# Does psychological distress and sleep disturbance mediate the link between socioeconomic position and inflammation?

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A thesis submitted for the degree of Doctorate in Clinical Psychology (D Clin Psych)

School of Health and Social Care University of Essex April 2024

Word Count: 39,970

## Acknowledgements

I would like to extend my deepest thanks to my supervisors Dr Antonella Trotta, Dr Cara Booker and Professor Meena Kumari. The knowledge, guidance and support you have imparted to me has been greatly appreciated and has been instrumental throughout the various stages of this research.

Thank you also to my family and the unrelenting support from my mother and father that has been invaluable throughout this doctorate, I will always appreciate it.

I would lastly like to acknowledge my wife, Scarlet, for your constant support, encouragement and giving me the time to complete this research. I honestly could not have done this without you. Finally, to my three boys: Sebastian, Joshua and Benjamin.

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## Abstract

**Background:** Socioeconomic position is associated with health inequalities. One way of understanding these health disparities is through the role of chronic inflammation, as measured by inflammatory biomarkers, including C-reactive protein (CRP) and fibrinogen. However, it is not yet fully explained how socioeconomic position and inflammation are associated and whether other factors play a mediating role within this relationship.

**Objective:** The purpose of this study was to examine the potential mediating role of psychological distress (General Health Questionnaire; GHQ-12), sleep duration (*9 hours or more*, *7-8 hours*, *6-7 hours*, *6 hours or less*), and sleep quality (*very good, fairly good, fairly bad, very bad*) in the link between socioeconomic position and inflammation, using the inflammatory biomarkers, CRP and fibrinogen.

**Methods:** Secondary data analysis was completed using Understanding Society: the UK Household Longitudinal Study. Multiple linear and ordinal regression analyses were used to examine the association between socioeconomic position and inflammation, together with using mediation analysis to explore the potential mediating role of psychological distress and sleep quality and duration. A number of relevant covariates were adjusted for within each of the models.

**Results:** Lower educational attainment and household income were both associated with raised concentrations of both CRP and fibrinogen. Higher rates of psychological distress were associated with lower income but associations were not apparent for educational attainment. Psychological distress did not meet the prerequisite conditions to be a possible mediator. There was a significant association between lower educational attainment with sleep duration, Contrary to what was hypothesised, having no qualifications was associated with a greater

likelihood of falling into the *9 hours sleep or more* category. Similarly, being in the lowest income tertile was associated with *very good* sleep, compared to the highest income tertile. Sleep quality was a significant mediator in the association between both educational attainment and household income and CRP, although this is an unexpected finding, therefore more research is needed to explore this.

**Conclusion:** The overall findings demonstrate that socioeconomic disadvantage is associated with increased levels of CRP and fibrinogen, sleep quality was shown to have a potential mediating role, although more research is needed to further understand this relationship as it could constitute a type I error. Sleep and psychological distress are potential points of intervention to alleviate health inequalities. These findings also indicate the importance of welfare support to help attenuate inequalities relating to both physical and mental health.

## **Chapter One: Introduction**

## **Chapter Summary**

This chapter provides the introductory context for the present study by reviewing the association between socioeconomic position and inflammation. A definition of the construct of socioeconomic position will be provided by explaining the main indicators that combine to create this concept. These are income, occupation and educational attainment. There will be a discussion of the work of Karl Marx and Max Weber in order to provide the theoretical origins of the concept of socioeconomic position. There will be an introduction to the literature illustrating the association between socioeconomic position and inflammation together with an outline of the biomarkers of inflammation. The potential mediators that may explain the association between socioeconomic position and inflammation will also be outlined. The chapter concludes with a systematic review of the literature examining the association between socioeconomic position and inflammation. Lastly, the aims of the present study are outlined.

## **Socioeconomic Position**

*Defining Socioeconomic Position.* Socioeconomic position is characterised by both social and economic factors that guide which position people occupy within the structure of society (Krieger et al., 1997; Lynch & Kaplan, 2000). Socioeconomic position is a frequently used concept within health research. Researchers distinguish between various terms used to describe the construct of socioeconomic position. Therefore, depending on the research tradition, researchers will use concepts such as socioeconomic status, social stratification and social class to describe this concept.

The term socioeconomic position will be used within the present research. Krieger et al. (1997) described socioeconomic position as an "aggregate concept that includes both

resource-based and prestige-based measures, as linked to both childhood and adult social class position" (p. 345). Education, income and occupation are associated with varying degrees of resource and prestige and as such are commonly used measures of socioeconomic position.

## **Theoretical Origins**

The concepts that underpin the use of socioeconomic position within health research have their theoretical origin in the work of Karl Marx and Max Weber. The notion of social class originated from the work of the philosopher Karl Marx (1844). It is helpful to understand these historical origins in considering the development of the concept of socioeconomic position. Marx postulated that society has gradually moved from a feudal system, in which people were provided with land in return of services such as working for the landowner, into a capitalist society. Capitalism concerns an economic system in which there is private ownership of industry and it is operated for the pursuit of profit. The key tenets of capitalism are capital accumulation, competitive markets, private property and wage labour. Social class in its original meaning as conceived by Marx was in relation to classifying an individual or group as to (a) whether they had ownership or control over a business, or (b) whether the individual or group forms part of the labour force in such an establishment (Marx, 1844). In this way, the ruling classes, also referred to as the bourgeois, held power and used the working classes, also referred to as the proletariat, to produce goods (e.g. in factories) and to ultimately retain the profit for themselves. Marx maintained that capitalism created vast inequalities between the two classes.

Marx stated that the inequalities between social classes remain constant throughout time. This is in contrast to the occasionally accepted notion in some countries that there is social mobility, which refers to an individual, family or household moving between one class to another with relative simplicity (Muller & Pollak, 2015). However, theorists such as Marx maintain that belonging to a specific class or social strata remains constant from one generation to the next (Bowles & Gintis, 2002).

Max Weber, another social theorist, responded to Marx's social class and theory of social stratification by introducing further class divisions. Following Weber's death one of his famous works was published in 1922, *Wirtschaft und Gesellschaft* (translated as 'Economy and Society'), it contained an essay later translated into English as 'Class, Status and Party'. This remained a central contribution by Weber in which he maintained that social position was based on three separate domains: class, status and party or power (Weber, 1922). Class refers to having economic resources and is indicated through measures of income. Status is characterised by an individual possessing a degree of respect and standing within the community. Status is based on family background, lifestyle and social networks. The last domain, power, is associated with the political context and the ability for an individual or group to fulfil their aims and flourish despite the resistance of others (Liberatos et al., 1988; Weber, 1922). Weber's three domains of social class provide the basis for the most common measures of socioeconomic position found within the literature, which seek to examine occupation, education and income.

## **Indicators of Socioeconomic Position.**

#### Education

Education is a commonly used indicator of socioeconomic position. Rooted within Weberian theory, an individual's education is associated with the status domain (Liberatos et al., 1988). Predominantly, education is completed in early adulthood. Therefore, in examining socioeconomic position across the lifespan it can be used to measure early life socioeconomic position (Davey Smith et al., 1998). In terms of measurement, education can be both assessed as a continuous variable by examining the number of years of completed education, or as a categorical variable by evaluating key educational indicators across the life course such as completion of primary school, high school, further education and degrees. The two variables of measurement make distinct assumptions about how socioeconomic position is indicated (Liberatos et al, 1988). The continuous measure assumes that the number of years of completed education contributes equally to an individual's indicated socioeconomic position. Whilst, on the other hand, the categorical measure assumes that the level of educational achievement is more important than years spent completing education (Liberatos et al, 1988).

## Income

Income is a determinant of socioeconomic position, which measures those material resources that an individual possesses. Mostly referring to material resources of an individual or household's financial situation. Income is utilised to purchase commodities and services that allow an individual to flourish. Income has a dose response relationship with health, in such a way that those with a higher income tend to have better health outcomes than those on a lower income (Ecob & Davey Smith, 1999).

Income can be broken down into individual income or that of a household. Income encompasses several different monetary domains and includes wages, annual salary, profits and any earnings received. Furthermore, any type of benefit an individual is in receipt of also constitutes income (Ecob & Davey Smith, 1999).

A key feature of income is that it is cumulative over the life span (Lynch et al., 1997). In contrast to other indicators of socioeconomic position (e.g. education), income has the potential to change and because of this does not always remain the same, for example, if a person no longer meets the criteria to receive benefits or they must stop working for a given reason.

## **Occupation**

Occupation is a commonly used measure to determine socioeconomic position. Associated with Weberian theory, occupation demonstrates Weber's domains of class and status. Within the Western part of the world, occupation is a symbol of a person's role within society, their social standing and is strongly associated with income and educational attainment. In terms of measurement, invariably studies measure the current employment or the employment of the longest duration held by the individual to indicate their socioeconomic position. Occupation can also be seen as encompassing both employers and employees and is associated, albeit less frequently, with Marxist theory in going towards demonstrating the power inequalities within society between the two groups. Lower occupational level is associated with increased employment insecurity, which refers to the perceived fear of losing one's job (Mohr, 2000). Furthermore, lower paid employment is associated with less development opportunities and less decision latitude, which is a term that describes the employee's level of control over their work and conduct during their time at work (Karasek, 1979). The fear of loss of employment has a critical bearing on both physical and mental health, although the effect on an individual's mental health is larger than the effect on their physical health (Sverke et al., 2002).

## Socioeconomic Position and Health

Social and economic circumstances have an influence on health throughout the life course and as such are commonly referred to as the 'social determinants of health' (Braveman & Gottlieb, 2014). It has been well documented in the literature that disadvantaged socioeconomic position has been linked with poor health outcomes.

In a cohort study of 51, 417 adults from the United Kingdom it was found that the risks for moderate and severe depression were substantially increased amongst those people experiencing a more disadvantaged socioeconomic position (Lob et al., 2020). In a cross sectional study of 10, 800 adults from Poland, Finland and Spain it was found that socioeconomic position, indicated by education and level of household income, was inversely related to depression. Furthermore, the findings revealed that higher educational attainment (although not income) substantially reduced the risk of depression in each of the countries (Freeman et al., 2016). Indeed, it is well documented that there is a social gradient across the social determinants of health, accordingly there is a broad evidence base consistently maintaining that those with lower income and educational attainment are more likely to experience worse health outcomes than people with higher income and educational attainment (Braveman & Gottlieb, 2014). Disadvantaged socioeconomic position has been linked with poorer living standards, such as unsafe housing and difficulty accessing food, which have been associated with depression and suicidality (Chen et al., 2017; Madigan & Daly, 2023). Lower socioeconomic position is also associated with anxiety (Green & Benzeval, 2013) and risk of psychosis (Sweeney et al., 2015).

Socioeconomic position is also associated with poorer physical health outcomes and morbidity. Findings from a meta-analysis of 48 independent prospective cohort studies showed that people of a lower occupation were 46% more likely to die early than those of a higher occupation (Stringhini et al., 2017). Socioeconomic position has also been associated with specific diseases, including cardiovascular disease (Schultz et al., 2018) and diabetes (Tsalamandris, et al., 2019). The ways in which these social determinants influence health is multifaceted and involves interconnecting factors that are often occurring over an individual's life course (Bibby, 2018). Whilst there is mounting evidence demonstrating potential causal links from socioeconomic position to poor health, there is a plausible bidirectional relationship, in other words, poor health may lead to more socioeconomic disadvantage. Chronic health conditions can impact on an individual's employment status (Varekamp et al., 2013). Long-term health conditions such as diabetes and cardiovascular disease are both associated with unemployment, resulting in loss of potential income and greater financial precarity (Pedron et al., 2019).

## Inflammation

Inflammation refers to the body's process of fighting injuries and pathogens in order to heal itself. The nomenclature inflammation has its origins in the Latin word *inflammare*, meaning 'to cause to catch fire' (Merriam-Webster, n.d.). The Roman scholar Aulus Cornelius Celsus (c.25 BC – c. 50AD) first referred to inflammation as being indicated by heat, redness, swelling and pain (Scott, 2004).

During the inflammatory response, the immune system releases white blood cells which subsequently release cytokines (small proteins) which subside once the threat has passed (Hansel et al., 2010; Slavich, 2015). The inflammatory response is not static but rather fluctuates and is one that is activated in response to infections, such as disease (Slavich, 2014). Accordingly, temporary increases in inflammation are an adaptive process that are fundamental to survival. Inflammation can be categorised into two different types: acute inflammation and chronic inflammation. Acute inflammation is rapid and temporary and plays an important role in minimising the potential for infection or injury, it also contributes to regaining physiological homeostasis and subsequently there should be a resolution of the inflammatory process. On the other hand, chronic inflammation occurs when the immune response stays in the body for months or years, leaving it in a state of high alert (Straub, 2017). Chronic inflammation is linked with several diseases across the lifespan that are the leading causes of chronic illness and disability worldwide (Furman et al., 2019). The inflammatory response is also triggered by psychosocial factors, such as persistent stress or adverse experiences (Danese & Lewis, 2016; Lacey et al., 2020), in which case the psychological distress experienced in turn activates the sympathetic nervous system and subsequently invokes an inflammatory response (Bierhaus et al., 2003).

## Assessing Inflammation

Inflammation is assessed through blood sampling which aims to detect proteins that are made by the liver or the immune system. The most frequently assessed biomarkers are acute phase proteins, these include C-Reactive Protein (CRP) and Fibrinogen, and cytokines, these include TNF $\alpha$  and Interleukins 1 $\beta$ , 6, 8, 10 and 12 (Menzel et al., 2021). The current study will focus on the former type of inflammation, specifically CRP and fibrinogen. Given both CRP and fibrinogen have been shown in previous studies to be related to both physical and mental health (Danese & Lewis, 2016).

## CRP

CRP was discovered by two researchers, Tillet and Frances, in 1930 when they identified that CRP was found in the serum of patients with acute inflammation who were experiencing *Pneumococcus*. Located and expressed on the first chromosome, CRP is a cytokine that is produced by the liver and is increasingly secreted in response to the release of inflammatory cytokines (Pepys & Hirschfield, 2003).

There are many causes of increases in CRP. These include both acute and chronic conditions and, in terms of aetiology, can be both infectious and non-infectious. CRP is associated with physical illness and psychological distress (Bierhaus et al., 2003).

CRP levels are determined through blood sampling. Importantly, there are certain medications that interfere with CRP levels, such as statins (e.g. Atorvastatin and Fluvastatin) and non-steroidal anti-inflammatory drugs (e.g. Ibuprofen) that can decrease CRP levels

(Kandelouei et al., 2022; Nehring et al., 2023). Conversely, if a person has a recent injury or illness this can result in rapidly increasing CRP levels (Nehring et al., 2023).

## Fibrinogen

Fibrinogen is glycoprotein complex produced in the liver and is a marker of inflammation. It is the precursor of fibrin and plays a critical role in influencing blood viscosity. It additionally plays an important role by facilitating the process of coagulation (Fibrinogen Studies Collaboration, 2004). Fibrinogen additionally reflects inflammation in the body. Increased levels of fibrinogen are associated with physical illness and psychological distress (Martins-de-Souza et al., 2014).

#### Socioeconomic Position and Inflammation

A large body of research has shown an important relationship between socioeconomic position and inflammation. As such, greater socioeconomic disadvantage is associated with increased concentrations of inflammatory biomarkers, including CRP (Alley et al., 2006; Davillias et al., 2017; Farmer et al., 2021; Kershaw et al., 2010) and fibrinogen (Kim et al., 2016; Steptoe et al., 2003). Inflammation is hypothesised to be a critical underlying mechanism linking socioeconomic position with health outcomes (Miller et al., 2011).

Lower socioeconomic position has been shown to be associated with poorer physical and mental health outcomes (Lago et al., 2018), increased psychological distress in both adults (Talala et al., 2011) and children (Kelly et al., 2017) and symptoms of depression (Azizoddin et al., 2017; Hoebel et al., 2017). A systematic review by Nazmi and Victoria (2007) of cross sectional and cohort studies found that of the studies reviewed rates of CRP were heightened in people that were experiencing persistent stress related to low socioeconomic position. The more years of education a person has completed has been associated with lower levels of CRP (Wu et al., 2002). Furthermore, higher income families have been found to display lower levels of CRP (Jousilahti et al., 2003). Many studies have shown an inverse relationship between socioeconomic position and inflammatory biomarkers. A study by Hemingway et al. (2003), using the civil service employment grade (which measures social position as relating to occupation by assessing job control and job demands), found that lower status occupations had increased levels of CRP and Interleukin-6 (IL-6). Although this association was reduced when taking into account waist-hip ratio. It is clear from the broad evidence base available that there are many other factors that need to be taken into consideration when delineating the relationship between socioeconomic position and inflammation.

#### Socioeconomic Position and Inflammation: Potential Mediators

A key area of epidemiological research has focussed on examining the relationship between socioeconomic position and inflammation and has sought to identify potential mediators (Muscatel et al., 2018). A mediator is an intervening variable that may explain the relationship between the dependent and independent variable (VanderWeele, 2016). Further research should examine the potential mediating role of other factors within the link between socioeconomic position and inflammation. A key finding is that higher body mass index (BMI) is associated with enhanced levels of inflammation (Murray et al., 2015). However, being overweight cannot exclusively explain inflammation in the body, given that obesity is also strongly associated with adverse outcomes such as increased psychological distress (Sarwer & Polonsky, 2016) and poor sleep (Beccuti & Pannain, 2011). Psychological distress and poor sleep are also experiences that are commonly related to socioeconomic position (Cohen et al., 2007), and associated with increased inflammation (Irwin et al., 2006). Taken together these findings suggest that difficulties with sleeping and psychological distress may play a mediating role in the link between socioeconomic position and inflammation.

## **Psychological distress**

## Defining the Concept of Psychological Distress

Psychological distress is a term used to refer to a group of experiences that are characterised by emotional discomfort and associated somatic symptoms (Goldberg & Goodyear, 2005). Specifically, psychological distress is defined as:

A set of painful mental and physical symptoms that are associated with normal fluctuations of mood in most people. In some cases, however, psychological distress may indicate the beginning of major depressive disorder, anxiety disorder, schizophrenia, somatization disorder, or a variety of other clinical conditions. It is thought to be what is assessed by many putative self-report measures of depression and anxiety (American Psychological Association Dictionary, Psychological Distress section).

Acute stress is characterised by stress that occurs for minutes or hours, whereas chronic stress occurs for days, weeks or months (Olff, 1999). Generally, psychological distress is as an experience that fluctuates in terms of the degree to which the emotional discomfort is experienced and is invariably a transitory experience. Psychological distress can result from psychosocial sources and is a key feature of many mental health difficulties (Goldberg & Goodyear, 2005). When psychological distress is assessed to be high, this indicates the individual is more likely to be experiencing difficulties with their mental health and may be characteristic of mental health difficulties such as anxiety and depression (Cuijers et al., 2009). Psychological distress is usually assessed through self-report measures. The General Health Questionnaire-12 (GHQ-12; Goldberg &Goodyear, 2005) is a popular measure of psychological distress (see Appendix A). The measure assesses concepts key to psychological distress such as cognitive function, self-esteem and low mood.

## **Psychological Distress and Inflammation**

There is evidence that there is communication between the neuroendocrine ('stress' mediating) and immune systems (Quan & Banks, 2007). There is a broad body of research showing that psychological distress has been associated with increased levels of inflammation (Maes et al., 1998). Similarly, stressful life experiences can result in heightened levels of inflammation (Slavich & Irwin, 2014). Endocrine and immune system dysregulation can be a consequence of persistent exposure to stressors, which results in increased inflammation (Rohleder, 2014).

Several studies have shown associations between increased levels of inflammatory biomarkers and depression (Valkanova et al., 2013). It was shown that levels of CRP could predict whether medication or a behavioural intervention may be the more suitable treatment for people with depression (Raison et al., 2018). The specific biomarkers that are indicated to be increased within people with depression include IL-1, IL-6, TNF- $\alpha$ , CRP and fibrinogen. Although, findings remain equivocal which has resulted in a need for research to elaborate further on the association between inflammation and depression and to delineate the precise links between inflammation and depression on certain types of symptoms (Rothermundt et al., 2001). Sin et al. (2015) maintained that everyday stressors are related to both increased levels of inflammation and individuals that experience low mood when encountering stressors are more likely to display increased inflammation. On the other hand, some studies

have found no link between psychological distress and inflammatory biomarkers (Linkas et al., 2022).

There is some evidence suggesting that inflammation is more associated with the somatic symptoms of depression as compared to affective symptoms (DellaGioa & Hannestad, 2010). In this study, patients received a course of a pharmaceutical therapy known as Interfon- $\alpha$  (IFN- $\alpha$ ), which is a drug used to treat a range of conditions including Leukeamia and hepatitis B. IFN- $\alpha$  is known to induce inflammation. In the first weeks, patients who received this drug presented with slowed motor movements, fatigue, altered sleep and changes in their appetite, which were non-responsive to antidepressants. Interestingly, some of these patients additionally developed affective symptoms associated with depression such as low mood or loss of enjoyment. These affective symptoms were reduced in response to antidepressant medication, unlike the somatic symptoms, which it is suggested were associated with the pro-inflammatory properties of IFN- $\alpha$ .

## Pathways from Psychological Distress to Inflammation

Given the association between psychological distress and inflammation, there has been significant interest to examine this relationship further and to delineate the potential pathways from the experience of stress to elevated levels of inflammation.

The main pathways associated with the stress response are the hypothalamic-pituitaryadrenal axis and the autonomic nervous system (Ader et al., 1995). In what follows, is a summary of how each process functions in response to psychological distress.

**The Hypothalamic-Pituitary-Adrenal Axis (HPA).** The HPA plays an integral role when a person experiences psychological distress by releasing cortisol. The HPA is regulated by a circadian rhythm, this is why cortisol levels are likely to be heightened in the morning and lower at night (Ramamoorthy et al., 2016). During the experience of psychological stress,

the hypothalamic paraventricular nucleus both synthesises and releases corticotrophin releasing hormone (CRH) into the hypophyseal portal system. The hypophyseal portal system is a system of blood vessels located at the base of the brain that connects the hypothalamus with the anterior pituitary, which refers to one of the two lobes of the pituitary gland. The main function of this system is to rapidly transport hormones from the hypothalamus to the anterior pituitary. Once CRH has been secreted it begins the release of adrenocorticotropic hormone (ACTH). Subsequently cortisol is released, which is one of the main glucocorticoids, from the zona fasciculata layer of the adrenal cortex (Thau et al., 2022). One of the key functions of glucocorticoids is to control immunologic processes (Chrousos & Gold, 1992) and recent research has shown evidence that glucocorticoids have a proinflammatory impact on the immune system (Elenkov, 2008).

When cortisol is released in small amounts, cortisol can actually have an adaptive function through boosting immunity by limiting inflammation (Cleveland Clinic, n.d.). The central nervous system has its own immune cells, known as microglia, circulated within the brain. During a response to a cortisol peak, microglia initiate both glucocorticoid and mineralocorticoid receptors (Calcia et al., 2016). Furthermore, increased cytokines, accumulation of monocytes and macrophages and elevated microglia have been detected in response to exposure to psychological stress (Johnson et al., 2005).

The autonomic nervous system (ANS). The autonomic nervous system plays an important role in moderating those automatic functions such as regulating heart rate, breathing, blood pressure and digestion. The ANS also plays an integral role in regulating the body's response to psychological stress. The parasympathetic nervous system is a subdivision of the autonomic nervous system. The sympathetic nervous system signals to the body to be on alert, contrastingly, the parasympathetic nervous system sends signals to the body which facilitate a return to normal activity levels (Schmidt & Thews, 1989). Colloquially, the two systems are respectfully referred to as the 'fight/flight' and 'rest and digest' responses (Schmidt & Thews, 1989).

When psychological stress is experienced the hypothalamus releases corticotrophinreleasing hormone (CRH). This activates the noradrenergic centres in the brain stem and spinal cord. The locus coeruleus is activated in the pons of the brain stem. The locus coeruleus increases sympathetic activity through activation of the  $\alpha$ 1-adrenocepters on preganglionic sympathetic neurons and reduces parasympathetic activity through the activation of  $\alpha$ 2-adrenocepters on preganglionic parasympathetic neurons.

Neurotransmitters Involved in the Sympathetic and Parasympathetic Nervous System. Neurotransmitters are chemicals with the principle function to allow neurons to communicate with each other. The neuron releases a chemical that travels to the target cell across a synapse (Sheffler et al., 2022). The main neurotransmitters involved with the sympathetic nervous system are acetylcholine, norepinephrine and epinephrine. Contrastingly, the parasympathetic nervous system utilises acetylcholine as the principle neurotransmitter. In most circumstances, once a stressful situation has passed, the parasympathetic nervous system is activated which releases hormones to relax the body and mind in order to regain a state of homeostasis. In contrast, when the stress experienced is persistent, the sympathetic nervous system proceeds to be activated with the exception of the parasympathetic nervous system providing its compensating role. Therefore, when stress is persistent there is an increase in catecholamine's (norepinephrine and epinephrine) and a decrease in levels of acetylcholine (Chrousos & Gold, 1992). This lessens the impact of the parasympathetic nervous system providing its fundamental counterbalancing role.

#### Socioeconomic Position, Psychological Distress and Inflammation

Symptoms of psychological distress are common within the general population (Goldberg & Goodyear, 2005). Although the extent to which psychological distress is experienced varies within and across countries of Europe, which suggests important demographic and economic factors that may influence the development and maintenance of psychological distress (Schlax et al., 2019).

A number of studies have found that elevated levels of CRP and fibrinogen are associated with psychological distress (Wium-Andersen et al., 2013; Muscatell et al., 2020; Lazzarino et al., 2015; Goldman-Mellor et al., 2010), sleep disturbances (Patel et al., 2009; Meier-Ewert et al., 2004; Grandner et al., 2013) and socioeconomic position (Lubbock et al., 2005; Berger et al., 2019; Davillas et al., 2017).

## **Sleep Quality, Sleep Duration and Inflammation**

Sufficient sleep in terms of quality and duration is associated with improved psychological wellbeing, augmented energy levels and reduction in fatigue and exhaustion (Silva-Costa et al., 2015). Indeed, good quality sleep plays a fundamental role in maintaining mental and physical health (Chokroverty, 2010). Contrastingly, sleep problems refer to disturbances of subjective sleep quality and sleep duration (Lallukka et al., 2012; Patel et al., 2008). Both sleep quality and sleep duration are commonly used measures within studies examining the relationship between sleep and health outcomes (Bei et al., 2016). A long sleep duration refers to sleeping 9 hours or more (Ren et al., 2019) and a short sleep duration refers to a sleep of under seven hours (Centres for Disease Control and Prevention, n.d.). A long sleep duration is associated with higher levels of CRP and IL-6 (Irwin et al., 2016). Similarly, a short sleep duration is associated with cardiovascular disease, coronary heart disease and obesity (Itani et al., 2017). Poor sleep quality has also been linked to having an adverse impact on health and has been linked to a greater likelihood of having a stroke, heart attack and increased risk of obesity (Wu et al., 2020). Sleep is modifiable and as such is susceptible for intervention that can have potential positive health outcomes (Irwin et al., 2015). Markers of inflammation such as CRP, interleukin 6 (IL-6) and fibrinogen have all been associated with poor sleep, specifically elevated inflammatory markers have been linked with sleep disturbances (Bjurström et al., 2017; Dowd et al., 2011; Dzierzewski et al. 2020).

The mechanisms that may influence the relationship between sleep and inflammation include the hypothalamus-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS). Both of these systems modify the basal gene expression profile toward release of greater proinflammatory cytokines (Slavich & Irwin, 2014). There is an existing understanding that sleep problems are associated with psychological distress (Zhang et al., 2022). The relationship between sleep and inflammatory cytokines and some inflammatory cytokines encourage sleep (Veler, 2023). However, in contrast to this evidence base there are several studies that report no association between sleep and inflammatory biomarkers (Irwin et al., 2016; Patel et al., 2009). One explanation of this is that elevated levels of inflammation can lead to an individual utilising sleep as a key form of recovery (Irwin et al., 2016).

## Links to Clinical Practice

Given the evidence suggesting that inflammation is linked with psychological distress, sleep quality and sleep duration, there is an increasing interest in exploring these relationship further due to the clinical applications of these findings. Psychological interventions aim to reduce an individual's distress and improve sleep, such interventions for depression have been shown to attenuate inflammation (Thornton et al., 2009). Previous studies indicated that increased inflammation is a precursor to worse outcome of antidepressants (Liu et al., 2019). However, it is complicated to delineate the effects of the medication on levels of inflammation (Strawbridge et al., 2020). In contrast with psychotropic medication, psychological therapies have shown to be associated with reducing inflammation. In a Randomised Control Trial (RCT) completed by O'Toole et al. (2018), maintained that psychological therapy attenuated levels of CRP. On the contrary, a review of 12 studies reported that psychological therapies showed no significant improvement on inflammation levels in individuals with depression. This suggests that findings are inconsistent within this area, one way of understanding these variations in outcomes is the heterogeneity between individuals with mental health problems. Furthermore, inflammation appears to be more associated with the somatic depression which some authors maintain is suggestive of a sub-category of individuals who are more likely to have both somatic symptoms and elevated levels of inflammation (Strawbridge et al., 2020).

## **Policy context**

The NHS 2022/23 priorities and operational planning guidance proposes a role for Integrated Care systems in addressing health inequalities. This indicates that individuals from socioeconomically deprived backgrounds and particular ethnic groups are more likely to experience worse health outcomes than individuals who are more socioeconomically advantaged. There is a drive for services to provide targeted strategies that will be both supportive and ultimately aim to take preventative action as the main aim of their approach. A key critique of government approaches to addressing health inequalities has been the absence of implementing a long-term, systemic approach to reducing health inequalities. Although the NHS has also been criticised for not having a substantial focus on alleviating health inequalities (The King's Fund, 2018).

## **Systematic Review**

## **Overview**

Within this section, a systematic narrative review of the literature on socioeconomic position and inflammation is presented. The aim of the review was to increase understanding of the association between socioeconomic position and inflammation. The review aimed to identify gaps in the literature, particularly in considering the mediating factors in the relationship between socioeconomic position and inflammation.

## Rationale and Objective

There has been a great amount of literature examining the links between socioeconomic position and inflammation. The extent to which inflammatory markers are associated with socioeconomic position varies across studies. This is possible due to variance in methodological concepts, demographic characteristics of the study population and the use of different measures of what constitutes socioeconomic position (Muscatell et al., 2020). The findings within this research area remain inconsistent. Therefore, it is difficult to determine the strength of the association between socioeconomic position and inflammation from a single study. Recent studies reviewing socioeconomic position and inflammation have focused on a North American population (Muscatell et al., 2020) or on socioeconomic position in childhood (Milaniak & Jaffee, 2019).

The current review involved completing a narrative synthesis following the steps outlined by Popay et al. (2006). This broadly included development of a theory of how the intervention works, forming a preliminary synthesis of the findings of the included studies, examining relationships between the findings of the studies and evaluation of the reliability of the synthesis. A meta-analysis was not undertaken due to methodological heterogeneity of the studies, for example, the heterogeneity of criteria used to measure socioeconomic position. Instead, a descriptive synthesis was completed.

Therefore, the objective of this review was to describe and synthesise findings of all studies that examined the association between inflammation and socioeconomic position in both adulthood and childhood. The research questions were:

1. To examine whether socioeconomic position is related to inflammation.

#### **Search Strategy and Information Sources**

A systematic review of the literature was completed using Medline and PsychINFO between the years 2000 and 2023. In creating a search strategy, the Population, Intervention, Comparison, Outcome (PICO) framework (Richardson et al., 1995) was used to ascertain the key themes that were intended to be reviewed and to generate key words and synonyms to be searched for. A broad set of search terms were highlighted, each relating respectively to socioeconomic status and inflammation. To uphold sensitivity, the set of search terms for each concept were combined with the Boolean operator OR and were then combined using the Boolean operator AND. The databases were each searched using the following key words: "Socioeconomic status", "socioeconomic position", "social position" AND "inflammation" OR "CRP" OR "C-Reactive Protein" OR "Fibrinogen" OR "Biomarker". These terms were used given they comprise the main themes and objective of the literature review, whilst also having been used successfully in other peer reviewed literature reviews based on similar research areas (e.g. Muscatell et al. 2018). The broader terms relating to socioeconomic position were chosen specifically. Initial searches were carried out using the individual elements comprising socioeconomic position (i.e. 'education' and 'income'), however, this resulted in a plethora of other research areas which were subsequently

identified by the database but were clearly irrelevant to the aim of the literature review, therefore it was decided that these terms were to be excluded as search terms.

The search of the literature on Medline and PsychINFO and was carried out between 17th July 2023 and 31st July 2023. To ensure the review was up to date as possible, a final search was performed on 2nd August 2023 on all three databases. A manual search was conducted by examining reference lists of both selected articles and those cited in systematic reviews on the similar area to identify any additional papers relevant to the present review criteria.

## Eligibility Criteria

Studies examining the relationship between socioeconomic position and inflammation were identified. Titles and abstracts were screened against the following inclusion criteria:

- 1) Empirical study
- 2) Measured socioeconomic position
- 3) Measured inflammation
- 4) Peer reviewed

Articles were screened against the following exclusion criteria:

- 1) Conference extracts and dissertations
- 2) Not an empirical study
- 3) Non-human sample

## Selection Process

Following initial database searches, articles were retrieved and merged together using Microsoft Excel and any duplicate records of the same article were removed. The author independently examined each article by title and abstract to ascertain whether the article was relevant to the review objective. At this stage in screening articles were included if it was unclear as to whether they met the eligibility criteria, this was with the view that they could be removed at a later stage following further scrutiny. Finally, the full text was retrieved and examined to see if it met with eligibility criteria for inclusion in the final review (see Figure 1).

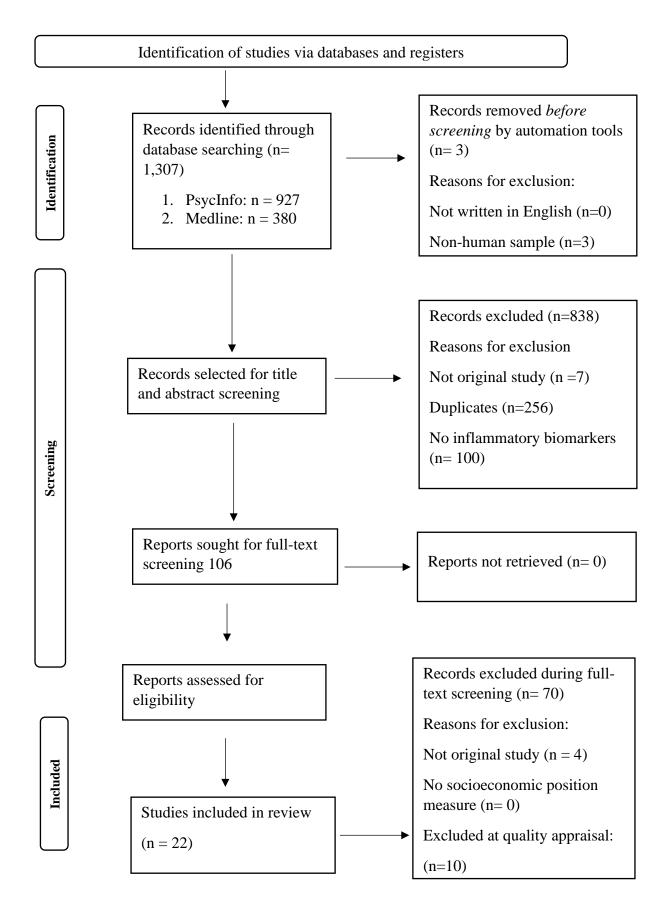
## **Data Collection Process**

The author independently extracted data from all eligible studies and any queries regarding study eligibility were discussed and resolved through discussion with colleagues until a consensus was reached. The data extracted from each study was put into tabular format.

## Results

1307 articles were selected for title and abstract screening, and subsequently 106 articles were selected for full text screening (see Figure 1). A total of 22 papers were included in the review. Table 1 presents general characteristics of each of these studies together with their main findings.

Figure 1 PRISMA Flow Chart



**Study Characteristics.** Characteristics of the studies are outlined in table 1; this included 22 studies exploring the links between socioeconomic position and inflammation. All studies were published between 2001 and 2020 and were completed in different countries: United States (n= 12), Brazil (n=1), Canada (n=1), United Kingdom (n=4), Mexico (n=1), Canada (n=1), Japan (n= 1) and Finland (n=1). Three studies (Clark et al., 2012; Gallo et al., 2012; Paul et al., 2008) included exclusively female participants. Two studies (Jousilahti et al., 2003; Ishizaki et al., 2001) included exclusively male participants. The ethnicity of participants was given in two studies (Chen et al., 2015; Clark et al., 2012), with the remaining studies reporting the percentage of the sample as people who identified as non-white which ranged from zero to 100%.

The majority of the studies included data that was gathered as part of larger projects (Appleton et al., 2012; Boylan & Ryff, 2013; Camelo et al., 2014; Clark et al., 2012; Davillas et al., 2017; Dowd et al., 2007; Janicki-Deverts et al., 2008; Jousilahti et al., 2003; Koster et al., 2006; Loucks et al., 2005; Muennig et al., 2007; Mwendwa et al., 2013; Owen et al., 2002; Steptoe et al., 2003; Zhang et al., 2008). Six studies were independent and recruited participants from the community (Chen et al., 2015; Gallo et al., 2012; Hostinar et al., 2015; Ishizaki et al., 2001; Paul et al., 2008; Thomas et al., 2005).

Socioeconomic position was measured primarily through self-report questionnaire in 10 of the studies (Davillas et al., 2017; Gallo et al., 2012; Janicki-Deverts et al., 2008; Jousilahti et al., 2003; Koster et al., 2006; Loucks et al., 2005; McDade et al., 2006; Owen et al., 2002; Steptoe et al., 2003; Thomas et al., 2005). Socioeconomic position was also measured through interview, in four of the studies a face-to-face interview was utilised (Appleton et al., 2012; Camelo et al., 2014; Dowd et al., 2007; Zhang et al., 2008) and one study used a telephone based interview (Boylan & Ryff, 2013). Two studies indicated socioeconomic position by educational attainment (Boylan & Ryff, 2013; Louks et al., 2005) and three utilised household income (Chen et al., 2015; Clark et al., 2012; Paul et al., 2008). One study indicated socioeconomic position through individual salary of each participant (Ishizaki et al., 2001) and one study used employment grade (Steptoe et al., 2003). The remaining studies utilised both household income and educational attainment as indicating socioeconomic position (Davillas et al., 2017; Dowd et al., 2007; Gallo et al., 2012; Janicki-Deverts et al., 2008; Jousilahti et al., 2003; Koster et al., 2006 (ownership of financial assets); Muennig et al., 2007; Mwendwa et al., 2013; Owen et al., 2002; Zhang et al., 2008).

Three studies measured socioeconomic position by parental educational attainment, household income and parental occupation (Appleton et al., 2012; Camelo et al., 2014; Hostinar et al., 2015).

Table 1	1
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Main characteristics of the 22 included studies of the systematic review.

Publication	Sample size ( <i>N</i> )	Non- white (%)	Country	Age	Male/Female ratio (%)	Measures of socioeconomic position	Markers of inflammation	Key findings
Appleton et al. (2012)	430	20	United States	42	41/59	Household income in childhood, parental educational attainment and occupation	CRP	<ul> <li>Increased levels of inflammation were associated with education, household income and occupation.</li> <li>Adult inflammation was observed to be higher in those adults who were exposed to lower socioeconomic position as children.</li> <li>Disparities in CRP may have developmental origins in childhood. Socioeconomic position was a moderating factor in linking child emotional functioning and adult CRP.</li> <li>There was no evidence that childhood emotional functioning mediated the link between early life socioeconomic position and adult CRP.</li> <li>Adult BMI mediated part of this relationship between educational attainment of the parent with CRP.</li> </ul>

Publication	Sample size ( <i>N</i> )	Percent non-white	Country	Age	Male/Female (%)	Measures of socioeconomic position	Markers of inflammation	Key findings
Boylan and Ryff (2013)	1054	93	United States	58	55/45	Education	CRP, IL-6	• Smoking, alcohol consumption and physical activity were not significant in explaining this relationship between inflammation and socioeconomic position.
Camelo et al. (2014)	13,371	47.8	Brazil	52	NR	Childhood: parental education. Adulthood: household income and occupational social class	CRP	<ul> <li>Lower childhood socioeconomic position was associated with higher levels of CRP. Higher levels of inflammation were associated with increased adverse social circumstances during the life course.</li> <li>Health risk behaviours accounted for 13.4% of the effect of socioeconomic position on CRP for men and only 4.4% for women. Alcohol consumption was associated with higher CRP levels amongst men who were socioeconomic disadvantaged position.</li> </ul>
Chen et al. (2015)	7,943	57	Canada	55	49/51	Household income	CRP, IL-6	• As socioeconomic position lowered, shift and persist strategies were associated with lower CRP and IL-6.
Clark et al. (2012)	24, 664	5	United States	53	0/100	Household income	CRP, fibrinogen, sICAM	<ul> <li>Household income was associated with higher levels of fibrinogen and sICAM. More favourable state-level socioeconomic circumstances were correlated with lower CRP.</li> <li>High levels of CRP were found amongst women with a low-income in the most deprived states.</li> </ul>

Publication	Sample size ( <i>N</i> )	Percent non-white	Country	Age	Male/Female (%)	Measures of socioeconomic position	Markers of inflammation	Key findings
Davillas et al. (2017)	7, 943	NR	United Kingdom	NR	2163out of 7943 females	Education and household income	CRP, fibrinogen	<ul> <li>In terms of age, socioeconomic inequalities in CRP emerged in 30s, increased up to mid-50s or early 60s and decreased with age. Lower fibrinogen levels were associated with those with higher income and educational attainment.</li> <li>Health related behaviours played a role in the relationship, with BMI playing the greatest role. Smoking and healthy diet was most important at early and later middle life compared to older ages.</li> </ul>
Dowd et al. (2007)	931	0	United States		58.1	Education	CRP	<ul> <li>CRP was not found to be associated with socioeconomic position.</li> <li>CRP strongly related to health-related behaviours such as smoking and obesity.</li> </ul>
Gallo et al. (2012)	284	100	Mexico	49	0/100	Education, household income	CRP, IL-6, sICAM	<ul> <li>Lower educational attainment and income were associated with increased levels of CRP, IL-6 and sICAM-1.</li> <li>Obesity and dietary fat were implicated in this association.</li> </ul>
Hostinar et al. (2015)	360	35	Canada	36	44/56	Parental occupational status, current occupational status	CRP, IL-6	<ul> <li>Lower early life and current socioeconomic position were associated with increased levels of inflammation.</li> <li>Lower socioeconomic position in early life was associated with harsher family climate and socioeconomic position disadvantage in adulthood was associated with perceived stress.</li> <li>Found no evidence that smoking, alcohol consumption or physical activity played an explanatory role.</li> </ul>

Publication	Sample size (N)	Percent non-white	Country	Age	Male/Female (%)	Measures of socioeconomic position	Markers of inflammation	Key findings
Ishizaki et al. (2001)	4,334	0	Japan	NR	100/0	Annual income	Fibrinogen	<ul> <li>Higher levels of fibrinogen were shown in groups of a lower socioeconomic position.</li> <li>No clear relationships between job strain or job demands and fibrinogen levels.</li> </ul>
Janicki- Deverts et al. (2008)	1,117	37	United States	40	56/44	Household income, education	CRP	<ul> <li>Unemployment predicted higher levels of CRP aged 33-45 years old during 3 year follow up on initial CRP measurement.</li> <li>The association between unemployment and CRP was independent of BMI, income and education.</li> <li>Poor health behaviours such as smoking status, alcohol intake and physical activity accounted for 20% of this association.</li> <li>Chronic psychological distress is hypothesized to account for the remaining variance.</li> </ul>
Jousilahti et al. (2003)	1,503	NR	Finland	NR	100/0	Education, household income	CRP, fibrinogen, serum amyloid A protein (SAA)	<ul> <li>Levels of CRP, fibrinogen and SSA all decreased with increasing socioeconomic position. The inequalities in CRP and fibrinogen remained significant after taking into account smoking status and waist-to-hip ratio.</li> <li>The association between higher levels of inflammation and lower socioeconomic position was particularly pertinent among men below 60 years of age.</li> </ul>

Publication	Sample size ( <i>N</i> )	Percent non-white	Country	Age	Male/Female (%)	Measures of socioeconomic position	Markers of inflammation	Key findings
Koster et al. (2006)	3,044	1264 out of 3044	United States	74	49/51	Education, income and ownership of financial assets	CRP, IL-6, TNF-α	<ul> <li>Lower socioeconomic position was associated with elevated levels of CRP, IL-6 and TNF-α.</li> <li>Health related behaviours, such as smoking, alcohol consumption and obesity explained a significant part of this association.</li> <li>Adjustment for heart diseases, lung disease and diabetes accounted for less of the association.</li> </ul>
Loucks et al. (2006)	2,729	NR	United States	62	47/53	Education	CRP, IL-6, sICAM	• Lower educational attainment was associated with elevated levels of CRP, IL-6, sICAM.
McDade et al. (2006)	188	NR	United States	59	NR	Education	CRP	• Lower educational attainment was associated with higher levels of CRP. Waist circumference, latency to sleep, smoking and perceived stress were independently associated with elevated levels of CRP.
Muenning et al. (2007)	10, 524	NR	United States		NR	CRP, fibrinogen	CRP, fibrinogen	<ul> <li>Fibrinogen was not associated with socioeconomic position. Increased levels of CRP was associated with lower household income and educational attainment.</li> <li>This relationship persisted after controlling for age, dietary behaviours, smoking, exercise, BMI, gender, and ethnicity. Hypothesised stress differences between socioeconomic positions may play a role.</li> </ul>

Publication	Sample size ( <i>N</i> )	Percent non- white	Country	Age	Male/Female	Measures of socioeconomic position	Markers of inflammation	Key findings
Mwendwa et al. (2013)	198	100	United States	45	NR	Education, income	CRP, IL-6	<ul> <li>Increased levels of hostility and depression were associated with elevated levels of CRP.</li> <li>Dispositional depression and CRP was more associated with greater hostility and lower educational attainment.</li> </ul>
Owen et al. (2003)	240	0	United Kingdom	40	NR	Occupational status, employment, income, education	CRP	• Lower educational attainment and income was associated with increased levels of CRP, independent of sex, body mass, age, waist-hip ratio, smoking, alcohol consumption.
Paul et al. (2008)	219	100	United States	31	0/100	Income	CRP	<ul> <li>Lower income was associated with increased levels of CRP. Perceived stress and social support were not significantly related to CRP.</li> <li>Stressful life events were a predictor of CRP levels.</li> </ul>
Steptoe et al. (2003)	221	0	United States	50	NR	Grade of employment	Fibrinogen	<ul> <li>Fibrinogen was higher in low employment grade groups compared to high employment grade groups. Women had higher fibrinogen levels than men.</li> <li>Men with low job control revealed greater fibrinogen levels in response to acute stress compared to those with high job control.</li> </ul>
Thomas et al. (2005)	208	0	United Kingdom	12	51/49	Free school meal eligibility, indices for deprivation (income, education and employment)	CRP, fibrinogen, homocyst(e)ine (Hcy)	• Elevated levels of fibrinogen and CRP were found in higher socioeconomic group.

Publication	Sample size ( <i>N</i> )	Percent non- white	Country	Age	Male/Female (%)	Measures of socioeconomic position	Markers of inflammation	Key findings
Zhang et al. (2008)	792	50	United States	53	50/50	Education, income	CRP	• Increased levels of CRP were associated with lower educational attainment and income, although sex, ethnicity, BMI and smoking were shown to be significant in this relationship.

*Note.* CRP, C-Reactive Protein; IL-6, Interleukin 6; NR, Not Reported; sICAM, Soluble Intercellular Adhesion Molecule; SAA, serum amyloid A protein; TNF-α, Tumor Necrosis Factor Alpha; BMI, Body Mass Index

Methodological Quality Assessment. Several tools were considered to assess the quality of the studies included within the present review. These included the National Institutes of Health (NIH) quality assessment tool for cross sectional studies, the Appraisal Tool for Cross-Sectional Studies (AXIS) and the Joanna Briggs Institute (JBI) Checklist for Analytical Cross-Sectional Studies. All of these tools are recommended tools for assessing the methodological quality of cross-sectional studies. Out of these tools, the JBI (Moola et al., 2020) is the preferred one (Ma et al., 2020). The JBI is an international, not-for-profit researching and development organisation based in Adelaide, South Australia. Therefore, it creates appraisal checklists that assess the methodological quality of health care studies. The purpose of the JBI is to evaluate the methodological quality of a study and to assess the risk of potential bias within the study. The JBI is comprised of eight questions that are described in below (see Categories). The process of using the JBI checklist involved considering whether the study met eight predetermined questions which are outlined within the tool. The questions are answered with a response of either 'yes', 'no', 'unclear' or 'not applicable'. A response of 'yes' indicates that the criterion was met and a response of 'no' indicates the criterion was not met.

For further information (see Appendix C) regarding the process of quality appraisal including a detailed explanation of each of the eight items together with a description of the information which was used to judge each individual question (i.e. the minimum criteria to give a 'yes' response).

**Categories.** Table 2 outlines the quality check performed and the extracted characteristics pertaining to the quality questions. A review of the quality check is outlined in further detail and the eight checklist items are grouped into the following categories: Sample (checklist items 1 and 2), Reliable Measure (checklist items 3 and 4), Inclusion of

Confounding Factors (checklist items 5 and 6) and measurement of the outcome (checklist items 7 and 8).

Table	2
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Quality Assessment using the JBI Checklist for Analytical Cross-Sectional Studies

	Were the criteria for inclusion in the sample clearly defined?	Were the study subjects and the setting described in detail?	Was the exposure measured in a valid and reliable way?	Were objective, standard criteria used for measurement of the condition?	Were confounding factors identified?	Were strategies to deal with confounding factors stated?	Were outcomes measured in a valid and reliable way?	Was appropriate statistical analysis used?
Appleton et al. (2012)	Yes	Yes	Yes	Yes	Unclear	Yes	Yes	Yes
Boylan and Ruff (2013)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Camelo et al. (2014)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Chen et al. (2015)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Clark et al. (2012)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Davillas et al. (2017)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

 Table 2 (Continued)

#### Were Were the criteria Were the study Was the Were confounding Were strategies Were outcomes Was appropriate for inclusion in subjects and the objective, factors identified? to deal with measured in a valid statistical analysis exposure the sample clearly setting measured standard confounding and reliable way? used? defined? described in factors stated? in a valid criteria used detail? and for reliable measurement way? of the condition? Yes Dowd et Yes Yes Yes Yes Yes Yes Yes al. (2007) Gallo et Yes Yes Yes Yes Yes Yes Yes Yes al. (2012)Hostinar Yes Yes Yes Yes Yes Yes Yes Yes et al. (2015) Ishizaki Yes Yes Yes Yes Yes Yes Yes Yes et al. (2001) Yes Janicki-Yes Yes Yes Yes Yes Yes Yes Deverts et al. (2008)

	Were the criteria for inclusion in the sample clearly defined?	Were the study subjects and the setting described in detail?	Was the exposure measured in a valid and reliable way?	Were objective, standard criteria used for measurement of the condition?	Were confounding factors identified?	Were strategies to deal with confounding factors stated?	Were outcomes measured in a valid and reliable way?	Was appropriate statistical analysis used?
Jousilahti et al. (2003)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Koster et al. (2006)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Loucks et al. (2006)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
McDade et al. (2006)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Muenning et al. (2007)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

	Were the criteria for inclusion in the sample clearly defined?	Were the study subjects and the setting described in detail?	Was the exposure measured in a valid and reliable way?	Were objective, standard criteria used for measurement of the condition?	Were confounding factors identified?	Were strategies to deal with confounding factors stated?	Were outcomes measured in a valid and reliable way?	Was appropriate statistical analysis used?
Owen et al. (2003)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Steptoe et al. (2003)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Thomas et al. (2005)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Zhang et al. (2008)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

# **Results of Quality Assessment**

Sample (checklist items 1 and 2). Predominantly the studies had a representative sample in terms of age, gender and ethnicity. However, some studies had a similar sample in terms of certain participant demographics, a result of having a homogenous sample is that it may affect the overall generalisability of the findings. Two studies had exclusively male participants (Janicki-Deverts et al., 2008; Jousilahti et al., 2003). Three studies (Clark et al., 2012; Gallo et al., 2012; Paul et al., 2008) included female only participants. Three studies had an exclusively white, middle-aged sample (Owen et al., 2002; Steptoe et al., 2003., Thomas et al., 2005). Four of the studies had a relatively small sample size which may affect the generalisability of the findings (Gallo et al., 2012; Hostinar et al., 2015; Owen et al., 2002; McDade et al., 2006;).

**Reliable measure (checklist items 3 and 4).** All of the studies included within the review utilised robust recommended indicators of socioeconomic position as outlined by the American Psychological Association (APA, 2007). In regards to inflammatory biomarkers, all of the studies used recommended inflammatory biomarker assays.

Inclusion of confounding factors (checklist items 5 and 6). In terms of taking into consideration confounding variables, all of the studies included within the review took into consideration potential confounding effects of individual demographics and health behaviours. Some studies were identified because they did not include particular confounding factors. Chen et al. (2015) did not include health behaviours such as smoking and alcohol consumption as potential covariates. Four studies did not take into consideration alcohol consumption or smoking status of participants as confounding factors within their analysis (Loucks et al., 2006; Jousilahti et al, (2003); Mwendwa et al., 2013).

Medications such statins, HRT and anti-inflammatory medications have been shown to influence inflammation (Kandelouei et al., 2022; Nehring et al., 2023). Seven studies did not take into consideration a participant's medication use as a confounding factor (Appleton et al., 2012; Boylan et al., 2020; Chen et al., 2015; Ishizaki et al., 2001; Mwendwa et al., 2013; Paul et al. 2008; Steptoe et al., 2003). No studies took into account use of HRT or contraception with the exception of Zhang et al. (2008). All of the studies used some form of multivariate analysis to take into consideration the covariates measured.

**Statistical analysis (checklist items 7 and 8).** All the studies used multivariate regression models, with the exception of one study that used ANOVA (Thomas et al., 2005).

# Synthesis of Systematic Review Findings

The following paragraphs summarise the narrative findings of the systematic review in regard to the research questions.

## Socioeconomic Position and Inflammatory Biomarkers

Socioeconomic position was associated with elevated levels of CRP in all studies (see table 1), except from Dowd et al. (2007) who found no association between socioeconomic position and CRP. Of the six studies that examined fibrinogen, all but one (Muenning et al. 2007) found that fibrinogen increased in relation to lower socioeconomic position. A table depicting the effect sizes and confidence intervals of the studies included in the systematic review which show the relationship between socioeconomic position and markers of inflammation included in the systematic can be found in Appendix D. In terms of effect sizes, greater effect sizes were found in studies that measured socioeconomic position by household income. Furthermore, larger effect sizes were shown in studies. All of the studies except from Dowd et al. (2007 and Muenning et al. (2007) had consistent medium to large effect sizes. The smallest effect sizes were -0.02 found in Dowd et al. (2007). The highest effect size was 0.48 which was reported by Paul et al. (2008) examining lower household income and increased concentrations of CRP.

Three studies explored levels of CRP in adulthood after examining their socioeconomic position in childhood (Appleton et al., 2012; Camelo et al., 2014; Hostinar et al, 2015). Of these studies, all three studies found that CRP was most elevated among those adults who had been exposed to a disadvantaged socioeconomic position in childhood. Appleton et al. (2012) found that those children with emotional difficulties who grew up in lower socioeconomic environment were more likely to have higher levels of CRP in adulthood. They found no evidence suggesting that emotional difficulties mediated the link between CRP and socioeconomic position. These findings suggest that inequalities in CRP may have their origins in childhood. Although, one study found that childhood socioeconomic position was not a critical period in which exposure to adverse circumstances results in lasting physiological impact (Camelo et al., 2014).

Two studies explored macro level socioeconomic conditions together with individual socioeconomic position (Clark et al. 2012; Gallo et al. 2012). The findings of Clark et al. (2012) revealed the importance of considering both micro and macro socioeconomic conditions through examining both individual socioeconomic position and state level deprivation. Using a large sample based in the United States, the authors found that higher levels of wealth and labour productivity and lower levels of state-level poverty and income equality were associated with lower levels of CRP. Personal household income was associated with sICAM-1 and fibrinogen than more macro state-level conditions. Whilst the sample size was large (n= 26, 029), a limitation is that the sample comprised only of female participants and due to the cross sectional design causal relationships between socioeconomic position and inflammatory biomarkers cannot be inferred. Contrastingly, Gallo et al. (2012) found neighbourhood socioeconomic circumstances were not statistically significant in the

link between socioeconomic position and inflammation, although a key limitation of this study was a small sample size.

## Mediators in the Association between Socioeconomic Position and Inflammation

Metabolic Alterations. Loucks et al. (2006) found that after adjusting for systolic blood pressure, diastolic blood pressure, total cholesterol, high density lipoprotein cholesterol ratio and cardiovascular disease, there was still an inverse relationship between educational attainment and income and levels of CRP and sICAM. Although lower childhood socioeconomic position was associated with higher levels of CRP in adult life, this association was not independent of adulthood socioeconomic position. There was a linear relationship between CRP increasing with the number of adverse social circumstances during a person's life. The metabolic alterations were the most substantial mediator between socioeconomic position and CRP, these included obesity, hypertension, cholesterol, hypertriglyceridemia and diabetes. Although, this mediation path accounted for less than half (49.5%) of the total effect of cumulative socioeconomic position on CRP among women, but only 20.2% among men. Koster et al. (2006) found that prevalent diseases that are associated with inflammation (including heart disease, diabetes and mellitus) explained less of the association between socioeconomic position and inflammation.

**Unemployment.** Janiki-Deverts et al. (2008) found that employment was associated with inflammation. Within this longitudinal study, the authors reported that having a history of unemployment during the three years following a CRP measurement was associated with elevated levels of CRP five to eight years later. This association was independent of BMI, ethnicity, age, educational attainment or income. Depression and health-related behaviours, including smoking, alcohol consumption and physical activity, accounted for approximately 22% of this association. Persistent psychological distress was not included in the analysis

and the authors maintain that this may have played a substantial role within this relationship, given the links between chronic psychological stress and levels of inflammation.

**Health Related Behaviours.** There were a number of health related behaviours that were implicated to be important in the relationship between socioeconomic position and inflammation. Smoking status was found to have relative importance in two studies (Loucks et al., 2006; Jousilahti et al. (2003). A number of studies demonstrated that BMI and waist to hip ratio were a significant factor in the association between socioeconomic position and inflammation (Jousilahti et al. (2003).

Loucks et al. (2006) found after adjusting for smoking and BMI there was still an inverse association between socioeconomic position (educational attainment and income) and levels of CRP and sICAM. Contrastingly, Hostinar et al. (2015) found no evidence that smoking, alcohol consumption or physical activity played an explanatory role in the association between socioeconomic position and inflammation. This study did have strict eligibility criteria in regards to health, which may have indirectly influenced the study sample by limiting the range of unhealthy behaviours to be included.

Koster et al (2006) found that health-related behaviours such as smoking, drinking alcohol and obesity explained a substantial part of the association between socioeconomic position and inflammation. Obesity and dietary fat were implicated in the association between socioeconomic position and inflammation (Gallo et al. 2012). Consistent with the previous studies, Davillas et al. (2017) found that health related behaviours were important in explaining the relationship between socioeconomic position and inflammation. Accounting for BMI, smoking status, physical activity and healthy diet partly explained the association between socioeconomic position and CRP. BMI exerted the most substantial influence in differences in CRP and educational attainment. Camelo et al. (2014) found that health risk behaviours accounted for 13.4% of the effect of socioeconomic position on CRP for men and only 4.4% for women, suggesting differences between men and women in terms of health-risk behaviours. Excessive alcohol consumption was associated with higher CRP levels and amongst men of a lower income. The prevalence of smoking and other health-risk behaviours were higher among men than compared to women. However, a substantial part of the association between socioeconomic position and inflammation could not be explained through health related behaviours or metabolic alterations, which suggests that other pathways, may play an important part. Psychological factors such as psychological distress were not included in the analysis and could be an important mediator as suggested by previous studies (Matthews et al., 2010).

Interestingly, in contrast to the other studies included within this review, Davillas et al. (2017) examined whether there were age-specific patterns between inflammation and socioeconomic position by examining differences across the adult age span. The authors found that heterogeneity in age was important in determining the role of health related behaviours. There was little evidence of age differences in the explanatory role of BMI and physical activity in the link between educational attainment and CRP. Smoking and healthy diet were most important at early and later middle life compared to older ages. In the case of fibrinogen, variances in BMI were less important in explaining income differences in fibrinogen as individuals got older, whereas physical activity was more relevant as individuals got older. For example, this disparity was shown through 33% of the income differences in fibrinogen were explained by the role of BMI at the age of 25 in comparison to 8% at the age of 70.

The study by Koster et al. (2006) used a sample of older adults (aged 70-79) and reported that low socioeconomic position was associated with elevated levels of IL-6, CRP and TNF- $\alpha$ . Health related behaviours, such as smoking, alcohol consumption and obesity,

accounted for the largest part of the association between socioeconomic position and inflammation.

**Psychological Factors.** A limited number of studies (n=3) examined psychological factors having a potentially mediating role within the relationship between socioeconomic position and inflammation. In the following section, those studies will be outlined.

Chen et al (2018) reported that those individuals with a lower socioeconomic position who utilised a shift-and-persist strategy had lower levels of inflammation. Therefore, by adopting a psychological approach that attenuates the perceptions of stress and reduces the inflammatory cytokine production. A similar finding was reported in children of a disadvantaged socioeconomic position; shift-and-persist was related to lower levels of CRP and IL-6.

Hostinar et al. (2015) investigated the concept of self-control. The authors defined self- control as "the ability to control one's impulses and abstain from gratifying immediate demands or desires". Self-control has been linked to the adoption and maintenance of healthrelated behaviours such as smoking, excessive alcohol use and obesity (Bogg & Roberts, 2012). These behaviours have been linked to inflammation (Elisia et al., 2020; Ellulu et al., 2017). Lower early life and current socioeconomic position was associated with increased levels of inflammation. Analysis revealed that lower self-control was important in this association.

Hostinar et al (2015) found that lower socioeconomic position in early life and lower socioeconomic position in adulthood was associated with perceived stress, both of which were related to a reduction in self-control (ability to control one's impulses) which was associated with abdominal adiposity (i.e. body fat) and linked with increased inflammation. This association was independent of participants current socioeconomic position, ethnicity, gender or age. The findings should be viewed tentatively given the cross-sectional design of the study, this precludes a definitive causal inference to be made.

One study explored whether differences in anger expression mediated the link between socioeconomic position and inflammation. Boylan and Ryff (2020) found that differences in level of anger management moderated the association between low educational attainment. Higher educational attainment was associated with elevated levels of IL-6 and fibrinogen and expression of anger, as shown through high trait anger, anger-out and low anger control. Whereas, individuals with lower educational attainment and with greater anger control had lower levels of IL-6.

## The Current Study

Using the *Understanding Society* data, a number of studies have explored associations between inflammatory biomarkers and socioeconomic position (e.g. Davillas et al., 2017), although no studies from this data set have examined psychological distress, sleep duration and sleep quality as potential mediators linking the association between socioeconomic position and inflammation.

Given the broad body of previous research showing the associations between socioeconomic position and inflammation, it is vital that current research considers the factors that potentially mediate this relationship. This may offer further understanding of the relationship between socioeconomic position and inflammation and offer a complimentary psychological perspective alongside one that is biological in nature.

## **Study Aim**

Based on previous findings showing that increased levels of inflammatory biomarkers are associated with psychological distress (Muscatell et al., 2020), socioeconomic position (Berger et al., 2019) and sleep disturbances, in terms of sleep quality and sleep duration, (Grandner et al., 2013), this study aimed to examine the potential mediating role of psychological distress, sleep quality and sleep duration in the link between socioeconomic position and inflammation, using the inflammatory biomarkers, CRP and fibrinogen.

Using data from *Understanding Society*: the UK Household Longitudinal Study, this research aimed to determine (1) whether socioeconomic position measured by educational attainment and income is associated with increased rates of inflammation, as measured by the inflammatory biomarkers CRP and fibrinogen (2) whether psychological distress mediates the link between socioeconomic position and inflammation and, (3) whether sleep quality or sleep duration mediates the relationship between socioeconomic position and inflammation.

# Hypotheses

Firstly, it will be hypothesised that increased levels of CRP and fibrinogen will be present in those with lower educational attainment and lower household income. Secondly, it will be hypothesised that psychological distress, sleep quality and sleep duration will be potential mediators in the link between socioeconomic position and inflammation.

## **Chapter Two: Methods**

## **Epistemological Position**

Epistemology is concerned with studying the nature, origin and range of knowledge. It is a derivative of the ancient Greek word episteme, which is defined as pertaining to "knowledge, understanding, skill and scientific knowledge" (Oxford Dictionaries, 2014). Ontology is a branch of philosophy that is characterised by the study of the beliefs about reality within the material, social, cultural and political contexts (Richards, 2003). Epistemology and ontology, whilst being two separate branches of philosophy, are related in so far as they both posit assumptions relating to the existence of entities and the acquisition of knowledge. Therefore, the relation between epistemology and ontology is important in taking into consideration both knowledge and the milieu in which it is produced (Gruba & Lincoln, 1994) Considering the ontological belief system is fundamental given it steers the researcher into making particular epistemological assumptions. According to Gruba and Lincoln (1994) if a singular truth is assumed the standpoint of the researcher is to be one of objectivity to allow for the acquisition of knowledge without the researcher's own bias having an impact on the process and findings of the research. The positivist approach is based on a standpoint that research is conducted from a position of objectivity (Aliyu et al., 2014).

The positivist-empiricist theory of knowledge is based on Francis Bacon's contention that knowledge about the natural world can be acquired through observation. Historically, the empiricist standpoint has been the predominant paradigm within psychological research, as exemplified through the work of the American philosopher and psychologist William James (Leahey, 2003). The empiricist standpoint maintains that true knowledge is arrived at through sensory experience (i.e. through application of the sense organs) and that reality is measurable in this way. The positivist tradition arose from the enlightenment period and was expounded by Auguste Comte in the early 19<sup>th</sup> century (Richards, 2003). Its theoretical assumptions are based on understanding the social world through the notion that knowledge can be acquired and as such, knowledge claims can be justified through verified data and general laws. In addition, through the conduction of experimentation it is maintained that the findings are invariably observable and quantifiable.

Emerging in the 1960s and 70s, the postpositivism stance was born out of a critique of positivism. Postpositivism is based on the ontological position of critical realism which makes the assumption that the social world exists independent of the researcher. Central to this stance is that knowledge of the social world can be only asserted imperfectly given the effects of possible biases of the researcher such as background knowledge can influence what is observed (Deluca et al., 2008). Therefore, within psychological research conclusions are based on probability as opposed to a singular truth. The work of Karl Popper (1959) was instrumental in asserting the principle hypothesis should be falsified rather than the positivist notion of verification of the hypothesis.

By taking such an approach, the investigator can examine a phenomenon in such a way without significantly influencing it or being influenced by it (Sale et al., 2002). The aim is to investigate a given phenomenon with the view to reveal causal effects or correlational relationships that are generalizable to a wider population and, therefore, affords the opportunity for the proposal of predictions to be made about a given population. An important component of this approach is that objective truths or facts can be delineated from subjective beliefs and values. The concept of validity is critical to this theoretical position and factors that may compromise validity are mitigated through scientific rigour, the purpose of which is to reduce the impact of subjective values and biases that may influence the research outcomes (Gruba & Lincoln, 1994). The quantitative positivist approach utilises methods that are rooted in statistical analysis, inferential statistics, structured research protocol, standardised psychometric tests and objective assays (Park et al., 2020). Given the main consideration that research findings should be applied to the wider population, a key requirement is that participant samples must be large enough to support claims of generalisability and ensure representativeness of findings (Park et al., 2020).

Given the present study is examining experiences of psychological distress and related sleep disturbances (sleep quality and sleep duration) it is important to acknowledge that the traditional view of mental health problems. This traditional position invariably proposes that mental health problems are discrete disease entities that can be classified by researchers (Bentall, 2003). The degree of validity and reliability of these putative mental disorders remains equivocal (Bental, 2010). Central to these debates is that disorders can be understood from one standpoint to be discrete biological entities and from another standpoint to be social constructs (Zachar & Kendler, 2017) that are shaped by the social and cultural context in which they are conceptualised (Kirmayer, 2005). This has resulted in mental health problems or psychological distress being understood as resulting from a complex interconnection between biology, psychology and socio-environmental factors, such an approach is known as the biopsychosocial paradigm and was first conceived by Engel (1977). In other words, as opposed to attributing a mental health problem as an entity that is located within an individual's body, rather a mental health problem or an individual's psychological distress is conceptualised as being closely linked to the individual's biological make up, psychology, social circumstances and the surrounding environment (Arnold & Wade, 2015). Given these assumptions, the current research was positioned within the postpositivist epistemological paradigm given it was rooted within empiricism and objectivism, whilst also

taking into consideration the possible biases of the researcher which can influence the research which is being undertaken.

#### Design

Given the assumptions of the postpositivist approach, a quantitative research design was utilised to address the aims of the current study. The design of the current study utilised a cross-sectional correlational analysis based on secondary data analysis collected as part of *Understanding Society*: the UK Household Longitudinal Survey (UKHLS; Buck and McFall, 2012; University of Essex, 2013). The UKHLS data set was selected for the current study given it affords the opportunity of a large and representative sample size and contains the variables of interest to the aims of the study.

# **Participants**

The current study used data from *Understanding Society:* the UK Household Longitudinal Study (Buck and McFall, 2012; University of Essex, 2013). The *Understanding Society* sample is comprised of 40,000 households in the United Kingdom, a stratified clustered sample of households recruited in 2009-2010, the former British Household Panel Survey (BHPS; which was conducted from 1991-2009), the Ethnic Minority Boost Sample and the Immigrant and Ethnic Minority Boost Sample. Information is gathered about members of the household; young people aged 10-15 are asked to complete a questionnaire and all adults are invited to participate in an interview. The objective of *Understanding Society* is to provide longitudinal data on various domains such as health and wellbeing, education, employment and social life.

In 2010-2012 direct health assessments and collection of blood samples were included in the overall survey. The health assessment entailed participant information being gathered regarding measures of specific health risks and key features that may be precipitating factors to chronic health conditions. Additionally, the blood samples have been used to measure a range of biomarkers. The term biomarker refers to "a characteristic that is objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or pharmacologic responses to a therapeutic intervention." (National Institutes of Health Biomarkers Definitions Working Group, 1998). The biomarker data from *Understanding Society* were collected by NatCen on behalf of the Institute for Social and Economic Research (ISER) and was funded by the Economic and Social Research Council. They can be made available through the United Kingdom Data Service (SN 7251).

Over a period of two years, the nurse health assessments in Wave 2 were completed with a subset of the General Population Sample (GPS) and included blood samples being taken alongside other anthropometric measurements including height, weight and percentage of body fat. The nurse health assessments were completed approximately five months following the main survey interview. All participants eligible to take part in the nurse health assessment had to be aged 16 or over, were not pregnant and had taken part in the main interview and living in England, Scotland or Wales. The nurse health assessments were not conducted in Northern Ireland or with the ethnic minority boost sample. The nurse interviewers were members of the Royal College of Nurses (RCN), the Royal College of Midwives (RCM) or registered on the Nursing and Midwifery Council (NMC) and were competent in venepuncture (McFall et al., 2012). More information is available from: https://www.understandingsociety.ac.uk/documentation/health-assessment/user-guide/

Data collection began in May 2010 for eligible participants interviewed in January 2010. At wave 2, 80% of households took part in the GPS and of them 90% of individuals took part (Knies, 2016). Of the 26,961 of those individuals who were eligible to take part in the nurse heath assessment at wave 2, 15,591 participated in the nurse health assessment. Participants were eligible for blood sampling on the condition that the participant had no clotting or bleeding disorder, such as haemophilia or low platelets. A stroke caused by a clot,

a myocardial infarction or an embolus, history of thrombophlebitis, a deep venous thrombosis were all not included as a clotting disorder. Exclusion criteria also included those respondents who volunteered that they were HIV positive or had hepatitis B or C. Lastly, people who have ever had a fit or were taking anticoagulant medications, such as warfarin, were excluded from giving a blood sample. However, Aspirin was not considered as an anticoagulant medication (Benzeval et al., 2014).

The interview was conducted with Computer Assisted Personal Interviewing (CAPI). The average duration of the nurse visit was approximately one hour if all the measurement samples were taken. Participants received leaflets about the specific measures and blood sampling procedure. All participants were given time to read the leaflets and consent form before signing the consent document. Participants were free to consent or not to any of the measures within the nurse visit (McFall et al., 2014).

For the present research, following similar studies (e.g. Davillas et al., 2017), participants were included if they were aged 25 years and above, this is because those adults were more likely to have completed their full time education and earning their own income.

## Variables

The current study used secondary data analysis of data collected as part of the UKHLS, therefore all inflammatory assays, measures of sleep duration and sleep quality and psychological distress were assessed as part of routine data collection for *Understanding Society* (Buck and McFall, 2012).

# **Biomarkers**

# Blood sampling and analysis

Non-fasting blood samples were taken from participants and labelled with their date of birth and a unique serial number. Samples were stored at the Fisher BioServices storage facility and processed which involved separating plasma and serum and storing them in freezers at -80 degrees Celsius. The duration from blood collection to the samples being processed was on average 2.4 days and 90% of the samples were processed within 90 days (Benzeval et al., 2014).

The blood samples were analysed by the Newcastle Upon Tyne Hospitals NHS Foundation Trust (NUTH). Between December 2013 and July 2014, batches of blood samples for 2000 participants were sent to NUTH sites. On arrival, each sample's bar code was scanned and subsequently assigned a unique bar code in accordance with the NUTH system. The analysis of serum was completed on a single Roche machine that needed 2x250uL of serum. The results were transmitted electronically from the analysers into a patient management information system and exported and transferred to ISER (Benzeval et al., 2014). Predominantly samples were processed within four days.

All analyses were completed in line with the Standard Operating Procedures by Health Care Professions Council (HCPC) Registered Biomedical Scientists. Internal Quality Controls (IQC) were undertaken on each machine at regular intervals per day. External Quality Assurance (EQA) systems were established to monitor all tests. Both internal and external quality assessments were regularly reviewed by the quality team and any trends identified were communicated through internal governance protocol; any non-conformities with EQAs were identified and investigated (Benzeval et al., 2014).

**CRP.** Following collection, CRP (mg/L) was analysed from serum utilising the N latex CRP mono assay on the Behring Nephelometer II Analyser (Dade Behring, Milton Keynes, UK). Intra and inter assay coefficients of variation were less than 2% (Benzeval et al., 2014). The cut off for CRP of systemic inflammation will be defined as CRP >3mg/L levels (Pepys & Hirschfield, 2003). This delineation was based on the Centres for Disease Control and Prevention and the American Heart Association (Pearson et al., 2003). Given inflammation levels increase during times of recent infection, participants with CRP levels higher than 10mg/L will be excluded (Pearson et al., 2003). CRP is influenced by the following medication: anti-inflammatory medications, statins and contraception and hormone replacement therapy. This was accordingly taken into account in the analysis and these medications were considered as covariates (see covariates) to be analysed. The CRP data was tested for normality using visual evaluation of Q-Q plots and histograms along with a Kolmogorov-Smirnov test. CRP was not normally distributed and to reduce skewness the CRP values were log transformed.

**Fibrinogen.** Fibrinogen is glycoprotein complex produced in the liver and is a marker of inflammation. Fibrinogen (g/L) was analysed from citrate plasma samples utilising an adjustment of the Clauss thrombin clotting method of the IL-ACS-TOPS analyser. Intra and inter assay coefficients of variation were less than 7% (Benzeval et al., 2014). There are no established cut off points to demonstrate clinical significance and this data will be continuous. The lower limit of the assay was 0.5g/L. Fibrinogen can be influenced by the following medication: contraception and hormone replacement therapy (Benzeval et al., 2014). This was taken into consideration when analysing these data (see covariates). The fibrinogen data was tested for normality using visual evaluation of Q-Q plots and histograms along with a Kolmogorov-Smirnov test. Fibrinogen values were not normally distributed and to reduce skewness the fibrinogen values were log transformed.

## **Psychological Distress**

Psychological distress is a key feature of many mental health problems and refers to experiences of anxiety, depression, stress and somatic symptoms (Goldberg & Goodyear, 2005). Symptoms of psychological distress are common within the general population as well as diagnosable mental health problems (Goldberg & Goodyear, 2005).

Psychological distress was measured using the 12-item General Health Questionnaire (GHQ-12; Goldberg et al., 1997), which asked participants "questions regarding the way you have been feeling recently". The GHQ-12 comprises the following questions: "Have you recently been able to concentrate on whatever you're doing", "Have you recently lost much sleep over worry", "Have you recently been feeling you were playing a useful part in things", "Have you recently felt capable of making decisions about things", "Have you constantly felt under strain", "Have you recently felt you couldn't overcome your difficulties", "Have you recently been able to enjoy your normal day-to-day activities", "Have you recently been able to face up to your problems", "Have you recently been feeling unhappy or depressed", "Have you recently been losing confidence in yourself", "Have you recently been thinking of yourself as a worthless person", "Have you been feeling reasonably happy, all things considered" (see Appendix A).

For negatively phrased responses, the items on the scale were scored in the following manner: 1 = Not at all, 2 = No more than usual, 3 = Rather more than usual, 4 = Much more than usual. For positively phrased responses, the items on the scale were scored in the following manner: 1 = More than usual, 2 = Same as usual, 3 = Less than usual, 4 = Much less than usual. According to the scoring method of GHQ items, higher values represent higher psychological distress. The caseness scoring was used to measure psychological distress. In accordance with the GHQ-12, a cut off score of  $\geq 3$  is used as a demarcation indicating at least moderate psychological distress (Goldberg et al., 1997). The GHQ-12 has

been robustly validated as a measure of psychological distress (Anjara et al., 2020; Lundin et al., 2016; Werneke et al., 2000).

# **Sleep Quality and Duration**

Participants were asked about their sleep habits as part of Understanding Society through the 'Self-Completion Adult Module' questionnaire, which collated sleep related data through self-report measures on sleep duration and sleep quality, this was asked of participants every three years (see Appendix B). In terms of sleep duration respondents were asked: "How many hours of actual sleep did you usually get a night during the last month", the responses produced continuous data based on hours of sleep. Sleep duration was divided into four categories: *6 hours or less*, *6-7 hours*, *7-8 hours*, *9 hours or more*. In terms of sleep quality respondents were asked: "During the last month, how would you rate your sleep quality overall". Respondents were invited to choose a category that most appropriately described their quality of sleep, the categories were: "*Very good*", "*Fairly good*", "*Fairly bad*", "*Very bad*". These questions are items from the Pittsburgh Sleep Quality Index (Buysse et al., 1989).

## Socioeconomic position

## Household Income

Socioeconomic position was assessed by both equivalised monthly household income and educational attainment, being two of the main indicators of socioeconomic position (APA, 2007). Family income is a measure of current socioeconomic position, whilst an individual's educational attainment is invariably determined earlier in life and therefore may attenuate the role of any possible effects of health shocks on socioeconomic position (Van Kippersluis et al., 2010). All adult participants were asked about all sources of income including earnings from principle jobs and second jobs, welfare benefits, state and private pensions and separate sources of income (Fisher et al., 2019). Monthly household income was subsequently divided into tertiles between low, middle and high (reference group), as has been done previously (Davilias et al., 2017).

The requirements of a household are assumed to change as a household increases in size. Accordingly, it is important to take into consideration the principle of economies of scale in consumption in regards to a household, this refers to the combined consumption of goods within a household. For example, the expenditure on energy bills will not increase five times for a household with five members than for a single person (Angus & Deaton, 1998). Through the use of equivalence scales each household type was allocated a value which corresponds to its needs. Important factors that are taken into consideration are the size of the household and the age of its members, for instance if the household member is an adult or a child (Atkinson et al., 1995; Burniaux et al., 1998). The income variable was equivalised using the modified OECD scale (Haagenars et al., 1994). This allocates a value of 1 to the first household member and subsequently 0.5 to each additional adult household member and finally 0.3 to each child living within the household.

## **Educational Attainment**

Participants were asked about current and past educational attainment, this comprised of measuring the highest educational attainment. Respondents indicated which of the following applied to them: no qualifications, GCSE/basic qualifications, A-level, other higher degree, degree (reference group).

# Covariates

The strength of associations were tested after fully adjusting for a number of possible confounding effects of covariates. These covariates were considered in terms of variables that have been included as relevant covariates in previous studies, together with variables that are typically associated with increased inflammatory biomarkers.

Demographic characteristics were included as relevant covariates. Age was treated as a continuous variable. Sex was reported as either male or female. Respondents were asked to provide their ethnicity which was assessed by the question: "what is your ethnicity?" Within the current study, ethnicity was treated as categorical variable. Respondents were categorised into five groups: White (reference group), Black/Black British, Asian/British Asian, Mixed Background/Other. Labour status was self-reported and included unemployed, employed (reference group), self-employed, student, retired and other.

Increased inflammation is linked with daily cigarette smoking, alcohol use and exercise (Crews et al., 2006). Smoking status was categorised as 'previous smoker' (reference group), 'current smoker' and 'never smoked'. Alcohol consumption was categorised as 'everyday', 'weekly' (reference group), 'monthly', 'less often', 'never drink'. Participation in exercise has been shown to be associated with levels of inflammation (Esteghamati et al., 2012) and was considered as a covariate, it was combined into a categorical variable of either 'yes' (reference group) or 'no'.

The use of anti-inflammatory medication will also comprise the covariates included in the analysis, as these have been known to influence inflammatory levels in the body (Danese et al., 2011). Furthermore, the use of Hormone Replacement Therapy (HRT), contraception and statins have also been shown to influence inflammation levels. Those taking such medications will be compared to those not taking them. Such covariates are included in past research examining levels inflammation (Davillas et al., 2017; Trotta et al., 2021). Body Mass Index (BMI) has been known to be associated with increased levels of inflammation through the production of pro-inflammatory cytokines by adipocytes (Brummet et al., 2013). BMI is calculated from measurements of height and weight. Height was measured using a stadiometer and body weight was measured by a scale (Tanita BF 522). BMI was calculated as the weight (kilograms) over the square of height (meters). Respondents were required to take off their socks, shoes and any bulky clothing that may affect the final weight measurement (Institute for Social and Economic Research, 2022). BMI (kg/m2) was measured and being overweight was defined as a BMI greater than or equal to 25, BMI was considered as a continuous variable.

#### **Statistical Analysis**

All statistical data analysis was completed using IBM SPSS Statistics version 29.00. *Characteristics of the Sample* 

Wave 1 sleep quality, sleep duration and psychological distress data was merged with Wave 2 nurse assessment data. The basic demographic characteristics of respondents were recorded together with the key variables of interest: psychological distress, sleep quality and duration, measures of socioeconomic position (household income and educational attainment) and levels of CRP (mg/L) and fibrinogen (g/L). During the nurse visit at their home, some respondents did not volunteer to give a blood sample or were not eligible to do so. In the preliminary stages of data analysis, this resulted in two groups of respondents who took part in the broader nurse health visit during wave 2: those that gave blood and those that did not give blood. Those respondents that did provide a blood sample constituted the analytical sample. To examine for differences in the means (e.g. age, ethnicity) between the two groups preliminary analyses were conducted using an independent samples t-test for all data identified as continuous and a Chi-square for all data identified as categorical.

CRP and fibrinogen data is not normally distributed and was log transformed to improve normality of their distributions and allow for statistical analysis, as has been done in previous studies (e.g. Trotta et al, 2021). CRP and fibrinogen was identified as continuous data. The biomarker data was positively skewed as indicated from inspection of Normal Q-Q Plots and histograms. A Kolmogorov-Smirnov test indicated that the CRP data did not follow a normal distribution D(8537) = .332, p = 0.001. A Kolmogorov-Smirnov test indicated that the fibrinogen data did not follow a normal distribution D(8537) = .079, p = 0.005.

The analysis of the association between socioeconomic position and CRP and fibrinogen was conducted using multiple regression with the exclusion of the mediating variables. Given the concept of socioeconomic position is derived from educational attainment and household income, in order to conduct the linear regression, binary indicator (dummy) independent variables were recoded to indicate the multiple categories of educational attainment (e.g. degree, other degree, A-Level etc.) Statistical significance was set at a cut off of p < 0.05. Covariates were fully adjusted for in each model.

Linear regression was used to explore levels of inflammation based on socioeconomic position, whilst controlling for covariates. This included the two measures of socioeconomic position (household income and educational attainment) being added to the model, together with the selected covariates to be analysed.

## **Mediation Analysis**

Psychological distress and sleep (quality and duration) were tested as potential mediating variables in the association between socioeconomic position and inflammation (see Figures 2 and 3). Accordingly, mediation analysis was proposed to test this relationship (Baron & Kenny, 1986). Psychological distress and both sleep duration and sleep quality were suggested to function as mediating variables given that they met the three conditions outlined by Baron and Kenny (1986). These conditions were, firstly, differences in socioeconomic position (independent variable) were associated with differences in the proposed mediators of psychological distress and sleep duration and quality (path A). Secondly, differences in psychological distress and both sleep duration and quality were associated with differences in the markers of inflammation (path B). Lastly, when pathways A and B are controlled for then a previous significant relationship between socioeconomic position and markers of inflammation (path C) should be significantly decreased. If the pathway C is not zero it suggests that there potentially multiple mediators, it is well documented that within psychological research a more feasible goal is to pursue mediators that significantly attenuate the relationship between the independent and dependent variables rather than remove it entirely (Baron & Kenny, 1986).

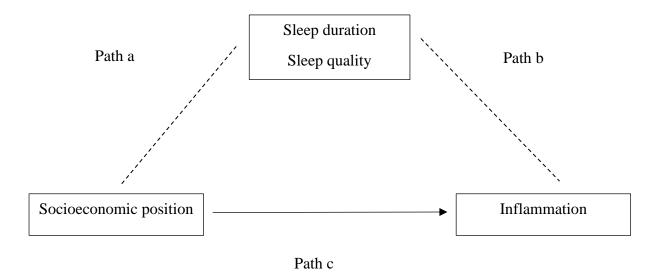
## Mediation model

Socioeconomic position was identified as the independent variable and CRP and Fibrinogen were identified as the dependent or outcome variables. To elucidate the mediation relationship, a conceptual illustration of each model depicts the two pathways leading to the outcome variable (figures 2 and 3). The first model (figure 2) suggests the potential mediating pathway of sleep duration and sleep quality in the relationship between socioeconomic position and inflammation. The second model (figure 3) suggests the potential mediating pathway of psychological distress in the relationship between socioeconomic position and inflammation. Therefore, it is proposed that there are two pathways leading to the biomarkers of inflammation. There is a direct pathway (path c) from socioeconomic position into the biomarkers of inflammation, CRP and fibrinogen. In the mediator pathway (path b), psychological distress and sleep (quality and duration) were set as two separate mediators going leading to CRP and fibrinogen. Finally, there is a pathway (path a) from socioeconomic position into the two separate mediators, psychological distress and sleep (quality and quantity). To explore if the indirect effect is significant, a Sobel test was completed using unstandardised coefficients and standard errors (Sobel, 1986). In the instance where there was more than one dummy variable capturing a construct, the Sobel test was applied to only one level of the dummy variable.

71

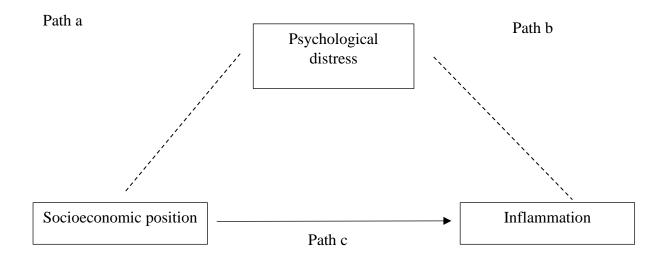
## Figure 2

Mediation model exploring the potential mediating roles of sleep duration and sleep quality on the relationship between socioeconomic position (separately measured by household income and educational attainment) and inflammation (separately measured by CRP and fibrinogen).



# Figure 3

Mediation model exploring the potential mediating roles of psychological distress on the relationship between socioeconomic position (separately measured by household income and educational attainment) and inflammation (separately measured by both CRP and fibrinogen).



# **Ethical Considerations**

Following the initial interview, participants were sent a letter and leaflet by post outlining details of the nurse health assessment (Mcfall et al., 2014). These resources can be found at https://www.understandingsociety.ac.uk/documentation/mainstage/fieldwork-documents.

Initial contact was made with participants by telephone and the health assessment was explained as part of the overall survey. Within this contact, any non-clinical questions were answered and the next steps were outlined in that the nurse would be making contact with the participant to schedule an appointment. During the visit, participants were given additional information about specific measures and the blood sampling procedure. The participant was given time to read the leaflet and consent form before signing it or giving oral consent to a procedure. Participants were aware that their participation was voluntary and they were free to give or withdraw their consent at any time to any procedure (Mcfall et al., 2014). The written consent form can be found at

https://www.understandingsociety.ac.uk/documentation/mainstage/fieldwork-documents.

Following blood sampling, participants received a copy of their signed consent form and were provided with information regarding how they could withdraw their consent to blood sample storage if they wished to do so after completion of the nurse visit.

All participants eligible to take part in the nurse visit had to be aged 16 or above and gave informed written consent for their blood to be taken and stored for future scientific analysis. The UKHLS was granted ethical approval by the University of Essex Ethics Committee and the nurse visit data collection by the National Research Ethics Service (10/H0604/2). Details of *Understanding Society* can be found at https://www.understandingsociety.ac.uk/

Approval from the National Research Ethics Service was obtained for the collection of biosocial data by trained nurses in Waves 2 and 3 of the main survey. (Understanding Society - UK Household Longitudinal Study: A Biosocial Component, Oxfordshire A REC, Reference: 10/H0604/2). Further details on specific measures and specific project instructions are available online

https://www.understandingsociety.ac.uk/documentation/mainstage/fieldwork-documents.

For the present research, all data remained confidential and no identifiable characteristics of participants were included in the study or in future dissemination of findings. The dataset was stored securely using an encrypted file on a password-protected computer to ensure confidentiality was not breached.

## Dissemination

A journal article is planned to be written based on the findings of the current study. It is intended that the research findings will be published in a journal related to the area of study. The favoured preference currently is in a journal such as *Brain, Behaviour and Immunity*, given that it is orientated to the area under study and achieves a high impact factor (12.84). To ensure that the clinical implications of the study are disseminated the findings will be presented at the annual University of Essex Health and Social Care Research Conference. This will be a proficient way of communicating the findings to researchers and clinical psychologists. The findings will be considered to be presented to mental health services in Essex.

#### **Chapter Three: Results**

### **Chapter Summary**

This chapter outlines the results of the current study. Descriptive statistics of both the full sample and analytical sample are provided. Subsequently, the main findings of the regression analyses are outlined regarding the proposed mediation models, as depicted in Figures 2 and 3. The results in the subsequent pages are conveyed in the following manner. Firstly, regression analyses examining the direct pathway (path c) from socioeconomic position into CRP and fibrinogen were completed. Then, independent regression analyses were competed examining the pathway (path b) from psychological distress and sleep (quality and duration) into CRP and fibrinogen. Finally, regression analyses were completed to examine the pathway (path a) from socioeconomic position into the two separate mediators, psychological distress and sleep (quality and quantity).

#### **Descriptive Statistics**

To examine whether there were differences found in the demographic characteristics of those without to those with inflammatory biomarker data, chi-square and t-tests were completed.

*Full Sample.* The full sample was comprised of a total of 14, 905 respondents from whom blood samples were collected as part of the biomarker survey. In this broader sample the average age was 50 years old (range 16 - 101 years) and consisted of 6,470 men (43.4%) and 8,435 women (56.6%). The average BMI was 28.02 (range 13-64). From this broader sample, there were 9,006 respondents who had CRP (5,899 respondents did not have a CRP value) and 9,181 respondents with fibrinogen (5724 respondents did not have a Fibrinogen value). The full sample was subsequently restricted to 25 years + to focus on adults who have

more likely completed their full-time education. This resulted in 13,767 respondents both with and without inflammatory biomarker data.

*Respondents without Inflammatory Biomarker Data.* There were 4,965 respondents who did not have either CRP or fibrinogen data. Of the respondents who did not give inflammatory biomarker data, 2,151 were men and 2,814 were women (see Table 3). The average age of this participant group was 52 years old (range 25 – 97 years). The average BMI was 28.37. In terms of sex, there were no significant differences between the male and female within either sample. There were differences between level of educational attainment between those with and without inflammatory biomarkers. The without inflammatory biomarker group differed from the analytical sample in terms of ethnicity, educational attainment, employment status, alcohol use and participation in exercise (see Table 3 for exact p values).

*Analytical Sample.* There were 8,802 respondents with either one or both inflammatory biomarkers. The sample consisted of 3,848 men and 4,954 women. The mean age was 53.29 years old (range 25 – 101 years). There were 8,697 (63.2%) respondents with CRP data and 8,642 with fibrinogen data. The mean CRP value was 3.27 and the mean fibrinogen value was 2.81. Sample characteristics are outlined in Table 3.

## Table 3

# Characteristics of both those without inflammatory biomarker data and those with

Baseline characteristic	Without biomarker sample (n= 4,965)	Biomarker sample (n=8,802)	Value of relevant comparative statistic	p-value
	N (%) or Mean	N (%) or Mean		
Gender			$X^{2}(1)=.189$	.664
Male	2,151 (43.3)	3848 (43.7%)		
Female	2,814 (56.7)	4954 (56.3%)		
Age	52.09	53.29	t (1)=203.088	.001
Ethnicity <sup>a</sup>			X <sup>2</sup> (3)=65.582	.001
White	4,478 (90.2)	8,266 (93.9)		
Black/Black British	115 (2.3)	94 (1)		
Asian/British Asian	212 (4.3)	224 (2.4)		
Mixed Other	92 (1.9)	107(1.3)		
Educational Attainment			$X^{2}(4)=21.438$	.001
Degree	1,809 (35.3)	3,198 (36.3)		
A-Level	802 (15.6)	1447 (16.4)		
GCSE	942 (18.4)	1,671 (19)		
Other	648 (12.6)	1070 (12.2)		
No Qualifications	922 (18.0)	1,408 (16)		
Income <sup>c</sup> (£)				
High	1,627 (31.8)	3,013 (34.2)		
Middle	1,654 (32.3)	2,976 (33.8)		
Low	1,839 (35.9)	2,807 (31.9)		
Employment			X <sup>2</sup> (5)=43.592	.001
Unemployed	250 (5.0)	335 (3.8)		
Student	26 (0.5)	42 (0.5)		
Employed	2,284 (46.0)	4,275 (48.5)		
Self-employed	363 (7.3)	669 (7.6)		
Retired	1,377 (27.7)	2,620 (29.8)		
Other	631 (12.7)	857 (9.7)		

# inflammatory biomarker data (analytical sample)

Baseline characteristic	Without biomarker sample (n= 4,965)	Biomarker sample (n=8,802)	Value of relevant comparative statistic	p-value
	N (%) or Mean	N (%) or Mean	1	
Inflammatory Biomarkers				
CRP (mg/L)	n/a	3.27		
Fibrinogen (g/L)	n/a	2.81		
Quality of Sleep			$X^{2}(3)=.818$	.845
Very good	1,056 (21.3)	1,921 (21.8)		
Fairly	2,308 (46.5)	4,159 (47.3)		
Fairly bad	826 (16.6)	1,471 (16.7)		
Very bad	207 (4.2)	332 (3.8)	$X^2 (3 = 1.096)$	.137
Duration of sleep (hours)	1,596 (32.1)	2,954 (33.6)	× ×	
6 hours or less	1,335 (26.9)	2,534 (28.8)		
6-7 hours	1,043 (21)	1,714 (19.5)		
7-8 hours	247 (5)	391 (4.4)		
9 hours +				
Psychological Distress	1.85	1.61	t (13) = 1.512	.131
(GHQ-12)	28.34	28.20		
BMI			$X^{2}(1)=11.956$	.001
Medication				
Contraception or	68 (1.4)	183 (2.1)	$X^{2}(1)=479$	.489
HRT	916 (18.4)	1,616 (18.4)	$X^{2}(1)=5.820$	.016
Statins	308 (6.2)	622 (7.1)		
Anti-inflammatories				
Smoking status	1,995 (40.2)	3,380 (39.1)	$X^{2}(2)=.904$	.342
Never smokers	2,970 (59.8)	8,323 (60.5)		
Former smokers	989 (32.3)	1,648 (31.3)		
Current smokers				
Alcohol	407 (8.2)	785 (8.9)	$X^{2}(4) = .904$	.001
Everyday	2,022 (40.7)	4,040 (45.9)		
Weekly	1,021 (20.6)	1,792 (20.4)		
Monthly	408 (8.2)	693 (7.9)		
Less often	117 (2.4)	132 (1.5)		
Never drink			$X^{2}(1)=66.784$	.001
Exercise	3,597 (72.4)	6,821 (77.5)		
Yes	1,368 (27.6)	1,981 (22.5)		
No	,/	,,		

*Note.* <sup>a</sup>White= British/Scottish/Welsh Northern Irish, Gypsy or Irish Traveller, Any Other White Background; Black/Black British= Caribbean, African, Any Other black Background; Asian/British Asian= Indian, Pakistani, Bangladeshi; Chinese, Any other Asian background; Mixed/Other=White and Black Caribbean, White and Black African, White and Asian, Any Other Mixed Background, Any Other Background Not Stated. CRP; C-reactive protein. Mg/l, Milligrams per litre. GHQ-12; General Health Questionnaire -12. BMI; Body Mass Index. HRT; Hormone Replacement Therapy.

### **Regression Analyses**

*Socioeconomic Position and CRP.* The following regression analyses consisted of examining the direct pathway (path c), firstly, from educational attainment to CRP and, secondly, from household income to CRP (see Table 4).

The regression model exploring educational attainment and CRP was significant F(24, 8421) = 91.775, p < .001, explaining 20.7% ( $R^2 = .207$ ) of the variance in levels of CRP. Compared to the highest level of education, CRP was raised in those with the lowest level of educational attainment following adjustment for all covariates (B = .093, SE=0.15, p<.001).

The regression model exploring household income and CRP was significant, F(24, 8421) = 91.052, p < .001, explaining 20.6% ( $R^2 = .206$ ) of the variance in CRP. In a similar manner, compared to those in the highest income tertile, both middle (B=.042, SE=.013, p<.001) and lowest (B=.061, SE=.012, p<.001) income tertiles were associated with higher concentrations of CRP. The coefficient was larger for those with a lower household income compared to a middle household income.

In terms of covariates for both educational attainment and household income models, following full adjustment, several variables remained independently associated with CRP (see table 4 for the specific levels of significance regarding the covariates specific to each model). Men were associated with lower levels of CRP compared with women. Taking antiinflammatory medication and contraception or HRT was associated with elevated CRP compared with those not taking these medications. In contrast, taking statins was associated with lower concentrations CRP, compared with not being on this medication. There was an association between those that have never smoked and lower levels of CRP, compared to people who were former smokers. Compared with previous smokers, current smoking was associated with higher levels of CRP and never smoking was associated with lower levels of CRP and never smoking was associated with lower levels of CRP and never smoking was associated with lower levels of CRP and never smoking was associated with lower levels of CRP and never smoking was associated with lower levels of CRP and never smoking was associated with lower levels of compared with lower levels of CRP and never smoking was associated with lower levels of compared with lower levels of CRP and never smoking was associated with lower levels of compared wit CRP. Not participating in exercise was associated with higher CRP compared to those who do exercise. BMI and age were also associated with higher levels of CRP.

# Table 4Regression analysis results for socioeconomic position and CRP

	Educational	Attainment	Household Income	
	Coefficient	Standard Error	Coefficient	Standard Error
Primary Variables				
<b>Educational Attainment</b>				
( <b>Ref=Degree</b> )				
A-Level	.020	.011		
GCSE	.023	.014		
No Qualifications	.093***	0.15		
Household Income				
(Ref=High income)				
Middle household income			.042***	.013
Low household income			.061***	.012
Covariates				
Gender (Ref=Female)				
Male	033***	.010	033*	.010
Age	.005***	.001	.005***	.001
Ethnicity (Ref=white)				
Black/Black British	008	.048	016	.048
Asian/British Asian	.041	.033	.033	.033
Mixed background/Other	.063	.045	. 054	.045
Medication				
Contraception/HRT	.197***	.034	.202***	.034
Anti-inflammatories	.005	.003	.005	.003
Statins				
Smoking status				
(Ref=previous smoker)				

Ec	Educational Attainment		Household Income		
Co	efficient Standard Erro	or Co	efficient S	Standard Error	
Current smoker	.130***	0.14	.132*	** .014	
Never smoked	024*	0.11	024	l* .011	
Alcohol consumption					
(Ref=Weekly)					
Everyday	.016	.018	.01	5.018	
Monthly	.023	.013	.020	.013	
Less often	.015	.019	.01	5.019	
Never drink alcohol	.023	.042	.020	.042	
BMI	.033***	.001	.034*	** .001	
Exercise (Ref= Yes- exercis	se)				
No exercise	.067***	.013	.07	5.013	

**Table 4** (Continued)

\*p≤0.05 \*\* p≤0.01 \*\*\*p≤0.001

*Socioeconomic Position and Fibrinogen.* The following regression analyses consisted of examining the direct pathway (path c), firstly, from educational attainment to fibrinogen and, secondly, from household income to fibrinogen (see Table 5).

The regression model exploring educational attainment and fibrinogen was significant, F(25, 8361) = 65.008, p < .001, explaining 16.3% ( $R^2 = .160$ ) of the variance in fibrinogen. Compared to the highest level of education, fibrinogen was raised in those with the lowest level of educational attainment (B = .006, SE=.003, p=.036) following adjustment of all covariates.

The regression model exploring household income and fibrinogen was significant, F(23, 8363)=70.870, p<.001), explaining 16.3% ( $R^2$ =.163) of the variance in fibrinogen. Compared to those in the highest income tertile, both middle and lowest income tertiles were associated with higher concentrations of fibrinogen (B=.008, SE=.003, p=.003).

In terms of covariates for both educational attainment and household income models, following full adjustment, several variables remained independently associated with fibrinogen (see table 5 for the specific levels of significance regarding the covariates specific to each model). Compared with women, men were associated with having lower levels of fibrinogen. Compared to those who previously smoked, current smokers were associated with higher levels of fibrinogen. Compared to drinking alcohol weekly, drinking alcohol everyday was associated with lower fibrinogen, whereas drinking alcohol monthly was associated with higher fibrinogen. Those with a higher BMI had higher levels of fibrinogen compared to those who do exercise. There were no associations between ethnicity and fibrinogen in either model. In the household income model, taking statins was associated with higher fibrinogen in either of the models.

# Table 5Regression analysis results for socioeconomic position and fibrinogen

	Educational Attainment		Househol	d Income
	Coefficient	Standard	Coefficient	Standard
	Coefficient	Error		Error
Educational Attainment				
(Ref=Degree)				
A-Level	.003	.001		
GCSE	.004	.003		
No Qualifications	.006*	.003		
Household Income				
(Ref=High household				
income)				
Middle household income			.005*	.002
Low household income			.008*	.003
Gender (Ref=Female)				
Male	015***	.002	015***	.002
Age	.002***	.000	.002***	.000
Ethnicity (Ref=white)				
Black/Black British	.004	.009	.003	.009
Asian/British Asian	.012	.006	.011	.006
Mixed background/Other	.014	.008	.013	.008
Medication				
Contraception/HRT	010	.006	009	.006
Statins	.004	.003	.004	.003

## Table 5 (Continued)

	Educational A	ttainment	Hou	Household Income		
	Coefficient	Standard Error	Coefficient	Standard Error		
Anti-inflammatories	.001	.004	.001	.004		
Smoking status						
(Ref=previous smoker)						
Current smoker	.035***	.003	.034***	.003		
Never smoked	004	.002	003	.002		
Alcohol consumption						
(Ref=Weekly)						
Everyday	012***	.003	012***	.003		
Monthly	.010***	.002	.010***	.002		
Less often	.006	.004	.005	.004		
Never drink alcohol	.003	.008	.003	.008		
BMI	.004***	.000	.004***	.000		
Exercise (Ref= Yes-						
exercise)						
No exercise	.009***	.002	.009***	.002		

\*p $\leq 0.05$  \*\* p $\leq 0.01$  \*\*\*p $\leq 0.001$ 

## Socioeconomic Position and Psychological Distress

Table 6 depicts the regression models for both socioeconomic position and psychological distress.

Socioeconomic Position and Psychological Distress. The regression model exploring educational attainment and psychological distress was significant, F(24, 7817)=20.134, p<.001), explaining 58% ( $R^2=.058$ ) of the variance in psychological distress. Educational attainment was not associated with psychological distress, whilst an association was apparent when household income was examined as a measure of socioeconomic position (B=.340, SE=.095, p<.001). Thus, those in the lowest household income tertile had raised psychological distress compared to those in the highest household income tertile. The model was significant, F(23, 7818)=21.422, p<.001), explaining 59% ( $R^2=.059$ ) of the variance in the outcome variable.

In terms of covariates for both models, following full adjustment, several variables remained independently associated with psychological distress (see Table 6 for the specific levels of significance regarding the covariates specific to each model). In both models, men were associated with lower psychological distress compared with women. Within the educational attainment model, there were significant associations with higher psychological distress found in those taking contraceptive medication or HRT compared to not taking these medications. Within the household income model, there were significant associations with psychological distress and taking anti-inflammatory medication and statins, when compared to those who were not taking these medications, respectively. Contrastingly, within the education model, only being a current smoker was associated with higher levels of psychological distress compared to those who previously smoked. In terms of alcohol consumption within the educational attainment model, both those that drank alcohol monthly and those that drank alcohol less often were associated with having higher psychological distress compared to those that drank alcohol weekly. Whereas, in the household income model, those that drank alcohol less often were associated with having higher psychological distress when compared to those that drank alcohol weekly. Those who did not participate in

exercise were associated with higher levels of psychological distress compared with those who do participate in exercise. A higher BMI was associated with higher levels of psychological distress in both models.

## Table 6

Regression analysis results for socioeconomic position and psychological distress

	Educational Attainment		Household Income	
	Coefficient	Standard	Coefficient	Standard
		Error		Error
<b>Educational Attainment</b>				
(Ref=Degree)				
A-Level	.111	.098		
GCSE	.112	.097		
No Qualifications	048	.111		
Household Income				
(Ref=High household				
income)				
Middle household income			.148	.085
Low household income			.340***	.095
Gender (Ref=Female)				
Male	325***	.074	309***	.074
Age	011*	.004	010*	.004
Ethnicity (Ref=white)				
Black/Black British	124	.392	136	.392
Asian/British Asian	.128	.252	.078	.252
Mixed background/Other	.379	.320	.367	.320
Medication				
Contraception/HRT	.916***	.244	.946	.244
Statins	.041	.100	.027***	.100

## Table 6 (Continued)

	Educational Attainment		Househ	old Income
	Coefficient	Standard Error	Coefficient	Standard Error
Anti-inflammatories	.506	.019	.504***	.139
Smoking status				
(Ref=previous smoker)				
Current smoker	.591***	.100	.557***	.100
Never smoked	102	.078	093	.078
Alcohol consumption				
(Ref=Weekly)				
Everyday	.104	.122	.122	.122
Monthly	.173*	.087	.155	.087
Less often	.341*	.130	.315*	.130
Never drink alcohol	137	.295	175	.295
BMI	.026***	.006	.025***	.006
Exercise (Ref= Yes-				
exercise)				
No exercise	.598***	.093	.552***	.092

\*p≤0.05 \*\* p≤0.01 \*\*\*pp≤0.001

## **Socioeconomic Position and Sleep Duration**

Table 7 depicts the ordinal regression model results for both educational attainment and household income for sleep duration.

*Socioeconomic Position and Sleep Duration.* In the ordinal regression model exploring educational attainment and sleep duration ('6 hours or less', '6-7 hours', '7-8

hours' and '9 hours or more'), there was a significant association between educational attainment and sleep duration, however, these were in directions that were not hypothesized. Having no qualifications was associated with an increased likelihood of falling within the '9 hours or more' sleep category, compared to those with the highest level of qualifications (B=.300, SE=.066, p<.001). Overall, the educational attainment model accounted for approximately 0.7% of the variance in sleep duration, McFadden's pseudo-R<sup>2</sup>= .007. Contrastingly, there were no significant associations between sleep duration and household income.

In terms of covariates for both models, following full adjustment a number of variables remained independently associated with sleep duration (see Table 7 for the specific levels of significance regarding the covariates specific to each model). Unless it is indicated, these covariates were significant in both the educational attainment and the household income model of sleep duration. Men had lower sleep duration compared with women. Being a current smoker was associated with higher sleep duration compared with being a previous smoker. A higher BMI was associated with a lower sleep duration. In terms of ethnicity, people who identified as Black or Black British, Asian or British Asian and a Mixed background were associated with an increased likelihood of falling within the '9 hours or more' sleep category, compared to those who were White.

	Educational Attainment		Househol	Household Income	
	Coefficient	Standard	Coefficient	Standard	
		Error		Error	
<b>Educational Attainment</b>					
(Ref=Degree)					
A-Level	.075	.058			
GCSE	.154*	.056			
No Qualifications	.300***	.066			
Household Income					
(Ref=High household					
income)					
Middle household income			002	.048	
Low household income			.044	.051	
Gender (Ref=Female)					
Male	.117*	.041	.108*	.041	
Age	001	.002	001	.001	
Ethnicity (Ref=white)					
Black/Black British	.869***	.197	.795***	.196	
Asian/British Asian	.643***	.132	.594***	.132	
Mixed background/Other	.384*	.178	.344	.178	
Medication (Ref = Not					
taking stated medication)					
Contraception/HRT	.204	.136	.204	.136	
Statins	.006	.056	.018	.056	

## **Table 7** (Continued)

	Educational Attainment		Household Income	
	Coefficient	Standard Error	Coefficient	Standard Error
Anti-inflammatories	009	.078	.006	.078
Smoking status				
(Ref=previous smoker)				
Current smoker	.276***	.056	.310***	.056
Never smoked	.067	.044	.057	.044
Alcohol consumption				
(Ref=Weekly)				
Everyday	.056	.071	.063	.071
Monthly	084	.050	071	.050
Less often	.000	.076	.030	.075
Never drink alcohol	156	.171	119	.170
BMI	021***	.004	020***	.004
Exercise (Ref= Yes-				
exercise)				
No exercise	.120*	.052	.198***	.098

## Socioeconomic Position and Sleep Quality

Table 8 depicts the ordinal regression model results for both educational attainment and household income for sleep duration.

*Socioeconomic Position and Sleep Quality.* An ordinal regression analysis was used to examine the relationship between educational attainment and sleep quality ('very bad',

'fairly bad', 'fairly good' and 'very good'). Similar to associations with sleep duration and educational attainment, associations were apparent with measures of socioeconomic position and sleep quality. However, these were also in directions that were not hypothesised. Overall, the educational attainment model accounted for approximately 30% of the variance in sleep quality, McFadden's pseudo- $R^2$ =.030. Thus, having no qualifications was associated with an increased likelihood of falling within the 'very good sleep' category (*B*=.211, SE= .074, p=.004), compared to the highest level of qualifications. Similarly, being in the lowest income tertile was associated with very good sleep (*B*=.197, SE= .060, p=.001), compared to the highest income tertile. Overall, the household income model accounted for approximately 30% of the variance in the outcome, McFadden's pseudo-R<sup>2</sup>=.030.

In terms of covariates for both models, following full adjustment a number of variables remained independently associated with sleep quality (see Table 8 for the specific levels of significance regarding the covariates specific to each model). Unless it is indicated, these covariates were significant in both the educational attainment and the household income model of sleep quality. A higher BMI was associated with a higher likelihood of falling within the lower category of sleep quality. Compared with women, men were more likely to have increased likelihood of falling within the lower sleep category. In terms of ethnicity, compared with White respondents, Black British respondents were associated with having 'very good sleep'. Drinking alcohol less often was associated with falling within the very good category of sleep quality, compared to drinking alcohol weekly. Not participating in exercise was associated with an increased likelihood of falling within a higher sleep quality, compared with those that do participate in exercise.

# Table 8

Regression analysis results for socioeconomic position sleep quality and covariates

	Educational Attainment		Household Income	
	Coefficient	Standard Error	Coefficient	Standard Error
Educational Attainment				
(Ref=Degree)				
A-Level	.091	.065		
GCSE	.064	.063		
No Qualifications	.211*	.074		
Household Income				
(Ref=High household				
income)				
Middle household income			.046	.054
Low household income			.197***	.060
Gender (Ref=Female)				
Male	250	.047	249***	.047
Age	.013	.002		
Ethnicity (Ref=white)				
Black/Black British	644**	.241	704**	.241
Asian/British Asian	007	.160	036	.160
Mixed background/Other	.311	.202	.283	.202
Medication (Ref = Not				
taking stated medication)				
Contraception/HRT	.320*	.152	.341*	.152
Statins	.133*	.064	.136*	.063

## Table 8 (Continued)

	Educational Attainment		Household Income	
	Coefficient	Standard Error	Coefficient	Standard Error
Anti-inflammatories	.357***	.087	.362***	.087
Smoking status				
(Ref=previous smoker)				
Current smoker	.348***	.063	.353***	.063
Never smoked	061	.050	062	.050
Alcohol consumption				
(Ref=Weekly)				
Everyday	.000	.079	.000	.079
Monthly	.010	.056	.005	.056
Less often	.260	.084	.259**	.084
Never drink alcohol	306	.201	309	.201
BMI	022***	.004	023***	.004
Exercise (Ref= Yes- exercise)				
No exercise	.239***	.058	.252***	.058

\*p≤0.05 \*\* p≤0.01 \*\*\*pp≤0.001

## **Psychological Distress and Inflammation**

Table 9 depicts the regression model for psychological distress on both CRP and fibrinogen..

*Psychological Distress and Inflammation.* A multiple linear regression was conducted to examine the relationship between psychological distress and levels of CRP. The CRP model was significant, F(22, 7730)=88.827, p<.001), explaining 20.2% ( $R^2$ =.202) of the variance in

the CRP. However, there was no significant association between psychological distress and CRP.

A multiple linear regression was conducted to examine the relationship between psychological distress and levels of fibrinogen. The model was significant, F(20, 7685)=74.628, p<.001), explaining 16.3% ( $R^2$ =.163) of the variance in the outcome variable. Similarly, there was no significant association with psychological distress and fibrinogen.

In terms of covariates for both models, following full adjustment a number of variables remained independently associated with psychological distress (see Table 9 for the specific levels of significance for each model). Unless it is indicated, these covariates were significant in both CRP and the fibrinogen models of psychological distress. Men had lower levels of CRP but not in the fibrinogen model (B=-.044, SE=.011, p<.001). Those that did not exercise had higher levels of CRP compared to those that do participate in exercise. Respondents of a Mixed ethnic background were associated with higher levels of fibrinogen (B=.021, SE=.009, p=.019), as compared with those respondents that were White. In contrast, there were no significant associations between ethnicity and CRP. In terms of medication, taking anti-inflammatory medication (B=.115, SE=.020, p<.001) and taking contraceptive medication and HRT was associated with higher levels of CRP (B= .184, SE=.036, p<.001), when compared to those not on these medications, respectively. In contrast, only statins were significantly associated with higher levels of fibrinogen compared to those not on this medication. Compared to those who previously smoked, being a current smoker was associated with higher levels of CRP and fibrinogen (B=.143, SE=.015, p<.001), whereas never smoking was associated with lower levels of CRP (B=-.026, SE=.011, p<.022). Age was associated with higher rates of CRP and fibrinogen (B=.005, SE=.001, p<.001). A higher BMI was associated with higher levels of CRP and fibrinogen (B=.034, SE=.001, p<.001). Compared to those who drink alcohol weekly, those that drink alcohol monthly (B = .012,

SE=.002, p<.001) and drank alcohol less often (B=.009, SE=.004, p=.015) was associated with having higher levels of fibrinogen.

# Table 9

Regression analysis results for psychological distress, CRP and fibrinogen

	CRP		Fibrinogen	
	Coefficient	Standard	Coefficient	Standard
	Coefficient	Error	Coefficient	Error
Psychological distress	.001	.002	.000	.000
(GHQ-12)	.001	.002	.000	.000
Gender (Ref=Female)				
Male	039***	.011	017	.002
Age	.005***	.001	.002***	.000
Ethnicity (Ref=white)				
Black/Black British	.007***	.058	001	.011
Asian/British Asian	.034	.037	.007	.007
Mixed background/Other	.059	.048	.021*	.009
Medication (Ref = Not				
taking stated medication)				
Contraception/HRT	.182***	.036	010	.007
Statins	079***	.015	.007*	.003

## **Table 9** (Continued)

	CF	RP	Fibrinogen	
	Coefficient	Standard Error	Coefficient	Standard Error
Anti-inflammatories	.111***	.020	.002	.004
Smoking status				
(Ref=previous smoker)				
Current smoker	.140***	.015	.036***	.003
Never smoked	025*	.012	003	.002
Alcohol consumption				
(Ref=Weekly)				
Everyday	.012	.018	.005	.007
Monthly	.023	.013	.012***	.002
Less often	.020	.019	.009*	.004
Never drink alcohol	.033	.043	.008	.008
BMI	.034***	.001	.004***	.000
Exercise (Ref= Yes-				
exercise)				
No exercise	.083***	.013	.010***	.003

\*p≤0.05 \*\* p≤0.01 \*\*\*p≤0.001

## Sleep Quality and Sleep Duration and Inflammation

Table 10 depicts the regression model for sleep duration, sleep quality and CRP and table 11 depicts the regression model for sleep duration, sleep quality and fibrinogen.

*Sleep Quality and Inflammation.* The regression model exploring sleep quality and levels of CRP was significant, F(23, 8422)=94.099, p<.001), explaining 20.4% ( $R^2=.204$ ) of the

variance in levels of CRP. An association between CRP and sleep quality was apparent such that poor sleep quality was associated with raised levels of CRP compared to high quality sleep. Both 'fairly bad' (B=.038, SE=.015, p<.005) and 'very bad' sleep (B=.054, SE=.027, p<.005) were associated with raised levels of CRP compared to high quality sleep.

The regression model exploring sleep quality and levels of fibrinogen was significant, F(23, 8364)=72.848, p<.001), explaining 16.1% ( $R^2=.161$ ) of the variance in fibrinogen. However, in contrast to CRP, sleep quality did not significantly contribute to the model and there was no association with sleep quality and fibrinogen.

In terms of covariates for both models, following full adjustment a number of variables remained independently associated with CRP and fibrinogen (see Table's 10 and 11 for the specific levels of significance for each model). Unless it is indicated, these covariates were significant in both sleep duration and sleep quality models of CRP and the fibrinogen. Compared with women, men had lower levels of CRP and fibrinogen. There were no significant associations between ethnicity and each inflammatory marker. In terms of medication, those taking statins had lower rates of CRP compared with those not taking statins, contrastingly, those taking contraceptive medication and HRT and anti-inflammatory medication had higher concentrations of both inflammatory markers, compared to those not taking these respective medications. Compared to those who previously smoked, higher concentrations of both CRP and fibrinogen were associated with being a current smoker, whereas never smoking was associated with lower levels of both inflammatory markers. There were no significant associations between levels of alcohol consumption and concentrations of CRP and fibrinogen within either model. Not participating in exercise was associated with higher levels of CRP and fibrinogen, compared to those that do participate in exercise. Age and BMI were associated with higher rates of CRP and fibrinogen.

Sleep Duration and Inflammation. A multiple linear regression was conducted to examine the relationship between sleep duration and levels of CRP. The model was significant, F(22, 8423)=97.281, p<.001), explaining 20.3% ( $R^2=.203$ ) of the variance in the outcome variable. In a similar manner to sleep quality, a longer sleep duration was associated with a higher level of CRP and this association was specific as associations were not apparent for fibrinogen. The regression model exploring sleep duration and levels of fibrinogen was significant, F(22, 8364)=72.695, p<.001), explaining 16.1% ( $R^2=.161$ ) of the variance in the fibrinogen.

In terms of covariates for both models, following full adjustment a number of variables remained independently associated with CRP and fibrinogen (see tables 10 and 11 for the specific levels of significance for each model). Unless it is indicated, these covariates were significant in both sleep duration models of CRP and fibrinogen. Compared with women, men had lower levels of CRP and fibrinogen (B=-.044, SE=.015, p<.001). Both age and BMI were associated with higher rates of CRP and fibrinogen. There were no significant associations between ethnicity and CRP and fibrinogen. In terms of medication, those taking statins had lower rates of CRP compared with those not taking statins, contrastingly, those taking contraceptive medication and HRT (B=.209, SE=.036, p<.001) and anti-inflammatory medication (B=.105, SE=.021, p<.001), had higher rates CRP, compared to those not taking these respective medications. There were no associations with medication with fibrinogen in the sleep duration model. Compared to those who previously smoked, higher levels of CRP and fibrinogen were associated with being a current smoker. There were no significant associations between alcohol consumption and levels of CRP in the sleep duration model. Not participating in exercise was associated with higher levels of CRP and fibrinogen compared to those that do participate in exercise.

# **Table 10**Regression analysis results for sleep duration, sleep quality and CRP

	Sleep Duration		Sleep	Sleep Quality		
	Coefficient Standard Error		Coefficient	Standard Error		
Sleep Duration (Ref=6-7						
hours)						
6 hours or less	.008	.011				
7-8 hours	.025	.013				
9 hours or more	.067***	.024				
Sleep Quality (Ref=Very						
good)						
Fairly good			.007	.011		
Fairly bad			.038**	.015		
Very bad			.054*	.027		
Gender (Ref=Female)						
Male	039***	.010	042***	.010		
Age	.005***	.001	.005***	.000		
Ethnicity (Ref=white)						
Black/Black British	014	.049	003	.049		
Asian/British Asian	.049	.033	.048	.033		
Mixed background/Other	.055	.045	.056	.045		
Medication (Ref = Not						
taking stated medication)						
Contraception/HRT	.199***	.034	.191***	.034		
Statins	087***	.014	083***	.014		

# Table 10 (Continued)

	Sleep Duration		Sleep Quality	
	Coefficient	Standard Error	Coefficient	Standard Error
Anti-inflammatories	.115***	.020	.113***	.020
Smoking status				
(Ref=previous smoker)				
Current smoker	.143***	.014	.142***	.014
Never smoked	027*	.011	026*	.011
Alcohol consumption				
(Ref=Weekly)				
Everyday	.009	.017	.010	.018
Monthly	.021	.012	.023	.013
Less often	.020	.019	.020	.019
Never drink alcohol	.035	.042	.040	.042
BMI	.034***	.001	.034***	.001
Exercise (Ref= Yes- exercise)				
No exercise	.087***	.013	.089***	.013

\*p $\leq 0.05$  \*\* p $\leq 0.01$  \*\*\*p $\leq 0.001$ 

## Table 11

	Sleep Duration		Sleep	quality
	Coefficient Standard Error		Coefficient	Standard Error
Sleep Duration (Ref=6-7				
hours)				
6 hours or less	.002	.002		
7-8 hours	.005	.003		
9 hours or more	001	.005		
Sleep Quality (Ref=Very				
good)				
Fairly good			.001	.002
Fairly bad			.003	.003
Very bad			.004	.005
Gender (Ref=Female)				
Male	016***	.002	016***	.002
Age	.002***	.000	.002***	.000
Ethnicity (Ref=white)				
Black/Black British	.004	.009	.004	.009
Asian/British Asian	.012	.006	.012*	.006
Mixed background/Other	.014	.009	.014	.008
Medication (Ref = Not				
taking stated medication)				
Contraception/HRT	010	.006	010	.006
Statins	.005	.003	.004	.003

Regression analysis results for sleep duration, sleep quality and fibrinogen

#### Table 11 (Continued)

	Sleep Duration		Sleep Quality		
	Coefficient	Standard Error	Coefficient	Standard Error	
Anti-inflammatories	.001	.002	.001	.004	
Smoking status					
(Ref=previous smoker)					
Current smoker	.036***	.003	.036***	.003	
Never smoked	004	.002	004	.002	
Alcohol consumption					
(Ref=Weekly)					
Everyday	012***	.007	012***	.003	
Monthly	.010***	.002	.010***	.002	
Less often	.006	.004	.006	.004	
Never drink alcohol	.004	.008	.004	.008	
BMI	.004***	.000	.004***	.000	
Exercise (Ref= Yes- exercise)					
No exercise	.011***	.002	.011***	.002	

\*p<u><0.05</u> \*\* p<u><0.01</u> \*\*\*pp<u><0.001</u>

## Mediation Analyses: Socioeconomic position, Sleep Quality and CRP

Based on the results of these regression analyses, two models were tested (see table 12) and one model involving sleep duration was estimated given it met the preconditions that were needed to proceed with mediation analysis. Therefore, it is estimated that in this model sleep duration may have been a possible mediator in the relationship between educational attainment and CRP. Although as discussed there were important associations found

regarding psychological distress, psychological distress did not meet the prerequisite requirements for mediation.

The regression model (model 1) exploring household income, sleep quality and CRP was significant, F(21, 8424)=102.901, p<.001), explaining 20.4% (R<sup>2</sup>=.204) of the variance in CRP. Those with 'fairly bad' sleep were associated with raised levels of CRP compared to high quality sleep (B=.036, SE=.015, p=.027). Those in the lowest household income tertile (B=.074, SE=.013, p<.001) had raised CRP compared to those in the highest household income tertile

The regression model (model 2) exploring educational attainment, sleep quality and CRP was significant, F(22, 8423)=98.255, p<.001), explaining 20.4% ( $R^2$ =.204) of the variance in CRP. Those with 'fairly bad' sleep were associated with raised levels of CRP compared to high quality sleep (B=.036, SE=.015, p<.005). Those with no qualifications (B=.091, SE=.015, p<.001) had raised CRP compared to those in the highest educational attainment. Covariates were adjusted for in both model 1 and 2 and are depicted in table 12.

Model 1 examined sleep quality as a mediator between the lowest household income tertile and CRP. In this model, sleep quality was significantly associated with CRP, though the association with lowest household income remained significant and the Sobel test indicated that the indirect effect of sleep quality was significant (Sobel= 2.8441253, SE= 0.00512565, p= 0.00445335). (Aroian= 2.81175509, SE= 0.00518466, p=0.0049272). This suggests that sleep quality is a significant mediator in the association between household income and CRP.

Model 2 examined sleep quality as a mediator between educational attainment and CRP. In this model, sleep quality was significantly associated with CRP, though the association with educational attainment remained significant and the Sobel test indicated that

the indirect effect of sleep quality was significant (Sobel= 2.5805376, SE= 0.0074407, p= 0.00986466). (Aroian= 2.55229381, SE= 0.00752304, p= 0.01070162). This suggests that sleep quality is a significant mediator in the association between educational attainment and CRP.

## Table 12.

Sleep quality as a mediator between income and education and CRP

Model 1			Model 2		
Predictor: Income			Predictor: Education		
Mediator: Sleep quality			Mediator: Sleep quality		
	В	SE		В	SE
Primary variables			Primary variables		
Household Income			Education		
Middle household income	.046	.027	A-Level	.019	.014
Lowest household income	.074***	.013	GCSE	.013	.013
			No qualifications	.091***	.015
Sleep Quality			Sleep Quality		
Fairly good	.007	.011	Fairly good	.006	.011
Fairly bad	.036*	.015	Fairly bad	.037**	.015
Very bad	.046	.027	Very bad	.051	.027
Covariates			Covariates		
Gender			Gender		
Male	039***	.010	Male	040***	.010
Age	.005***	.000	Age	.005***	.000
Ethnicity			Ethnicity		
Black/Black British	005	.048	Black/Black British	.004	.048
Asian/British Asian	.037	.033	Asian/British Asian	.047	.033
Mixed Background/Other	.051	.045	Mixed Background/Other	.061	.045

Mode	el 1		Model 2		
Predictor: Income			Predictor: Education		
Mediator: Sleep quality			Mediator: Sleep quality		
Covariates			Covariates		
Medication			Medication		
Contraception/HRT	.199***	.034	Contraception/HRT	192***	.034
Statins	088***	.014	Statins	088***	.000
Anti-inflammatories	.112***	.020	Anti-inflammatories	.111***	.020
Smoking status			Smoking status		
Current smoker	005	.048	Current smoker	.134***	.014
Never smoked	.037	.033	Never smoked	024*	.011
Alcohol consumption			Alcohol consumption		
Everyday	.014	.018	Everyday	.014	.018
Monthly	.019	.013	Monthly	.023	.013
Less often	.013	.019	Less often	.015	.019
Never drink alcohol	.031	.042	Never drink alcohol	.033	.042
BMI	.033***	.001	BMI	.033***	.00
Exercise			Exercise		
No exercise	.078***	.013	No exercise	.074***	.013

#### **Chapter Four: Discussion**

#### **Chapter Summary**

This chapter provides an overview of the findings of the current study. Relevant theory and literature was utilised to discuss the findings. This chapter also outlines the strengths and limitations of the current study. Potential clinical implications and impact on policy is outlined. Finally, conclusions are drawn from the current study and the author gives a reflexive account.

### **Summary of Findings**

The main objective of the current study was to examine the potential mediating role of psychological distress and sleep duration and sleep quality in the association between socioeconomic position and inflammation, using the inflammatory biomarkers, CRP and fibrinogen. Although other studies have found that socioeconomic position is linked with levels of inflammation, less is known about the potential underlying mechanisms of this association. The current study addresses this gap in the literature by examining sleep (duration and quality) and psychological distress as possible mediators in the association between socioeconomic position and inflammation. Using data from *Understanding Society*: the UK Household Longitudinal Study, this research aimed to determine whether socioeconomic position, as measured by educational attainment and household income, was associated with increased rates of inflammation, as measured by the biomarkers, CRP and fibrinogen. Secondly, the current study aimed to determine whether (a) psychological distress mediates the link between socioeconomic position and CRP and fibrinogen, and (b) whether sleep quality and sleep quantity mediate the link between socioeconomic position and CRP and fibrinogen. A range of relevant covariates were adjusted for within each analysis.

Overall, the findings of the study supported the first hypothesis. Compared to the highest level of education, both CRP and fibrinogen was raised in those with the lowest educational attainment following adjustment for all covariates. Similarly, compared to those in the highest income tertile, both middle and lowest income tertiles were associated with higher concentrations of both CRP and fibrinogen.

Educational attainment was not associated with psychological distress, however, when household income was used as a measure of socioeconomic position, those with the lowest household income had raised psychological distress compared to those in the highest household income. However, there was no association of either CRP and fibrinogen and psychological distress following adjustment for all covariates.

In terms of sleep, there were no associations between sleep duration and household income. There was a significant association between educational attainment and sleep duration, however, these were in the opposite directions than were hypothesised. Having no qualifications was associated with an increased likelihood of falling within the '9 hours or more' sleep category, compared to the highest level of qualifications. However, a longer sleep duration was associated with a higher level of CRP, although these associations were not evident for fibrinogen.

Similar to associations with sleep duration and educational attainment, associations were apparent with measures of socioeconomic position and sleep quality. However, these were in directions that were not hypothesised. Thus, having no qualifications was associated with an increased likelihood of falling within the 'very good sleep' category, compared to the highest level of qualifications. Similarly, being in the lowest income tertile was associated with 'very good sleep', compared to the highest income tertile.

Psychological distress did not meet the prerequisite criteria to be a possible mediator, given it was not associated with either CRP or fibrinogen. However, the results do demonstrate that sleep quality may mediate the relationship between raised concentrations of CRP and socioeconomic position, when measured either by educational attainment or by household income and following adjustment for all covariates.

#### **Discussion of findings**

# Socioeconomic Position and Inflammation

Research has shown that socioeconomic position is associated with the biomarkers of inflammation, including CRP (Alley et al., 2006; Davillias et al., 2017; Farmer et al., 2021) and fibrinogen (Kim et al., 2016; Steptoe et al., 2003). In recent years, inflammation has been maintained to be an important aspect in the relationship between those who are disadvantaged in terms of education and income and health outcomes (Miller et al., 2011).

Based on the systematic review of the literature completed as part of this thesis, it is generally demonstrated that a more disadvantaged socioeconomic position, whether it is measured by income, educational attainment or occupation, is associated with higher levels of inflammation. This may explain why socioeconomic position is associated with poor health outcomes, thus compared to individuals of a more advantaged socioeconomic position. For instance, individuals of a lower socioeconomic position are more likely to experience increased rates of serious physical health conditions such as cardiovascular disease (Schultz et al., 2018) and diabetes (Tsalamandris et al., 2019). These health disparities are also shown for psychological wellbeing. Research has shown that there is a higher risk of depression

amongst people of a lower socioeconomic position (Lob et al., 2020). In fact, previous research has indicated that there is an inverse relationship between socioeconomic position and mental health, for example, higher education considerably decreased the risk of experiencing depression (Freeman et al., 2016).

The current findings revealed that both CRP and fibrinogen were elevated in those with the lowest level of educational attainment compared to those with the highest level of education. The association with educational attainment with CRP and fibrinogen was independent of demographic factors such as age, sex and ethnicity and health related behaviours such as alcohol consumption, smoking status, exercise, BMI and medication use (anti-inflammatory medication, statins, HRT and contraception). The coefficients pertaining to educational and income were larger compared to the fibrinogen coefficients. This may suggest that the impact of level of educational attainment and household income on CRP is more substantial than compared to fibrinogen. These findings suggest educational attainment is a considerable factor that underlies the dipartites in raised concentrations of CRP. Previous research has demonstrated similar findings showing that education attainment exerts a power over CRP levels than compared to income (Dinwiddie et al., 2015). One way of understanding these findings is that an individual's educational attainment allows for the opportunity to engage with educational facilitates. This can promote the likelihood of securing an occupation that occasions the opportunities to increase income and thus allowing access to nutritious food, leisure activities and health care, all of which may alleviate stress and reduce behaviours that are adverse to health (Dinwiddie et al., 2010).

A similar relationship was shown when socioeconomic position was measured by household income. Compared to those in the highest income tertile, both middle and lowest income tertiles were associated with higher concentrations of both CRP and fibrinogen. The coefficient was larger for those with a lower household income compared to a middle household income. The association with educational attainment with CRP was independent of demographic factors, alcohol consumption, smoking status, exercise, BMI and medication use. Therefore, the current study aligns with the extant literature (e.g. Davillias et al., 2017; Kim et al., 2016) and provides evidence showing that a lower socioeconomic position is associated with increased inflammation, as measured by CRP and fibrinogen. It is important to consider that whilst there is evidence of an association between socioeconomic position and inflammation, it cannot be determined which direction the association is travelling. It is feasible that both may influence each other over time (Pedron et al., 2019; Varekamp et al., 2013).

#### Socioeconomic Position and Psychological Distress

The current results showed that educational attainment was not significantly associated with psychological distress. This is in contrast to a growing body of literature illustrating that the more years of education a person has or a higher level of educational attainment is linked with overall better psychological wellbeing compared to individual's with lower educational attainment (Avendano et al., 2020; Mirowsky & Ross, 2003). This may be due to the virtue of possessing an education increases the chances of psychological wellbeing through better skills and resources (Ross & Zhang, 2008). Although, in accordance with the current findings, there are exceptions to this where no evidence has been found that educational attainment is related to better mental health (Dahmann & Schnitzlein, 2019). One explanation of this is that educational attainment is different in the UK now than it used to be. There has been increases in overall educational attainment because of policy changes. Currently policy is being developed referred to as The Advanced British Standard which states that some form of numeracy and literacy will be studied until the age of 18. It is possible these changes have broken the link between educational attainment and psychological distress.

In the current study, when household income was used as a measure of socioeconomic position, those with the lowest income had higher rates of psychological distress compared to those with the highest household income. As discussed previously, psychological distress is a broad term that conceptualises stress, anxiety and depression and is an important characterisation of any mental health problem (Goldberg & Goodyear, 2005). There is a large evidence base showing that together with psychological distress, lower income is related to depression (Chen et al., 2017), anxiety (Green & Benzeval, 2013), suicide (Madigan & Daly, 2023) and psychosis (Sweeney et al., 2015). It is important to consider that whilst there is evidence of an association between household income and psychological distress, it cannot be determined which direction the association is travelling. Both household income and psychological distress may influence each other over time. Lower income may contribute to increased psychological distress due to the anxiety in relation to financial precarity (Sun & Houle, 2020; Ryu & Fan, 2023). Indeed, there is a perceived association between low household income and higher psychological distress (McMillan et al., 2010), for a review see Thomson et al. (2022). It is also important to note that the relationship between household income and psychological distress can be bidirectional. For example, high psychological distress may result in a lower income through being unable to maintain employment and being unable to address financial burdens (Fenn et al., 2014).

Broadly, there are two main approaches to delineating the pathways between income and psychological distress and mental health. These are social causation and social selection (also referred to as social drift). The social causation hypothesis posits that the financial stress, poor living conditions and hardship linked with low income results in an exacerbation of psychological stress and development of depression, anxiety and other mental health problems (Kessler et al., 1997; Wickham et al., 2017). Contrastingly, the social selection hypothesis maintains that individuals experiencing high levels of psychological distress and mental health problems are more likely to be faced with a loss of employment with related loss of income, lower income employment and hospital admissions related to mental health (Dohrenwend et al., 1992). Recent research has provided evidence that both these mechanisms may act simultaneously (Jin et al., 2020).

There is a preponderance of studies consistently demonstrating the vital role of material factors in elucidating the socioeconomic position gradient in mental health (for a meta-analysis see, Thompson et al., 2022). Household income has been most robustly associated with the capability to purchase goods and services that foster health (Subramanian et al., 2002). Poverty and low income can result in stressors related to financial precarity and uncertainty regarding having enough money to purchase essential goods such as food, heating and issues around securing adequate housing. Furthermore, low-income communities are more likely to experience difficulties with poor schooling systems, higher rates of crime and violence and poor infrastructure, all of these factors have a negative impact on psychological wellbeing (Shule & Bolte, 2015).

Together with the health implications of being unable to buy health enhancing goods, being in a financial position of low income, where it is not possible to achieve material needs might also result in high psychological distress through a process of social comparison (Marmot & Wilkinson, 2001). Social comparison theory refers to individual's evaluating their own worth in regards to how they compare to others in their social environment (Festinger, 1954). Indeed, material goods offer more than satisfying essential requirements, such as purchasing food and heating, being capable of purchasing and being a consumer of goods and services affords the opportunity to express a social identity (Marmot & Wilkinson, 2001). Indeed, Marx (1942) and Weber (1922) espoused this perspective in their conceptualisation of socioeconomic position and its relation to power and prestige. An individual's self-image is fortified through the acquisition of possession of material goods (Marx, 1942; Weber, 1922). Income is a representation of social status and success, similar to how being in a financially disadvantaged position might be stigmatising (Foster, 2021). Although this theoretical lens focuses on Western ideas of materialism, it has been indicated that materialism occurs in various other countries around the world (Belk, 1988), although it is the given expression of materialism that tends to be culturally specific (Ger & Belk, 1996).

#### Socioeconomic Position and Sleep Duration

There were no significant associations between sleep duration and household income. This is represents an anomaly and is not consistent with the literature which usually demonstrates an association between an adequate sleep duration (7-9 hours) and higher income (e.g. Mukherjee et al., 2015). Evidence suggests that an optimum sleep pattern is between seven and 9 hours, less than 7 hours is considered inadequate sleep and a sleep duration that exceeds 9 hours is deemed to be a too long sleep (Ren et al., 2019). When educational attainment was used as a measure of socioeconomic position, however, the current results did reveal a significant association but in the reverse direction as to what was initially hypothesised. Thus, having no qualifications was associated with an increased likelihood of having a longer sleep duration (9 hours or more), compared to those with a higher level of education. These findings align with a recent study conducted by Berry-Caban et al. (2023) which showed that those with a sleep duration of 9 hours or more had lower educational attainment, reported substantial life discontent and experienced poorer overall health. Furthermore, these findings are consistent with previous studies that have shown that individuals with less education report longer sleep durations (e.g. Basner et al., 2014).

These disparities in education and sleep duration might be understood as being related to type of employment. For instance, people with less education have a greater likelihood of working non-standard hours such as shift work, which has been shown to effect sleep adversely and leads to a longer sleep duration (Kecklund & Axelsson., 2016). As discussed previously, within the current study it was found that both education and income were associated with psychological distress. This is finding is important in that an increased risk of psychological distress has been shown to be more prominent in those sleeping for both shorter and for longer periods of time (Vestergaard et al., 2024). It is important to consider that there is a bidirectional relationship between socioeconomic position and sleep duration.

### Socioeconomic Position and Sleep Quality

The current findings revealed that having no qualifications was associated with an increased likelihood of having better quality sleep (i.e. 'very good sleep' category), compared to those with the highest level of qualifications. Correspondingly, being in the lowest income tertile was associated with very good sleep, compared to those in the highest income tertile. These findings are in contrast with the extant literature (e.g., Patel et al., 2010) which invariably shows that lower income and higher educational attainment is associated with better sleep quality.

# Sleep and Inflammation

**Sleep Duration.** Consistent with the previous findings, the current results indicate that a long sleep duration of 9 hours or more is associated with higher concentrations of CRP, although this was not apparent for fibrinogen. Therefore, this indicates a specificity in the link between CRP as a biomarker of inflammation and a long sleep duration. In contrast, the impact of a short sleep duration was not found to be statistically significant after full adjustment of covariates. The biological pathways that underpin the association between long sleep duration (9 hours or more) and CRP are still not fully explained. This perhaps reflects the inconsistent results in the evidence base on the link between sleep duration and levels of inflammation. For instance, the Longitudinal Whitehall II study showed that a shorter sleep duration was associated with raised concentrations of CRP (Ferrie et al., 2013). Previous studies have shown that a long sleep duration (9 hours or more) is associated with high levels of CRP (Garbarino et al., 2021). A meta-analysis conducted by Irwin et al., (2016) found that evidence from 72 studies that sleep disturbance and longer sleep duration were related to elevated CRP and IL-6. Furthermore, epidemiologic research has provided evidence that both a short and long duration of sleep are related to raised levels of CRP (Patel et al., 2009). The mechanisms linking long sleep duration to increased CRP are not conclusive. However, it has been postulated that a greater sleep duration could be caused by an undiagnosed medical condition, such as risk of stroke, or a comorbid condition that is related to fatigue or tiredness (Wen et al., 2016). In terms of psychological explanations, depression is characterised lethargy and sleeping for longer amounts of time, such as excessive sleep is referred to as hypersomnia (Lopez et al., 2017). Sleeping for longer durations may be related to an effort to avoid psychological distress and cope with depressed mood (Lopez et al., 2017). Indeed, those that report a long sleep duration report more symptoms in accordance with depression (Lee at al., 2020). Furthermore, depression has been associated with inflammation (Valkanova et al., 2013). It is important to consider that there is a bidirectional relationship between sleep duration and inflammation.

#### **Psychological Distress and Inflammation**

Psychological distress was not associated with either CRP or fibrinogen. This perhaps reflects the extant literature which conveys inconsistent findings on the relationship between psychological distress and inflammatory biomarkers, with some studies reporting an association (Baek et al., 2019; Purnichi et al., 2017) and others not (Linkas et al., 2022). Furthermore, there may be other factors that influence this relationship such as adverse life experiences, there is large body of studies showing that the experience of trauma with psychological distress is associated with increased levels of inflammatory biomarkers, including CRP and IL-6 (Lawn et al., 2022). A prospective study by Purnichi et al., (2017) found that fibrinogen levels were associated with psychological distress and depression, participants who scored highly in terms of psychological distress had raised levels of fibringen, compared to those who scored lower for psychological distress, although the authors do caution that the effect size was quite small. One explanation for not finding an association between psychological distress and inflammation within the current study is that some of the studies reporting the link between psychological distress and inflammation have been completed with exclusively clinical populations (e.g. Yuan et al., 2019), in contrast to the current study which comprised of a large sample of the general population. A further explanation to take into consideration is that of chronicity, as suggested by Dantzer et al. (2008) who maintained that a potential factor in the link between inflammation and mental health is the long duration of elevated rates of inflammation over a period of time which might then have more impact on the brain. Another factor to take into consideration is that it has been reported that elevated levels of inflammatory biomarkers have been associated with the somatic symptoms of depression (DellaGioa & Hannestad, 2010). However, the GHQ-12 does not comprehensively measure somatic symptoms associated with psychological distress and depression such as changes in appetite, lack of energy and loss of libido (Tylee & Gandhi, 2005). It is important to consider that there is a bidirectional relationship between psychological distress and inflammation (Beurel et al., 2020).

### Mediation of sleep quality

In contrast to psychological distress, the results do demonstrate that sleep quality may mediate the relationship between raised concentrations of CRP and socioeconomic position, when measured either by educational attainment or by household income and following adjustment for all covariates. People with lower educational attainment and in the lowest household income tertile were more likely to rate the quality of their sleep as 'very good'. Therefore, the indication that 'good quality' mediates the link between CRP and lower educational attainment and household income is an unpredicted finding. As discussed previously, a longer sleep (9 hours or more) is associated with poorer health outcomes and increased inflammation. There are some possible reasons for this unexpected finding of how it appears that 'very good' sleep mediates the link between socioeconomic position and inflammation. It is important to consider that the evidence of mediation could constitute a type I error, given that only one of the 12 possible models that could have been tested had ultimately met all of the preconditions and shown evidence of mediation.

A type I error is characterised by erroneously stating that the study found significant differences and the null hypothesis is rejected, or in other words it refers to a false positive. If indeed, this was a type I error it would mean that 'very good' sleep quality was not a mediator between the relationship between CRP and socioeconomic position. A reason for this result is that the variable 'very good' sleep was acting as a proxy variable for something else that might be the true mediating variable. This may have occurred given that the construct of sleep quality was examined using a single item question.

Future research should examine how the single item sleep disturbance question varies by the other sleep disturbance questions or the sleep duration categories. This may highlight if those people subjectively rating their sleep as 'very good' may have other sleep behaviours that would be expected to mediate the relationship between CRP levels and socioeconomic position. This could be completed by running the cross tabulation function in SPSS to quantitatively examine the relationship and possible correlations between 'sleep quality' and the other variables in the data set, such as 'sleep duration' and other items measuring sleep quality such as 'waking during the night', use of sleeping pills, taking a long time to get to sleep. A possible explanation for this unexpected finding is that despite subjectively reporting 'very good' quality of sleep, respondents may have additionally experienced sleep disturbances such as broken sleep, waking up tired, and a taking a long time to get to sleep, all of which have been associated with increased inflammatory biomarkers (Bjurström et al., 2017; Dowd et al., 2011; Dzierzewski et al. 2020).

To test this relationship further, future research should consider utilising additional questions of the Pittsburgh Sleep Quality Index (Buysse et al. 1989), these include questions such as 'How many minutes does it take to get to sleep each night', 'Waking up in the middle of the night or early in the morning' and 'Have to wake up to use the bathroom.

# Sleep quality and inflammation

Previous studies have shown that poor sleep quality is associated with raised CRP (Wilson et al., 2015; Lee et al., 2020).

The current findings confirmed these previous results, both 'fairly bad' and 'very bad' sleep were associated with raised levels of CRP compared to 'very good' sleep quality. However, the association was specific, as there was no apparent association when fibrinogen was the measure of inflammation. Some studies have shown that fibrinogen is linked with a longer sleep duration (Hale et al., 2012).

These findings are important given that raised CRP is as an associated risk factor depression (Valkanova et al., 2013) together with physical health conditions such as for

coronary heart disease (Amezcua-Castillo et al., 2023) and diabetes (Kanmani et al., 2019). The mechanisms that may underpin the relationship between poor sleep and health include the sympathetic nervous system and proinflammatory responses. It has been well documented that sleep performs an essential function in physiological recouperation (Hamilton et al., 2007). During periods of good sleep, the sympathetic nervous system is deactivated whilst the para-sympathetic nervous system is triggered resulting in the release of growth hormone. This allows the body to be in a state sometimes referred to as 'rest and digest', as discussed previously (Schmidt & Thews, 1989). This is essential in facilitating the recovery of psychological stress related to daily life (Van Reeth et al., 2000). In contrast, if a person is experiencing prolonged periods of poor quality sleep the sympathetic nervous system is initiated and this results in the release of inflammatory cytokines.

There are a number of explanations as to why poor quality of sleep may raise concentrations of inflammation. For example, typically there is an elevation in acute inflammation when someone is awoken from sleep (Ranjbaran et al., 2007), however, if a person has an enduring time of disturbed sleep and low sleep quality this results in a proliferation of proinflammatory and anti-inflammatory cytokines (Frey et al., 2007). Taken together these finding point to an explanation of why poor sleep quality is associated with increased inflammation (Huang et al., 2017). Another factor to take into account is the circadian clock, which plays an integral role in facilitating homeostasis and is coordinated with the 24-hour day (Lowrey et al., 2004). Interestingly, circadian rhythms exist in predominantly all of the body's cells and are coordinated by a central clock that resides in the suprachiasmatic nucleus (SCN) which is located in the hypothalamus region of the brain (Ko & Takahashi, 2006). Accordingly, poor sleep quality may increase inflammation by persistent disturbance of these cellular parts (Irwin et al., 2016).

#### **Discussion of covariates**

Consistent with previous studies, there were a number of variables that were included as covariates that were independently shown to be related to increased concentrations of CRP and fibrinogen. Compared with women, men were associated with having significantly lower levels of CRP and fibrinogen. Previous findings have shown that women do tend to have higher rates of CRP when compared to men (Khera et al., 2005). In a longitudinal study of adults aged 20 to 74 years, over a period of 11 years CRP was raised in women compared with men (Kranjac et al., 2021). A putative mechanism suggested to be involved in sex differences of CRP is that there are differences in the link between CRP and obesity in women. Specifically, as body fat (or adiposity) increases so too does CRP in women compared with men (Khera et al., 2005). It was also found that those who did not participate in exercise were associated with having raised CRP and fibrinogen. This confirms previous findings that have demonstrated that lack of exercise and sedentary lifestyle is correlated with higher rates of CRP (Esteghamati et al., 2012) and exercise has been shown to be associated with reduce rates of inflammatory biomarkers, including CRP (Zheng et al., 2019).

Increasing BMI was associated with higher concentrations of CRP and fibrinogen as has been extensively reported previously (e.g. Ellulu et al., 2017). Being a current smoker was associated with higher concentrations of CRP compared to those who previously smoked. This confirms previous findings of higher rates of CRP being found in those who currently smoke (Elisia et al., 2020).

### Strengths

Understanding Society is a both nationally and internationally renowned dataset, it offers very large sample sizes and has been frequently utilised to facilitate impact on government policy, together with contributing to and developing a greater understanding of how social problems, such as health inequalities, can be understood and addressed (Harding et al., 2022). The large sample size of the current study may lend itself to better generalisability of findings (Dunn et al., 2015). The current study uses secondary data analysis which has many benefits. It afforded the opportunity to analyse a large data set that typically would not be within the scope of the current study being completed by a single researcher (Dunn et al., 2015).

In terms of measurement of variables, the current study uses both educational attainment and household income as measures of socioeconomic position. This allows for a broader indication of socioeconomic position. Furthermore, as well as self-report measures for sleep and psychological distress, it uses biomarker data as an outcome which is not affected by reporting biases and allows for further understanding of the relationship between psychological and physical health. A further strength regarding methodology is that the data set was collected utilising standardised measures that have been validated.

### Limitations

Firstly, a key limitation of the current study is that the study design was crosssectional, this means that causation can be inferred but it cannot be assertively determined and it is not possible to ascertain whether there is a causal relationship between the associations between socioeconomic position, psychological distress, sleep duration, sleep quality and inflammatory biomarkers. For example, inflammation may affect a person's health which may then result in changes to their income. Secondly, whilst a number of covariates were adjusted for in the analyses, it was not possible to include childhood trauma as a covariate given it is not obtained as part of data collection within *Understanding Society*. Childhood adverse life experiences have been linked to inflammation (Danese & Lewis, 2016; Lacey et al., 2020). Therefore, it is possible that the impact of childhood trauma may have been an unaccounted for confounding variable. Thirdly, it is known that inflammatory biomarkers indicate different characteristics of inflammation, for example, synergistic effects of inflammatory biomarkers or anti-inflammatory cytokines, both of which play a role in counteracting the aspects of inflammation, such as Interleukin-4, Interleukin-10 and Interleukin-13 (Libby, 2007). Therefore, the inflammatory biomarkers used within the current study do not capture all aspects of inflammation. The measure of sleep quality may be limited. A broader measure that captured experiences such as difficulty falling asleep may have yielded slightly different findings. Another shortcoming is that the study relied on selfreport measures of sleep and other health related behaviours, these may have been subject to some degree of measurement error. Therefore, certain 'desirable' behaviours may be over reported whilst 'undesirable' behaviours are potentially under reported. Thus, characterising a social desirability bias, which is a shortcoming of self-report measures.

Another limitation to be considered is related to the quality appraisal of the studies to be included within the systematic review. The tool may have lacked sensitivity when it was used to appraise the quality of studies for the systematic review. Whilst the JBI is a robust checklist for assessing the quality of studies there are some shortcomings to this tool and its administration within the present study. The tool has been used for approximately 20 years, however, through this time there have been changes in how the risk of bias can be examined in recent years, this has resulted in a critique being directed at the tool in that it may be outdated. A recent review by Barker et al. (2023) has suggested that to improve the tool's sensitivity in detecting bias there should be a number of amendments made to it, specifically in accordance with the recent progresses in the synthesis of evidence, for example reporting in line with the PRISMA (2020).

### Implications of the study

The findings of the current study have various important implications for clinical practice, policy and future research.

# **Policy Making Decisions**

The findings have important implications for policy makers aiming at addressing socioeconomic disparities in health. The results are particularly significant as they demonstrate a vital link between lower educational attainment and lower household income with increased levels of inflammation. Inflammation is a key link to health outcome (Miller et al., 2011) and linked to both mental health, such as depression (Valkanova et al., 2013), and physical health conditions, such as diabetes (Kanmani et al., 2019). This finding has important implications for policy. The cost of living in the United Kingdom has increased and inflation was at its peak level in April 2022. Food, non-alcoholic beverages, electricity and gas were the goods and services that were impacted most by inflation (Joseph Rowntree Foundation, 2024). However, incomes are not increasing commensurately with inflation, in fact, any small increases are being overshadowed by inflation. It has been shown that 4.3 million households were not able to purchase essential goods in 2022/23 (Joseph Rowntree Association, 2024). According to Karjalainen and Levell (2022), those families that have a lower household income will be considerably more be impacted by increased inflation. For instance, heating bills are substantially impacted by inflation, and it has been shown that these families are paying a great proportion of their budget on basic requirements such as buying food and paying energy bills.

In terms of policy, income is receptive to policy change and intervention (Thomson et al., 2022). These findings may inform policy making decisions at a population based level that may help to decrease socioeconomic inequalities in health. Therefore, policy makers may

benefit from these findings when considering the psychological impact associated with lower income and the importance of implementing a sufficient financial support plan to protect against the detrimental impact of socioeconomic inequalities and the corresponding health inequalities associated with them (Thomson et al., 2022). This suggests the importance of a joint up approach at both the individual level (e.g. therapeutic intervention) together with larger economic and social policy interventions.

## Informing Psychological Interventions

The current research identifies the potential psychological impact of disparities in socioeconomic position, in terms of sleep and psychological distress. This is important as both sleep and psychological distress are responsive to intervention. There are important contributions which add to the evidence base showing the value of exploring socioeconomic factors with clients. An integral aspect within clinical psychology practice is formulation and it is characterised by a process of creating a hypothesis or understanding of the beginnings of a person's distress (Johnstone & Dallos, 2013). These findings support the importance of including socioeconomic factors within either a team formulation or when co-creating a formulation with a client to capture the potential social determinants of mental health. Given socioeconomic position is linked with mental health and comorbid physical health conditions, as previously discussed. This study demonstrates the importance of identifying these factors in order to address them, which might have consequences on reducing the overall burden of ill health, for example, in order helping to access the appropriate financial support.

This study has shown associations between socioeconomic position, psychological distress and inflammation. Thus, the current findings have provided support and contributed to the broad evidence base maintaining that the immune system and inflammation is influenced by behavioural, social and neurocognitive influences (Slavich & Cole, 2013).

There is a lot of evidence showing that a range of psychological interventions can have an impact on reducing inflammation, such as Acceptance and Commitment Therapy (Järvelä-Reijonen et al., 2020). A systematic review of 23 studies maintained that CBT was involved with reducing inflammation in people with depression (Lopresti (2017). A meta-analysis of more than 40 studies that psychological interventions such as CBT and other psychotherapies were effective in enhancing immunity and reducing levels of inflammation (Shields et al., 2020). The findings also contribute to the literature on psychological interventions for sleep, given that sleep is amenable to intervention. Cognitive Behavioural Therapy for Insomnia (CBTi) is a first line intervention that is comprised of relaxing techniques, identification and management of unhelpful thinking patterns and sleep restriction (van der Zweerde et al., 2019). The findings provide a rationale for the use of interventions for sleep such as CBTi. Furthermore, within the current literature it is known that there is a need for more research in relation to vulnerable groups for whom CBTi is directed, this may include inequalities in terms of education and income (Muench et al., 2022). The current findings make a contribution to understanding the links between sleep and people with a lower education and household income. Therefore, this research contributes to the theories on sleep difficulties and gives further contribution to the literature in consideration of the findings on the associations between socioeconomic position and inflammation. This study offers evidence of how factors vital to psychological health, such as getting sleep that is adequate in both duration and quality is associated with inflammation. Thus showing a clear link between psychological and physiological health. Therefore, it is fundamental value that mental health services are sufficiently funded and available to access. This aligns with the NHS Long Term Plan that has identified a need for mental health services to better integrate both mental and physical health care (NHS Long Term Plan, 2019).

#### Reducing Stigmatisation and Evidence for a Biopsychosocial Approach to Health

These findings challenge the stance that mental health problems or psychological distress are inherent or 'located within' an individual, which may contribute to reducing the stigma attached to mental health problems attached to the person. They highlight the need for mental health difficulties to be regarded less like an 'illness' within society and for policy makers to take into consideration that psychological distress is characterised by a complex interplay between biological, social and psychological factors. As such providing evidence of the value in taking a biopsychosocial approach (Engel, 1977). Furthermore, these findings contribute to the evidence base of such theories that psychological distress occurs on a continuum, these findings lend support to the dimensional view of mental health that posits that people can experience psychological distress at varying degrees, which are related to social factors such as income and education disparities (British Psychological Society, 2017; Speed & Taggart, 2019).

# Services and Policy

As well as disseminating research findings to academic audiences, it is intended that the clinical implications of the current study will be disseminated to services that provide support for service users impacted by psychological distress. In terms of influencing general policies, the Policy Team of the British Psychological Society (BPS) might be contacted. The findings may reveal important clinical implications in highlighting the impact of the wider socio-political context on mental health. The findings help further understand and inform initiatives aimed at reducing inequalities in social position and informing targeted psychological interventions aimed at managing psychological distress.

### **Directions for Future Research**

Future studies may want to explore the potential associations between sleep and psychological distress. The current study design was cross sectional, therefore a recommendation for future research is to explore the relationship of these variables over a period of time, such a longitudinal analysis would afford the opportunity to examine changes to rates of inflammation over time. In addition, future research should examine the potential bidirectional relationships between measures of socioeconomic position and inflammatory biomarkers. Lastly, future research may benefit from exploring interactions between variables relevant to this area, such as examining whether demographical factors interact, for example, exploring the interactions between gender and socioeconomic position and distress.

## Self-Reflexivity

The following section is a reflective account outlining both my learning and my positioning in regards to this thesis using self-reflexivity. Self-reflexivity refers to the process of a researcher reflecting on their own research in terms of their own assumptions, biases, positioning and life experiences. An integral part of this is then considering how these might influence the research that is being undertaken (Potter & Hepburn, 2012). In terms of a reflexive position to my research, I am considering these reflections from what Burnham (1992) referred to as 'Approach', which denotes the manner in which a therapist or a researcher positions themselves within their work.

Given my current role as Trainee Clinical Psychologist, I am at present completing my final year specialist placement working within a psychotherapy team. As part of this role, I complete long-term psychodynamic psychotherapy with adults experiencing severe psychological distress and many of whom have attracted diagnoses such as personality disorder. A cornerstone of psychodynamic therapy is fostering a warm therapeutic relationship with the client through sitting alongside in a therapeutic manner with the view to help alleviate their psychological distress.

From my point of view as the therapist, the therapeutic process relies on me getting to know the client exceptionally well and taking notice of how they are making me feel and the feelings I might elicit in them. This work involves building an understanding, jointly with the client, over the course of several months (or years) with the intention to understand and make sense of their distress. In this way, psychodynamic psychotherapy is largely exploring individual experience at a phenomenological level, in terms of the person's own life history, their perceptions of self and others and the meanings they come to derive (Malan, 2003). Therefore, completing secondary data analysis of a large data set of over 8,000 people reflects a big contrast to this work and my own positioning as a therapist. With such a large data set, I think there is a reductive aspect to the idiosyncratic experience of the individuals taking part in the study, as such, the participants within the sample may appear as a list of numbers and figures within a data file. It has led me to reflect that a phenomenological perspective of these personal experiences (e.g. on the experience of distress or the experience of having a low income) is missing due to the nature of a quantitative research design.

Notwithstanding these considerations, I believe the choice to undertake a secondary data analysis afforded the opportunity to examine a range of variables I was interested in exploring, these variables included socioeconomic position, inflammation, psychological distress and sleep duration and quality. It allowed me to create a broad understanding of these associations within the general population, which may have greater generalisability and a better application of the findings given the size of the data set. It also afforded me the opportunity to work with a very large data set that I would otherwise not have access to due to practicalities of recruitment and financial reasons.

At first, when contemplating my thesis interests, I wanted to focus on a particular diagnostic category such as depression or schizophrenia. However, on reflection during my doctoral studies, I have taken into consideration that the phenomenon of psychological distress is substantially more than what can be described within a single diagnostic category or more than a characterisation limited to a discrete entity (Bentall, 2003, 2010). Accordingly, having spent an extensive period of time during this research considering psychological wellbeing as being related to socioeconomic position, I have noticed how this experience has now altered my perception of referrals to mental health services, which regularly convey a client in an individualistic position. Whilst such a position I believe is useful, I have developed an appreciation of the importance of holding a multiple lens perspective in terms of the factors related to understanding psychological distress and ultimately how wellbeing is to be improved. Furthermore, through completing this research, it has indicated the alternatives to a diagnostic conceptualisation of psychological distress. Particularly in how such experiences may be better characterised by taking into consideration biological, psychological and social factors. Included in such an approach, is the vital contextualisation of the person's distress within the wider familial, social and political systems in which they live. Frequently, I have noticed that factors relating to the person's life, be they from the present or past experiences, were relevant in making sense of their distress.

I have developed an understanding of the power imbalances that exist within society, particularly in social circumstances and the stressors that are a part of daily life. Together with taking into consideration how these factors can affect our psychological safety and wellbeing. On reflection, I think this is what led me to develop an interest in this area of research exploring socioeconomic circumstances, together with having an appreciation of the fundamental value of examining both the physical and psychological health impacts of these. I realise in writing this that making such a binary distinction is already separating the two areas as discrete entities. Through several years of clinical work, I have noticed that both physical and mental health are invariable intertwined, with one having the capacity to affect the other.

I am aware of my own socioeconomic position, as a Trainee Clinical Psychologist, I am in a relatively privileged position in terms of my own educational attainment and occupational income. It has made me more conscious of the language and the way in which I write about these concepts.

Language is one of the main forms through which we communicate. I have come to gain a deepened appreciation of how the language we use can influence the meaning we make of ourselves, others and our life experiences. I have found this particularly important when considering the terms I would use to describe groups within my research. As well as consulting language guidelines such as those from the American Psychological Association (APA) Publication Manual. It became important to endeavour to write in a strengths-based way that reduced bias and avoided a deficit-based language style which was based on what people do not have. However, although I have been very conscientious of this, I realise that it is with great difficulty to completely avoid language that may have negative connotations attached to it.

I have also reflected on the ways in which the findings of this study might be implemented, in line with what has been discussed already, I believe there is value in systemic change being undertaken such as welfare support from the state or targeted psychological interventions regarding alleviating psychological distress or improving sleep. I have also reflected on the other ways in which the findings might be utilised, particularly applications that might not align with my own attitudes and values. One of the main findings of the research demonstrates that high rates of inflammation are more prevalent in people who have lower educational attainment and low household income compared to people with high education and high household income. I have considered that such a finding as this could possibly be used in a way that a dominant single narrative is upheld, such as locating the origins of health as exclusively intrinsic, which could lead to the implementation of an exclusively biomedical intervention to these people in the absence of other systemic changes.

Through completing this thesis, I have come to develop a greater appreciation that research is an iterative process, rather than being linear in nature. I have come to gain an understanding that it is an ever-changing process. I recall first thinking about embarking on this research and those initial stages of considering key research questions and I have seen how my ideas have changed and developed over this time. I have noticed that I have needed to frequently make adaptations and consider how to overcome challenges at different stages of the research. Overall, I place a lot of value on what I have learnt throughout conducting this research.

#### Conclusion

Lower educational attainment and household income were both associated with raised concentrations of both CRP and fibrinogen. Higher rates of psychological distress were associated with lower income but were not apparent for educational attainment. Although, psychological distress did not meet the prerequisite conditions to be a possible mediator. There was a significant association between lower educational attainment with sleep duration, although these were in directions not hypothesised. Having no qualifications was associated with a greater likelihood of falling into the '9 hour or more' sleep category. Similarly, being in the lowest income tertile was associated with 'very good sleep', compared to the highest income tertile. Sleep quality was a significant mediator in the association between both

educational attainment and household income and CRP. However, it is important to consider that the evidence of mediation could constitute a type I error.

The overall findings demonstrate that socioeconomic disadvantage is associated with increased levels of CRP and fibrinogen. Furthermore, this finding contributes to delineating the underlying mechanisms linking socioeconomic position and inflammation. Sleep and psychological distress are both amenable to change, therefore may be a potential point of intervention to alleviate health inequalities, although additional research is needed to explore this further. Furthermore, at a population level these findings indicate the importance of welfare support to help attenuate inequalities relating to both physical and mental health.

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## Appendix A

#### General Health Questionnaire (GHQ-12; Goldberg & Goodyear, 2005)

In terms of scoring, for negatively phrased responses the items on the scale are: 1 = Not at all, 2 = No more than usual, 3 = Rather more than usual, 4 = Much more than usual. For positively phrased responses the items on the scale were: 1 = More than usual, 2 = Same as usual, 3 = Less than usual, 4 = Much less than usual.

According to the scoring method of GHQ items, responses of 3 = More than usual and 4 = Much more than usual (for negatively phrased items) and 3 = Less so than usual and 4 = Much less than usual (for positively phrased items) indicated psychological distress, with higher values representing higher psychological distress.

The caseness scoring was used to measure psychological distress. In accordance with the GHQ-12, a cut off score of  $\geq$  3 is used as a demarcation indicating at least moderate psychological distress (Goldberg et al., 1997).

#### Short General Health Questionnaire (GHQ 12)

Have you recently?

1.	Been able to concentrate on what you're doing?	Better than usual	Same as usual	Less than usual	Much less than usual
2.	Lost much sleep over worry?	Not at all	No more than usual	Rather more than usual	Much more than usual
3.	Felt you were playing a useful part in things?	More so than usual	Same as usual	Less useful than usual	Much less useful
4.	Felt capable of making decisions about things?	More so than usual	Same as usual	Less so than usual	Much less capable
5.	Felt constantly under strain?	Not at all	No more than usual	Rather more than usual	Much more than usual
6.	Felt you couldn't overcome your difficulties?	Not at all	No more than usual	Rather more than usual	Much more than usual
7.	Been able to enjoy your normal day-to-day activities?	More so than usual	Same as usual	Less so than usual	Much less than usual
8.	Been able to face up to your problems?	More so than usual	Same as usual	Less so than usual	Much less able
9.	Been feeling unhappy and depressed?	Not at all	No more than usual	Rather more than usual	Much more than usual
10	. Been losing confidence in yourself?	Not at all	No more than usual	Rather more than usual	Much more than usual
11	. Been thinking of yourself as a worthless person?	Not at all	No more than usual	Rather more than usual	Much more than usual
12	. Been feeling reasonably happy, all things considered	More so than usual	About same as usual	Less so than usual	Much less than usual;

## **Appendix B**

## The Pittsburgh Sleep Quality Index (PSQI; Buysse et al. 1989)

Instructions: The following questions relate to your usual sleep habits during the past month

only. Your answers should indicate the most accurate reply for the majority of days and

nights in the past month. Please answer all questions. During the past month,

1. When have you usually gone to bed?

2. How long (in minutes) has it taken you to fall asleep each night?

3. When have you usually gotten up in the morning?

4. How many hours of actual sleep do you get at night? (This may be different than the

number of hours you spend in bed) \_\_\_\_\_

5. During the past month, how often have you Not during Less than Once or Three or had

trouble sleeping because you... the past once a twice a more times

month (0) week (1) week (2) week (3)

a. Cannot get to sleep within 30 minutes

b. Wake up in the middle of the night or early morning

- c. Have to get up to use the bathroom
- d. Cannot breathe comfortably
- e. Cough or snore loudly
- f. Feel too cold
- g. Feel too hot
- h. Have bad dreams
- i. Have pain

j. Other reason(s), please describe, including how often you

have had trouble sleeping because of this reason(s):

6. During the past month, how often have you taken medicine

(prescribed or "over the counter") to help you sleep?

7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?8. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?Very Fairly Fairly Very

good (0) good (1) bad (2) bad (3)

9. During the past month, how would you rate your sleep

quality overall?

# Appendix C

## Analytical cross sectional studies Critical Appraisal Tool

Answers: Yes, No, Unclear or Not/Applicable

1. Were the criteria for inclusion in the sample clearly defined?

The authors should provide clear inclusion and exclusion criteria that they developed prior to recruitment of the study participants. The inclusion/exclusion criteria should be specified (e.g., risk, stage of disease progression) with sufficient detail and all the necessary information critical to the study.

2. Were the study subjects and the setting described in detail?

The study sample should be described in sufficient detail so that other researchers can determine if it is comparable to the population of interest to them. The authors should provide a clear description of the population from which the study participants were selected or recruited, including demographics, location, and time period.

3. Was the exposure measured in a valid and reliable way?

The study should clearly describe the method of measurement of exposure. Assessing validity requires that a 'gold standard' is available to which the measure can be compared. The validity of exposure measurement usually relates to whether a current measure is appropriate or whether a measure of past exposure is needed.

Reliability refers to the processes included in an epidemiological study to check repeatability of measurements of the exposures. These usually include intra-observer reliability and inter-observer reliability.

4. Were objective, standard criteria used for measurement of the condition?

It is useful to determine if patients were included in the study based on either a specified diagnosis or definition. This is more likely to decrease the risk of bias. Characteristics are another useful approach to matching groups, and studies that did not use specified diagnostic methods or definitions should provide evidence on matching by key characteristics

5. Were confounding factors identified?

Confounding has occurred where the estimated intervention exposure effect is biased by the presence of some difference between the comparison groups (apart from the exposure investigated/of interest). Typical confounders include baseline characteristics, prognostic factors, or concomitant exposures (e.g. smoking). A confounder is a difference between the comparison groups and it influences the direction of the study results. A high quality study at the level of cohort design will identify the potential confounders and measure them (where possible). This is difficult for studies where behavioural, attitudinal or lifestyle factors may impact on the results.

6. Were strategies to deal with confounding factors stated?

Strategies to deal with effects of confounding factors may be dealt within the study design or in data analysis. By matching or stratifying sampling of participants, effects of confounding factors can be adjusted for. When dealing with adjustment in data analysis, assess the statistics used in the study. Most will be some form of multivariate regression analysis to account for the confounding factors measured.

7. Were the outcomes measured in a valid and reliable way?

Read the methods section of the paper. If for e.g. lung cancer is assessed based on existing definitions or diagnostic criteria, then the answer to this question is likely to be yes. If lung cancer is assessed using observer reported, or self-reported scales, the risk of over- or under-reporting is increased, and objectivity is compromised. Importantly, determine if the measurement tools used were validated instruments as this has a significant impact on outcome assessment validity.

Having established the objectivity of the outcome measurement (e.g. lung cancer) instrument, it's important to establish how the measurement was conducted. Were those involved in collecting data trained or educated in the use of the instrument/s? (e.g. radiographers). If there was more than one data collector, were they similar in terms of level of education, clinical or research experience, or level of responsibility in the piece of research being appraised?

8. Was appropriate statistical analysis used?

As with any consideration of statistical analysis, consideration should be given to whether there was a more appropriate alternate statistical method that could have been used. The methods section should be detailed enough for reviewers to identify which analytical techniques were used (in particular, regression or stratification) and how specific confounders were measured.

## Appendix D

Publication	Measures of socioeconomic position	Markers of inflammation	ES (95% CI)
Appleton et al. (2012)	Household income, education	CRP	0.12 (0.03, 0.22)
Boylan and Ryff (2013)	Education	CRP IL-6	0.04 (-0.06, 0.14) 0.18 (0.07, 0.30)
Camelo et al. (2014)	Education, household income	CRP	0.12 (0.04, 0.23)
Chen et al. (2015)	Household income	CRP	0.29 (0.26, 0.33)
Clark et al. (2012)	Household income	CRP	0.05 (-0.02,0.00)
Davillas et al. (2017)	Education and household income	CRP Fibrinogen	0.32 (0.18, 0.46) 0.22 (0.08, 0.36)
Dowd et al. (2007)	Education	CRP	-0.02 (0.28, 0.34)
Gallo et al. (2012)	Education, household income	CRP IL-6,	0.21 (0.10, 0.33)
Hostinar et al. (2015)	Occupation	CRP	0.04 (-0.06, 0.14)
Ishizaki et al. (2001)	Income	Fibrinogen	0.12 (0.09, 0.16)
Janicki-Deverts et al. (2008)	Household income and education	CRP	0.21 (0.10, 0.33)
Jousilahti et al. (2003)	Education, household income	CRP	0.14 (0.05, 0.24)
Koster et al. (2006)	Education, household income	CRP	0.18 (0.11, 0.25)
Loucks et al. (2006)	Education	CRP	0.11 (0.07, 0.14)
McDade et al. (2006)	Education	CRP	0.21 (0.06, 0.37)

# Effect sizes and confidence intervals from studies examining the association between socioeconomic position and the biomarkers of inflammation.

Publication	Measure of socioeconomic position	Markers of inflammation	ES (95% CI)
Muenning et al. (2007	NR	CRP	0.05 (0.03, 0.07)
Mwendwa et al. (2013)	Education, income	CRP	0.12 (-0.02, 0.26)
Owen et al. (2003)	Education, income	CRP	0.14 (0.08, 0.21)
Paul et al. (2008)	Household income	CRP	0.49 (0.35, 0.62)
Steptoe et al. (2003)	Occupation	Fibrinogen	0.07 (-0.15, 0.28)
Thomas et al. (2005)	Free school meal eligibility, indices for deprivation	CRP	0.17 (-0.06, 0.41)
Zhang et al. (2008)	Educational attainment, household income	CRP	0.07 (-0.02, 0.16)