index, psychosocial distress, and other comorbid conditions), there were no significant differences in pain prevalence between adults with a high school education and adults with less than high school education or with any higher education degrees (i.e., Associates vocational, Associates academic, Bachelors, Masters or higher) (Zajacova et al., 2020). However, these results remove the potential portion of the effect of education on pain that operates through the covariates in the model.

We presented DAGs to depict the hypothesized causal relationships between different domains of SEP and chronic pain. Informed by these DAGs, we estimated the associations of five complementary SEP measures (i.e., educational attainment, social class, family income measured as percent of the federal poverty threshold [FPT], family members with interest income, and home tenure status) with high-impact chronic pain (HICP), and low-impact chronic pain (LICP).

By characterizing the relationships between various SEP domains and pain within a causal framework, we aimed to help improving the measurement and conceptualization of SEP in future research. Moreover, by using the “c-word” (i.e., causal) and the “t-word” (i.e., theory) (Hernán, 2018; Jones and Schooling, 2018) we hope to help moving the field of pain research beyond documenting socioeconomic pain inequities. This work will enable enhanced hypothesis generation and testing of causal mechanisms that inform population health interventions to reduce and eliminate socioeconomic pain inequities.

Specifically, we aimed to: b) use a DAG-informed approach to estimate the associations of educational attainment, family income, social class, family savings/investments and home tenure status with HICP, LICP, adjusting for sociodemographic characteristics.

## 5.2. Methods

### 5.2.1. Causal thinking for socioeconomic pathways of pain inequities

Based on previous conceptualizations of the causal pathways between SEP and health, we constructed DAGs including parental education or childhood SEP (unmeasured in the present study) as the furthest upstream measure of SEP, since they determine adult SEP and are also associated with adult pain outcomes net of adult SEP (Muthuri et al., 2018). Following early life SEP, we included a causal chain of education, occupation, and income (Lahelma et al., 2004; Singh-Manoux et al., 2002). Further downstream, we also included two measures of wealth (family savings/investments and home tenure status), which aimed to capture additional dimensions of material resources, financial stability, and possibly intergenerational markers of SEP.

In addition to being hypothesized as determinants of HICP, each SEP measure was also hypothesized to influence all other SEP measures that were downstream to it. Demographic characteristics are generally thought to be common causes of both economic circumstances and pain (Foley et al., 2021; Solar and Irwin, 2010). In effect, we conceptualized socioeconomic circumstances to be in the causal pathways between marginalized intersectional social positions and pain. These pathways and mechanisms of embodiment are discussed in more detail in Section 2. To produce the DAGs and verify adjustment sets for each exposure of interest, we used DAGitty (Textor et al., 2016).

#### 5.2.2. DAG-informed statistical analysis

We obtained prevalence ratios (PR) and 95% confidence intervals (CI) using Poisson regression for binary outcomes. Each of the models was built separately and adjusted using indicator variables for the minimal sufficient sets of variables determined by DAG graphical criteria (e.g., back-door pathways, Textor et al., 2016), or the closest alternative given available data. This included all demographics (except for marital status, not included in the education model), and other SEP variables hypothesized to be upstream of the primary SEP exposure for each model. Specifically, education was included in the models of social class, income, family savings or investments, and home tenure status; social class was included in the models of income, family savings or investments, and home tenure status; income was included in the models of family savings or investments, and home tenure status; family savings or investments included was included in the model of home tenure status.

All estimates and 95% CI were produced using NHIS-provided complex survey weights. Final person-level weights are roughly inversely proportional to the sampling probability of their address' cluster, correcting for non-response and other ratio-adjustments. (Centers for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCHS), 2018) Weights are also post-stratified by age, sex, and race/ethnicity to produce population estimates that reflect quarterly population estimates produced by the U.S. Census Bureau (Centers for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCHS), 2018). According to NCHS guidance, we divided weights by two to avoid doubling the size of population estimates produced by pooling two years of data (Centers for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCHS), 2018).

Unweighted sample sizes (n) and weighted population estimates (pop. est.) are shown for descriptive purposes. Data management and analyses were conducted in Stata Statistical Software, Release 14 (StataCorp, 2015), using the '*svy*' and '*mi*' suites to handle complex survey sampling weighting and multiple data sets imputed for family income, respectively. For each set of models for a given pain outcome, individuals with missing data for the non-imputed socioeconomic exposures, covariates or outcome were excluded from the analysis. Stata '*subpop*' commands were used to limit the sample according to the age inclusion criterion and complete data for each set of models, maintaining the survey design parameters necessary for appropriate variance estimation (Centers for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCHS), 2018). Figure 6 was produced using the R package ggplot2 (Wickham, 2016).

## 5.3. Results

### 5.3.1. Population characteristics

The overall prevalence of HICP among adults ≥25 years who had ever worked in civilian occupations in 2016-2017 was 8.3% (95% CI: 8.0, 8.7), affecting an estimated total of 16.6 million adults in this population. As it relates to sociodemographic distribution of HICP (Table 1), age is the most strikingly different between adults with HICP and the overall population. While each age category higher in age represented a smaller share of adults in the overall population, there was an inverted 'U'-shaped age pattern among adults with HICP, highest among adults 55-64 and 45-54 years. Although these age groups account for 38.9% of the overall population, they account for 52.0% of adults with HICP. There was a smaller proportion of adults in age categories 25-34 and 35-44 among adults with HICP than in the overall population. There was a slight over-representation of females among adults with HICP, as well as non-Hispanic (NH) White, NH-multiple races, and NH-

American Indian or Alaskan Native. US citizens and adults who live in the South region were also slightly over-represented among adults with HICP compared to the overall population.

The distribution of SEP measures is presented primarily to illustrate the proportion represented by each category (Table 1). Reference categories had the highest proportion of the overall population in each SEP measure, except for family members with savings or investments. Multiple imputation of family income due to missing or incomplete data was done by NHIS staff for 15.4% of the weighted sample (95% CI: 14.6, 16.2%).

**Table 1.** Distribution of demographic and socioeconomic characteristics by high-impact chronic pain status among adults ≥25 years who had ever worked in civilian occupations, NHIS 2016-2017.

| | **Overall** | **High-impact Chronic Pain** | |
|---|---|---|---|
| | | **No** | **Yes** |
| | pop. est.= 199,014,173 | pop. est.= 182,404,639 | pop. est.= 16,609,534 |
| **Demographics, % (95% CI)** | | | |
| **Age (years)** | | | |
| 25-34 | 20.3 (19.8, 20.9) | 21.5 (21.0, 22.1) | 6.9 (5.9, 8.0) |
| 35-44 | 18.8 (18.3, 19.3) | 19.5 (19.0,20.0) | 11.4 (10.1, 12.7) |
| 45-54 | 19.6 (19.2, 20.1) | 19.3 (18.8, 19.8) | 23.5 (21.8, 25.2) |
| 55-64 | 19.3 (18.8, 19.8) | 18.5 (18.0, 19.0) | 28.5 (26.8, 30.2) |
| 65-74 | 13.3 (12.9, 13.8) | 13.0 (12.6, 13.5) | 16.6 (15.3, 17.9) |
| 75-84 | 6.4 (6.1, 6.6) | 6.2 (5.9, 6.4) | 8.5 (7.7, 9.4) |
| 85+ | 2.2 (2.1, 2.4) | 2.0 (1.9, 2.2) | 4.5 (3.7, 5.3) |
| | | | |
| **Sex** | | | |
| Female | 51.2 (50.6, 51.8) | 50.6 (50.0, 51.2) | 58.0 (55.9, 60.0) |
| Male | 48.8 (48.2, 49.4) | 49.4 (48.8, 50.0) | 42.0 (40.0, 44.1) |
| | | | |
| **Ethnicity, Race** | | | |
| NH, White only | 66.7 (65.2, 68.2) | 66.4 (64.9, 67.9) | 70.1 (67.9, 72.4) |
| Hispanic | 14.3 (13.1, 15.6) | 14.5 (13.2, 15.7) | 12.5 (10.6, 14.4) |
| NH, Black or AA only | 11.1 (10.2, 11.9) | 11.1 (10.2, 11.9) | 11.3 (9.8, 12.8) |
| NH, Asian only | 5.6 (5.0, 6.1) | 5.9 (5.3, 6.5) | 1.9 (1.3, 2.5) |
| NH, multiple race | 1.5 (1.3, 1.7) | 1.4 (1.2, 1.5) | 2.7 (2.0, 3.4) |
| NH, AIAN only | 0.7 (0.4, 0.9) | 0.6 (0.4, 0.9) | 1.3 (0.8, 1.7) |
| NH, race not releasable | 0.2 (0.1, 0.2) | 0.2 (0.1, 0.2) | 0.2 (0.1, 0.4) |
| | | | |
| **Marital status** | | | |
| Married | 59.5 (58.7, 60.2) | 60.3 (59.6, 61.1) | 50.0 (48.0, 51.9) |
| Single/never married | 18.5 (18.0, 19.1) | 18.9 (18.3, 19.5) | 14.5 (13.2, 15.8) |
| Divorced | 13.2 (12.8, 13.5) | 12.5 (12.2 12.9) | 20.0 (18.5, 21.5) |
| Widowed | 6.5 (6.2, 6.7) | 6.0 (5.8, 6.3) | 11.1 (10.1, 12.0) |
| Separated | 2.4 (2.2, 2.5) | 2.2 (2.0, 2.3) | 4.5 (3.8, 5.2) |
| | | | |
| **US citizenship** | | | |
| Yes | 92.6 (92.1, 93.2) | 92.3 (91.7, 92.8) | 96.9 (95.9, 97.8) |
| No | 7.4 (6.8, 7.9) | 7.7 (7.2, 8.3) | 3.1 (2.2, 4.1) |

| | | | |
|---|---|---|---|
| **Region** | | | |
| South | 35.4 (33.3, 37.6) | 35.3 (33.1, 37.5) | 37.3 (34.4, 40.1) |
| West | 23.6 (21.7, 25.5) | 23.6 (21.7, 25.5) | 23.9 (21.3, 26.4) |
| Midwest | 22.6 (21.2, 23.9) | 22.6 (21.3, 24.0) | 21.9 (19.8, 23.9) |
| Northeast | 18.4 (16.7, 20.0) | 18.5 (16.8, 20.2) | 17.0 (15.1, 18.9) |
| **Socioeconomic position, % (95% CI)** | | | |
| **Educational attainment** | | | |
| Bachelor's degree or higher | 36.4 (35.3, 37.6) | 38.1 (37.0, 39.3) | 17.7 (16.2, 19.2) |
| Some college or Associate degree | 29.2 (28.5, 29.9) | 28.8 (28.1, 29.6) | 33.3 (31.4, 35.3) |
| High School Graduate | 20.9 (20.3, 21.5) | 20.5 (19.9, 21.1) | 24.7 (23.1, 26.3) |
| GED or equivalent | 2.8 (2.6, 3.0) | 2.6 (2.3, 2.8) | 5.7 (4.8, 6.5) |
| Less than High School | 10.7 (10.1, 11.3) | 10.0 (9.4, 10.5) | 18.6 (17.0, 20.1) |
| **Relational social class, NS-SEC** | | | |
| Managerial and professional | 43.6 (42.7, 44.6) | 44.8 (43.9, 45.7) | 30.9 (29.1, 32.6) |
| Intermediate occupations | 12.0 (11.6, 12.4) | 12.0 (11.5, 12.4) | 12.4 (11.1, 13.6) |
| Small employers and own account | 7.2 (6.8, 7.5) | 7.0 (6.7, 7.4) | 8.5 (7.4, 9.6) |
| Lower supervisory and technical | 14.5 (14.0, 14.9) | 14.1 (13.6, 14.6) | 18.6 (17.2, 20.1) |
| Semi-routine and routine workers | 22.7 (22.1, 23.4) | 22.1 (21.5, 22.8) | 29.6 (27.8, 31.4) |
| **Family income as percent of FPT** | | | |
| ≥400% FPT | 45.5 (44.4, 46.7) | 47.5 (46.3, 48.7) | 24.0 (22.2, 25.8) |
| 300-399% FPT | 13.2 (12.8, 13.7) | 13.4 (12.9, 13.9) | 11.2 (9.9, 12.5) |
| 200-299% FPT | 15.4 (14.9, 15.9) | 15.4 (14.8, 15.9) | 16.4 (14.8, 18.0) |
| 100-199% FPT | 16.6 (16.0, 17.1) | 15.6 (15.1, 16.2) | 26.6 (24.9, 28.4) |
| <100% FPT | 9.3 (8.8, 9.7) | 8.1 (7.7, 8.6) | 21.8 (20.3, 23.3) |
| **Family members w/ savings or investments** | | | |
| Any | 28.1 (27.0, 29.2) | 28.9 (27.7, 30.0) | 19.9 (18.4, 21.4) |
| None | 71.9 (70.8, 73.0) | 71.1 (70.0, 72.3) | 80.1 (78.6, 81.6) |
| **Home tenure status** | | | |
| Owned or being bought | 68.7 (67.9, 69.6) | 69.2 (68.3, 70.1) | 63.1 (61.2, 65.0) |
| Rented | 29.4 (28.5, 30.3) | 28.8 (28.1, 29.9) | 33.7 (31.8, 35.6) |
| Other arrangement | 1.9 (1.7, 2.0) | 1.8 (1.6, 1.9) | 3.2 (2.6, 3.8) |

HICP= High-impact chronic pain; AA=African American; AIAN=American Indian or Alaska Native; FPT=federal poverty threshold; Overall n=50,357; No HICP n=45,764; Yes HICP n=4,593; NS-SEC= U.K. National Statistics Socio-Economic Classification; Variables with categories without natural ordering, such as race, sex, marital status, region, and home tenure status are listed in order of descending frequency in adults without HICP.

### 5.3.2. Causal model of socioeconomic pain inequities in US adults

Separate DAGs are presented for each of the five socioeconomic exposures of interest, to depict which variables would be selected for adjustment in each DAG-informed model: education (Figure 2), relational social class (Figure 3), family income (Figure 4), family savings or investments (Figure 5), and home tenure status (Figure 6).

In the sequence in which they were presented above, SEP exposures of interest were arranged in a causal chain, forming an arc directed from left to right at the top of the DAGs. All SEP exposures were hypothesized to have a direct effect on pain, pictured in the center of the DAG. Each SEP concept was also hypothesized to influence other aspects of SEP that were downstream to them, pictured to their right, leading to potential indirect effects of SEP on pain.

Demographic variables, presented as a vertical stack at the bottom of the DAG, were hypothesized to be upstream of all measures of SEP and upstream of pain, except for marital status, which was hypothesized to be downstream of education. That is because most marriages and other changes in marital status occur later into educational attainment or after its completion in the United States (Payne, 2012).

The main exposure in each figure is pictured in a yellow rectangular node and marked with a small black triangle, while their descendants (i.e., consequences) are pictured in blue nodes. The outcome, which is marked with a small vertical black bar, is also pictured as a blue rectangular node, since by definition it is also a descendant of the exposure. Measured ancestors (i.e., causes) of both exposure and outcome which are not on the causal pathways of interest (i.e., measured confounders), are pictured as grey rectangular nodes.

Unmeasured confounders are pictured as a white elliptical. Arrows represent the direction of hypothesized causal pathways; green arrows are the causal pathways of interest, black arrows are pathways blocked by confounder adjustment, and pink arrows are biasing (i.e., unmeasured confounding) paths.

**Figure 1.** Socioeconomic pathways of pain inequities by education

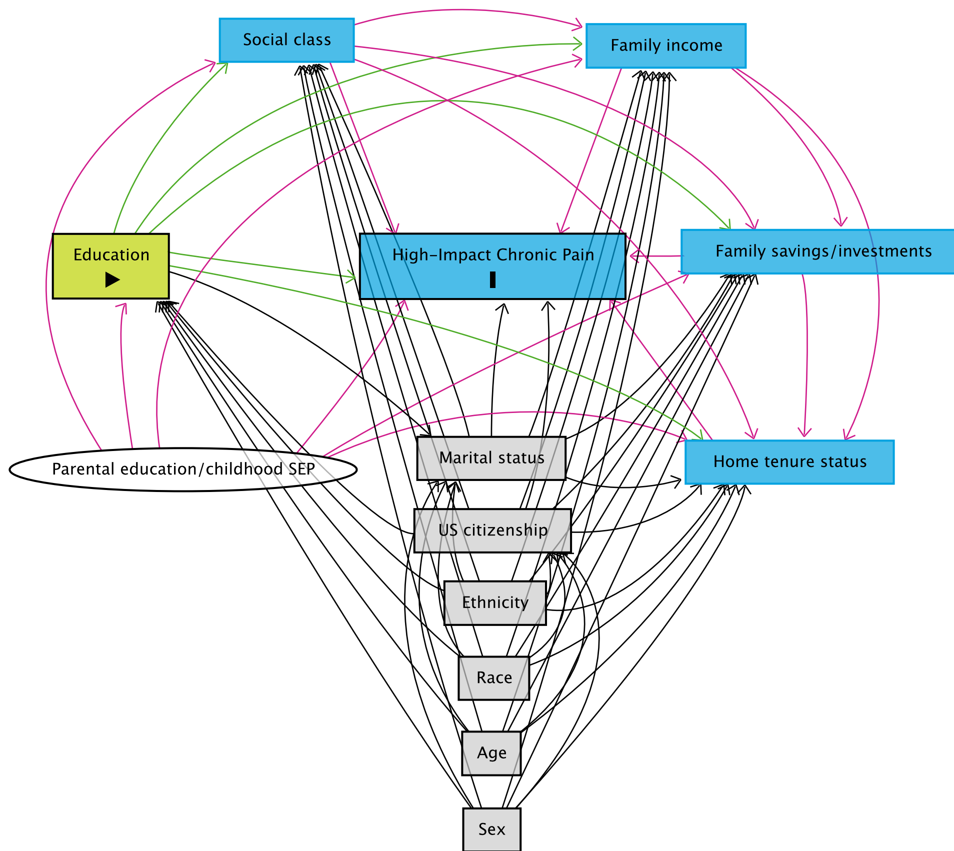


**Figure 2.** Socioeconomic pathways of pain inequities by relational social class

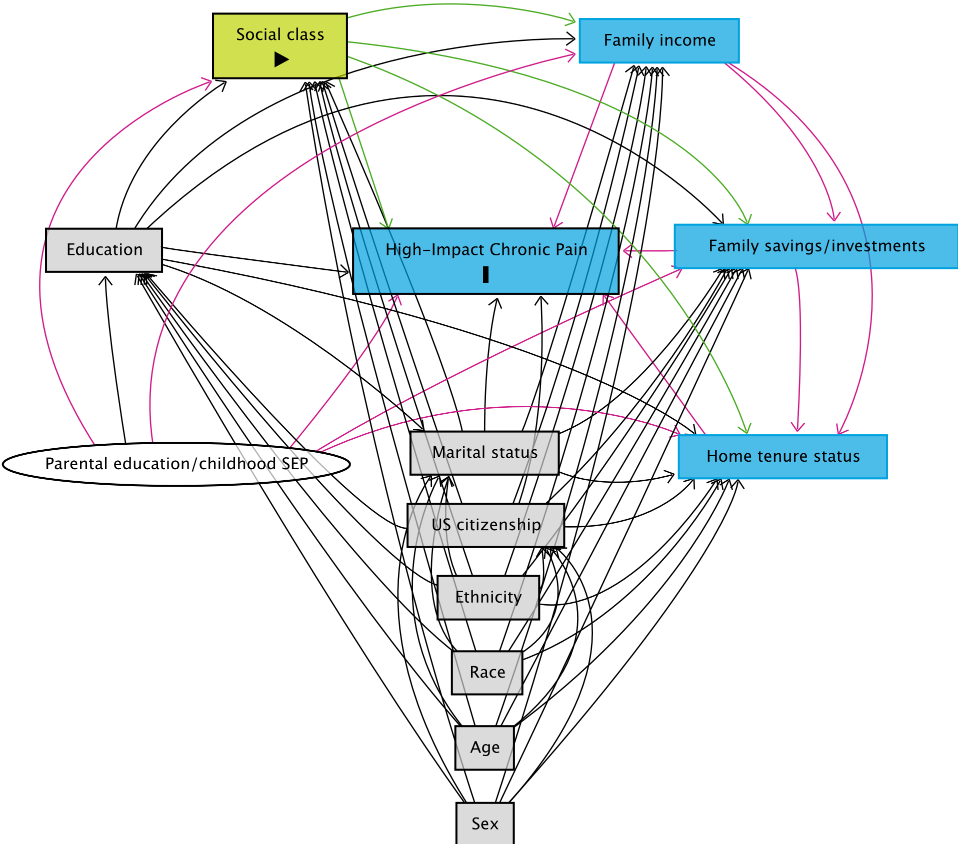


**Figure 3.** Socioeconomic pathways of pain inequities by family income

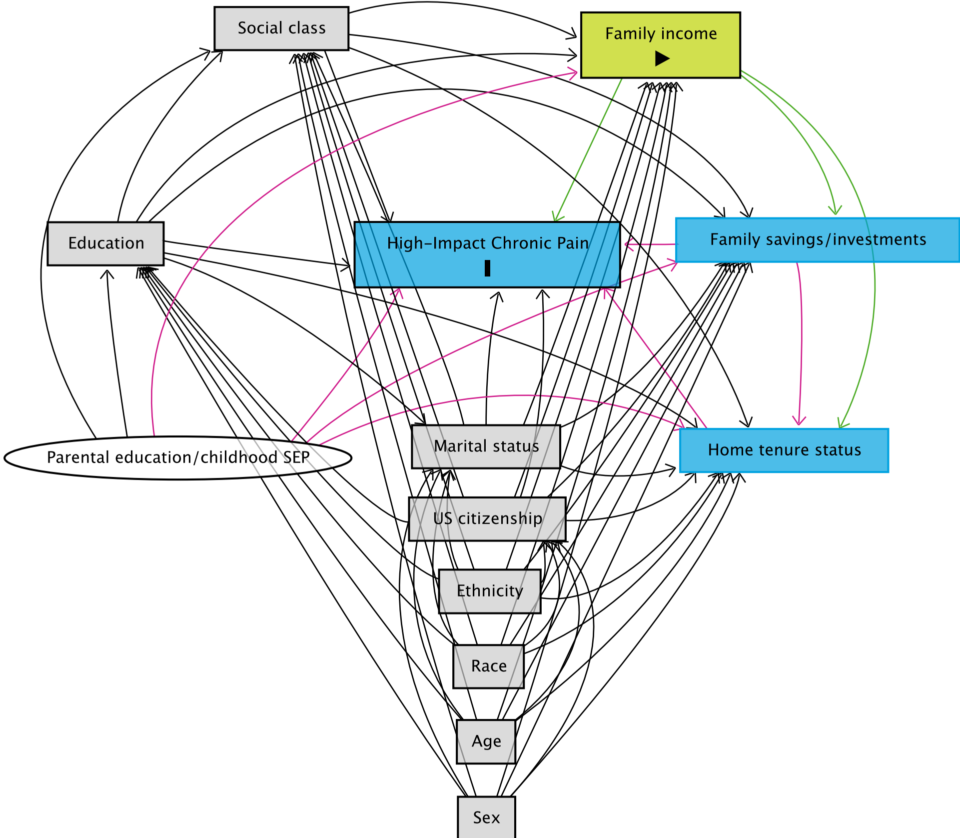


**Figure 4.** Socioeconomic pathways of pain inequities by family savings or investments

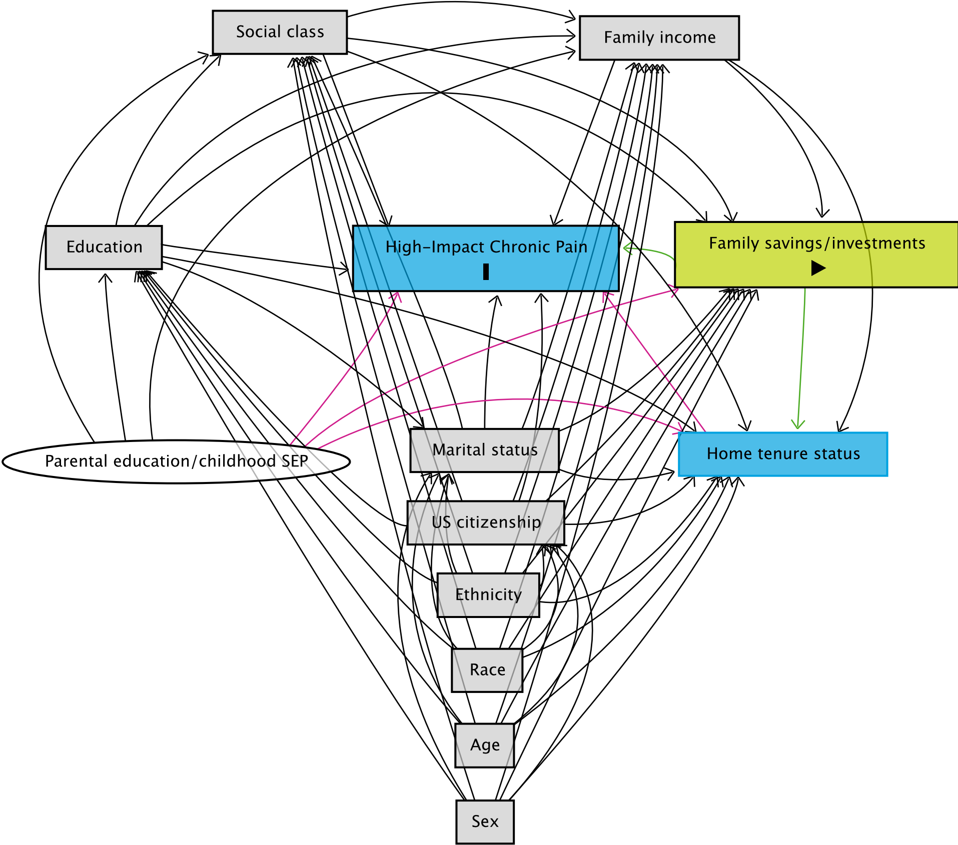
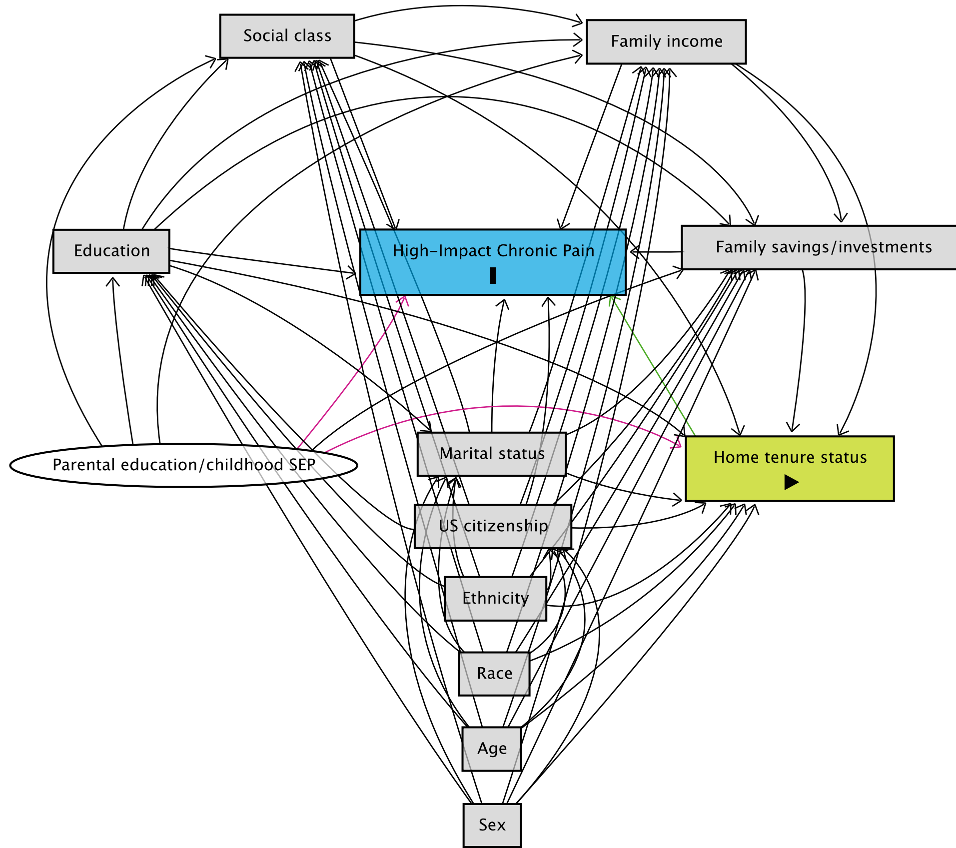


**Figure 5.** Socioeconomic pathways of pain inequities by home tenure status



#### 5.3.3. Socioeconomic inequities: HICP and LICP

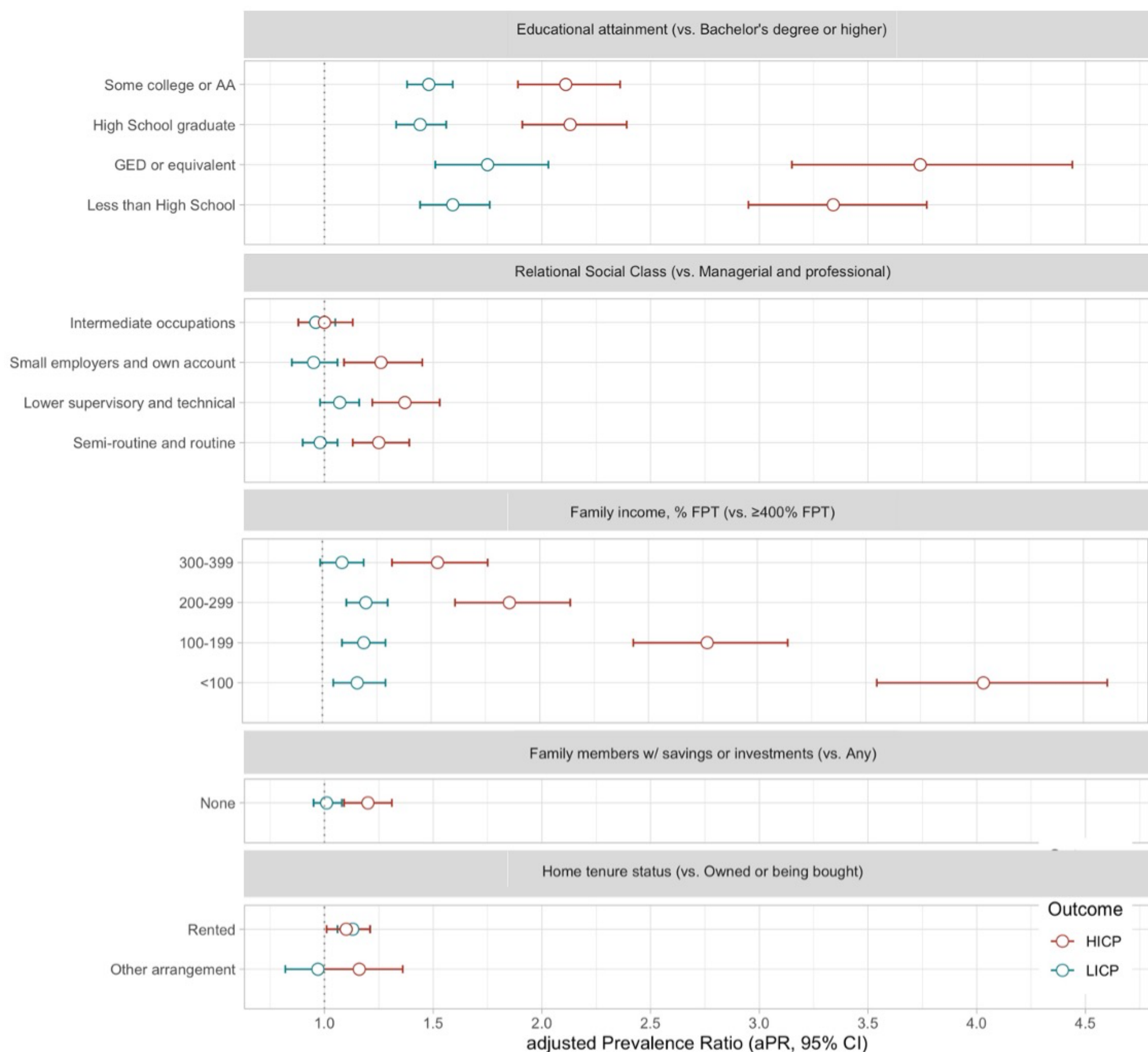
The association between socioeconomic position and pain differed substantially across exposures and outcomes (Figure 6; Suppl. Table 1). Although both HICP and LICP showed generally higher burden of pain with lower socioeconomic positions, the estimates for HICP were of markedly greater magnitude, especially for education and family income. For instance, the prevalence of HICP among adults with a GED or equivalent education was 3.74 times (95% CI: 3.15, 4.44) the prevalence of adults with a bachelor's degree or higher, adjusting for demographic characteristics. The largest estimate for LICP was for this same educational contrast; the prevalence of LICP in adults with GED or equivalent was 1.75 times (95% CI: 1.51, 2.03) the prevalence in adults with a bachelor's degree or higher

The prevalence of HICP among adults with a family income below the poverty threshold was approximately 4 times the prevalence among adults with a family income ≥400% FPT (aPR=4.04, 95% CI: 3.55, 4.61), adjusting for education, social class, and demographic characteristics. Differently than for HICP, where each category higher in the family income variable was associated with a stronger association, the aPRs for the association between family income and LICP were similar for most categories (ranging from aPR=1.09, 95% CI: 0.99, 1.19 to aPR=1.20, 95% CI: 1.11, 1.30).

The estimates for measures of family wealth showed more modest effect sizes after adjusting for education, social class, and family income. For example, adults who reported living in rented homes showed a similarly small elevation in burden of HICP (aPR=1.10, 95% CI: 1.01, 1.21) and LICP (aPR=1.13, 95% CI: 1.06, 1.21) relative to adults who owned or were buying their homes. Adults with no family members with savings or investments had a slightly higher burden of HICP than adults with had any (aPR=1.20, 95%CI: 1.09,

1.31). There was no difference in burden of LICP by the presence of family members with savings or investments (aPR=1.02, 95%CI: 0.95, 1.08). Likewise, in contrast to the association between social class and HICP (previously presented in Section 4), the estimates for the association between social class and LICP were very close to null.

**Figure 6.** Forest plot with estimates and 95% confidence intervals of the associations of socioeconomic characteristics with high-impact chronic pain and low-impact chronic pain in US adults ≥25 years who had ever worked in civilian occupations, NHIS 2016-2017.




AA=Associate's degree; FPT=Federal poverty threshold; HICP=High-impact chronic pain; LICP=Low-impact chronic pain.

### 5.4. Discussion

Our results comparing the associations between HICP and LICP are consistent with the literature suggesting that SEP is not only associated with chronic pain prevalence, but it is also associated with higher pain-related impact on activities (Janevic et al., 2017). We extend these findings to a large nationally representative sample adults ≥25 years. The large magnitude of estimates for association between SEP and HICP, especially family income and education, are striking. In contrast with the magnitude of LICP, estimates suggest that SEP may be more strongly associated with pain impact than with pain presence or duration (i.e., pain for 6 months or longer). These findings suggest that policy points of entry related to preventing unequal consequences (Diderichsen et al., 2001) of pain may be of special interest for future investigation.

We do acknowledge, however, that the exact time since chronic pain onset in our sample is unknown; we cannot rule out that adults with HICP have also had longer duration of their pain. The onset and progression of pain-related disability is not well characterized (e.g., chronic pain starts as LICP and progresses to HICP, vs. HICP starts as HICP). Additionally, some of the observed effects may be due to reverse causation, with HICP limiting SEP. This could be especially prominent for HICP relative to LICP due to potential impact on work. Likewise, the bias in our analysis due to effects of pain on SEP may be more relevant for downstream SEP variables due to probability of pain preceding the determination of that aspect of SEP. Although pain as a cause of SEP was not the focus of this analysis, this research avenue warrants follow-up. This causal direction would also have important implications on poverty prevention and interrupting intergenerational transmission of health inequities.

DAG-informed regression modeling is a powerful tool for stimulating causal thinking, identifying causal pathways of interest, selecting covariates for adjustment, and interpreting estimates. For instance, researchers often present mutually adjusted models for multiple exposures. However, if these exposures have causal relationships among them, the estimates adjusting for mediators would eliminate part of the total effect (Assari, 2013). Making these relationships explicit, such as by presenting DAGs, can prevent unintentional over-adjustment bias and help in the interpretation of controlled direct effects when presented intentionally. Despite our cross-sectional study design, some of our exposures of interest are consistently defined prior to others, forming well documented causal chains (e.g., education, occupation, income) (Mirowsky and Ross, 2003). Therefore, a DAG-informed approach supports estimation and interpretation of the total associations of interest.

Additional study limitations include imperfect or missing measurements of the constructs of interest. For example, the variable for family members with savings or investments was far from an ideal measure of family wealth. Instead of considering the actual amount of family assets in interest-bearing accounts, it may be subject to 'noise' due to family size and financial literacy/education. Childhood SEP, often measured as parental education, are thought to influence development with implications to life course neurological, immune, endocrine, cognitive, emotional, behavioral, and social outcomes, including socioeconomic position, stress reactivity, social support systems, mental health, and pain sensitivity (Denk et al., 2014; Eller-Smith et al., 2018; McLaughlin, 2016; Murray et al., 2020). Unfortunately, indicators of early life SEP are not available in the present data, leaving unmeasured confounding in our analyses. That is, the estimates for each of the measures of socioeconomic position also include differences in pain outcomes due to childhood SEP.

Although all models contain some unmeasured confounding by childhood SEP, the estimates for the associations between pain and more downstream socioeconomic exposures are hypothesized to be the least affected by this bias. For example, in the model for the association between home tenure status and pain, confounding by childhood SEP only occurs through direct effects of childhood SEP on pain and home tenure status. On the other hand, in the estimate for the association between education and pain, confounding by childhood SEP may occur through direct effects of childhood SEP on education and pain, but also through indirect effects of childhood SEP on social class, family income, family savings or investments, and home tenure status. Future analyses could employ quantitative bias analysis to estimate the magnitude of the associations by childhood SEP (or other unmeasured confounders) necessary to nullify the observed results.

While we did not aim to quantify racial pain inequities, we included race and ethnicity in our analyses to adjust for confounding by differences in life experiences, and social conditions between groups in our racialized society, which has exposed people to different risks and opportunities based on arbitrary physical appearance characteristics, to justify power hierarchies and uphold White Supremacy (Boyd et al., 2020; Ford and Airhihenbuwa, 2010; Jones, 2018). These categories serve as crude proxy measures for exposure to racism and have no bearing on biological differences other than those that result from centuries of exposure to systems of oppression and privilege (Boyd et al., 2020). Nevertheless, racialization has well-documented impact on socioeconomic position in the United States (Bailey et al., 2020), as well as on the experience and treatment of pain (Brown et al., 2018; Dugan et al., 2017; Meints et al., 2019).

Racial and ethnic categories contain people with distinct ancestry, national origin, cultural and ethnic background, therefore, subgroups may experience systematically different racialization depending on place and time. However, sample size limitations and data confidentiality precluded more fine-grained disaggregation in analyses, with implications for health equity (e.g., 'no data, no problem'). For example, one of the OMB standard race categories, 'Native Hawaiians or other Pacific Islander', was completely suppressed from the NHIS 2016-2017 data, merged with other racial groups, or presented as 'race not releasable'.

Finally, socioeconomic position and racism are entangled in a network of mutually reinforcing systems of privilege and oppression, such as sexism, ableism, ageism, heterosexism, gender binarism, nativism, etc. (Krieger, 2020). These interactions result in profound differences in the exposures and impact of social determinants of health within and between social groups due to intersectionality (Crenshaw, 1991; Jackson et al., 2016). The differences in pain inequities at the intersections of race, sex and socioeconomic position are further explored in Section 7.

## 5.5. Appendix

### 5.5.1. DAGitty code for causal models of socioeconomic pain inequities

```
dag {
bb=”0,0,1,1”
“Family income” [pos=”0.679,0.114”]
“Family savings/investments” [pos=”0.805,0.333”]
“High-Impact Chronic Pain” [outcome,pos=”0.540,0.337”]
“Home tenure status” [pos=”0.778,0.557”]
“Marital status” [adjusted,pos=”0.539,0.553”]
“Parental education/childhood SEP” [latent,pos=”0.292,0.565”]
“Social class” [pos=”0.375,0.108”]
“US citizenship” [adjusted,pos=”0.540,0.630”]
Age [adjusted,pos=”0.539,0.867”]
Education [exposure,pos=”0.234,0.337”]
Ethnicity [adjusted,pos=”0.542,0.707”]
Race [adjusted,pos=”0.541,0.787”]
Sex [adjusted,pos=”0.539,0.948”]
“Family income” -> “Family savings/investments” [pos=”0.816,0.229”]
“Family income” -> “High-Impact Chronic Pain”
“Family income” -> “Home tenure status” [pos=”0.923,0.292”]
“Family savings/investments” -> “High-Impact Chronic Pain” [pos=”0.699,0.338”]
“Family savings/investments” -> “Home tenure status” [pos=”0.796,0.390”]
“Home tenure status” -> “High-Impact Chronic Pain”
“Marital status” -> “Family income” [pos=”0.593,0.451”]
“Marital status” -> “Family savings/investments” [pos=”0.667,0.497”]
“Marital status” -> “High-Impact Chronic Pain” [pos=”0.539,0.438”]
“Marital status” -> “Home tenure status” [pos=”0.641,0.596”]
“Marital status” -> “Social class” [pos=”0.507,0.442”]
“Parental education/childhood SEP” -> “Family income” [pos=”0.238,0.229”]
“Parental education/childhood SEP” -> “Family savings/investments” [pos=”0.510,0.464”]
“Parental education/childhood SEP” -> “High-Impact Chronic Pain” [pos=”0.460,0.423”]
“Parental education/childhood SEP” -> “Home tenure status” [pos=”0.565,0.456”]
“Parental education/childhood SEP” -> “Social class” [pos=”0.088,0.258”]
“Parental education/childhood SEP” -> Education [pos=”0.235,0.408”]
“Social class” -> “Family income” [pos=”0.521,0.054”]
“Social class” -> “Family savings/investments” [pos=”0.712,0.155”]
“Social class” -> “High-Impact Chronic Pain”
“Social class” -> “Home tenure status” [pos=”0.684,0.251”]
“US citizenship” -> “Family income” [pos=”0.627,0.375”]
“US citizenship” -> “Family savings/investments” [pos=”0.678,0.496”]
“US citizenship” -> “High-Impact Chronic Pain” [pos=”0.591,0.511”]
“US citizenship” -> “Home tenure status” [pos=”0.678,0.634”]
“US citizenship” -> “Social class” [pos=”0.440,0.293”]
“US citizenship” -> Education [pos=”0.441,0.636”]
Age -> “Family income” [pos=”0.674,0.363”]
Age -> “Family savings/investments” [pos=”0.646,0.606”]
Age -> “Home tenure status” [pos=”0.698,0.711”]
```

```
Age -> “Marital status” [pos=”0.444,0.715”]
Age -> “Social class” [pos=”0.405,0.271”]
Age -> “US citizenship” [pos=”0.656,0.744”]
Age -> Education [pos=”0.420,0.694”]
Education -> “Family income” [pos=”0.356,0.102”]
Education -> “Family savings/investments” [pos=”0.521,-0.001”]
Education -> “High-Impact Chronic Pain” [pos=”0.272,0.334”]
Education -> “Home tenure status” [pos=”0.618,0.428”]
Education -> “Marital status” [pos=”0.383,0.394”]
Education -> “Social class” [pos=”0.256,0.217”]
Ethnicity -> “Family income” [pos=”0.657,0.293”]
Ethnicity -> “Family savings/investments” [pos=”0.657,0.538”]
Ethnicity -> “Home tenure status” [pos=”0.656,0.728”]
Ethnicity -> “Marital status” [pos=”0.502,0.642”]
Ethnicity -> “Social class” [pos=”0.417,0.241”]
Ethnicity -> “US citizenship” [pos=”0.589,0.695”]
Ethnicity -> Education [pos=”0.449,0.673”]
Race -> “Family income” [pos=”0.666,0.321”]
Race -> “Family savings/investments” [pos=”0.653,0.565”]
Race -> “Home tenure status” [pos=”0.672,0.722”]
Race -> “Marital status” [pos=”0.475,0.706”]
Race -> “Social class” [pos=”0.423,0.299”]
Race -> “US citizenship” [pos=”0.619,0.721”]
Race -> Education [pos=”0.431,0.679”]
Sex -> “Family income” [pos=”0.686,0.359”]
Sex -> “Family savings/investments” [pos=”0.666,0.603”]
Sex -> “Home tenure status” [pos=”0.738,0.657”]
Sex -> “Marital status” [pos=”0.421,0.707”]
Sex -> “Social class” [pos=”0.435,0.535”]
Sex -> “US citizenship” [pos=”0.681,0.743”]
Sex -> Education [pos=”0.415,0.726”]
}
```

### 5.5.2. Supplemental Tables

**Supplemental Table 1.** Associations of measures of socioeconomic position with high and low impact chronic pain in US adults ≥25 years who had ever worked in civilian occupations, NHIS 2016-2017.

| | High-Impact Chronic Pain (HICP) | Low-Impact Chronic Pain (LICP) |
|---|---|---|
| | aPR (95% CI) | |
| **Educational attainment** | | |
| Bachelor's degree or higher | 1.00 (Reference) | |
| Some college or Associate degree | 2.11 (1.89, 2.36) | 1.48 (1.38, 1.59) |
| High School Graduate | 2.13 (1.91, 2.39) | 1.44 (1.33, 1.56) |
| GED or equivalent | 3.74 (3.15, 4.44) | 1.75 (1.51, 2.03) |
| Less than High School | 3.34 (2.95, 3.77) | 1.59 (1.44, 1.76) |
| **Social Class, NS-SEC** | | |
| Managerial and professional | 1.00 (Reference) | |
| Intermediate occupations | 1.00 (0.88, 1.13) | 0.96 (0.88, 1.05) |
| Small employers and own account owners | 1.26 (1.09, 1.45) | 0.96 (0.86, 1.07) |
| Lower supervisory and technical occupations | 1.37 (1.22, 1.53) | 1.07 (0.98, 1.16) |
| Semi-routine and routine workers | 1.25 (1.13, 1.39) | 0.98 (0.90, 1.06) |
| **Family income as percent of FPT** | | |
| ≥400% FPT | 1.00 (Reference) | |
| 300-399% FPT | 1.53 (1.32, 1.76) | 1.09 (0.99, 1.19) |
| 200-299% FPT | 1.86 (1.61, 2.14) | 1.20 (1.11, 1.30) |
| 100-199% FPT | 2.77 (2.43, 3.14) | 1.19 (1.09, 1.29) |
| <100% FPT | 4.04 (3.55, 4.61) | 1.16 (1.05, 1.29) |
| **Family members with savings or investments** | | |
| Any | 1.00 (Reference) | |
| None | 1.20 (1.09, 1.31) | 1.01 (0.95, 1.08) |
| **Home tenure status** | | |
| Owned or being bought | 1.00 (Reference) | |
| Rented | 1.10 (1.01, 1.21) | 1.13 (1.06, 1.21) |
| Other arrangement | 1.16 (0.99, 1.36) | 0.97 (0.82, 1.15) |

aPR: adjusted prevalence ratio; CI: confidence interval. NS-SEC= NS-SEC= U.K. National Statistics Socio-Economic Classification. High-impact and low-impact chronic pain models n=50,357; pop. est.=199,014,173. Prevalence ratio estimates were obtained using survey-weighted Poisson regression. Models were adjusted for race, ethnicity, sex, marital status, US citizenship, region, as well as all socioeconomic characteristics listed above them on the table (i.e., social class model adjusted for education; family income model adjusted for education and social class; family members with savings or investments model adjusted for education, social class, and family income; home tenure status model adjusted for education, social class, family income, and family members with savings or investments).

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# 6. Socioeconomic pain inequities in US adults: Site-specific pains

## 6.1. Introduction

The anatomically defined structure of healthcare specialty training in Western countries has likely contributed to a fragmentation of the study of pain literature. Despite growing interdisciplinary collaboration in the field, and evidence that comorbidity between pain at different body sites in the same individuals are quite common, a substantial proportion of the pain literature remains fragmented into single site-specific pain siloes. Mechanisms that are often used to explain overlap in different pain disorders include individual differences in central pain processing (e.g., deficit in endogenous pain modulation, cognitive, affective, and behavioral).

As in investigations into the etiology of pain as a whole, less attention has been devoted to the role of social and contextual determinants in shaping the burden pain comorbidity patterns. Our conceptual framework presented in Section 2 has detailed pathways through which different aspects of SEP, for example, may contribute as 'causes of causes' of pain. SEP may contribute to individual vulnerability to pain (such as the differences in central pain processing mentioned above), but also to differential exposure to pain, and differential impact of pain. We aimed to characterize the distribution of site-specific pains among adults with and without HICP, and quantify the associations between SEP and site-specific pains, using a DAG-informed regression approach.

## 6.2. Methods

We estimated the association between SEP and site-specific pains (i.e., low back pain, severe headaches or migraines, facial pain, limiting arthritis, and number of site-specific pains [0-5]) informed by the causal model of socioeconomic pain inequities presented in

Section 5.3.2. We maintained the five SEP exposure variables and DAG-informed covariate selection presented for the analysis of the primary outcome of HICP in Section 5.

We obtained prevalence ratios (PR) and 95% confidence intervals (CI) using Poisson regression for binary outcomes. Additionally, we investigated the association between the five measures of SEP and having higher numbers of site-specific pains as an ordinal outcome. We tested for violations of the proportional odds assumption for each category of the exposures (i.e., that the estimated prevalence odds ratios [POR] of having a higher number of site-specific pains were the same for all values of the outcome [>0, >1, >2, >3, >4]) using the Brant test (Long and Freese, 2014). Since there was statistical evidence that the assumption had been violated for all models, we used generalized ordinal logistic regression (Williams, 2006; Liu and Koirala, 2012), which relaxes the proportional odds assumption. We obtained POR and 95% CI, for having more than zero, one, two, three, and four site-specific pains by levels of SEP, adjusting for covariates.

All estimates and 95% CI are produced using NHIS-provided complex survey weights. Due to technical limitations, the Brant test was performed in non survey weighted models. Figure 1 was produced using the R package eulerr (Larsson, 2020), and Figures 2-6 were produced using the R package ggplot2 (Wickham, 2016).

## 6.3. Results

### 6.3.1. Site-specific pains and HICP

There was a high degree of overlap between site-specific pains, especially among adults with HICP (Figure 1). Low back pain was the most common site-specific pain, among adults with and without HICP (Table 1). Limiting arthritis and facial pain were especially more

common among adults with HICP than without HICP. Among adults with HICP, only 6.0% did not report any of the five assessed site-specific pains (low back pain, neck pain, severe headaches or migraines, facial pain, limiting arthritis), compared to 58.9% among adults without HICP. Likewise, 50.2% of adults with HICP reported having pain in 3 to 5 of these sites, compared to 6.4% of adults without HICP (Table 1).

### 6.3.2. Socioeconomic inequities: site-specific pain outcomes

The magnitude of the associations between measures of SEP and site-specific pain was generally similar between pain outcomes. Facial pain and limiting arthritis estimates (Table 2) were consistently slightly above the estimates for low back pain and severe headaches and migraines (Table 3); the largest effect sizes were for adults with GED or equivalent education (facial pain aPR= 2.09, 95% CI: 1.70, 2.71; limiting arthritis aPR=2.02, 95% CI: 1.78, 2.29) and adults with family income <100% FPT (facial pain aPR=2.34, 95% CI: 1.98, 2.77; limiting arthritis aPR=2.48, 95% CI: 2.27, 2.70). The small non-gradient patterns in education and social class were mostly maintained. For social class, the only category whose 95% CIs did not include the null value across all pain sites (i.e., pain burden higher than adults in 'managerial and professional occupations') was 'lower supervisory and technical occupations' (ranging from aPR=1.10, 95%CI: 1.01, 1.20 to aPR=1.29, 95%CI: 1.11, 1.51).

**Figure 1.** Area-proportional Euler diagrams of prevalence and overlap of site-specific pains by high-impact chronic pain status

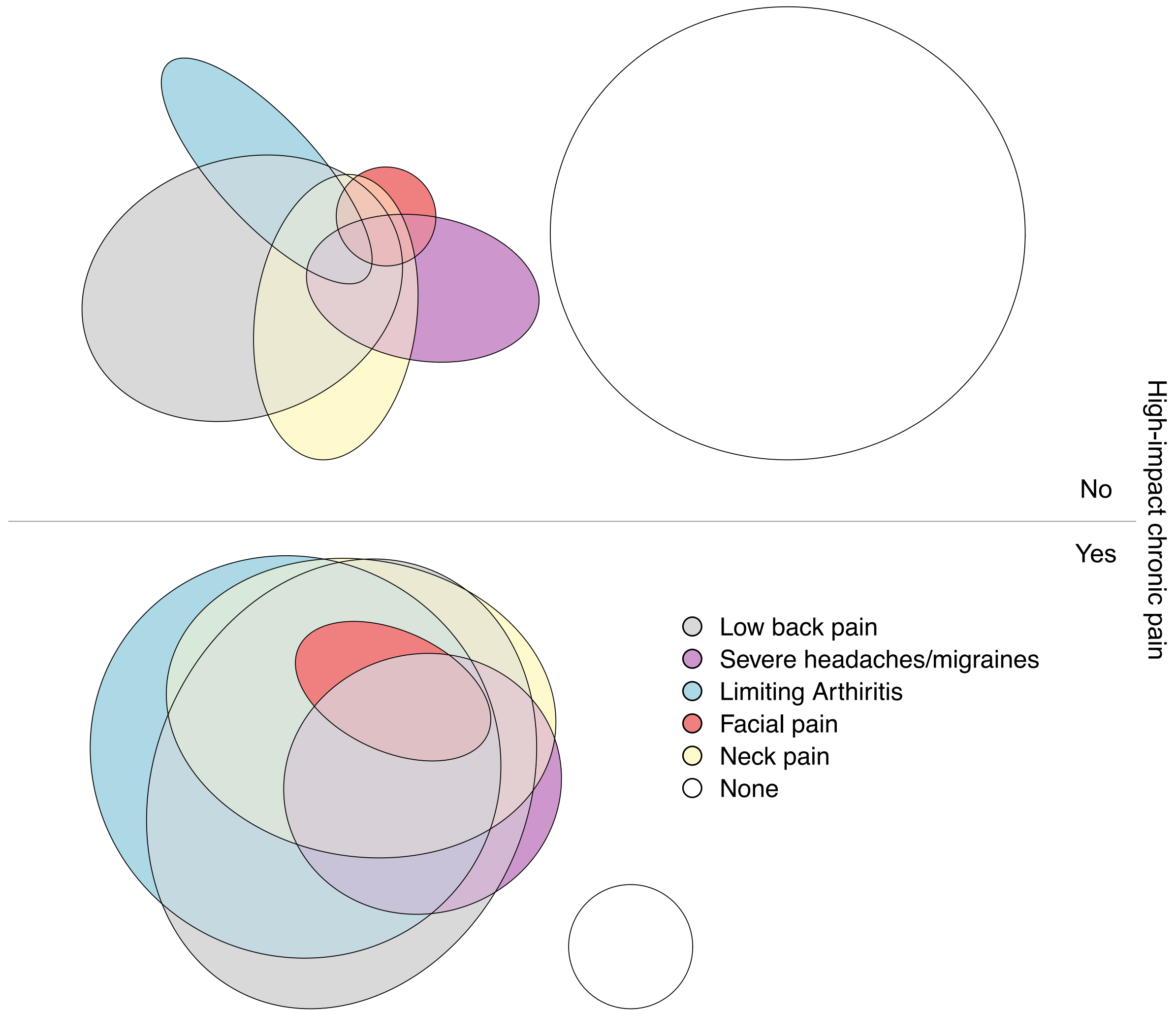


**Table 1.** Distribution of site-specific pains by high-impact chronic pain status among adults ≥25 years who had ever worked in civilian occupations, NHIS 2016-2017.

| | Overall | High-impact Chronic Pain | |
|---|---|---|---|
| | | No | Yes |
| | pop. est.= 199,014,173 | pop. est.= 182,404,639 | pop. est.= 16,609,534 |
| **Site-specific pain, % (95% CI)** | | | |
| **Low back pain** | | | |
| Yes | 30.6 (30.0, 31.3) | 26.6 (26.0, 27.2) | 74.7 (73.1, 76.4) |
| No | 69.4 (68.7, 70.0) | 73.4 (72.8, 74.0) | 25.3 (23.3, 26.9) |
| **Severe headaches or migraines** | | | |
| Yes | 15.0 (14.5, 15.4) | 13.1 (12.7, 13.6) | 35.2 (33.4, 37.0) |
| No | 85.0 (84.6, 85.5) | 86.9 (86.4, 87.3) | 64.8 (63.0, 66.6) |
| **Facial pain** | | | |
| Yes | 4.6 (4.4, 4.9) | 3.6 (3.4, 3.8) | 15.9 (14.6, 17.1) |
| No | 95.4 (95.1, 95.6) | 96.4 (96.2, 96.6) | 84.1 (82.9, 85.4) |
| **Neck pain** | | | |
| Yes | 16.5 (16.0, 17.0) | 13.3 (12.9, 13.8) | 51.1 (49.1, 53.0) |
| No | 83.5 (83.0, 84.0) | 86.7 (86.2, 87.1) | 48.9 (47.0, 50.9) |
| **Limiting arthritis** | | | |
| Yes | 15.2 (14.7, 15.7) | 9.9 (9.5, 10.3) | 73.6 (71.9, 75.2) |
| No | 84.8 (84.3, 85.3) | 90.1 (89.7, 90.5) | 26.4 (24.8, 28.1) |
| **Number of site-specific pains** | | | |
| 0 | 54.5 (53.7, 55.3) | 58.9 (58.1, 59.7) | 6.0 (5.0, 7.0) |
| 1 | 23.6 (23.0, 24.1) | 23.9 (23.3, 24.5) | 19.4 (17.9, 21.0) |
| 2 | 12.0 (11.6, 12.4) | 10.9 (10.5, 11.3) | 24.4 (22.7, 26.1) |
| 3 | 6.2 (5.9, 6.5) | 4.5 (4.2, 4.7) | 25.8 (24.0, 27.5) |
| 4 | 2.9 (2.7, 3.1) | 1.6 (1.4, 1.7) | 17.3 (15.8, 18.8) |
| 5 | 0.8 (0.7, 0.9) | 0.3 (0.2, 0.3) | 7.1 (6.2, 8.1) |

Overall n=50,357; Without high-impact chronic pain (HICP) n=45,764; with HICP n=4,593.
CI=Confidence interval.

**Table 2.** Associations of measures of socioeconomic position with low back pain and limiting arthritis in US adults ≥25 years who had ever worked in civilian occupations, NHIS 2016-2017.

| | Facial pain | Limiting arthritis |
|---|---|---|
| | aPR (95% CI) | |
| **Educational attainment** | | |
| Bachelor's degree or higher | 1.00 (Reference) | |
| Some college or Associate degree | 1.43 (1.32, 1.60) | 1.45 (1.35, 1.56) |
| High School Graduate | 1.00 (0.89, 1.16) | 1.42 (1.31, 1.53) |
| GED or equivalent | 2.09 (1.70, 2.71) | 2.02 (1.78, 2.29) |
| Less than High School | 1.55 (1.31, 1.82) | 1.91 (1.74, 2.09) |
| **Social Class, NS-SEC** | | |
| Managerial and professional | 1.00 (Reference) | |
| Intermediate occupations | 1.01 (0.87, 1.17) | 1.00 (0.91, 1.09) |
| Small employers and own account workers | 1.11 (0.88, 1.40) | 1.09 (0.98, 1.20) |
| Lower supervisory and technical occupations | 1.29 (1.11, 1.51) | 1.18 (1.09, 1.28) |
| Semi-routine and routine workers | 1.09 (0.94, 1.26) | 1.13 (1.06, 1.21) |
| **Family income as percent of FPT** | | |
| ≥400% FPT | 1.00 (Reference) | |
| 300-399% FPT | 1.34 (1.12, 1.59) | 1.31 (1.20, 1.43) |
| 200-299% FPT | 1.53 (1.30, 1.81) | 1.43 (1.32, 1.55) |
| 100-199% FPT | 1.81 (1.54, 2.12) | 1.84 (1.71, 1.99) |
| >100% FPT | 2.34 (1.98, 2.77) | 2.48 (2.27, 2.70) |
| **Family members with savings or investments** | | |
| Any | 1.00 (Reference) | |
| None | 1.06 (0.94, 1.20) | 1.02 (0.96, 1.09) |
| **Home tenure status** | | |
| Owned or being bought | 1.00 (Reference) | |
| Rented | 1.18 (1.04, 1.34) | 1.10 (1.03, 1.18) |
| Other arrangement | 1.12 (0.86, 1.47) | 1.10 (0.97, 1.25) |

aPR: adjusted prevalence ratio; CI: confidence interval. NS-SEC= NS-SEC= U.K. National Statistics Socio-Economic Classification. Facial pain model n=50,397; limiting arthritis model n=50,374. Prevalence ratio estimates were obtained using survey-weighted Poisson regression. Models were adjusted for race, ethnicity, sex, marital status, US citizenship, region, as well as all socioeconomic characteristics listed above them on the table (e.g., social class model adjusted for education, family income model adjusted for education and social class, etc.)

**Table 3.** Associations of measures of socioeconomic position with severe headaches or migraines and facial pain in US adults ≥25 years who had ever worked in civilian occupations, NHIS 2016-2017.

| | Severe headaches or migraines | Low back pain |
|---|---|---|
| | aPR (95% CI) | |
| **Educational attainment** | | |
| Bachelor's degree or higher | 1.00 (Reference) | |
| Some college or Associate degree | 1.43 (1.33, 1.53) | 1.40 (1.34, 1.47) |
| High School Graduate | 1.37 (1.26, 1.48) | 1.40 (1.33, 1.47) |
| GED or equivalent | 1.85 (1.60, 2.14) | 1.84 (1.69, 1.99) |
| Less than High School | 1.77 (1.61, 1.95) | 1.61 (1.52, 1.71) |
| **Social Class, NS-SEC** | | |
| Managerial and professional | 1.00 (Reference) | |
| Intermediate occupations | 0.92 (0.84, 1.00) | 0.98 (0.92, 1.04) |
| Small employers and own account workers | 0.93 (0.82, 1.05) | 1.06 (1.00, 1.14) |
| Lower supervisory and technical occupations | 1.10 (1.01, 1.20) | 1.15 (1.09, 1.21) |
| Semi-routine and routine workers | 1.00 (0.93, 1.08) | 1.06 (1.01, 1.11) |
| **Family income as percent of FPT** | | |
| ≥400% FPT | 1.00 (Reference) | |
| 300-399% FPT | 1.14 (1.03, 1.27) | 1.07 (1.01, 1.13) |
| 200-299% FPT | 1.16 (1.07, 1.27) | 1.15 (1.09, 1.21) |
| 100-199% FPT | 1.44 (1.33, 1.57) | 1.33 (1.26, 1.40) |
| >100% FPT | 1.72 (1.57, 1.89) | 1.55 (1.46, 1.51) |
| **Family members with savings or investments** | | |
| Any | 1.00 (Reference) | |
| None | 1.05 (0.98, 1.12) | 1.02 (0.98, 1.06) |
| **Home tenure status** | | |
| Owned or being bought | 1.00 (Reference) | |
| Rented | 1.13 (1.06, 1.20) | 1.09 (1.05, 1.13) |
| Other arrangement | 1.14 (0.98, 1.32) | 1.12 (1.02, 1.23) |

aPR: adjusted prevalence ratio; CI: confidence interval. NS-SEC= NS-SEC= U.K. National Statistics Socio-Economic Classification. Severe headaches or migraines model n=50,401; low back pain model n=50,393. Prevalence ratio estimates were obtained using survey-weighted Poisson regression. Models were adjusted for race, ethnicity, sex, marital status, US citizenship, region, as well as all socioeconomic characteristics listed above them on the table (e.g., social class model adjusted for education, family income model adjusted for education and social class, etc.)

### 6.3.3. Socioeconomic inequities: number of site-specific pains

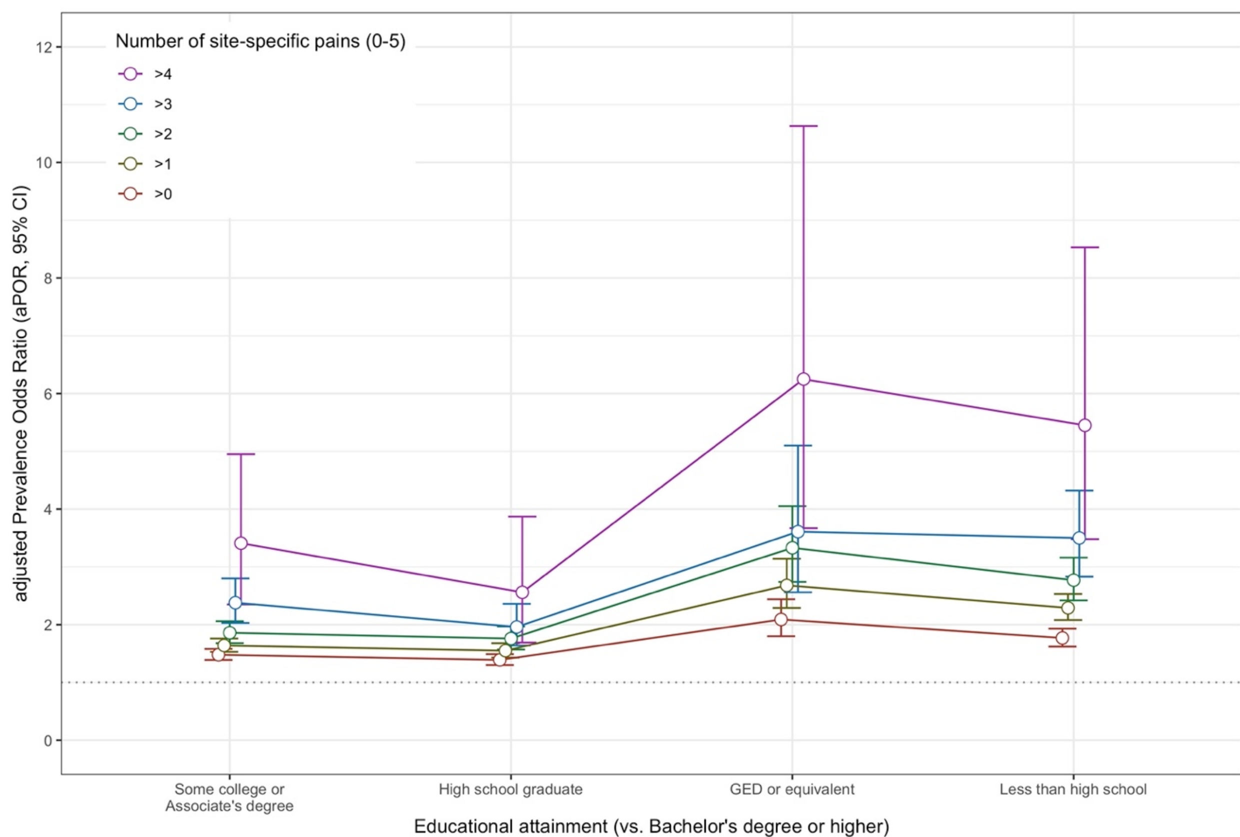
The Brant test for violation of the proportional odds assumption was significant for all levels of SEP ($p<0.001$), except for the 'other arrangement' category of home tenure status ($p=0.037$), which had the smallest sample size across all SEP categories. We plotted the adjusted prevalence odds ratios (aPOR) and 95% CI for having a higher number of site-specific pains than each of the cut points (0-4) by SEP (Figures 2-6). The magnitude of associations between SEP and number of site-specific pains was substantially greater for education (Figure 2) and family income (Figure 3) as compared to the other SEP measures. Generally, the association with number of site-specific pains was stronger (and less precise) for higher numbers of site-specific pains, and for lower SEP.

Adults in all levels of education below the reference category had higher odds of having more site-specific pains than adults with a bachelor's degree or higher, adjusting for demographic characteristics. For example, the odds of having more than four site-specific pains among adults with GED or equivalent education were 6.25 times (95% CI: 3.67, 10.63) the odds among adults with a bachelor's degree or higher. The odds of having more than four site-specific pains among adults with less than high school education were 5.45 times (95% CI: 3.48, 8.53) the odds among adults with a bachelor's degree or higher.

With regards to family income, all but one of the contrasts had higher odds of having more site-specific pains than adults with family income ≥400% FPT – the exception was adults with 300-399, whose odds of having more than four site-specific pains were no different than that of adults with ≥400% FPT (aPR=1.62, 95% CI: 0.87, 3.02). Estimates were higher for higher number of site-specific pains, especially for lower levels of family income. The magnitude of some of these estimates was very large. For example, the odds of having more

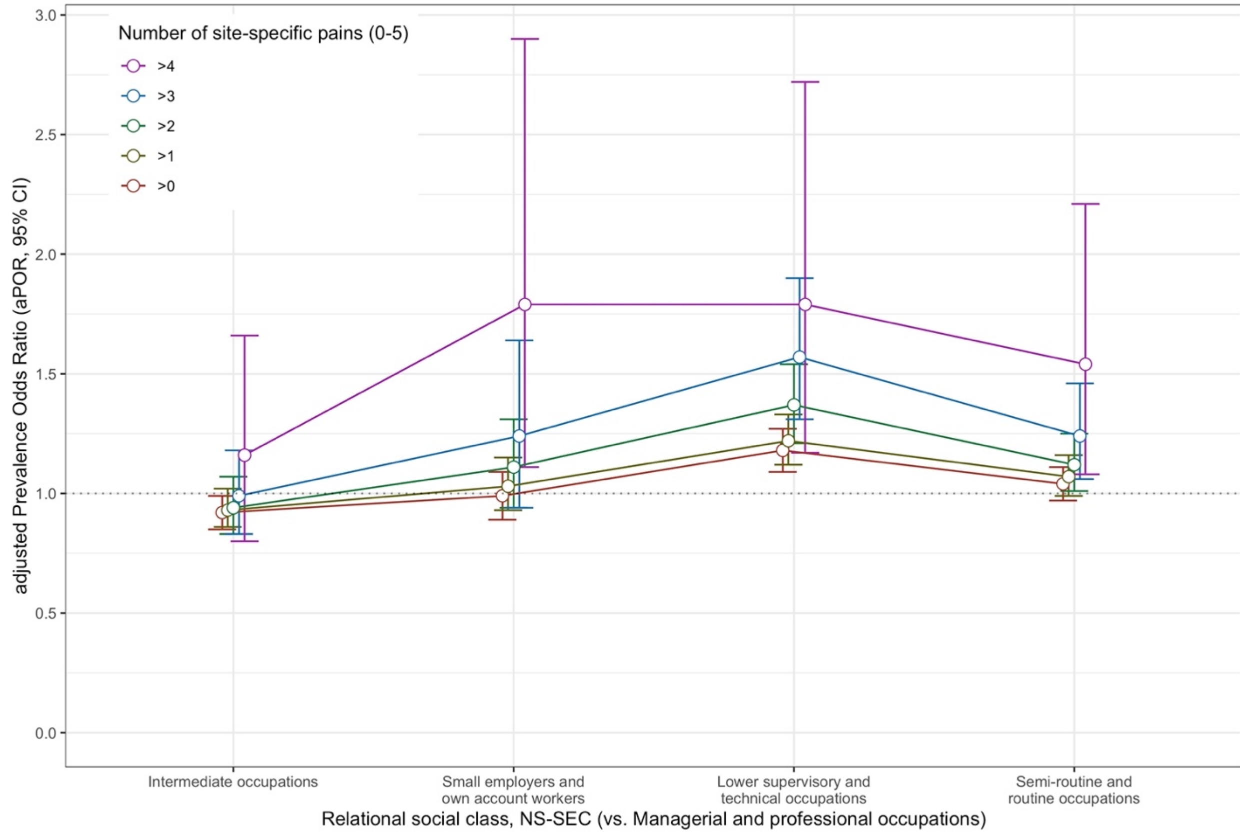
than three site specific pains among adults with family income <100% FPT were 4.17 times (95%CI: 3.39, 5.13) the odds among adults with family income ≥400% FPT, adjusting for demographic characteristics and education. The odds of having more than four site-specific pains among adults with family income <100% FPT were 7.73 times (95% CI: 4.98, 12.02) the odds among adults with family income ≥400% FPT.

**Figure 2.** Adjusted prevalence odds ratios of having more than 0-4 site-specific pains by categories of educational attainment, NHIS 2016-2017.



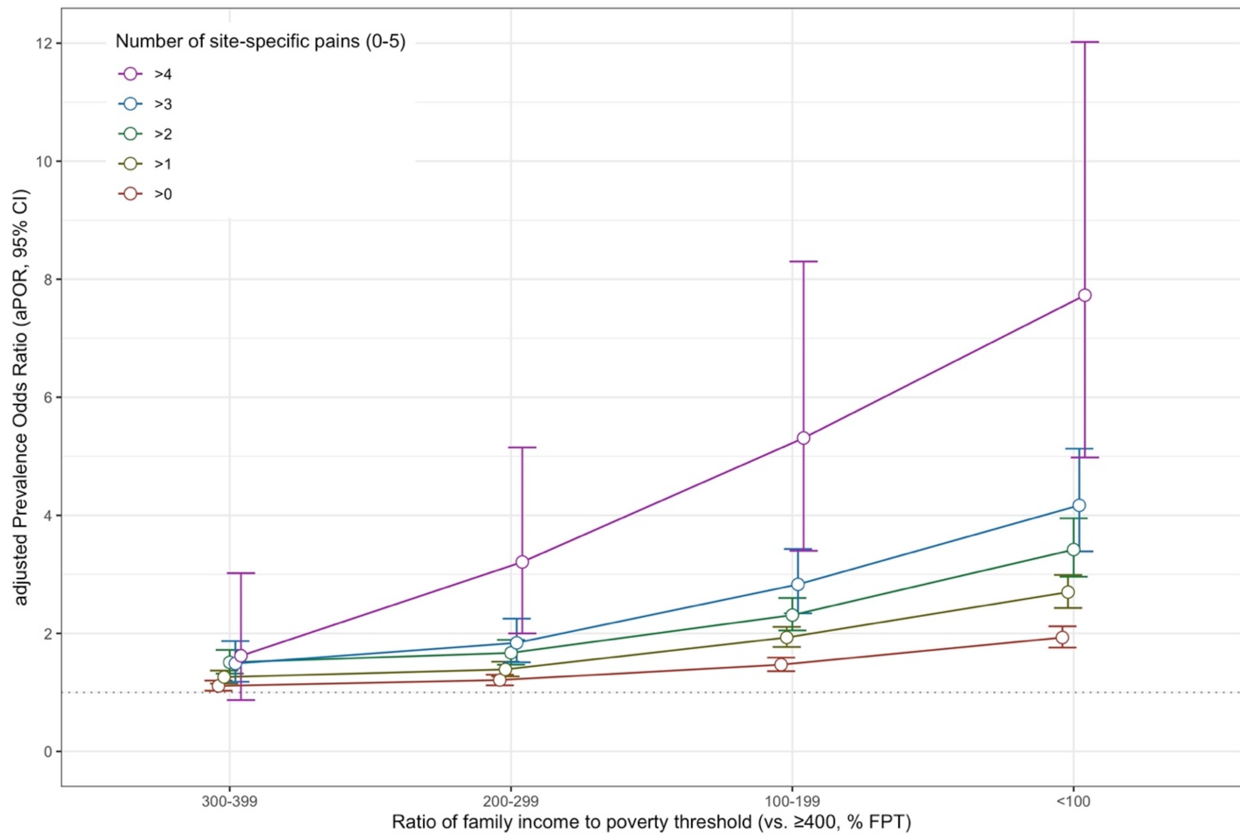

Brant test was significant for all levels of education, indicating that estimates for all cut points of number of site-specific pains [0-4] were *not* equal [i.e., violation of the proportional odds assumption] for all contrasts of educational attainment (vs. bachelor's degree or higher). CI=Confidence interval.

**Figure 3.** Adjusted prevalence odds ratios of having more than 0-4 site-specific pains by categories of relational social class, NHIS 2016-2017.



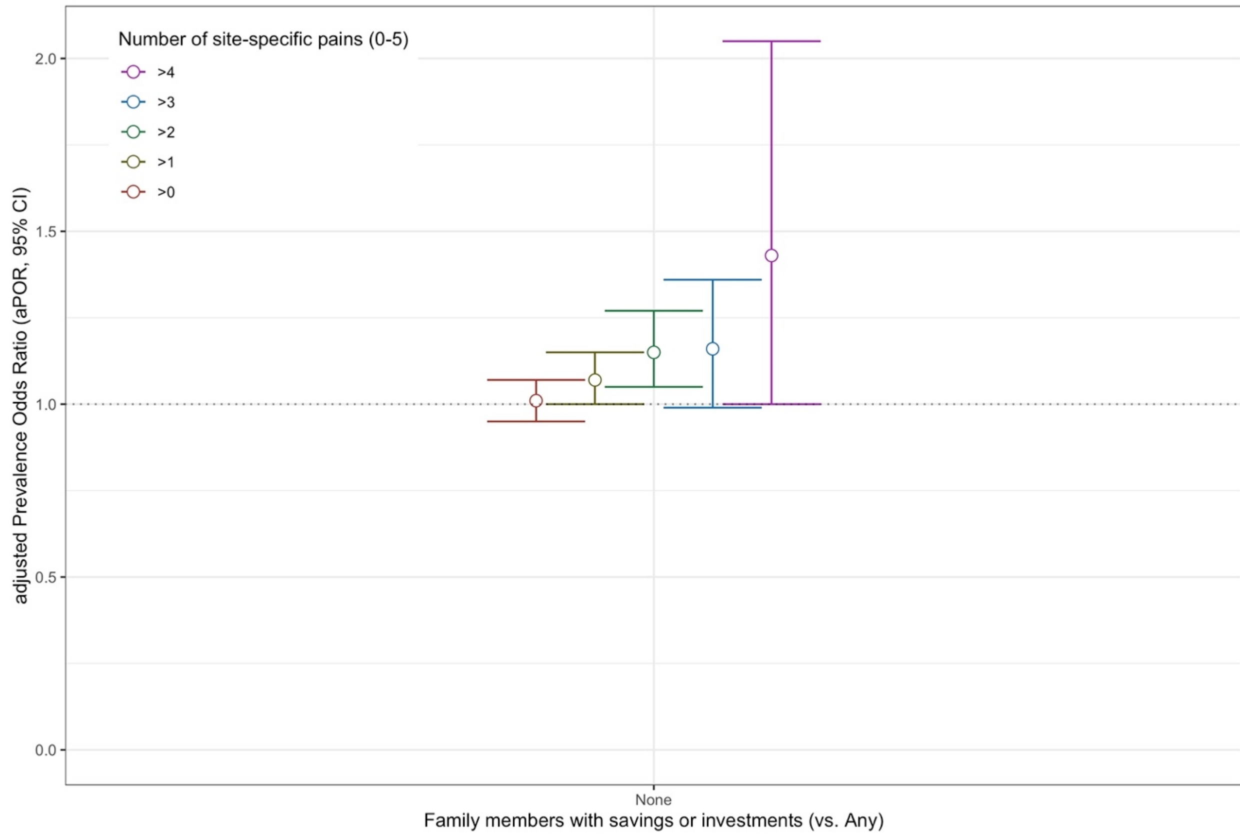

Brant test was significant for all levels of social class, indicating that estimates for all cut points of number of site-specific pains [0-4] were *not* equal [i.e., violation of the proportional odds assumption] for all contrasts of social class (vs. managerial and professional occupations). CI=Confidence interval.

**Figure 4.** Adjusted prevalence odds ratios of having more than 0-4 site-specific pains by categories of family income as a percentage of the federal poverty threshold (FPT), NHIS 2016-2017.



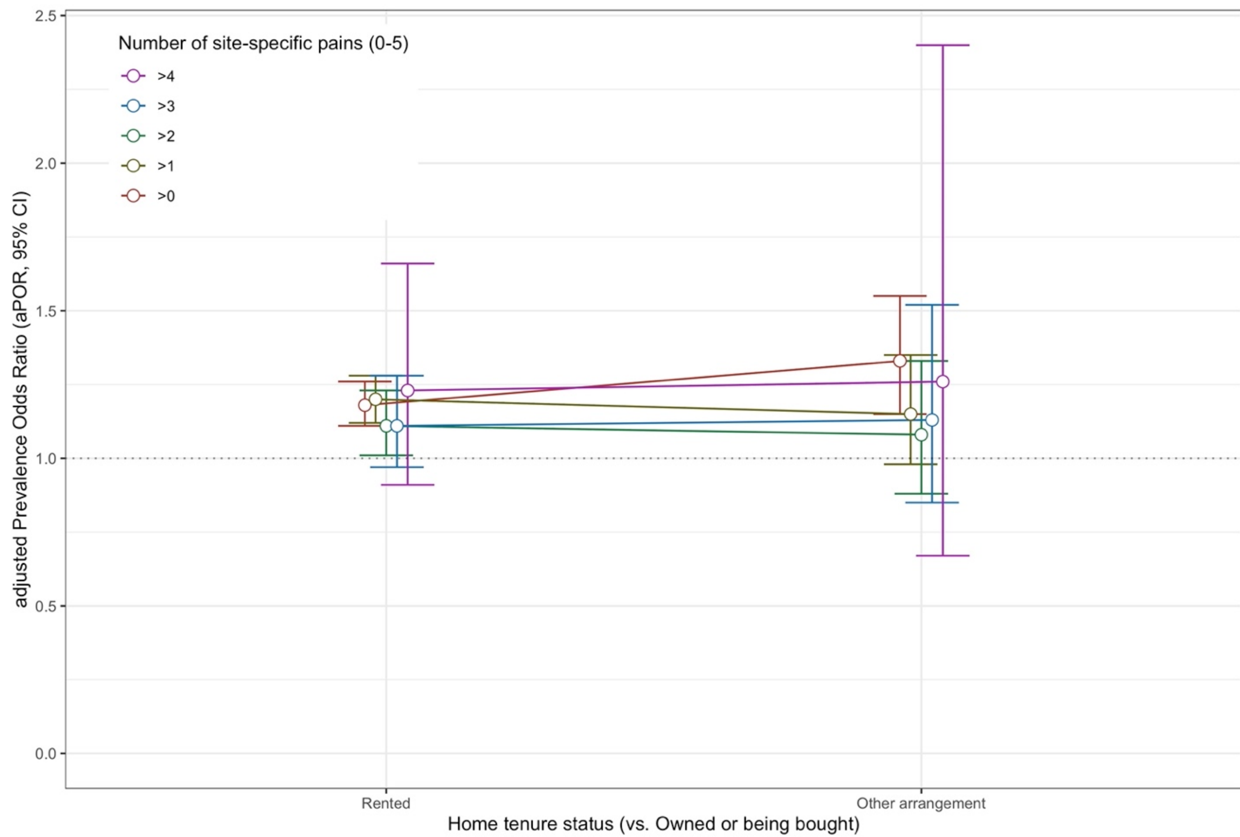

Brant test was significant for all levels of family income, indicating that estimates for all cut points of number of site-specific pains [0-4] were *not* equal [i.e., violation of the proportional odds assumption] for all contrasts of family income (vs. ≥400% FPT). CI=Confidence interval.

**Figure 5.** Adjusted prevalence odds ratios of having more than 0-4 site-specific pains by presence of family members with savings or investment, NHIS 2016-2017.




Brant test was significant, indicating that estimates for all cut points of number of site-specific pains [0-4] were *not* equal [i.e., violation of the proportional odds assumption] for the contrast of family members with savings or investments (none vs. any). CI=Confidence interval.

**Figure 6.** Adjusted prevalence odds ratio of having higher than 0-4 site-specific pains by categories of home tenure status, NHIS 2016-2017.




Brant test was significant for 'rented', but not for 'other arrangement'. A significant test indicates that estimates for all cut points of number of site-specific pains [0-4] were *not* equal [i.e., violation of the proportional odds assumption] for a contrast of a given category of the exposure with its reference category. CI=Confidence interval.

### 6.4. Discussion

There was strong evidence of socioeconomic inequities in the number of site-specific pains, with higher odds of having more site-specific pains among adults in lower SEP. The burden of number of site-specific pains was higher for lower income and lower education relative to their respective reference categories. Inequities were especially pronounced for the comparisons at higher number of site-specific pains (e.g., >3, >4).

In contrast, the effect sizes for single site-specific pains were small. Except for limiting arthritis (which had the shortest recall period of 30 days, vs. 3 months for other site-specific pain measures), site-specific pain measures did not specify minimum thresholds for pain frequency or impact. Therefore, we would expect them to be a mix of HICP, LICP, and non-chronic pain. Facial pain and limiting arthritis, which had greater representation among adults with HICP than in the overall population, presented generally higher estimates of the associations with SEP than low back pain and severe headaches or migraines.

Given the common overlap between these site-specific pains, especially among adults with HICP, and strong evidence of association between SEP and number of site-specific pains, the single site-specific estimates should be interpreted with caution. Importantly, that is also true of much of the literature that examines such outcomes. Based on estimates of the association between SEP and four site-specific pains (i.e., low back pain, severe headaches or migraines, facial pain, and limiting arthritis), our findings suggest that SEP is likely more strongly associated with number of site-specific pains and pain impact than with pain location.

Mutual adjustment for pain sites and/or number of site-specific pains in the efforts to handle their comorbidity would also be potentially problematic. The comparison within groups with the same status site-specific pain status assumes no interaction between them; however, we have presented evidence of stronger SEP inequities with each additional pain site. The issue of comorbidities – which also includes frequently associated non-painful conditions such as sleep and mood disorders – reinforces the need for methodological advances to enhance study of causal inference and interventions for multimorbidity.

Complex systems modelling has been suggested as a promising approach (Hammond et al., 2017)

Although there was statistical evidence (Brant test) of difference between estimates for different cut points of number of site-specific pains across all but one of the SEP categories, some of these differences did not seem to be qualitatively meaningful. In particular, future iterations of this work may consider whether home tenure status and family members with savings or investments would be adequately modelled with a 'conventional' ordinal logistic regression (i.e., proportional odds cumulative logit) for simplicity purposes.

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# 7. Intersectionality and social inequities in high-impact chronic pain

## 7.1. Introduction

In most of health research, social constructs such as race, socioeconomic position (SEP), and gender are conceptualized as individual characteristics, which often leads to the framing of health differences between social groups as individual differences. The term 'individual differences' may be appropriate to refer to inequalities due to characteristics that are randomly distributed across individuals and populations. Health inequities, in the other hand, are by definition avoidable, unnecessary, and unjust (Whitehead, 1992); such is the case of differences in health resulting from unequal distribution of risks and resources between socially defined groups due to power and privilege differentials.

The utility of reporting health inequity findings – especially when social group membership is measured at the individual level – is greatly bolstered by interpretations that highlight fundamental social causes of illness (Phelan et al., 2010). Otherwise, the individual framing of social differences in health obscures and protects the group and societal-level political, economic, and social structures that shape individual exposures, vulnerability, and resources to manage health (Bowleg, 2019). Presenting health inequities as individual differences without social context may also reinforce conceptions of biological essentialism (i.e., claims of inherent superiority and/or normality of dominant groups) (Krieger, 2020) and be used to promote narratives of ill health (and chronic pain) as a personal deficit (Adler and Stewart, 2010). Individual differences in pain have been defined as "between-person differences in the pain experience that are independent of the initiating stimulus" (Fillingim, 2017). While pain is determined by how one's biological endowments are "sculpted by a mosaic of factors that is completely unique to each individual at a given point in time" (Fillingim, 2017), social group memberships may be the bases for systematic

exposure to experiences and conditions that increase risk and prognosis of high-impact pain.

In Section 2, we presented a multilevel framework of pain etiology and impact, with an emphasis on the societal and systemic factors as the fundamental causes of large extant pain inequities. We highlighted pathways of embodiment through which the social context may shape individual psychological and biological risk and resilience factors for pain. This theoretical framework aims to contextualize these ‘individual differences’ and stimulate research revealing potential targets for intervention on social determinants of pain, health and social equity, at the levels of organization at which they operate (e.g., interpersonal, groups, communities, societies).

Health inequity-generating social structures are formed by mutually reinforcing systems of privilege and disadvantage (e.g., racism, sexism, classism, heterosexism, ableism, nativism, etc.) (Bowleg, 2012; Krieger, 2020). The concept of intersectionality accounts for combinations of social positions that one may embody, which simultaneously and interdependently shape life experiences and conditions according to hierarchies of power (Crenshaw, 1991; Jones, 2014). In intersectionality approaches, the highlighting of heterogeneity of experiences between people with different overlapping social positions speaks to the ‘uniqueness’ aspect of the ‘individual differences’ approach, with a critical lens pointing to the multilevel forces outside of the individual that are responsible for social stratification and health inequities.

The development of quantitative methods aligned with intersectionality principles is an area of active research (Bauer et al., 2021). In this section, we will describe the distribution

of high-impact chronic pain (HICP) in US adults at different intersections of social positions and explore novel quantitative methods to investigate intersectionality in HICP inequities. Specifically, we aimed to estimate joint HICP disparities by race/ethnicity and SEP and its components using intersectional decomposition analysis (Jackson et al., 2016). This method seeks to highlight unique disparity patterns that are obscured by investigating social determinants independently. Identifying patterns of interaction may help to clarify etiological mechanisms at play, as well as inform pathways for social change, policy, and population health interventions, prioritizing targets with greater potential impact for population health and health equity.

### 7.1.1. Intersectional decomposition analysis

The intersectional decomposition methods described by Jackson and colleagues (Jackson et al., 2016; Jackson and VanderWeele, 2019, 2018) highlight the role of multiple axis of marginalization in the observed disparity patterns. We use the term 'disparity' from here on to align with the language used by the authors, and to refer generally to differences in health that may or may not be aligned with power differentials between groups. Intersectional decomposition analysis aims to expose gaps in health outcomes across populations at different intersections of social positions.

The authors argue that most studies that aim to quantify intersectional effects rely on multiplicative interaction terms and overlook the absolute impact of disparities on population health (Jackson et al., 2016). Furthermore, studies often fail to report on the magnitude of disparities resulting from simultaneously belonging to more than one marginalized social category (Jackson et al., 2016). That is, (two-way) multiplicative interaction terms compare, in the relative scale, the magnitude of disparity along one axis

of marginalization between strata of (i.e., adjusting for) another axis of marginalization. Studies that focus on this measure may neglect to report the absolute risk or burden of illness among persons who belong to both marginalized categories, as well as its contrast with the those who do not belong to any of these categories. This contrast (in the additive scale), termed the *joint disparity* by Jackson et al., and its components quantify absolute differences in population health that would be expected in the absence of disparities.

Jackson et al. outline a simple approach to present the mean health outcomes for each category defined by two axes of marginalization, as well as measures of disparity in the absolute (i.e., additive) scale. Although the proposed measures can be obtained with straightforward statistical methods, the innovation of this approach is in the conceptualization of the additive interaction and the effect of each axis of marginalization among the referent group along the other axis as components of a joint intersectional disparity, as well as a framework for their interpretation under a population health equity lens. Intersectional decomposition is advocated as a descriptive method, which does not require assumptions about causal relationships between the social category labels and the outcomes in question (Jackson et al., 2016). Because it does not seek to identify or test specific causal mechanisms underlying disparities, it should be used in conjunction with additional theory, and qualitative and quantitative methods for the development of interventions to eliminate health disparities.

Nonetheless, the joint disparity and its components can have important policy value: they may reveal subgroups with disproportionately high or low disease burden, indicating directions for investigation into the social systems that interact to create such results. Further, they quantify the potential population health outcomes in the absence of each of

the disparity components (Jackson et al., 2016). We aimed to describe patterns of HICP distribution by race/ethnicity and SEP simultaneously, using the absolute measures of intersectional disparities proposed by the decomposition method, which speak to potential population impact of health equity interventions .

## 7.2. Methods

The original publication (Jackson et al., 2016) presents the decomposition method using four categories defined by combinations of two binary variables (i.e., race, black vs. white; and early life SES, low vs. high). We use similar intersections between race/ethnicity and SEP but extend the application of the method to multiple categories (beyond binary variables) along these same two axes of marginalization.

The data source for this analysis is the same as presented in Section 3, the National Health Interview Survey (NHIS), combining years 2016 and 2017. We limited the analysis to adults ≥25 years, and excluded adults with missing data for the study variables, described below.

### 7.2.1. Key variables

**Our first social identity of interest was race/ethnicity, defined with the following groups:**

- Race/ethnicity:
    - Non-Hispanic White (Reference)
    - Non-Hispanic Black
    - Hispanic
    - Non-Hispanic Asian

We sought to expand the "coverage" of racialized categories of US adults going beyond the original black and white dichotomy presented in the original decomposition example (Jackson et al., 2016). Additionally, we balanced this expansion with parsimony in the number of categories (and model parameters), to retain the interpretability that comes with the simplicity of their approach. The variable we selected for this analysis is a recode based on self-reported ethnicity (Hispanic or Latino vs. Not Hispanic or Latino) and self-reported "primary race". That is, when asked what race or races the respondent considered themselves to be, persons who selected more than one out of the sixteen options (i.e., White, Black/African American, Indian [American], Alaska Native, Native Hawaiian, Guamanian or Chamorro, Samoan, Other Pacific Islander, Asian Indian, Chinese, Filipino, Japanese, Korean, Vietnamese, Other Asian, or Some other race) was asked about which one of these groups would they say best represents their race.

The category 'Hispanic' includes adults of all races who reported being of Hispanic or Latino ethnicity. Sensitivity analysis conducted for Section 5 analysis revealed that approximately 88.4% of adults who reported Hispanic or Latino ethnicity selected 'White' as their only racial category. We excluded adults who were in the race/ethnicity category of 'Non-Hispanic All other race groups' due to their small sample size and ambiguous interpretation. The use of self-reported "primary race" may enhance the validity of this measure in capturing the impact of racism on health, to the extent that people primarily identify with the racial category that corresponds to their racialized experiences and exposures in life.

The Non-Hispanic White category was selected as the reference category for race/ethnicity since it represents the racial group with the most power and privilege in

our society. Their social context could be conceptualized as the counterfactual for racial/ethnic minorities in the absence of racism.

SEP is represented primarily by family income, based on the magnitude of associations estimated in Sections 4-6. As secondary analyses, we substitute education for SEP. Each of them is discussed below:

- Family income as percent of the US Census Bureau federal poverty threshold (FPT) (referred to below as 'family income'):
    - ≥400% FPT (reference)
    - 200-399% FPT
    - 100-199% FPT
    - <100% FPT

Family income categories were defined with cutoff points based on previous literature (Persmark et al., 2020; Singh et al., 2017). Respondents are asked about income and earnings from all sources (e.g., wages, salaries, Social Security, or retirement benefits, help from relatives, etc.) for themselves and each member of the family before taxes during the past calendar year (Division of Health Interview Statistics National Center for Health Statistics (NCHS), 2017). After responding to a series of questions about income and earnings from different sources, respondents are then asked to estimate the total combined family income, used in the calculation of the family income as a percent of FPT. The more detailed income responses are not used to estimate total combined income in NHIS, but they serve to improve participant recall (Division of Health Interview Statistics National Center for Health Statistics (NCHS), 2017). Respondents that did not report exact values for family income were asked about progressively detailed questions about categories of

family income, which helped to improve the precision of imputation (Division of Health Interview Statistics National Center for Health Statistics (NCHS), 2017).

The category of ≥400% of the FPT was selected as the reference category for family income since it represents the group with the most power and privilege in our society. Their social context and resources could be conceptualized as the counterfactual SEP for the groups with lower family income in the absence of socioeconomic disadvantage.

- Educational attainment:
    - Bachelor's degree or higher (reference)
    - Some college or associate's degree
    - High school graduate
    - Less than high school, general educational development (GED) or equivalent

These categories were built based on a detailed variable with twenty-two levels of educational attainment. Cut off points were determined based on previous literature (Zajacova et al., 2020) and the results of our analysis in Section 5, where we combined categories with similar effect sizes (i.e., 'less than high school' and 'GED or equivalent') for the association between education and HICP. The category of bachelor's degree or higher was selected as the reference category for education since it represents the group with the most power and opportunities in our society. Their social context and resources could be conceptualized as the counterfactual SEP for the groups with lower educational attainment in the absence of educational inequities.

We investigated the intersections of race/ethnicity and SEP using family income and education separately. For each of these two measures of SEP, we obtained sixteen categories from combinations of four race/ethnicity and four SEP levels. These categories were used as a basis for comparison in the analysis, with Non-Hispanic White adults with the highest SEP considered the overall reference categories. The outcome of interest was high-impact chronic pain (HICP: 'yes', 'no'), defined as pain that limits life or work activities on 'most days' or 'every day' for 6 months or longer (Dahlhamer et al., 2018; Interagency Pain Research Coordinating Committee, 2016). Sex ('male', 'female') and age ('25-34 years', '35-44 years', '45-54 years', '55-64 years', '65-74 years', '75-84 years', '≥85 years'), were used as adjustment variables.

#### 7.2.2. Statistical analysis

Since our outcome of interest, HICP, was binary and not rare in many of the comparison groups, we fit log-binomial regression to model the associations between race/ethnicity (coefficients $\beta_{1\text{-}3}$) and SEP (either family income or education, coefficients $\beta_{4\text{-}6}$), interactions between race/ethnicity and SEP (coefficients $\beta_{7\text{-}15}$), adjusted for age (coefficients $\beta_{16}$) and sex . Indicator variables were used to model all variables flexibly, per the following equation:

$$Log(P\ HICP=1 \mid SEP,\ race/ethnicity*sex,\ age)=$$

$$\beta_0 + \beta_1 X_{Hispanic} + \beta_2 X_{NH\text{-}Asian} + \beta_3 X_{NH\text{-}Black} +$$

$$\beta_4 X_{SEP\ 1} + \beta_5 X_{SEP\ 2} + \beta_6 X_{SEP\ 3} + \cdot$$

$$\beta_7 X_{SEP\ 1*Hispanic} + \beta_8 X_{SEP\ 2*Hispanic} + \beta_9 X_{SEP\ 3*Hispanic} + \beta_{10} X_{SEP\ 1*NH\text{-}Asian} + \beta_{11} X_{SEP\ 3*NH\text{-}Asian} +$$

$$\beta_{12} X_{SEP\ 2*NH\text{-}Asian} + \beta_{13} X_{SEP\ 1*NH\text{-}Black} + \beta_{14} X_{SEP\ 2*NH\text{-}Black} + \beta_{15} X_{SEP\ 3*NH\text{-}Black} +$$

$$\beta_{16} X_{35\text{-}44\ years} + \beta_{17} X_{45\text{-}54\ years} + \beta_{18} X_{55\text{-}64\ years} + \beta_{19} X_{65\text{-}74\ years} + \beta_{20} X_{75\text{-}84\ years} + \beta_{21} X_{\geq 85\ years} + \beta_{22} X_{Female}$$

We then computed age and sex-adjusted predicted probabilities of HICP, and obtained the mean ($\hat{\mu}$) for each intersectional category of race/ethnicity and SEP. Finally, we computed differences in mean predicted probabilities between groups, corresponding to the joint disparities, referent race/ethnic disparities, referent socioeconomic disparities, and excess intersectional disparities (Jackson et al., 2016), described in detail below. We obtained standard error (SE) for each of these estimates using non-parametric bootstrap with 5000 replications. We flagged unreliable estimates according to 2017 National Center for Health Statistics (NCHS) data presentation standards for proportions (Parker et al., 2017).

The adjusted mean predicted probabilities of HICP ($\mu$) among adults ≥25 years were defined as follows:

$\mu_{00} = \mu$ for adults of non-Hispanic White race/ethnicity and highest SEP. The 'doubly privileged' category.

$\mu_{01} = \mu$ for adults of non-Hispanic White race/ethnicity and lower SEP.

$\mu_{10} = \mu$ for adults of minority race/ethnicity and highest SEP.

$\mu_{11} = \mu$ for adults of minority race/ethnicity and lower SEP. The 'doubly marginalized' category.

Generally, the first subscript represents race/ethnicity (0= reference category, non-Hispanic White; 1= race/ethnicity minority category). The second subscript represents SEP (family income: 0=reference category, ≥400% FPT; 1=lower income category; education: 0= reference category, bachelor's degree or higher, 1=lower education category). We compared all possible combinations of racial/ethnic minority and lower SEP categories (e.g., non-Hispanic Asian and family income 100-199% FPT; Hispanic and family income 200-399%

FPT, etc.) with the reference categories of Non-Hispanic White and highest SEP (≥400% FPT or bachelor's degree or higher).

Differences in group means were then defined as the following measures of disparity:

$\mu_{11} - \mu_{00}$ **= Joint disparity by race/ethnicity and SEP**

When considering two axes of marginalization, the joint disparity describes the total difference between those who belong to both disadvantaged categories (e.g., race/ethnic minority, lower SEP), relative to those who belong to both privileged categories (e.g., non-Hispanic White, highest SEP).

The burden of HICP across different intersectional social positions is further described by measures of disparity associated with belonging to one disadvantaged category while belonging to a privileged position along the other axis (e.g., non-Hispanic White and <100% FPT; non-Hispanic Black and bachelor's degree or higher) relative to the doubly privileged reference category:

$\mu_{01} - \mu_{00}$ **= Referent socioeconomic disparity**

The referent socioeconomic disparity measures differences in the outcome by SEP among those in the reference group for race/ethnicity, who are unlikely to be subject of racism in the U.S.

$\mu_{10} - \mu_{00}$ **= Referent race/ethnicity disparity**

The referent race/ethnicity disparity measures differences in the outcome by race/ethnicity among those in the reference group for SEP, who are in a privileged socioeconomic position in the U.S.

When subtracting the two referent disparities from the joint disparity, we obtain the excess intersectional disparity:

$(\mu_{11} - \mu_{00}) - [(\mu_{01} - \mu_{00}) + (\mu_{10} - \mu_{00})]$, which can be re-written as:

$\mu_{11} - \mu_{10} - \mu_{01} + \mu_{00}$ = **Excess intersectional disparity**

The excess intersectional disparity is a measure of additive interaction. It quantifies the component of the joint disparity that arises in the presence of both disadvantaged conditions, beyond what is accounted for by race/ethnicity and SEP independently. The excess intersectional disparity simultaneously refers to the difference in socioeconomic HICP disparity for adults of different race/ethnicities, and the difference in race/ethnicity HICP disparity for adults of difference SEP.

The disparity components are presented on their own as absolute differences in adjusted predicted probability of HICP, and as a percentage of the joint disparity. Positive disparity values reflect *worse* outcomes for the disadvantaged group, while negative disparity values reflect *better* outcomes for the disadvantaged group. Regardless of their sign and magnitude, the sum of the referent race/ethnicity disparity, referent SEP disparity, and excess intersectional disparity will be equal to the joint disparity. The sum of their percentage of the joint disparity will be equal to one.

As in the original references for the intersectional decomposition method (Jackson et al., 2016; Jackson and VanderWeele, 2019, 2018), survey weights were not applied. Additionally, we used a single imputation of family income (data set 1). These were due to technical limitations in the implementation of analysis of the present study. We compared

the distribution of our main variables of interest in weighted and unweighted samples to qualitatively assess the magnitude of potential selection bias.

## 7.3. Results

### 7.3.1. Sample characteristics

Of 54,418 sample adults aged ≥25 years in the NHIS 2016-2017, 100 (0.2%) had missing data on HICP, 195 (0.4%) had missing data on educational attainment, and 770 (1.4%) reported 'non-Hispanic other race'. There was no missing data for age, sex, and race/ethnicity. Some respondents had more than one of these exclusion criteria, and total exclusions corresponded to 1,054 adults or 1.9% of the eligible sample. The final study sample size was 53,364 adults. Family income was imputed by NHIS for approximately 16.5% of this sample (i.e., 12.3% income originally reported in categories, 4.2% reported no information for total family income).

The study population was made-up mostly of adults of non-Hispanic White race/ethnicity (72.6%) (Table 1). The reference categories for SEP also contained the greatest proportion of adults (family income ≥400% FPT=41.1%; bachelor's degree or higher education=34.4%). The reference category for joint disparities by race/ethnicity and family income, non-Hispanic White adults with family income ≥400% FPT, accounted for 33.5% of the sample. This was a greater proportion than all family income levels for all race/ethnicity minority categories combined. The reference category for joint disparities by race/ethnicity and education, non-Hispanic White adults with bachelor's degree or higher education, was also the largest category (26.9%), followed closely by non-Hispanic White adults with some college or associate's degree (22.4%). Non-Hispanic Black and Hispanic adults were mostly evenly distributed across SEP categories of family income and education, whereas non-

Hispanic White and non-Hispanic Asian adults had a steep gradient with higher proportion among higher SEP levels. The correlation between family income and education categories was 0.44.

**Table 1.** Distribution of adults ≥25 years across categories defined by the intersections of race/ethnicity and socioeconomic position.

| N (%) | NH-White | | NH-Black | | Hispanic | | NH-Asian | | Total | |
|---|---|---|---|---|---|---|---|---|---|---|
| *Family income, % FPT* | | | | | | | | | | |
| <100 | 3,157 | (5.9) | 1,398 | (2.6) | 1,385 | (2.6) | 271 | (0.5) | 6,211 | (11.6) |
| 100-199 | 6,129 | (11.5) | 1,488 | (2.8) | 1,670 | (3.1) | 374 | (0.7) | 9,661 | (18.1) |
| 200-399 | 11,558 | (21.7) | 1,643 | (3.1) | 1,678 | (3.1) | 664 | (1.2) | 15,543 | (29.1) |
| ≥400 | 17,895 | (33.5) | 1,363 | (2.6) | 1,347 | (2.5) | 1,344 | (2.5) | 21,949 | (41.1) |
| Total | 38,739 | (72.6) | 5,892 | (11.0) | 6,080 | (11.4) | 2,653 | (5.0) | 53,364 | (100.0) |
| *Education* | | | | | | | | | | |
| <HS or GED | 4,154 | (7.8) | 1,250 | (2.3) | 2,123 | (4.0) | 258 | (0.5) | 7,785 | (14.6) |
| HS Graduate | 8,298 | (15.5) | 1,433 | (2.7) | 1,273 | (2.4) | 429 | (0.8) | 11,433 | (21.4) |
| Some College or AA | 11,939 | (22.4) | 1,869 | (3.5) | 1,514 | (2.8) | 476 | (0.9) | 15,798 | (29.6) |
| ≥BS/BA | 14,348 | (26.9) | 1,340 | (2.5) | 1,170 | (2.2) | 1,490 | (2.8) | 18,348 | (34.4) |
| Total | 38,739 | (72.6) | 5,892 | (11.0) | 6,080 | (11.4) | 2,653 | (5.0) | 53,364 | (100.0) |

NH = non-Hispanic; FPT = federal poverty threshold; HS = High School; AA = associate's degree; ≥BS/BA = bachelor's degree or higher. Unweighted number of persons in study sample. Cells sum up to the total study sample size separately for each of the blocks of categories for family income and education.

There were qualitative (i.e., not tested statistically) differences in age and sex distributions between categories defined by race/ethnicity and SEP (Suppl. Table 2). The overall mean age (standard deviation [SD]) was 53.8 (16.9) years, generally lower for Hispanic adults and higher for non-Hispanic White adults of all socioeconomic positions. For non-Hispanic White and non-Hispanic Black adults, mean age was higher with lower categories of family

income from ≥400% down to 100-199% FPT, but the lowest family income category, <100% FPT, had the lowest mean age among family income categories for those race/ethnicities. In contrast, the lowest family income category had the highest mean age among non-Hispanic Asian adults. For all race/ethnicity categories, lower education categories had higher mean age, although this pattern was not as consistent among Hispanic adults.

Overall, 55.0% of respondents reported female sex (Suppl. Table 2). Across all race/ethnicity categories, proportion of female sex was consistently higher with lower family income, especially among non-Hispanic Black and Hispanic adults. Among non-Hispanic adults there was a similar inverse relationship between proportion of female sex and educational attainment. On the other hand, proportion female was generally higher among higher education categories for non-Hispanic Black, non-Hispanic White, and Hispanic adults.

#### 7.3.2. HICP burden across intersectional categories of race/ethnicity and SEP

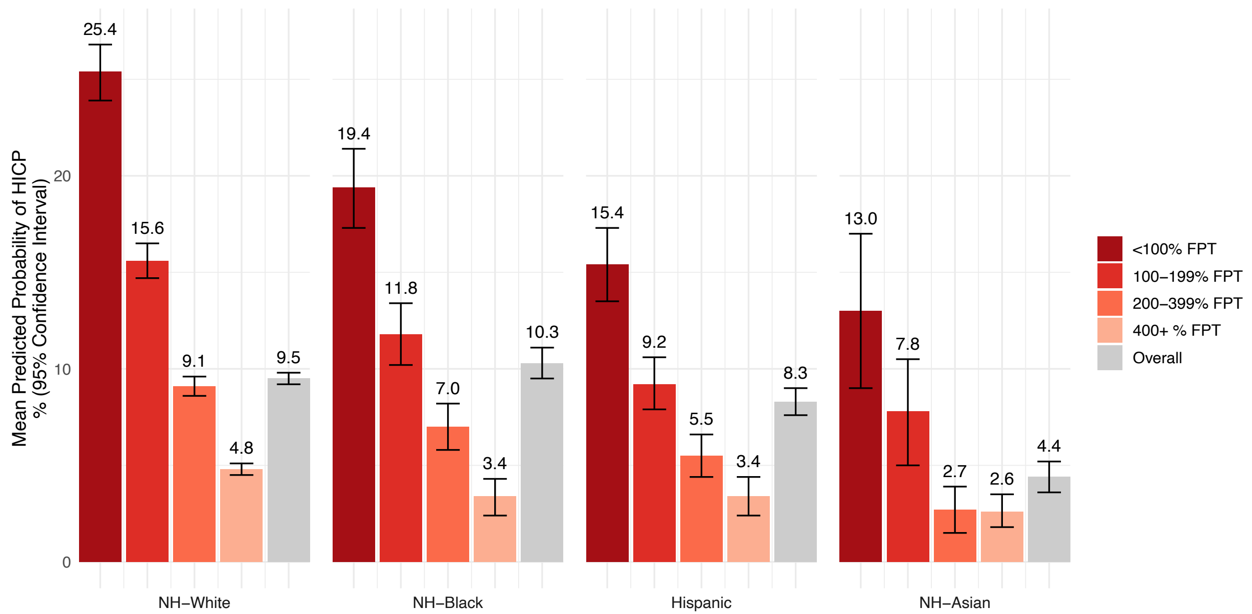
The overall mean predicted probability of HICP (standard error [SE]), adjusting for age and sex, was 9.2% (0.1). Since the present data are cross-sectional, we will refer to the means as the group burden (as opposed to risk, for example, which could be interpreted as implying a causal relationship). There was wide variation in HICP burden between groups defined by race/ethnicity and family income (Figure 1). Non-Hispanic Asian adults with bachelor's degree or higher had the lowest burden of HICP (2.3% [0.4]), while non-Hispanic White adults with family income ≤100% FPT had the highest burden of HICP (25.4% [0.8]). When combining adults by race/ethnicity regardless of SEP categories, non-Hispanic Black (10.3% [0.4]), non-Hispanic White (9.5% [0.2]), and Hispanic (8.3% [0.4]) adults had somewhat similar burden of HICP, while non-Hispanic Asian had markedly lower burden (4.4% [0.4]).

Categories with lower family income had higher burden of HICP overall and across all race/ethnicities.

For education, the burden of HICP was similar for high school graduates and adults with some college or associate's degree when combining all race/ethnicity categories (Figure 2). However, this resulted from opposite patterns between the Hispanic group and all other race/ethnicities. Among Hispanics there was a non-monotonic pattern, where the burden of HICP was higher among those with some college or associate's degree compared to high school graduates as well as those with bachelor's degree or higher. Conversely, high school graduates had higher burden of HICP than adults with some college or associate degrees among non-Hispanic White, non-Hispanic Black, and non-Hispanic Asian adults – within these race/ethnicities, there was a social gradient across all levels of education, with higher burden of HICP for lower education categories.

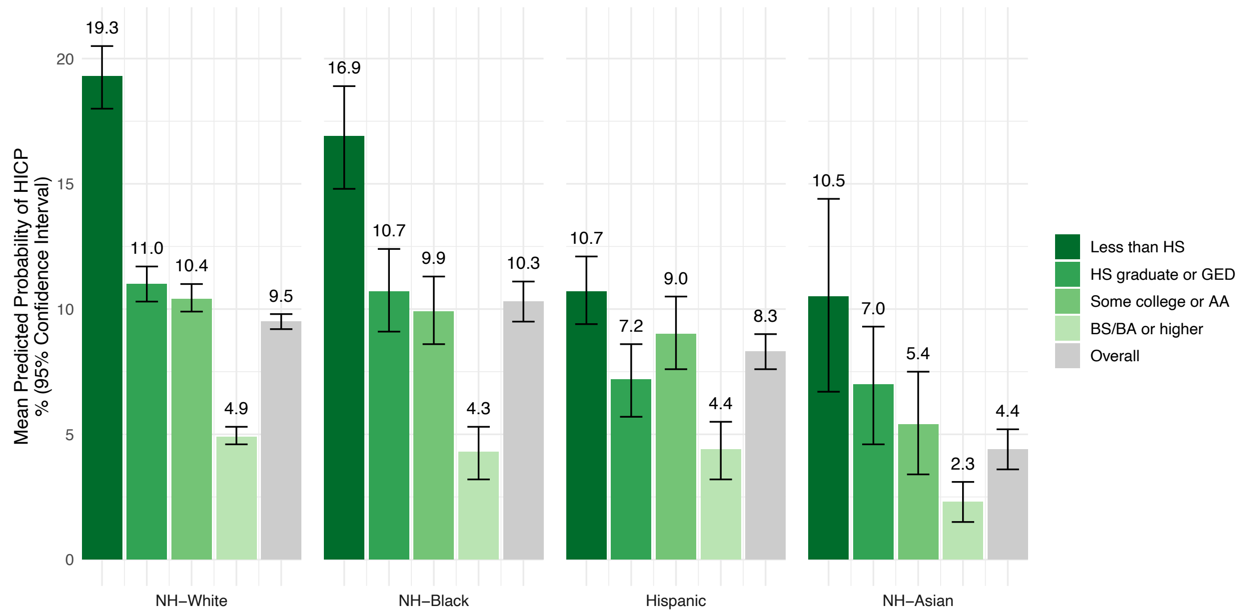
Non-Hispanic White adults had the highest burden across all levels of SEP, despite their relative race/ethnicity privilege.

**Figure 1.** Mean predicted probability of high-impact chronic pain by intersectional categories of race/ethnicity and family income as percent of the federal poverty threshold, adjusted for age and sex.




HICP = high-impact chronic pain; NH = non-Hispanic; FPT = federal poverty threshold. 95% confidence intervals obtained from bootstrap standard error with 5000 replications.

**Figure 2.** Mean predicted probability of high-impact chronic pain by intersectional categories of race/ethnicity and educational attainment, adjusted for age and sex.




HICP = high-impact chronic pain; NH = non-Hispanic; HS = High School; AA = associate's degree; BS/BA = bachelor's degree. 95% confidence intervals obtained from bootstrap standard error with 5000 replications.

#### 7.3.3. Decomposition of joint disparities by race/ethnicity and SEP

As hypothesized based on power and resource differentials, there were positive joint disparities in HICP for all race/ethnicity minority categories with lower family income levels, except for non-Hispanic Asian adults with family income 200-399% FPT (Table 2). That is, eight out of nine intersectional categories of minority race/ethnicity and family income less than 400% FPT, had an estimated higher burden of HICP than non-Hispanic White adults with family income ≥400% FPT. In contrast, the burden of HICP among non-Hispanic Asian adults with 200-399% FPT was 2.1 percentage points (SE=0.7) *lower* than the doubly privileged reference category. Joint disparities for all race/ethnicity minority categories were more pronounced for lower family income levels. The highest joint disparity

was a difference of 14.6 percentage points (1.1), with higher burden of HICP among non-Hispanic Black adults, relative to non-Hispanic White adults with family income ≥400% FPT. The magnitude of joint disparities was generally higher for non-Hispanic Black adults, followed by Hispanic, then non-Hispanic Asian adults.

When decomposing joint disparities into referent SEP, referent race/ethnicity and excess intersectional disparities, the components with the largest magnitude were referent SEP disparities (Table 2). That is, the largest disparities in burden of HICP were among adults in the referent race/ethnicity category (non-Hispanic White), comparing family income ≥ 400% FPT with lower family income (<100% FPT= 20.6% [0.8]; 100-199% FPT= 10.8% [0.5]; 200-399% FPT=4.3% [0.3]).

Referent race disparities were relatively small, with slightly lower burden of HICP for race/ethnicity minority categories with family income ≥400% FPT compared to non-Hispanic White adults with family income ≥400% FPT (Table 2). Among adults with referent family income (≥400% FPT), the burden of HICP was 2.2 percentage points (0.5) lower for Non-Hispanic Asian adults and 1.4 percentage points (0.5) lower for both non-Hispanic Black and Hispanic adults, relative to non-Hispanic White adults. When subtracting these referent disparities from the joint disparities, we obtain negative excess intersectional disparities that represent lower HICP burden than expected for race ethnicity minority categories, especially for non-Hispanic Asian and Hispanic adults and for lower levels of family income (Table 2). That is, adults of race/ethnicity minorities of lower family income had a lower burden of HICP than what would be expected based on the sum of disparities based on race/ethnicity and SEP alone.

For example, Hispanic adults with family income <100% FPT had a burden of HICP 10.6 percentage points (1.0) higher than non-Hispanic White adults with family income ≥400% FPT (joint disparity) (Table 2). The same family income contrast among the referent race category (non-Hispanic White) was associated with 20.6 percentage points (0.8) higher for adults with family income <100%FPT than adults with family income ≥400% FPT. If there were no race/ethnicity or intersectional disparities (only SEP disparities), we would expect the joint disparity comparing the burden of HICP between Hispanic adults of family income <100% FPT to the burden of HICP of non-Hispanic White adults of ≥400% FPT to be equal to the referent SEP disparity, 20.6 percentage points higher for Hispanic adults of family income <100% FPT. Since we observed a smaller joint disparity, referent race/ethnicity and excess intersectional disparities together are necessarily negative.

The same race/ethnicity contrast among the referent family income category (≥400%) was associated with 1.4 percentage points lower for Hispanic than non-Hispanic White adults (Table 2). If there were no SEP and no excess intersectional disparities (just race/ethnicity disparities), we would expect the burden of HICP between Hispanic adults of family income <100% FPT to the burden of HICP of non-Hispanic White adults of ≥400% FPT to be equal to the referent race/ethnicity disparity, 1.4 percentage points lower for Hispanic adults of family income <100% FPT. However, given the observed joint disparity (10.6 [1.0]), the referent SEP disparity (20.6 [0.8]) and the referent race/ethnicity disparity (-1.4 [0.5]), we conclude that the disparity in burden of HICP among Hispanic adults of <100% FPT relative to non-Hispanic White adults of ≥400% FPT is 8.6% points (1.4) lower than what we would have expected based on race/ethnicity and SEP disparities alone. This pattern is similar across minority race/ethnicity categories and non-referent income levels (Table 2)

**Table 2.** Absolute joint disparities in high-impact chronic pain by race/ethnicity and family income and its components.

| Difference in mean predicted probability of HICP, % (SE) | | Joint disparity | Referent SEP disparity | Referent race/ethnicity disparity | Excess intersectional disparity |
|---|---|---|---|---|---|
| Race/ ethnicity | Family income, % FPT | vs. NH-White ≥400% FPT | vs. ≥400% FPT, among NH-White | vs. NH-White, among ≥400% FPT | Joint minus referent disparities |
| NH-Black | <100 | 14.6 (1.1) | 20.6 (0.8) | -1.4 (0.5) | -4.6 (1.4) |
| | 100-199 | 7.0 (0.9) | 10.8 (0.5) | -1.4 (0.5) | -2.4 (1.1) |
| | 200-399 | 2.2 (0.7) | 4.3 (0.3) | -1.4 (0.5) | -0.7 (0.9) |
| Hispanic | <100 | 10.6 (1.0) | 20.6 (0.8) | -1.4 (0.5) | -8.6 (1.4) |
| | 100-199 | 4.4 (0.7) | 10.8 (0.5) | -1.4 (0.5) | -5.0 (1.0) |
| | 200-399 | 0.7 (0.6) | 4.3 (0.3) | -1.4 (0.5) | -2.2 (0.8) |
| NH-Asian | <100 | 8.2 (2.1) | 20.6 (0.8) | -2.2 (0.5) | -10.2 (2.2) |
| | 100-199 | 3.0 (1.4) | 10.8 (0.5) | -2.2 (0.5) | -5.6 (1.5) |
| | 200-399 | -2.1 (0.7) | 4.3 (0.3) | -2.2 (0.5) | -4.2 (0.8) |

Joint disparity by race/ethnicity and family income (measured as percent of the federal poverty threshold [FPT]) was calculated as the difference in predicted probability of high-impact chronic pain (HICP) relative to non-Hispanic (NH) White adults with family income ≥400% FPT. Joint disparity is decomposed into a) referent socioeconomic position (SEP) disparity – disparity by family income among NH-White adults, b) referent race/ethnicity disparity – disparity by race/ethnicity among adults with family income ≥400% FPT, and c) excess intersectional disparity – the joint disparity minus the sum of referent disparities. By definition, there is only 1 referent SEP disparity per SEP level (among NH-White adults), and 1 referent race/ethnicity disparity per race/ethnicity category (among adults with family income ≥400% FPT); however, these values are repeated in the table to show all three components of each joint disparity for a combination of minority race/ethnicity and SEP level. SE = standard error, obtained from non-parametric bootstrap estimation with 5000 replications.

**Table 3.** High-impact chronic pain disparity components as a proportion of joint disparity by race/ethnicity and family income.

| Proportion of joint disparity % (SE) | | Referent SEP disparity (=NH-White) | | Referent race/ethnicity disparity (≥400% FPT) | | Excess intersectional disparity | |
|---|---|---|---|---|---|---|---|
| Race/ ethnicity | Family income, % FPT | vs. ≥400% FPT, among NH-White | | vs. NH-White, among ≥400% FPT | | Joint minus referent disparities | |
| NH-Black | <100 | 141.3 | (11.7) | -9.8 | (3.7) | -31.5 | (11.6) |
| | 100-199 | 154.4 | (20.6) | -20.4 | (8.1) | -34.1 | (19.6) |
| | 200-399 | 196.6 | (149.0) | -65.1 | (50.3) | -31.5 | (118.9) |
| Hispanic | <100 | 194.7 | (20.2) | -13.1 | (5.3) | -81.6 | (19.6) |
| | 100-199 | 243.0 | (44.3) | -31.1 | (13.9) | -112.0 | (40.7) |
| | 200-399 | 614.5 | (44612.7) | -197.2 | (12300.8) | -317.3 | (32453.3) |
| NH-Asian | <100 | 251.5 | (91.6) | -26.8 | (12.1) | -124.6 | (81.9) |
| | 100-199 | 364.8 | (30917.4) | -74.1 | (6030.1) | -190.7 | (24899.8) |
| | 200-399 | -205.7 | (285.0) | 105.0 | (115.3) | 200.7 | (183.7) |

Proportions of joint disparity were calculated by dividing the disparity components (referent socioeconomic position [SEP] disparity, referent race/ethnicity disparity, and excess intersectional disparity) by the joint disparity for the corresponding combination of minority race/ethnicity and non-referent SEP (measured as percent of the federal poverty threshold [FPT]) and multiplied by 100. (E.g., the referent SEP disparity for adults with family income <100% FPT was 141.3% of [i.e., larger than] the joint disparity for non-Hispanic (NH) Black adults with family income <100% FPT relative to NH-White adults with family income ≥400% FPT). SE = standard error, obtained from non-parametric bootstrap estimation with 5000 replications. All but two of the estimates (NH-Black and Hispanic referent race disparity as a proportion of joint disparity at <100% FPT) are *unreliable* according to the new NCHS presentation standards (2017) for proportions. Consider omitting this table in the publication stage.

The estimates of disparity components as a proportion of the joint disparity were much less precise (Table 3). All but two of the estimates were *not* up to the new NCHS data presentation standards for reliability of proportions. They generally indicate that the joint disparity was composed primarily of positive SEP disparities (i.e., higher HICP burden with

lower family income). Negative excess intersectional disparities (i.e., lower HICP burden for minority race/ethnicities with lower family income) contributed the second largest proportion of joint disparities, except in non-Hispanic Black adults of family income 200-399%, where the referent race/ethnicity disparities was larger than the excess intersectional disparity. Finally, negative race/ethnicity disparities contributed with a smaller proportion of the joint disparity (i.e., lower HICP burden for race/ethnicity minorities).

Overall, the patterns observed for intersectional disparities by race/ethnicity and education were similar in many regards to those by race/ethnicity and family income. Differently than for family income, the magnitude of joint disparities of Hispanic and non-Hispanic Asian adults with the two lowest categories of education were very similar. The joint disparity for Hispanic adults with some college or associate's degree was greater than that of Hispanic high school graduates, when compared to non-Hispanic White adults with bachelor's degree or higher education. Although joint disparities by race/ethnicity and family income were generally larger than by race/ethnicity and education, contrasts of non-Hispanic Black and Hispanic adults with some college or associate's degree had larger joint disparities than the contrasts of those race/ethnicities with category that was also immediately below the reference (family income 200-399% FPT).

For non-Hispanic Black adults of all levels of education, the joint disparities were of similar magnitude to the referent SEP (Suppl. Table 3). Then, referent race/ethnicity and excess intersectional disparities were small, including two positive excess intersectional disparities for non-Hispanic Black adults with high school education and non-Hispanic

Black adults with some college or associate's degree. While referent race/ethnicity disparities among adults with bachelor's degree or higher were smaller than for race/ethnicity disparities among adults with family income ≥400% FPT for non-Hispanic Black and Hispanic adults, negative referent race/ethnicity disparities for non-Hispanic Asian adults with bachelor's degree or higher education were slightly larger (lower HICP burden) than for non-Hispanic Asians with family income ≥400% FPT.

The negative excess intersectional disparities for Hispanic adults with less than high school or GED, and Hispanic high school graduates were larger (lower HICP burden) than for non-Hispanic Asian adults with the same levels of education, when compared to the doubly privileged category. This was different than for the corresponding disparities with family income. On the other hand, the excess intersectional disparity for Hispanic adults with some college or associate's degree was smaller than the analogous contrast with family income, and smaller than the contrast of non-Hispanic Asian adults with the same level of education.

Similarly to the disparities by race/ethnicity and family income, the referent SEP disparities had the greatest relative contribution to the joint disparity for all groups (Suppl. Table 4). Referent race/ethnicity and excess intersectional disparities were either second or third largest relative contribution to the joint disparity. Once again, estimates of disparity components relative to the joint disparity had generally low precision, and all but one of the estimates were *not* up to the new NCHS data presentation standards for reliability of proportions.

#### 7.3.4. Parallel with multiplicative measures and stratification approach to intersectional disparities

When examining *total additive family income disparities* in HICP for minority race/ethnicity categories (Table 4), the total difference in burden of HICP relative to the reference SEP category (≥400% FPT) is equal to the sum of referent SEP (SEP disparities among non-Hispanic White adults) and the corresponding excess intersectional disparity (i.e., the additive interaction between SEP and race/ethnicity).

**Table 4.** Additive family income disparities in high-impact chronic pain, by race/ethnicity, and race/ethnicity disparities by levels of family income.

| % (SE) | NH-White | | NH-Black | | Hispanic | | NH-Asian | |
|---|---|---|---|---|---|---|---|---|
| *Family income* | | | | | | | | |
| <100% FPT | 20.6 | (0.8) | 16.0 | (1.2) | 11.9 | (1.1) | 10.4 | (2.1) |
| 100-199% FPT | 10.8 | (0.5) | 8.4 | (1.0) | 5.8 | (0.9) | 5.2 | (1.5) |
| 200-399% FPT | 4.3 | (0.3) | 3.6 | (0.8) | 2.1 | (0.8) | 0.1 | (0.8) |
| ≥400% FPT | Reference | | | | | | | |
| *Family income* | | | | | | | | |
| <100% FPT | Reference | | -6.0 | (1.3) | -10.0 | (1.3) | -12.4 | (2.2) |
| 100-199% FPT | | | -3.8 | (1.0) | -6.4 | (0.8) | -7.8 | (1.5) |
| 200-399% FPT | | | -2.1 | (0.7) | -3.6 | (0.6) | -6.4 | (0.7) |
| ≥400% FPT | | | -1.4 | (0.5) | -1.4 | (0.5) | -2.2 | (0.5) |

Percentages represent difference in mean predicted probabilities of high-impact chronic pain, adjusted for age and sex. SE = standard error, obtained from non-parametric bootstrap estimation with 5000 replications. Highlighted yellow cells represent the 'referent socioeconomic disparities', or income disparities among non-Hispanic (NH) White adults, the reference group for race/ethnicity. Highlighted blue cells represent the 'referent race/ethnicity disparities', or race/ethnicity disparities among adults with highest level of family income (≥400% of the federal poverty threshold [FPT]).

Likewise, for the *additive total race/ethnicity disparities* for non-referent family income categories (Table 4). The total difference in burden of HICP relative to the non-Hispanic White category is equal to the sum of the referent race/ethnicity disparity (among adults

with the highest SEP) and the corresponding excess intersectional disparity (i.e., the additive interaction between SEP and race/ethnicity).

These results are analogous to what is usually presented when investigating intersectional disparities with multiplicative interactions between two axes of marginalization using regression models for binary outcomes (log binomial, in our case). Exponentiated linear combinations of model coefficients can be interpreted as prevalence ratios (PR), or total disparities in the burden of HICP by one axis, along categories of the other axis (i.e., multiplicative family income disparities by categories of race/ethnicity, and race/ethnicity disparities by categories of family income, Table 5). These estimates of are the sum of multiplicative referent disparities and their corresponding multiplicative interaction term.

The model coefficients for race/ethnicity correspond to the *multiplicative* referent race/ethnicity disparities, or disparities in burden of HICP between race/ethnicity minorities and non-Hispanic White categories, among adults with the referent SEP category. The model coefficients for SEP correspond to the *multiplicative* referent SEP disparities, or disparities in the burden of HICP between lower SEP categories with the referent SEP categories, among non-Hispanic White adults.

The model coefficients for the interaction terms between SEP and race/ethnicity correspond to the *multiplicative* excess intersectional disparity, and it is sometimes shown (in its exponentiated form for multiplicative regression models) as a ratio of ratios. The *multiplicative* joint disparity in HICP by race/ethnicity and SEP could be obtained by the sum of the coefficients for the minority race/ethnicity (multiplicative referent race disparity), the SEP (multiplicative referent SEP disparity) and their interaction

(multiplicative excess intersectional disparity). Results from the education by race/ethnicity stratification approach analogous to Tables 4 and 5 in family income by race/ethnicity are available in the Appendix (Suppl. Tables 4 and 5).

**Table 5.** Multiplicative family income disparities in high-impact chronic pain, by race/ethnicity, and race/ethnicity disparities by levels of family income.

| aPR (SE) | NH-White | | NH-Black | | Hispanic | | NH-Asian | |
|---|---|---|---|---|---|---|---|---|
| *Family income* | | | | | | | | |
| <100% FPT | 5.42 | (0.24) | 5.77 | (0.89) | 4.57 | (0.72) | 4.51 | (1.03) |
| 100-199% FPT | 3.29 | (0.15) | 3.57 | (0.57) | 2.75 | (0.45) | 2.84 | (0.69) |
| 200-399% FPT | 1.96 | (0.09) | 2.13 | (0.36) | 1.71 | (0.30) | 1.00 | (0.29) |
| ≥400% FPT | Reference | | | | | | | |
| *Family income* | | | | | | | | |
| <100% FPT | Reference | | 0.78 | (0.05) | 0.70 | (0.05) | 0.54 | (0.09) |
| 100-199% FPT | | | 0.80 | (0.06) | 0.69 | (0.06) | 0.56 | (0.10) |
| 200-399% FPT | | | 0.80 | (0.07) | 0.72 | (0.08) | 0.33 | (0.08) |
| ≥400% FPT | | | 0.73 | (<0.01) | 0.83 | (0.12) | 0.65 | (<0.01) |

aPR = prevalence ratio of high-impact chronic pain, adjusted for age and sex; SE = standard error, obtained from non-parametric bootstrap estimation with 5000 replications; Highlighted yellow cells represent the 'referent socioeconomic disparities', or income disparities among non-Hispanic (NH) White adults, the reference group for race/ethnicity. Highlighted blue cells represent the 'referent race/ethnicity disparities', or race/ethnicity disparities among adults with highest level of family income (≥400% of the federal poverty threshold [FPT]).

Although we did not employ survey weights, the distribution of adults across intersectional categories was similar to the weighted distribution (Supplemental Table 6). Proportion of non-Hispanic Whites in the sample is slightly higher than in the weighted population estimates (72.5% vs. 66.6%), and the proportion of Hispanics is slightly lower (11.4% vs. 15.1%). The over-representation of non-Hispanic Whites in the sample is mainly observed in lower income categories. Overall proportion with ≥400% FPT family income is slightly lower in the sample than in the weighted populations estimates (41.1% vs. 44.4%), and this

under-representation is observed mostly among higher income race/ethnicity minority categories.

### 7.4. Discussion

Our findings highlight very wide variation in the mean predicted probability of HICP across intersectional categories defined by race/ethnicity and SEP, adjusting for age and sex. Socioeconomic inequities were strong across all race/ethnicity categories and continue to be an important subject for investigation of pain inequities. Indeed, SEP disparities were the main component of joint disparities in HICP when considering all 18 examined intersections of minority race/ethnicity and non-referent SEP (3 minority race/ethnicity * 3 family income categories + 3 minority race/ethnicity * 3 educational attainment categories).

Contrary to what would have been expected based on the social imbalance of power and privilege in our society, non-Hispanic White adults had higher burden of HICP than race/ethnicity minority adults across all levels of SEP. This is reflected in small negative referent race/ethnicity disparities for all minority race/ethnicities (i.e., among those in the highest SEP level, adults of minority race/ethnicity had slightly lower burden of HICP than adults of NH-White race/ethnicity).

Moreover, on average, adults in nearly all (16 of 18) examined doubly marginalized social positions by race/ethnicity and SEP had *lower* HICP burden than expected based on the sum of disparities by race/ethnicity alone and SEP alone. Negative excess intersectional disparities were the second largest component of joint disparities for most (13 of 18) intersections, with a greater contribution than the disparity by race/ethnicity alone. The additive interaction was especially large (i.e., associated with smaller burden of HICP) for

race/ethnicity minority categories with lower SEP. These findings may point to aspects of doubly marginalized social contexts that create group resilience in the face of oppression, rather than individual weakness or vulnerability as these groups are sometimes portrayed (Katz et al., 2020). Alternatively, there could be a relative lack of group resilience among NH-White adults, despite their many structural advantages.

As a comparison for the intersectional decomposition analysis, we presented a parallel with disparity measures that are often used to present intersectional disparities from regression models with multiplicative interactions (i.e., stratified effects). Those findings showed somewhat similar patterns such as larger SEP disparities for lower levels of income for all race/ethnicity categories, which were stronger for the contrasts with family income than with education. The magnitude of the disparities was also greater for non-Hispanic White adults, followed by non-Hispanic Black, Hispanic and non-Hispanic Asian at similar SEP levels. Additionally, we observed generally smaller burden of HICP among adults of minority race/ethnicity at most levels of SEP, and especially lower for non-Hispanic Asian adults. However, estimates of total multiplicative disparities combine the referent and excess interaction disparities from the decomposition analysis. This obscures how much of them are accounted for by race/ethnicity alone, by SEP alone and by their intersection.

Importantly, the omission of the joint disparities in the conventional presentation of stratified multiplicative effects meaningfully changes the overall interpretation of the results. In the decomposition analysis, we observed positive estimated joint disparities for 17 of the 18 intersectional categories of minority race/ethnicity and non-referent SEP (i.e., higher or similar burden of HICP relative to adults of non-Hispanic White race/ethnicity and highest SEP). Of note, 72.3% of adults of minority race/ethnicity were also part of lower

SEP categories (doubly marginalized; non-Hispanic Black=76.4%, Hispanic=78.1%, Non-Hispanic Asian=50.0%). Therefore, the joint disparity measures better reflect population race/ethnicity contrasts in the burden of HICP than contrasts which analytically equalize (by stratification) SEP level. Without the joint disparities, the perception of race/ethnicity minorities as being better off (i.e., all disparity measures by race/ethnicity showed lower burden of HICP among race/ethnicity minorities relative to non-Hispanic White adults of the same SEP), could have led to race/ethnicity disparities and potential social inequities being dismissed as unimportant. Furthermore, the decomposition approach also featured an opportunity to highlight potential strengths of adults in doubly marginalized social positions: the negative excess intersectional disparities contributed to less pronounced joint disparities in the burden of HICP disparities than expected based on the sum of SEP and race/ethnicity disparities alone.

Putting these findings in context, the U.S. opioid epidemic went through a peak of opioid-related deaths in the years of 2016-2017, when the data were collected (Scholl et al., 2018). While non-Hispanic White adults had the highest annual opioid-related death rate by race/ethnicity in 2017 (19.4 per 100,000), it was followed closely by the rate in non-Hispanic American Indian or Alaska Native (15.7 per 100,000) and non-Hispanic Black (12.9 per 100,000). Despite narratives of the opioid crisis that portray it as a problem of White lower and middle class, the increase in rate of opioid-related deaths between 2016-2017 was higher among non-Hispanic Black adults (Scholl et al., 2018). A recent intersectional analysis of prescription opioid misuse corroborates the need for more a more nuanced perspective on risk of opioid misuse (Persmark et al., 2020). A population-based strategy for pain prevention and care (including prevention of opioid misuse and overdose) needs to centrally address social inequities as key determinants of chronic pain, as outlined in the

National Pain Strategy and other related documents (Institute of Medicine (IOM), 2011; Interagency Pain Research Coordinating Committee, 2016).

Although quantitative methods for intersectional analysis are developing rapidly, the role of multiple marginalized social positions to pain disparities has received little attention. Such evidence may help to identify social structures and processes that result in the wide variation of HICP burden across intersectional categories. Population health interventions could then aim to address these social targets towards eliminating pain inequities. Specifically, intersectional decomposition analysis has three main attractive features. First, it highlights joint disparities, which describe the disparities that result from the experience of multiple marginalized positions. Joint disparities are generally not presented by intersectional approaches that describe disparities by one marginalizing social position along categories of another axis of marginalization. Second, the decomposition approach quantifies the components of joint disparities by race/ethnicity alone, by SEP alone and by their intersection. Each of these components may indicate any direction and magnitude of differences contributing to particularly high or low joint pain disparities, providing a more detailed picture of the role of interlocking social positions on the burden of HICP. Third, this method uses additive measures disparity to quantify the absolute differences in population health and HICP disparities that would be observed in the absence of each of the axis of marginalization separately and combined.

Although absolute measures of disparity are not an exclusive feature of the intersectional decomposition analysis, simple methods are presented for computing and interpreting such *descriptive* measures in a manner compatible with the cross-sectional nature of our data. The use of these measures allows for quantitative assessment of potential impact of

counterfactual scenarios on population pain burden (i.e., in the absence of disparities) without stringent assumptions about causal relationships between the variables of interest. This type of investigation speaks to the need and directions for causal inference work on social determinants of pain, as well as the magnitude of potential population health benefit of pain equity interventions.

In this analysis we expanded the number of intersectional categories relative to the intersectional decomposition method (Jackson et al., 2016). With this modification, our findings go beyond Black and White racial groups to the four largest race/ethnicity categories in the US. We also expanded the categorization by SEP from a binary measure of high and low early life socioeconomic status to four categories of educational attainment and four categories of family income (measured as percent of the federal poverty threshold [FPT], which account for survey year, family size, and number of related children under 18 years). The cut off points for both SEP categories were based on previous analyses of health and pain disparities, including our own work presented in Section 5.

Theoretically, our comparison *between* categories defined by social labels is primarily aligned with inter-categorical intersectional approaches (McCall, 2005), which use these socially defined groups to expose social systems that create inequities between them, especially for those who are in disadvantaged positions along more than one axis of marginalization. By expanding the number of categories investigated, we sought to manage the heterogeneity *within* these broad social groups and quantify differences in the burden of HICP at different, slightly more nuanced intersections. The concern with diversity of experiences within social positions is a feature of the intra-categorical intersectional approaches (McCall, 2005). Given the need to limit the regression model parameters and

interpretability when describing decomposition results, we only nod to intra-categorical intersectional theory. We acknowledge that our intersectional categories are simplistic and there may be additional axes of power and privilege that are omitted by our analysis.

For example, the correlation between family income and education was only 0.44, so it is possible that different patterns would have emerged if we defined intersectional categories with both education and family income as different aspects of SEP, in addition to race/ethnicity. Additionally, social class has been suggested as a potentially important aspect in 'deaths of despair' in the US (Navarro, 2019). Given the rural/urban differences in how the opioid epidemic has played out, this variable (only available in restricted data) should also be considered in future analysis. Upcoming iterations of this work could include investigation of HICP disparities using methods that are suitable to quantify heterogeneity within and between multidimensional intersectional categories, such as the multilevel analysis of individual heterogeneity and discriminatory accuracy (MAIHDA) (Evans et al., 2018).

If the burden of HICP was associated with the sampling probability, our results may be subject to selection bias and lack of generalizability to the US population. Novel methods to perform bootstrap in multiply imputed survey data may be explored in future iterations of this work (Comulada, 2015). Alternatively, we could conduct quantitative bias analyses to estimate the magnitude of differences that would have been necessary for our observed findings to be the results of substantial selection bias.

## 7.5. Appendix

### 7.5.1. Supplemental Tables

**Supplemental Table 1.** Age and sex distribution of adults ≥25 years across categories defined by the intersections of race/ethnicity and socioeconomic position.

| | | NH-White | NH-Black | Hispanic | NH-Asian | Overall |
|---|---|---|---|---|---|---|
| *Overall* | | | | | | |
| | Mean age (SD) | 55.3 (16.8) | 51.9 (16.3) | 47.6 (15.7) | 49.7 (16.8) | 53.8 (16.9) |
| | % Female | 54.0 | 60.1 | 56.9 | 53.9 | 55.0 |
| *Family income*, % FPT | | | | | | |
| ≥400 | Mean age (SD) | 54.0 (15.2) | 51.1 (14.5) | 46.9 (14.0) | 47.3 (15.1) | 53.0 (15.3) |
| | % Female | 50.1 | 51.3 | 50.0 | 51.1 | 50.3 |
| 200-399 | Mean age (SD) | 56.4 (17.8) | 51.9 (16.6) | 46.3 (15.5) | 51.0 (17.7) | 54.6 (17.8) |
| | % Female | 54.7 | 57.9 | 52.5 | 57.7 | 54.9 |
| 100-199 | Mean age (SD) | 58.5 (18.6) | 53.8 (17.4) | 48.6 (16.3) | 52.5 (18.2) | 55.8 (18.5) |
| | % Female | 60.2 | 62.9 | 58.7 | 55.1 | 60.2 |
| <100 | Mean age (SD) | 53.2 (16.9) | 50.9 (16.2) | 48.5 (16.7) | 54.8 (18.8) | 51.7 (16.9) |
| | % Female | 61.0 | 68.4 | 66.6 | 56.5 | 63.7 |
| *Education* | | | | | | |
| ≥BS/BA | Mean age (SD) | 52.3 (16.5) | 49.0 (15.5) | 45.6 (14.3) | 45.0 (15.2) | 51.0 (16.4) |
| | % Female | 53.7 | 60.5 | 56.0 | 51.3 | 54.2 |
| Some College or AA | Mean age (SD) | 54.7 (16.4) | 49.3 (15.7) | 44.6 (14.8) | 51.5 (16.6) | 53.0 (16.5) |
| | % Female | 55.4 | 62.9 | 59.2 | 54.6 | 56.6 |
| HS Graduate | Mean age (SD) | 59.1 (16.5) | 53.2 (16.2) | 46.6 (15.7) | 57.5 (16.2) | 56.9 (16.9) |
| | % Female | 53.5 | 56.5 | 55.0 | 57.6 | 54.2 |
| <HS or GED | Mean age (SD) | 60.3 (17.1) | 57.7 (16.5) | 51.4 (16.4) | 60.9 (16.8) | 57.5 (17.3) |
| | % Female | 51.7 | 59.7 | 56.9 | 60.9 | 54.7 |

NH = non-Hispanic; FPT= federal poverty threshold; HS = High School; AA = associate's degree; ≥BS/BA = bachelor's degree or higher; SD = standard deviation.

**Supplemental Table 2.** Absolute disparities in high-impact chronic pain by race/ethnicity and education.

| Absolute difference in HICP probability % (SE) | | Joint disparity | Referent SEP disparity | Referent race/ethnicity disparity | Excess intersectional disparity |
|---|---|---|---|---|---|
| Race/ ethnicity | Education | vs. NH-White, ≥BS/BA | vs. ≥BS/BA, among NH-White | vs. NH-White, among ≥BS/BA | Joint minus referent disparities |
| NH-Black | <HS or GED | 11.9 (1.1) | 14.3 (0.6) | -0.7 (0.6) | -1.7 (1.3) |
| | HS Graduate | 5.8 (0.9) | 6.1 (0.4) | -0.7 (0.6) | 0.4 (1.1) |
| | Some College or AA | 5.0 (0.7) | 5.5 (0.3) | -0.7 (0.6) | 0.2 (0.9) |
| Hispanic | <HS or GED | 5.8 (0.7) | 14.3 (0.6) | -0.6 (0.6) | -8.0 (1.1) |
| | HS Graduate | 2.2 (0.8) | 6.1 (0.4) | -0.6 (0.6) | -3.3 (1.0) |
| | Some College or AA | 4.1 (0.8) | 5.5 (0.3) | -0.6 (0.6) | -0.9 (1.0) |
| NH-Asian | <HS or GED | 5.6 (2.0) | 14.3 (0.6) | -2.6 (0.4) | -6.1 (2.1) |
| | HS Graduate | 2.0 (1.2) | 6.1 (0.4) | -2.6 (0.4) | -1.4 (1.3) |
| | Some College or AA | 0.5 (1.1) | 5.5 (0.3) | -2.6 (0.4) | -2.4 (1.2) |

Joint disparity by race/ethnicity and educational attainment is calculated relative to non-Hispanic (NH) White adults with family income ≥400% FPT. Joint disparity is decomposed into a) referent socioeconomic position (SEP) disparity – disparity by educational attainment among NH-White adults, b) referent race/ethnicity disparity – disparity by race/ethnicity among adults with family income ≥400% FPT, and c) excess intersectional disparity – the joint disparity minus the sum of referent disparities. By definition, there is only 1 referent SEP disparity per SEP level (among NH-White adults), and 1 referent race/ethnicity disparity per race/ethnicity category (among adults with family income ≥400% FPT); however, these values are repeated in the table to show all three components of each joint disparity for a combination of minority race/ethnicity and SEP level. HICP = high-impact chronic pain; HS = High School; AA = associate's degree; ≥BS/BA = bachelor's degree or higher; SE = standard error, obtained from non-parametric bootstrap estimation with 5000 replications.

**Supplemental Table 3.** High-impact chronic pain disparity components as a proportion of joint disparity by race/ethnicity and education.

| Proportion of joint disparity % (SE) | | Referent SEP disparity (=NH-White) | | Referent race/ethnicity disparity ≥BS/BA) | | Excess intersectional disparity | |
|---|---|---|---|---|---|---|---|
| NH-Black | <HS or GED | 120.1 | (12.0) | -5.6 | (5.0) | -14.5 | (12.4) |
| | HS Graduate | 104.5 | (17.4) | -11.5 | (10.7) | 7.1 | (18.8) |
| | Some College or AA | 109.9 | (17.2) | -13.4 | (12.3) | 3.5 | (19.4) |
| Hispanic | <HS or GED | 247.6 | (33.2) | -9.7 | (11.4) | -137.8 | (33.6) |
| | HS Graduate | 271.5 | (717.2) | -25.2 | (135.3) | -146.2 | (609.3) |
| | Some College or AA | 135.1 | (28.8) | -13.8 | (17.1) | -21.3 | (30.0) |
| NH-Asian | <HS or GED | 254.9 | (543.3) | -46.9 | (110.1) | -108.0 | (434.8) |
| | HS Graduate | 297.6 | (140332.9) | -129.2 | (69611.6) | -68.4 | (70723.4) |
| | Some College or AA | 1104.0 | (305427.9) | -527.2 | (156790.0) | -476.8 | (148677.9) |

Proportions of joint disparity were calculated by dividing the disparity components (referent socioeconomic position [SEP] disparity, referent race/ethnicity disparity, and excess intersectional disparity) by the joint disparity for the corresponding combination of minority race/ethnicity and non-referent SEP and multiplied by 100. (E.g., the referent SEP disparity for adults with <High School [HS] or GED education was 120.1% of [i.e., larger than] the joint disparity for non-Hispanic [NH] Black adults with <HS or GED education relative to NH-White adults with bachelor's degree or higher [≥BS/BA]). AA = associate's degree; SE = standard error, obtained from non-parametric bootstrap estimation with 5000 replications. All but one of the estimates (NH-Black referent race disparity as a proportion of joint disparity at <HS or GED education) are *unreliable* according to the new NCHS presentation standards (2017) for proportions. Consider omitting this table in the publication stage.

**Supplemental Table 4.** Additive educational disparities in high-impact chronic pain, by race/ethnicity, and race/ethnicity disparities by levels of education.

| % (SE) | NH-White | | NH-Black | | Hispanic | | NH-Asian | |
|---|---|---|---|---|---|---|---|---|
| *Education* | | | | | | | | |
| <HS or GED | 14.3 | (0.6) | 12.6 | (1.2) | 6.4 | (0.9) | 8.3 | (2.0) |
| HS Graduate | 6.1 | (0.4) | 6.5 | (1.0) | 2.8 | (0.9) | 4.7 | (1.3) |
| Some College or AA | 5.5 | (0.3) | 5.7 | (0.9) | 4.6 | (0.9) | 3.1 | (1.1) |
| ≥BS/BA | Reference | | | | | | | |
| *Education* | | | | | | | | |
| <HS or GED | Reference | | -2.4 | (1.2) | -8.5 | (0.9) | -8.7 | (2.0) |
| HS Graduate | | | -0.3 | (0.9) | -3.8 | (0.8) | -4.0 | (1.3) |
| Some College or AA | | | -0.5 | (0.7) | -1.4 | (0.8) | -5.0 | (1.1) |
| ≥BS/BA | | | -0.7 | (0.6) | -0.6 | (0.6) | -2.6 | (0.4) |

Percentages represent difference in mean predicted probabilities of high-impact chronic pain, adjusted for age and sex. SE = standard error, obtained from non-parametric bootstrap estimation with 5000 replications. HS = high school; AA = associate's degree; Yellow cells represent the 'referent socioeconomic disparities', or educational disparities among non-Hispanic (NH) White adults, the reference group for race/ethnicity. Blue cells represent the 'referent race/ethnicity disparities', or race/ethnicity disparities among adults with highest level of education (bachelor's degree or higher [≥BS/BA]).

**Supplemental Table 5.** Multiplicative educational disparities in high-impact chronic pain, by race/ethnicity, and race/ethnicity disparities by levels of education.

| aPR (SE) | NH-White | | NH-Black | | Hispanic | | NH-Asian | |
|---|---|---|---|---|---|---|---|---|
| *Education* | | | | | | | | |
| <HS or GED | 3.50 | (0.17) | 2.31 | (0.35) | 2.22 | (0.33) | 3.36 | (0.83) |
| HS Graduate | 1.98 | (0.10) | 3.43 | (0.49) | 1.66 | (0.28) | 2.27 | (0.55) |
| Some College or AA | 2.00 | (0.09) | 2.31 | (0.34) | 2.16 | (0.34) | 1.98 | (0.50) |
| ≥BS/BA | Reference | | | | | | | |
| *Education* | | | | | | | | |
| <HS or GED | Reference | | 0.88 | (0.06) | 0.63 | (0.04) | 0.54 | (0.10) |
| HS Graduate | | | 1.04 | (0.09) | 0.84 | (0.09) | 0.64 | (0.11) |
| Some College or AA | | | 1.04 | (0.08) | 1.08 | (0.09) | 0.56 | (0.11) |
| ≥BS/BA | | | 0.90 | (0.12) | 1.00 | (0.14) | 0.56 | (<0.01) |

aPR = prevalence ratio of high-impact chronic pain, adjusted for age and sex; SE = standard error, obtained from non-parametric bootstrap estimation with 5000 replications; HS = high school; AA = associate's degree; Yellow cells represent the 'referent socioeconomic disparities', or educational disparities among non-Hispanic (NH) White adults, the reference group for race/ethnicity. Blue cells represent the 'referent race/ethnicity disparities', or race/ethnicity disparities among adults with highest level of education (bachelor's degree or higher [≥BS/BA]).

**Supplemental Table 6.** Weighted population estimates of distribution of adults ≥25 years across categories defined by intersection of race/ethnicity and socioeconomic position.

| n (%), millions | NH-White | | NH-Black | | Hispanic | | NH-Asian | | Total | |
|---|---|---|---|---|---|---|---|---|---|---|
| *Family income, % FPT* | | | | | | | | | | |
| <100 | 9.2 | (4.3) | 4.9 | (2.3) | 6.1 | (2.9) | 1.2 | (0.6) | 21.5 | (10.1) |
| 100-199 | 19.3 | (9.1) | 5.9 | (2.8) | 9.0 | (4.2) | 1.9 | (0.9) | 36.0 | (16.9) |
| 200-399 | 40.6 | (19.1) | 7.6 | (3.6) | 9.2 | (4.3) | 3.3 | (1.5) | 60.6 | (28.5) |
| ≥400 | 72.9 | (34.3) | 7.0 | (3.3) | 7.9 | (3.7) | 6.8 | (3.2) | 94.6 | (44.5) |
| Total | 142.0 | (66.8) | 25.3 | (11.9) | 32.2 | (15.1) | 13.2 | (6.2) | 212.7 | (100.0) |
| *Education* | | | | | | | | | | |
| <HS or GED | 14.0 | (6.6) | 4.5 | (2.1) | 11.2 | (5.3) | 1.3 | (0.6) | 31.0 | (14.5) |
| HS Graduate | 30.0 | (14.1) | 6.4 | (3.0) | 6.9 | (3.2) | 2.0 | (0.9) | 45.3 | (21.3) |
| Some College or AA | 42.3 | (19.8) | 8.1 | (3.8) | 8.1 | (3.8) | 2.2 | (1.0) | 60.7 | (28.5) |
| ≥BS/BA | 56.0 | (26.3) | 6.4 | (3.0) | 6.0 | (2.8) | 7.7 | (3.6) | 76.1 | (35.7) |
| Total | 142.3 | (66.8) | 25.4 | (11.9) | 32.2 | (15.1) | 13.2 | (6.2) | 213.1 | (100.0) |

NH = non-Hispanic; FPT = federal poverty threshold; HS = High School; AA = associate's degree; ≥BS/BA = bachelor's degree or higher; SE = standard error, obtained from non-parametric bootstrap estimation with 5000 replications. Weighted population estimates in millions. Percentages of total population estimate sum up to 100 across the 16 categories of race/ethnicity and family income, and across the 16 categories of race/ethnicity and education.

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# 8. Final discussion

## 8.1. Theorical

Social determinants of chronic pain have been severely under researched despite decades of evidence about the existence of pain inequities, and periodic calls for a public health approach. We have identified an epistemological gap in pain theories – which have been lacking critical perspectives – as a major contributor to this stasis. We sought to bridge social theories with population health and causal inference methods, to advance the conceptualization and understanding of social determinants of chronic pain.

Informed by the previous literature, the current historical period, and intersectional social context of the United States, we elected to focus the applied portion of this work primarily on socioeconomic and race/ethnicity inequities in HICP. Both of these constructs have been considered less worthy of study due to being perceived as 'non-modifiable'. Part of our goals was to challenge this common narrative by contextualizing individual biological, psychological, and social factors in a multilevel causal framework shaped by intersectional determinants of (in)equity. Our conceptual framework provided the theoretical grounds for methodological and empirical investigations.

Throughout this piece, we aimed to present pain differences by SEP and race/ethnicities as the result of social conditions that are based on human-made systems of privilege and power (Krieger, 2020). as opposed to immutable characteristics that inherently predispose or protect against pain. Importantly, these upstream determinants represent opportunities for policy intervention, collective action, and population health promotion (Diderichsen et al., 2001). This broader understanding of multilevel social determinants a great extension of intervention targets from mainstream pain research, which is mainly concerned with

druggable targets, and biomedical interventions.

### 8.2. Methodological

Our DAG approach and use of the National Pain Strategy recommended HICP as the primary outcome overcame two common issues that have led to underestimation of pain inequities in previous publications: over-adjustment bias and failure to account for pain impact in outcome measures. The large nationally representative sample constituted another important strength of our study.

Consistent with the theory of fundamental causes (Link and Phelan, 1995), our analysis have shown that different components of SEP contribute to the burden of chronic pain through multiple pathways. Therefore, when SEP is conceptualized as a confounder, adjusting for one or two SEP variables is likely to result in residual confounding. Moreover, this and other approaches such as using SES index measures need to be careful not to perpetuate the conceptualization of SES as a non-modifiable individual characteristic. If assuming these variables are proxies for an underlying latent SES, socioecological theories may be used to avoid framing SES as inherent to the individual. Although they may have many advantages, composite SES measures can be detrimental to causal investigation of pain inequities. Specific targets for intervention might not be clear when presenting SEP as single complex lurking construct.

### 8.3. Empirical

Some of the main findings in this analysis include the large association between lower SEP – especially family income and education – and HICP, as well as higher number of site-specific pains. These included estimates in the 4-8-fold range relative to the reference

categories. Estimated inequities were markedly smaller for single site-specific pains, and low-impact chronic pain. Of note, adults with HICP had a high degree of comorbidity between site-specific pains, thus the population value of commonly performed analyses by pain sites in isolation is unclear. These findings suggest that socioeconomic pain inequities are likely driven primarily by pain impact rather than by pain duration or location. Furthermore, lower SEP was strongly associated with higher number of pain sites, warranting further investigation into processes that may lead to multi-site or spreading of pain as potential contributors to pain impact and socioeconomic pain inequities.

When examining their joint disparities, there was evidence of similar or higher burden of HICP for nearly all doubly marginalized intersections examined (vs. NH-White with highest level of family income or education). We argued that the joint disparity is a more relevant population measure of HICP disparities since over 70% of adults of minority race/ethnicity were also in a non-reference category of family income or education. That is, the doubly marginalized position better reflects the reality of most adults of race/ethnicity minorities. In contrast, the results of the commonly used stratification approach to interaction (e.g., reporting the effect of race/ethnicity by categories of SEP, and the effect of SEP by categories of race/ethnicity) would have reported a slightly lower burden of HICP for minority race/ethnicity categories by categories of SEP, and similarly higher burden of HICP for lower SEP by categories of race/ethnicity.

Additionally, our newly developed measure of relational social class raises the possibility of using labor and occupational policies to prevent and mitigate pain inequities. Our analysis identified three relational social classes with significantly higher burden of HICP: lower supervisory and technical occupations, small employers and own account workers, and

semi-routine and routine workers. Together, these classes account for 44.4% of adults ≥25 years who had ever worked in civilian occupations. Although theses association were of smaller magnitude than for some of the other measures of SEP, large population effects are not ruled out: the hypothesized inequity-generating mechanisms of exploitation and domination are ubiquitous and potentially amenable to policy interventions. Evaluation of the effects of policies on pain outcomes may consider programs directed at low-income families (e.g., EITC, Medicaid expansion, bolsa-família [Brazil], etc.).

In summary, we produced theorical, methodological, and subject-matter empirical evidence that may serve as the foundations for a research program in social epidemiology of chronic pain and pain inequities. These contributions also add to the broader fields of pain research and epidemiology, with findings that highlight the great need for and potential societal benefit of a population health equity approach to chronic pain prevention and management.