Chapter 5

Coronary Heart Disease

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1 Introduction

Social determinants of health are factors that are primarily responsible for health inequities. This chapter discusses social determinants of health from the perspectives of socioeconomic status (SES) and coronary heart disease (CHD). We are particularly concerned with the pathways through which socioeconomic inequalities are translated into CHD and the strategy for preventing CHD.

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K. Kondo (ed.), *Social Determinants of Health in Non-communicable Diseases*,
Springer Series on Epidemiology and Public Health,
https://doi.org/10.1007/978-981-15-1831-7_5

2 Overview of SES and Coronary Heart Disease

CHD is a broad disease category and consists of several conditions, the most prevalent being myocardial infarction and angina pectoris. CHD affects the vascular system supplying the heart muscle and is attributed to build-up of atheromatous plaques that cover the lining of the coronary arteries. CHD is the leading cause of death over the long term in highly developed and industrialized Western countries, and many studies have been conducted on its causes and prevention. In Japan, CHD is the second-leading cause of death behind malignant neoplasm and has accounted for many cases of heart disease in recent years. In addition, acute myocardial infarction was one of four diseases targeted by regional medical care plans of Japan in fiscal year 2008. Risk factors for CHD are shown in Table 5.1. In addition to these risk factors, SES is also an important factor in CHD.

SES comprises factors such as income, education, and occupation (which includes being employed or unemployed and position at work). Low SES is associated with large increases in CHD risk in high-income countries [1]. For example, in countries like the USA and the UK, which are presumably at a more advanced stage of the cardiovascular disease (CVD) epidemic, there is an inverse relationship between SES and CVD mortality rates [2]. As the prevalence of CHD increases in industrialized and developing countries, it affects the more affluent classes initially and then percolates through the social classes [2]. Although the data are somewhat old, in developing countries like India and Hong Kong, this phenomenon is at the growing stage, and there is a higher CVD prevalence associated mainly with the high-income classes [2]. In Japan, whose population does not have distinctly large economic differentials, the “CHD epidemic percolation” postulation is less evident [2]. In a study on civil servants in Toyama Prefecture, no direct association between CHD and SES was observed [3]. In addition, in a Jichi Medical School Study, educational background was shown to be unrelated to CHD [4]. In Japan, with a

Table 5.1 Risk factors for coronary heart disease

• Hypertension
• Hyperlipidemia
• Diabetes
• Smoking
• Excessive alcohol intake
• Being physically inactive
• Unhealthy diet
• Being overweight or obese (accumulation of visceral fat)
• Excessive stress
• Aging
• Being male (for women, post-menopause)
• Genetics

continuous and a marked rise in total cholesterol, in sharp contrast to a constant fall in total cholesterol in other developed countries, age-adjusted CHD mortality declined between 1980 and 2008 [5]. Although there may be some protective factors unique to Japan, it is better to describe the relationship between SES and CHD.

CHD is caused by atherosclerosis through luminal narrowing or precipitating thrombi in the coronary artery walls that obstruct blood flow to the heart. Atherosclerosis is a lipoprotein-driven disease that leads to plaque formation at specific sites of the arterial tree through intimal inflammation, necrosis, fibrosis, and calcification. Although most plaques remain asymptomatic (subclinical disease), some become obstructive (stable angina), and others elicit acute thrombosis and may lead to an acute coronary syndrome. Rupture of thin-cap fibroatheroma and subsequent thrombosis may occur spontaneously, but, in some cases, a temporary increase in emotional or physical stress provides the final trigger for the event [6].

Although a direct association between SES and CHD has not been demonstrated in Japan [7], associations between some risk factors for CHD and some aspects of SES were reported in the Jichi Medical School Study. Namely, the study found that male white-collar workers have lower plasma fibrinogen concentrations than male blue-collar workers [8]. Many foreign studies have demonstrated not only a direct association between CHD and SES but also an association between CHD risk factors and SES; in particular, these studies have demonstrated that hypertension, hyperlipidemia, and diabetes mellitus are frequently observed among low-SES classes [9]. Lower SES is also reported to be associated with higher blood pressure, higher waist circumference, higher triglyceride levels in blood, and lower concentration of high-density lipoprotein cholesterol [10].

Thus, SES is considered to affect CHD under certain conditions. One reason for the unclear association between the two in Japan may be the lack of factors related to low-income and unemployed individuals in surveys [7]. Although the prevention of lifestyle-related diseases at the individual level is already one of the initiatives of Health Japan 21, prevention based on social aspects remains almost completely unexplored. Therefore, our objective in this chapter is to demonstrate hints for efficiently preventing CHD based on social aspects. We also discuss the mechanism of the link between SES and CHD as well as the possibility of preventing CHD based on aspects of SES. Using PubMed for literature searches, we referred to Western studies in which the association between CHD and SES was relatively clear. We searched for literature using the terms “{(cardiovascular heart disease) or (coronary heart disease) or (ischemic heart disease)} and {(socioeconomic) or (socio-economic)}.” We referred to reviews for general outlines and to original publications for individual research results.

3 Pathway of the Effect of SES on CHD

This chapter concerns the pathways through which SES is translated into CHD, highlighting the likely role of psychosocial processes. Figure 5.1 shows the possible pathways.

3.1 Health Behaviors and Access to Health Promotion Resources and Medical Care

Studies in the UK and Sweden indicated that low-SES classes are generally characterized by undesirable health behaviors, such as a high prevalence of smoking, unhealthy diet, obesity, and lack of physical activity [11, 12]. Low-SES classes also seldom use health-promotion resources such as health information and health checkups¹³ and tend to have an insufficient primary care [14]. Low-SES classes tend to delay hospitalization following cardiovascular events after CHD onset [14]. These behaviors are easily linked to exacerbation of symptoms and death. Thus, among low-SES classes, the multitude of risk factors and insufficient access to medical care and health resources are considered to have negative effects on the onset and prognosis of CHD.

Fig. 5.1 Pathways of the effect of socioeconomic status on coronary heart disease

3.2 *Psychosocial and Biological Pathways*

Psychosocial factors related to SES can influence CHD risk via stimulation of neuroendocrine, autonomic, and immune processes. Psychosocial characteristics observed in low-SES individuals include chronic stress, large numbers of life events, depression, anger, hostility, and social isolation; these characteristics are risk factors for CHD through this psychosocial pathway [15]. Conversely, in high-SES individuals, the effect of stress is alleviated by greater senses of control, mastery, and perceived control [15].

The stress reaction is mainly regulated by an axial system consisting of two neuroendocrine systems: the hypothalamic–pituitary–adrenocortical (HPA) system and the sympathetic adrenomedullary (SAM) system. In response to physiological and psychogenic stressors, the HPA axis orchestrates the systemic release of glucocorticoids while the SAM system operates through adrenaline (Adr) and noradrenalin (NA) signals. Although these substances possess an anti-stress effect, chronically increased secretion causes damage to organisms. Prolonged high levels of cortisol (Cort), a representative glucocorticoid, result in the onset of diabetes mellitus because of its gluconeogenic effect. Chronically high levels of Cort increase visceral fat, thereby increasing blood pressure [16]. Cort also increases oxidative stress [17], thereby increasing the risk of CHD. Cort has an anti-inflammatory effect, which normally works to prevent CHD. However, because chronically high levels of Cort result in low resistance, Cort ceases to exert a sufficient effect on target organs. This may, in turn, stimulate the immune system and cause inflammation [18]. Enhancement of the SAM system, on the other hand, causes Adr and NA to increase blood pressure, blood glucose, and inflammatory response [19]. Therefore, prolonged, chronically high levels of Adr and NA increase the risk of CHD.

We now discuss evidence for how differences in psychosocial characteristics caused by SES affect CHD. We discuss the psychological aspects, first followed by social aspects.

3.2.1 **Depression**

Prospective cohort studies provide strong evidence that depression is an independent etiological and prognostic factor for CHD [20]. The high prevalence of depression in patients with CHD supports a strategy of screening for depressive symptoms in CHD patients [21].

Dysregulation of the HPA axis function is frequent in major depression, and hypercortisolemia can be a mediating factor in the relationship between depression and CHD development [22]. In addition, patients with major depression have been found to exhibit increased biomarkers of inflammation in both the periphery and the brain [18]. Patients with major depression exhibit higher plasma interleukin-6 levels following exposure to a psychological stressor than nondepressed, healthy control participants [18], thus explaining depressed individuals' susceptibility to CHD onset.

Inflammatory cytokine levels are also increased in individuals with metabolic syndrome [23]. Therefore, inflammatory cytokines may have a synergistic effect on CHD. Furthermore, depression has been associated with heightened blood pressure and NA to behavioral tasks [24, 25]. This result suggests that the SAM system is also a factor contributing to CHD.

3.2.2 Hostility and Anger

Well-established associations have been documented between elevated levels of hostility/anger and CHD. Hostility increases blood pressure, catecholamines, platelet activation, and daily Cort secretion [26, 27]. Thus, both HPA and SAM systems are pathways for exacerbation of CHD. In addition, anger has been reported to promote oxidation in the body [28], indicating that oxidation may be associated with the pathway through which anger affects CHD.

3.2.3 Work Environment

The Job Demand Control (JDC) model is a well-known theoretical perspective regarding workload and work-related stress [29]. The JDC model emphasizes job demand and job control (decision latitude) with four working conditions: high-strain jobs (high demands and low controls), low-strain jobs (low demands and high controls), active jobs (high demands and high controls), and passive jobs (low demands and low controls). According to this model, individuals in lower positions experience higher strain [29].

The high strain group is reported to possess characteristics associated with CHD risk factors, such as enhancement of the SAM system [30] and higher levels of Cort in the blood [31]. Although many reports have demonstrated a relationship between high strain and blood pressure, there is also counterevidence that hypertension is not the link between high strain and CHD [32]. Therefore, there is some uncertainty regarding the mechanism of the link between high strain and CHD.

In mental stress-testing studies, which demonstrate the effect of a sense of control, participants demonstrate a positive relationship between strain and systolic blood pressure when exposed to uncontrollable stress; however, no such association is observed for controllable stress [33]. These findings demonstrate that uncontrollable stress exerts a more harmful effect on CHD, even when the nature of the stress is identical.

3.2.4 Social Support

Social support is a factor that can exert a beneficial effect on CHD. Support in the workplace reduces the risk of CHD by decreasing heart rate during sleep, work, and leisure [15]. In a three-factor model in which “support” is added to the (two-factor) JDC model, CHD prevalence and mortality are high among the

high-demand/low-control/low-support group [23]. Social support is also a buffer factor against stress and depressive mood outside the workplace [34]. Therefore, social support should indirectly reduce CHD risk factors.

3.2.5 Summary of Psychosocial Pathway

Psychosocial factors can affect CHD through the effects of SES, as described above. As one's chronic psychosocial state affects responses to acute stress, different responses to the same acute stress are observed for different SES. For example, in acute stress tests, participants with low SES exhibit larger responses in terms of biological indicators (blood pressure, heart rate, etc.) than do participants with high SES [15].

3.3 *Parents and Childhood*

Some studies have indicated that one's parents' and one's own SES during childhood will affect CHD in the future. For example, Swedish and American epidemiological studies demonstrated an association between childhood SES and CHD in late middle age and onward [35, 36]. On the contrary, this association has been denied in Finnish and British studies [37, 38]. A separate British epidemiological study concluded that while SES has a major direct effect on CHD beginning in adulthood, childhood social environment affects CHD through employment and social status [39].

Thus, although the relationship between CHD and childhood SES appears not to be strong, childhood experiences can influence adulthood behavioral and psychosocial factors (smoking, lack of exercise, hostility, occupational strain, unhealthy mental state, etc.), thereby becoming a factor for CHD [1]. In addition, infants with low birth weight are reported to be predisposed to CHD because of the effect of birth weight on subsequent biological indicators such as a blood coagulation system, cholesterol levels, blood pressure, insulin resistance, and abnormal glucose tolerance [40]. Considering that mothers with low SES experience higher rates of babies with low birth weight, there may be a pathway through which birth weight is indirectly associated with CHD risk. Furthermore, stressful environments during pregnancy and in childhood diminish the functioning of the HPA and SAM systems, which may result in a subsequent predisposition for CHD [41].

3.4 *Genetic Factors*

CHD has a strong hereditary component. MCP-1, MCP-4, MIP-1, RANTES, and other genes associated with atherosclerosis have been identified as risk factors [42]. Although there has been no verification in relation to accumulation of higher-risk

genes according to the social gradient, and, thus, genetic involvement in the association between SES and CHD is unknown, it is possible that exposure to exacerbating factors for low SES is involved in gene expression or epigenetic processes.

4 Hints on CHD Prevention

Socioeconomic inequalities in CHD occur in most Western countries and are of major concern to public health authorities. This may become a crucial problem in Japan as well. The World Health Organization cites “social and economic environment,” “physical environment,” and “individual characteristics and behaviors” as determinants of health. Specific examples are shown below [43].

- Income and social status: Higher income and social status are linked to better health. The magnitude of the gap between the richest and poorest people tends to reflect the differences in health.
- Education: Low education levels are linked with poor health, more stress, and lower self-confidence.
- Physical environment: Safe water and clean air; healthy workplaces; and safe houses, communities, and roads all contribute to good health.
- Employment and working conditions: People who are employed are healthier, particularly those who have more control over their working conditions.
- Social support networks: Greater support from families, friends, and communities are linked to better health.
- Culture: Customs and traditions and the beliefs of the family and community all affect health.
- Genetics: Inheritance plays a part in determining lifespan, healthiness, and the likelihood of developing certain illnesses.
- Personal behavior and coping skills: Balanced eating, keeping active, smoking, drinking, and how we deal with life’s stresses and challenges all affect health.
- Health services: Access to and use of services that prevent and treat disease influence health.
- Gender: Men and women experience different types of diseases at different ages.

These determinants of health can be applied in the prevention of CHD and the improvement of health [9].

Many of the above items are associated with social and economic conditions. This suggests that we should introduce CHD-prevention strategies that have seldom been conducted in Japan, focusing on socioeconomic factors.

4.1 Intervention Through Education

Educational programs for the prevention of CHD have been carried out in the USA, Finland, Australia, Switzerland, South Africa, and Germany. These educational programs, which aim to reduce CHD risk factors, included local publicity campaigns, the establishment of designated smoking areas, and the use of mass media; these various programs have all generally achieved favorable results [9]. Although factors such as the spread of statin-based medicine and the improvement of social supports through intervention may be effective, broad education can also improve SES (in which education is a crucial factor).

4.2 Social Security Systems

Material wealth is strongly related to CHD throughout life, while poverty has harmful effects not only on CHD but on overall health. Thus, social security systems take on major significance. In fact, societies with well-developed social security systems are considered to be healthier than market-oriented ones. Specifically, countries in the former group (Austria, Sweden, Norway, Denmark, and Finland) boast lower infant mortality rates and lower percentages of poor children than countries in the latter group (Belgium, Germany, the Netherlands, France, Italy, Switzerland, the UK, Ireland, the USA, and Canada) [44]. This finding demonstrates that given country health policies affect health and disease, including CHD. Therefore, such policies are an important factor in disease prevention. Considering that childhood SES affects health in adulthood [45], the improvement of SES through the development of social security systems is a highly viable candidate for CHD prevention.

4.3 Support for Critical Periods in Life

Thirteen critical periods of the life course have been identified during which people are especially vulnerable to social disadvantage: fetal development; birth; nutrition, growth, and health in childhood educational career; leaving the parental home; entering labor market; establishing social and sexual relationships; job loss or insecurity; parenthood; episodes of illness; labor market exit; and chronic sickness [13]. During such adverse times, adequate support must be provided to maintain health and prevent illness. In addition to providing support on an individual level, providing support to the whole society may also be effective.

5 Summary

In developed Western countries, CHD is generally prevalent among low-SES classes. As we have introduced, the mechanism of this association is affected by many factors, such as a life course from birth to old age, lifestyle habits, and psychosocial stress. In Japan, there are still few empirical studies on the association between SES and CHD; therefore, this relationship is not properly understood. Lifelong studies are necessary to devise strategies for improving SES and preventing CHD [46]. Socio-epidemiological studies will be needed to advance plans for evidence-based medical care in Japan.

References

1. Clark AM, DesMeules M, Luo W, Duncan AS, Wielgosz A. Socioeconomic status and cardiovascular disease: risks and implications for care. *Nat Rev Cardiol*. 2009;6:712–22.
2. Khor GL. Cardiovascular epidemiology in the Asia-Pacific region. *Asia Pac J Clin Nutr*. 2001;10:76–80.
3. Sekine M, Chandola T, Martikainen P, Marmot M, Kagamimori S. Socioeconomic inequalities in physical and mental functioning of British, Finnish, and Japanese civil servants: role of job demand, control, and work hours. *Soc Sci Med*. 2009;69:1417–25.
4. Honjo K, Tsutsumi A, Kayaba K, Jichi Medical School Cohort Study Group. Socioeconomic indicators and cardiovascular disease incidence among Japanese community residents: the Jichi Medical School Cohort Study. *Int J Behav Med*. 2010;17:58–66.
5. Sekikawa A, Miyamoto Y, Miura K, et al. Continuous decline in mortality from coronary heart disease in Japan despite a continuous and marked rise in total cholesterol: Japanese experience after the Seven Countries Study. *Int J Epidemiol*. 2015;44:1614–24.
6. Bentzon JF, Otsuka F, Virmani R, Falk E. Mechanisms of plaque formation and rupture. *Circ Res*. 2014;114:1852–66.
7. Kagamimori S, Gaina A, Nasermoaddeli A. Socioeconomic status and health in the Japanese population. *Soc Sci Med*. 2009;68:2152–60.
8. Hirokawa K, Tsutsumi A, Kayaba K, Jichi Medical School Cohort Group. Occupation and plasma fibrinogen in Japanese male and female workers: the Jichi Medical School Cohort study. *Soc Sci Med*. 2009;68:1091–7.
9. Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation*. 1993;88:1973–98.
10. Manuck SB, Phillips JE, Gianaros PJ, Flory JD, Muldoon MF. Subjective socioeconomic status and presence of the metabolic syndrome in midlife community volunteers. *Psychosom Med*. 2010;72:35–45.
11. Rosengren A, Orth-Gomér K, Wilhelmsen L. Socioeconomic differences in health indices, social networks and mortality among Swedish men. A study of men born in 1933. *Scand J Soc Med*. 1998;26:272–80.
12. Marmot MG, Smith GD, Stansfeld S, et al. Health inequalities among British civil servants: the Whitehall II study. *Lancet*. 1991;337:1387–93.
13. Raphael D. Social exclusion—including processes of material deprivation, lack of participation in common societal activities, and exclusion from decision-making and civic participation—is the means by which low income causes cardiovascular disease. In: Raphael D, editor. *Social justice is good for our hearts*. Toronto: CSJ Foundation for Research and Education; 2002. p. 21–34.

14. Morrison C, Woodward M, Leslie W, Tunstall-Pedoe H. Effect of socioeconomic group on incidence of, management of, and survival after myocardial infarction and coronary death: analysis of community coronary event register. *BMJ*. 1997;314:541–6.
15. Steptoe A, Marmot M. The role of psychobiological pathways in socio-economic inequalities in cardiovascular disease risk. *Eur Heart J*. 2002;23:13–25.
16. Dhabhar FS, McEwen BS. Bidirectional effects of stress and glucocorticoid hormones on immune function: possible explanations for paradoxical observations. In: Ader R, Felten DL, Cohen N, editors. *Psychoneuroimmunology*, vol. 2. 3rd ed. San Diego, CA: Academic; 2001. p. 301–29.
17. Yamaji M, Tsutamoto T, Kawahara C, et al. Serum cortisol as a useful predictor of cardiac events in patients with chronic heart failure: the impact of oxidative stress. *Circ Heart Fail*. 2009;2:608–15.
18. Pace TW, Miller AH. Cytokines and glucocorticoid receptor signaling. *Ann N Y Acad Sci*. 2009;1179:86–105.
19. Flierl MA, Rittirsch D, Nadeau BA, Chen AJ, Sarma JV, Zetoune FS, McGuire SR, List RP, Day DE, Hoesel LM, Gao H, Van Rooijen N, Huber-Lang MS, Neubig RR, Ward PA. Phagocyte-derived catecholamines enhance acute inflammatory injury. *Nature*. 2007;449:721–5.
20. Hemingway H, Marmot M. Evidence based cardiology: psychosocial factors in the aetiology and prognosis of coronary heart disease. Systematic review of prospective cohort studies. *BMI*. 1999;318:1460–7.
21. Lichtman JH, Bigger JT Jr, Blumenthal JA, et al. Depression and coronary heart disease: recommendations for screening, referral, and treatment: a science advisory from the American Heart Association Prevention Committee of the Council on Cardiovascular Nursing, Council on Clinical Cardiology, Council on Epidemiology and Prevention, and Interdisciplinary Council on Quality of Care and Outcomes Research: endorsed by the American Psychiatric Association. *Circulation*. 2008;118:1768–75.
22. Jokinen J, Nordström P. HPA axis hyperactivity and cardiovascular mortality in mood disorder inpatients. *J Affect Disord*. 2009;116:88–92.
23. Grippo AJ, Johnson AK. Stress, depression and cardiovascular dysregulation: a review of neurobiological mechanisms and the integration of research from preclinical disease models. *Stress*. 2009;12:1–21.
24. Light KC, Kothandapani RV, Allen MT. Enhanced cardiovascular and catecholamine responses in women with depressive symptoms. *Int J Psychophysiol*. 1998;28:157–66.
25. Kirschbaum C, Prussner JC, Stone AA, et al. Persistent high cortisol responses to repeated psychological stress in a subpopulation of healthy men. *Psychosom Med*. 1995;57:468–74.
26. Suarez EC, Kuhn CM, Schanberg SM, et al. Neuroendocrine, cardiovascular, and emotional responses of hostile men: the role of interpersonal challenge. *Psychosom Med*. 1998;60:78–88.
27. Shimbo D, Chaplin W, Kuruvilla S, Wasson LT, Abraham D, Burg MM. Hostility and platelet reactivity in individuals without a history of cardiovascular disease events. *Psychosom Med*. 2009;71:741–7.
28. Tsuboi H, Hamer M, Tanaka G, Takagi K, Kinae N, Steptoe A. Responses of ultra-weak chemiluminescence and secretory IgA in saliva to the induction of angry and depressive moods. *Brain Behav Immun*. 2008;22:209–14.
29. Karasek R, Baker D, Marxer F. Job decision latitude, job demands, and cardiovascular disease: a prospective study of Swedish men. *Am J Public Health*. 1981;71:694–705.
30. Collins SM, Karasek RA, Costas K. Job strain and autonomic indices of cardiovascular disease risk. *Am J Ind Med*. 2005;48:182–93.
31. Maina G, Bovenzi M, Palmas A, Larese Filon F. Associations between two job stress models and measures of salivary cortisol. *Int Arch Occup Environ Health*. 2009;82:1141–50.
32. Kivimäki M, Head J, Ferrie JE, Shipley MJ, Steptoe A, Vahtera J, Marmot MG. Hypertension is not the link between job strain and coronary heart disease in the Whitehall II study. *Am J Hypertens*. 2007;20:1146–53.

33. Steptoe A, Cropley M, Joeekes K. Job strain, blood pressure, and responsivity to uncontrollable stress. *J Hypertens*. 1999;17:193–200.
34. Veiel HO. Buffer effects and threshold effects: an alternative interpretation of nonlinearities in the relationship between social support, stress, and depression. *Am J Community Psychol*. 1987;15:717–40.
35. Vagero D, Leon D. Effect of social class in childhood and adulthood on adult mortality. *Lancet*. 1994;343:1224–5.
36. Kittleson MM, Meoni LA, Chu NY, Ford DE, Klag J. Association of childhood socio-economic status with subsequent coronary heart disease in physicians. *Arch Intern Med*. 2006;166:2356–61.
37. Lynch JW, Kaplan GA, Cohen RD, et al. Childhood and adult socioeconomic status as predictors of mortality in Finland. *Lancet*. 1994;343:524–7.
38. Wannamethee SG, Whincup PH, Shaper G, et al. Influence of fathers' social class on cardiovascular disease in middle-aged men. *Lancet*. 1996;348:1259–63.
39. Marmot M, Shipley M, Brunner E, Hemingway H. Relative contribution of early life and adult socioeconomic factors to adult morbidity in the Whitehall II study. *J Epidemiol Community Health*. 2001;55:301–7.
40. Forouhi N, Hall L, McKeigue P. A life course approaches to diabetes. In: Kuh D, Ben-Shlomo Y, editors. *A life course approach to chronic disease epidemiology*. Oxford: Oxford University Press; 2004. p. 165–88.
41. Latendresse G. The interaction between chronic stress and pregnancy: preterm birth from a biobehavioral perspective. *J Midwifery Womens Health*. 2009;54:8–17.
42. Sheikine Y, Hansson GK. Chemokines and atherosclerosis. *Ann Med*. 2004;36:98–118.
43. World Health Organization. The determinants of health. <http://www.who.int/hia/evidence/doh/en/index.html>. Accessed 4 July 2018.
44. Navarro V, Shi L. The political context of social inequalities and health. In: Navarro V, editor. *The political economy of social inequalities: consequences for health and quality of life*. Amityville, NY: Baywood; 2002. p. 403–18.
45. Poulton R, Caspi A, Milne BJ, et al. Association between children's experience of socioeconomic disadvantage and adult health: a life-course study. *Lancet*. 2002;360:1640–5.
46. Ben-Shlomo Y, Kuh D. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. *Int J Epidemiol*. 2002;31:285–93.

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