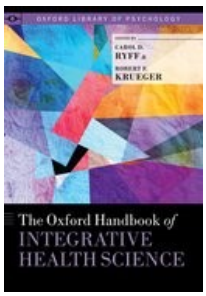

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CHAPTER

34 The Educational Gradient in Physiological Dysregulation: A Cross-Country Investigation

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Abstract

The chapter first reviews extant literature on educational gradients in physiological dysregulation. Prior studies suggested there is an inverse relationship between education and risk, although the association may be weaker at the oldest ages and stronger among whites than blacks; the educational gradient may differ by country; and sex differences in the educational gradient may depend on the context. The chapter then presents new comparative analyses of the relationship between physiological dysregulation and education based on data from five countries (United States, England, Russia, Costa Rica, and Taiwan). Large educational differences were found in dysregulation in Russia, US white men, US black women, and English white women. The finding that the educational differential among US women is larger for blacks than for whites appears to be sensitive to how one defines "high" education. Using race-specific cutoffs, the education gradient did not differ significantly between black and white women.

Keywords: education, physiological dysregulation, selective attrition hypothesis, whites, blacks, black women, white women

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Introduction

The relationship between socioeconomic status (SES) and health (that a higher position in the social hierarchy along one or more of multiple dimensions is associated with better health) is widely observed among modern higher income countries (Avendano et al., 2010; Dow & Rehkopf, 2010; National Research Council, 2011). This relationship generally is found whether it is based on income, education, wealth, or occupational status.

The literature on the association between SES and health, however, is not unambiguous. Some work has questioned the strength of the relationship in middle-income or more recently developed countries, and there is some evidence that even among higher income countries the relationship may not be universal (Hirokawa, Tsutusmi, & Kayaba, 2006; Wilkinson, 1996).

A number of explanations have been proposed for the variation in findings. Age at observation may affect the relationship. The effects of SES on health may increase with age if advantages accumulate over life, or they may decrease as biological frailty increases with age and survivors become more selected (Smith & Goldman, 2007; Zajacova, Goldman, & Rodriguez, 2009). In other words, if selective attrition is operating, we would expect the relationship to become attenuated with age, whereas if advantage accumulates over time, we would expect the relationship to become stronger. Social inequality may also be a factor. Some work—albeit contested—suggests that income inequality is associated with higher mortality (Ash & Robinson, 2009; De Vogli, Mistry, Gnesotto, & Cornia, 2005; Lynch et al., 1998; but see also Deaton & Lubotsky, 2009; Deaton & Paxson, 2004; Lynch, ↳ Smith, Harper, & Hillemeier, 2004; Lynch, Smith, Harper, Hillemeier, & Ross, 2004; Lynch, Smith, Kaplan, & House, 2000). Yet another explanation suggests that the position of a country in the epidemiological transition may be a factor (Weinstein, Glei, & Goldman, 2013). At early stages of the epidemiological transition, when infectious disease dominates chronic conditions, one might expect, for example, that better nutrition across all social classes would diminish the relationship between position in hierarchy and health; at later stages, obesity might become a more important factor. And, of course, variation in analytical techniques may result in the appearance of different associations.

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Over the past two decades, the widespread (and increasing) practice of collecting biological specimens as part of psychosocial and demographic surveys has provided the opportunity to better understand the pathways through which position in social hierarchies operates to affect health. Here, we focus on the relationship between one indicator of SES—education—and measures of physiological dysregulation (sometimes denoted "allostatic load"). We focus on education for several reasons. As noted by Zajacova, Montez, & Herd (2014, p. 974), there are multiple advantages to using education as the measure of SES: Education is determined earlier in life than income and occupation; it is available for adults of all ages and employment statuses; and it is less prone to reverse causation (i.e., health decline that causes reductions in income or wealth). Importantly for studies of aging, education is less likely to change in later adulthood than other measures (e.g., income or occupational status) of position in social hierarchies (Goldman, 2001).

Allostatic load is a concept introduced by McEwen and Stellar (1993) and is intended to capture the cumulative physiological costs of chronic stress: "We define allostatic load as the cost of chronic exposure to heightened neural or neuroendocrine response resulting from repeated or chronic environmental challenge that an individual reacts to as being particularly stressful" (McEwen & Stellar, 1993, p. 2093). Although originally formulated with the effects of stressful experience in mind, much of the literature on allostatic load has examined the relationship between some operationalization of the concept, typically a simple sum of out-of-range biological markers and position in hierarchy (see discussion that follows), where SES is assumed to be a surrogate indicator of stressful experience (Gruenewald et al., 2012;

Gustafsson, Janlert, Theorell, Westerlund, & Hammarstrom, 2011; Kubzansky, Kawachi, & Sparrow, 1999; T. Seeman et al., 2008; T. E. Seeman et al., 2004).

Especially in the context of this chapter, where we focus on biological dysregulation as a function of education, not measures of stress, we prefer the terms *physiological dysregulation* or *biological risk*. Measures of cumulative dysregulation (or allostatic load) typically incorporate biological indicators across multiple physiological systems; they are often operationalized as counts of indicators that are found to be out of "normal" operating range. Other formulations have made use of a continuous value for each biomarker by computing a *Z*-score measure of dysregulation (Seplaki, Goldman, Glei, & Weinstein, 2005); the results are generally similar regardless of the formulation (Glei, Goldman, Shkolnikov, Jdanov, Shkolnikova, et al., 2013; Glei, Goldman, Wu, & Weinstein, 2013). Given that allostatic load is theorized to capture cumulative risk, it is not unreasonable to expect that it would increase with age. However, it is important to recognize that some of its components do not necessarily increasingly deviate from what we think of as normal operating ranges with age; the relationship may be complex.

In this chapter, we first review the extant literature on educational gradients in physiological dysregulation. Our review of the literature follows the language (allostatic load or physiological dysregulation) used by the authors of the articles we summarize here. Our new analyses for this chapter (presented further in the chapter) use the term *physiological dysregulation*. We focus our review on studies that examined educational differences in a biological risk score or on inflammatory markers, particularly C-reactive protein (CRP), a measure of inflammation that is now recognized as an important clinical marker associated with elevated risk for major coronary events (Pearson et al., 2003). Second, we follow the discussion of prior work with a presentation of the new analyses of the relationship between physiological dysregulation and education. Our primary goal is to provide a consistent analytical approach across five countries controlling for age and stratifying by sex and, for US women, race. We focus on three outcomes for which we have data in all five countries: an overall measure of physiological dysregulation, a subscore that summarizes metabolic and cardiovascular (CV) risk, and a measure of inflammation: CRP. We use data from biosocial surveys in the United States, England, Russia, Costa Rica, and Taiwan and, to the extent possible, construct comparable
p. 481 measures in order to ↳ present analyses of the relationship between physiological dysregulation and education.

These countries represent five different regions of the world (North America, Latin America, Western Europe, Eastern Europe, and East Asia); with the exception of Russia, they share similar life expectancy: 78.9 years in the United States; 80.6 years for England/Wales; 78.7 years in Costa Rica; 78.8 years in Taiwan; and 68.9 years in Russia as of 2010 (University of California, Berkeley [USA] & Max Planck Institute for Demographic Research [Germany], 2016; World Bank, 2015). Yet, they differ in terms of the spread of universal education and the timing of increases in educational attainment, the nature of the healthcare system, and cultural norms that shape health behaviors, such as physical activity and smoking.

There also have been differences in the timing and pace of demographic/epidemiological transitions and in how economic change has evolved. For example, although Taiwan and Russia shared similar life expectancy in 1970 (68.7 for Taiwan vs. 68.8 for Russia), Taiwan gained 6.0 years in life expectancy between 1970 and 1994, whereas expectation of life *declined* by 5.0 years in Russia during the same period (University of California, Berkeley [USA] & Max Planck Institute for Demographic Research [Germany], 2016). The net gain in life expectancy between 1970 and 2014 was so much larger for Taiwan (+10.9 years) than for Russia (+2.1 years) that life expectancy of Taiwanese (79.6 years) in 2014 exceeded that of Russians (70.9) by nearly 9 years (University of California, Berkeley [USA] & Max Planck Institute for Demographic Research [Germany], 2016).

With respect to economic change, Russia has transitioned from a centrally planned to a market-based economy: The social, economic, and political changes that have ensued (e.g., erosion of social norms and

cohesion after the collapse of the former Soviet Union and the subsequent psychological stresses) have been implicated in the mortality reversal. Increased inequality within Russian society has created a polarization between a few "haves" with enormous wealth and a large population of "have nots" who are increasingly embittered and alienated (Field, 1995; Shkolnikov et al., 2004). In contrast, Taiwan's transformation from an agricultural to an industrial-oriented economy over recent decades has resulted in rapid economic growth.

Finally, we offer some thoughts about intercountry variation and directions for future research.

Prior Analyses of the Relationship Between Education and Biological Risk

We organize our review of the literature by looking first at analyses of a single country. Much—indeed most—of the research on educational gradients in biological risk has focused on a single country at a time; most of that research has been based on US samples. Thus, we start with the United States, where much of the research has focused, then review studies of other individual countries. Finally, we review multicountry analyses. Most—albeit not all—of these studies document an inverse relationship between risk and education.

One of the earliest studies of the effects of education on allostatic load was conducted by Kubzansky and colleagues (1999) using data from men who participated in the Normative Aging Study (United States; n = 818, aged 42–88). They looked at both education and hostility and reported that lower levels of education and higher levels of hostility were associated with higher allostatic load, but when both education and hostility were included in their model, the effects of education were attenuated.

Teresa Seeman was an early adapter of the allostatic load framework and has continued to explore its dimensions using multiple sources of data. Seeman and her colleagues (Arun Karlamangla, Tara Gruenewald, Peifung Hu, Eileen Crimmins, among others) have published a series of articles that examined the relationship between education and other indicators of SES and allostatic load (sometimes denoted "biological risk" in their analyses). In an article (T. Seeman et al., 2008) using NHANES data (n = 15,578, aged 20+) to explore biological risk profiles, she and her colleagues found significant inverse gradients in education and income with both individual markers of biological risk as well as summary scores across systems; consistent with an "age-as-leveler" hypothesis, they found that the relationships were attenuated with older age. National Health and Nutrition Examination Survey (NHANES) data (n = 2,879–5,215 depending on the marker; aged 25+ except for fibrinogen, which was based on ages 40+) were also used by Muennig, Sohler, and Mahato (2007) in an analysis of links between education and individual biomarkers; they found the expected relationships between education and high-density lipoprotein (HDL) cholesterol and CRP, although homocysteine, while inversely related to income, was not significantly related to education, and low-density lipoprotein (LDL) cholesterol and fibrinogen were also not related.

p. 482 Using data from the Midlife in the United States (MIDUS) 2 study (n = 1,008, aged 35–85), Gruenewald and colleagues (2012) also found large differences in allostatic load related to SES as measured by education, income, and financial difficulties. Consistent with these results, Karlamangla et al. (2005), in analyses of data from the Coronary Artery Risk Development in Young Adults (CARDIA: n = 4,149, aged 18–40) study, found that both the participant's and his or her parents' educational level and financial hardship were inversely associated with baseline risk among women and with change in risk among both men and women. Another article by T. E. Seeman et al. (2004) examined data from the MacArthur Studies of Successful Aging (n = 657, aged 70–79). While they found an inverse relationship between baseline allostatic load and educational attainment, it just missed being statistically discernible (p ~ .054).

Numerous studies have examined the relationship between education and individual markers of risk in various analyses of health outcomes and sources of data. For example, Albert, Glynn, Buring, and Ridker (2006), in an analysis of SES and CV events based on the Women's Health Study (*n* = 22,688 women, mean age = 54.1, standard deviation [SD] = 7.1), found that education was inversely associated with body mass index (BMI) and hypertension. Koster et al. (2005, 2006) found inverse links to triglycerides, interleukin 6 (IL-6), CRP, tumor necrosis factor alpha (TNF-α), and having a BMI below 25 (data from the Dynamics of Health, Aging, and Body Composition study: *n* = 3,066, aged 70–79). Friedman and Herd (2010) found inverse links to inflammation (MIDUS data: *n* = 704, aged 35–86). Based on data from the Multi-Ethnic Study of Atherosclerosis (MESA; *n* = 6,599, aged 45–84), Ranjit et al. (2007) found that education was inversely associated with inflammation (as measured by CRP and IL-6) among both whites and blacks, but not among Chinese or Hispanic Americans; Pollitt et al. (2008) found that low education was associated with higher levels of inflammation among white but not black participants in the study (*n* = 12,681, aged 45–64); and Gruenewald, Cohen, Matthews, Tracy, and Seeman (2009) found inverse educational gradients in CRP and IL-6 among whites (but not blacks) using data from the CARDIA study (*n* = 3,549, aged 37–55).

More recent analyses of US data revealed similar inverse associations between allostatic load, inflammation, and education. For example, Upchurch and her colleagues (2015) used longitudinal data from the Study of Women's Health Across the Nation (SWAN: *n* = 2,063, aged 42–52) to investigate links between SES and allostatic load. They concluded that, for both whites and blacks, lower education was associated with higher allostatic load. Merkin, Karlamangla, Roux, Shrager, and Seeman (2014) used different longitudinal data (MESA: *n* = 6,135, aged 45–84) to assess the relationship between SES and allostatic load over time. They reported that high adult education was associated with a significantly lower increase in allostatic load among participants who had lower baseline allostatic load; they found no association among those who had higher allostatic load at baseline. Brummett and her colleagues (2013) used data from the National Longitudinal Study of Adolescent to Adult Health (AddHealth: *n* = 11,371, aged 24–32) and focused on CRP in a study of mediators of the association between SES and CRP. Their results indicated a modest association between SES (including education) and CRP among whites, but not among blacks. As in the study by Gruenewald et al. (2009), Deverts, Cohen, Kalra, and Matthews (2012) used data from the CARDIA study (*n* = 2,658, aged 25–37) to assess longitudinally the association between SES and CRP. Their findings showed a significant inverse relationship over the course of 13 years. Finally, a recent article by Howard and Sparks (2015) used four waves of NHANES data (*n* = 6,990, aged 25 and above) to explore whether racial/ethnic differences in allostatic load are evident across all educational strata. They found that the race/ethnicity differential was not consistent across all levels of education: Levels of allostatic load did not differ among low-education individuals but were seen for individuals with at least a college degree.

To summarize, at least among white US residents, most—but not all—previous studies reported an inverse relationship between educational attainment and both summary measures of physiological dysregulation and markers of inflammation. Results for blacks were less consistent.

Fewer analyses are based on non-US data, and even fewer studies reported results from multiple countries. Comparisons across multiple countries are important because countries differ in many respects that are relevant for health and for the effects of education on health in particular. These differences include, for example, the timing and pace of demographic/epidemiological transitions; differences in the transition
p. 483 from agrarian-based to an industrialized/urbanized economy; and ↳ differences in the spread of universal education and the timing of increased educational attainment. For example, in Taiwan, rapid increases in educational attainment over time have created a generation gap (Thornton, Fricke, Lang, & Chang, 1994); in the early 1950s, only 10% of girls and 24% of boys aged 12–14 attended junior high school, but by 1985 the corresponding percentages had reached 90% for both sexes (Fricke, Chang, & Yang, 1994). There are also differences in the nature of the healthcare systems and differences in cultural norms that directly affect health and may be strongly related to education.

Several of these studies were conducted using data from Taiwan (Dowd & Goldman, 2006; Hu, Wagle, Goldman, Weinstein, & Seeman, 2007; Weinstein, Goldman, Hedley, Yu-Hsuan, & Seeman, 2003). These studies (*n* = 101, aged 67–94 for Weinstein et al., 2003; *n* = 972–1023, aged 54+ for the other two studies) reported an inverse relationship between education and allostatic load. Dowd and Goldman (2006) further found an inverse relationship between education and a subscore of allostatic load (based on CV and metabolic markers) among women but not among men.

Glei, Goldman, Shkolnikov, Jdanov, Shalnova, et al. (2013) examined data from Russia (*n* = 1,495, aged 55+); their findings showed large educational disparities in biological risk, particularly for standard CV and metabolic risk factors and for inflammation (there was no evidence that the relationship differed by sex). An analysis of Polish men (*n* = 3,887, aged 25–60) (Lipowicz, Szklarska, & Malina, 2014) reported an inverse relationship between allostatic load (11 markers) and education. A recent study by Robertson and Watts (2016) that used data from the Scottish Health Survey (*n* = 1,834, aged 18+) also demonstrated an inverse association between education and allostatic load, but they reported that the relationship widened with age, becoming statistically discernible at ages 35 to 44, then narrowing at older ages (75 and above).

Finally, Osler and colleagues (2000) used a CV risk score that comprised many of the same factors as an allostatic load score to examine the relationship between education and those risk factors in Denmark (*n* = 6,695, aged 30–60); they found that not only did more highly educated persons have lower risk scores, but also that the trend over time showed an increasing differential—a change that they attributed primarily to decreases in smoking among the more highly educated. By contrast, Rosero-Bixby and Dow (2009) found that Costa Ricans (*n* = 2,827, aged 60+) with no education were the *least* likely to exhibit metabolic syndrome, a reversal of the "classic" education–health pattern, potentially linked to higher SES individuals being more likely to adopt a Western diet, resulting in those who are better off being overnourished.

A few studies using data from outside the United States have examined the association between education and inflammation. Rosvall, Engstrom, Janzon, Berglund, and Hedblad (2007), in an analysis of data from a subcohort of the Malmö Diet and Cancer Study (Sweden: *n* = 3,921, aged 45–65), found an inverse relationship between education and CRP. Another study of the relationship between inflammation and education was published by Gimeno et al. (2008). Using data from the Cardiovascular Risk in Young Finns Study (*n* = 1,484, aged 24–39), they explored whether differences in CRP by education were determined early or late in life. They found that in childhood (when SES was measured by parental education and occupational status) there were no differences in CRP, but in adulthood they documented an inverse relationship between the participant's education and CRP. An earlier study of the same Finnish data (*n* = 2,290, aged 24–39) (Kivimaki et al., 2005) was consistent with those results: Education was associated in early adulthood with levels of CRP. However, Rosero-Bixby and Dow (2009) found no significant relationship between education and CRP in Costa Rica (*n* = 2,827, aged 60+).

Comparisons across multiple countries pose a number of challenges with respect to comparability of measures. It is not surprising, therefore, that only a few studies emerged from our review of the literature. A comparison of England and the United States (individuals aged 40–70) (Banks, Marmot, Oldfield, & Smith, 2006) examined both self-reported health outcomes (diabetes, hypertension, all heart diseases, myocardial infarction, stroke, lung disease, and cancer) and a set of biomarkers (glycosylated hemoglobin [HbA_{1c}], blood pressure, CRP, fibrinogen, and HDL cholesterol) as a function of education. They reported sharp educational gradients in both England and the United States for both the self-reported health outcomes and the biomarkers, but a steeper gradient and worse levels in the United States.

A study by Goldman, Turra, Rosero-Bixby, Weir, and Crimmins (2011) compared the relationship between
p. 484 education and a variety of individual biomarkers (BMI, waist circumference, blood pressure, glucose, ↳ HbA_{1c}, total cholesterol [TC], triglycerides, dehydroepiandrosterone sulfate [DHEA-S], and cortisol) across three countries: Taiwan (n = 1,023, aged 54+), Costa Rica (n = 2,750, aged 60+), and the United States (n =

6,886, aged 53+). They found nonsystematic associations in Taiwan and Costa Rica (indeed, for Costa Rica they found higher risk associated with higher education), but statistically significant associations in the United States (data on only 6 of the 10 indicators were available for the United States): For US women—except for cholesterol—all the biomarkers were significantly associated with education; for US men, HbA_{1c} and both diastolic blood pressure (DBP) and systolic blood pressure (SBP) measures were significant. In all, they found that "the results for Costa Rica and Taiwan challenge the commonly held assumption that more educated individuals have healthier biological profiles than their less educated peers" (Goldman et al., 2011, p. 312).

Finally, Cornman, Glei, Ryff, and Weinstein (2015) compared biological risk in Taiwan (n = 976, aged 53+) and the United States (n = 1,014, aged 34–84) according to SES (education, income, and subjective social status). Biological risk was operationalized as four scores: a summary score of overall physiological dysregulation and three subscores reflecting CV and metabolic risk, hypothalamic–pituitary–adrenal (HPA) and sympathetic nervous system function, and inflammation. They found that among men, the magnitude of the association between education and biological risk (overall, CV/metabolic, inflammation, and neuroendocrine) was somewhat stronger in the United States (MIDUS) than Taiwan (Social Environment and Biomarkers of Aging Study, SEBAS). Among women, the educational disparities were generally similar in the two countries. They concluded that the educational gap tended to be larger for men than women, especially in the United States.

Conclusions Based on Prior Work

In sum, previous studies of the association between education and biological risk suggest that

1. There is an inverse relationship between education and risk, although the association may be stronger in middle age than at the oldest ages (e.g., selective attrition hypothesis) and stronger among whites than blacks.
2. The educational gradient may differ by country (stronger in the United States, weaker in Costa Rica and Taiwan).
3. Sex differences in the educational gradient may depend on the context, and disparate results have been reported even for the same country (Cornman et al., 2015; Goldman et al., 2011).

New Analyses From Five Countries

Data

We used data from five biosocial surveys, each of which was based on a nationally representative sample of older adults: Wave 2 (2004–2006) of the MIDUS study; the 2006 wave of the SEBAS in Taiwan; Wave 1 (2004–2006) of the Costa Rican Study on Longevity and Healthy Aging (CRELES); Wave 2 (2004–2005) of the English Longitudinal Study of Aging (ELSA); and Wave 1 (2006–2009) of the survey on Stress, Aging, and Health in Russia (SAHR).

The data from MIDUS 2 were based on the biomarker subsample (n = 1,255, ages 34–86, mean = 56.9, SD = 11.8). The SEBAS data were restricted to those who participated in the 2006 exam (n = 1,036, ages 53–97, mean = 66.3, SD = 10.0). The CRELES data were based on those who provided a blood specimen (n = 2,694, aged 60–109, mean = 76.3, SD = 10.1). Data from ELSA were restricted to the subsample that participated in the nurse visit and provided a usable blood specimen (n = 6,228, ages 52+,[1] mean = 66.0, SD = 9.5). The data

from SAHR were based on those who completed the baseline survey (n = 1,800, ages 55–92, mean = 68.3, SD = 7.8).

After excluding those with missing data for education or one of the biomarkers in this analysis (2% in MIDUS; 2% in SEBAS; 15% in CRELES; 22% in ELSA; 3% in SAHR), we were left with 1,226 respondents from MIDUS (United States), 1,017 from SEBAS (Taiwan), 2,297 from CRELES (Costa Rica), 4,834 from ELSA (England), and 1,744 from SAHR (Russia). Because our review of the literature suggested that we might find variation in the relationship by race, we further subdivided the US and English samples by race. The vast majority of the English sample was white (98%) with very few nonwhites (33 men and 31 women). The US sample included 72 black men and 144 black women, with very few of some other race (including those reporting mixed race); Hispanics are included among the racial group with which they most closely identify themselves. Because of the small nonwhite sample sizes, we have restricted our analyses for England to whites, and for the US, we have conducted separate analyses for white men, white women, and black women.

Measures

Physiological Dysregulation

To measure physiological dysregulation (PD), we used a set of nine biomarkers that were available for all five countries. They included eight CV/metabolic markers (i.e., SBP, DBP, HDL, the ratio TC/HDL, triglycerides, HbA_{1c}, BMI, and waist circumference) and one inflammatory marker (CRP). In CRELES, some of the specimens were not analyzed using a high-sensitivity assay for CRP; thus, the lower detection limit was very high (2.98 m/L). In order to make the values comparable across countries, we bottom coded all CRP values to 2.98 m/L. Consequently, 38% of respondents in CRELES, 67% of those in SAHR, 82% of those in SEBAS, 70% of those in MIDUS, and 64% of those in ELSA were coded as having CRP $\leq$ 2.98 m/L.

In the first step of the analysis, we transformed the individual biomarkers to better approximate a normal distribution. We used a log transformation for SBP, ratio TC/HDL, HDL, triglycerides, BMI, HbA_{1c}, and CRP. The other two markers (DBP and waist circumference) remained untransformed. Then, we reverse coded ln(HDL) so that higher values reflect greater risk. Next, we standardized each of the transformed markers (based on the pooled distribution). Then, we computed a Z-score summary measure for CV/metabolic risk factors by computing the average across the eight markers (α = .71). An overall Z-score measure of PD was based on the average all nine biomarkers (α = .69).

Given the truncated values for CRP, the distribution was heavily skewed even after applying a log transform. Thus, when we modeled CRP individually, we dichotomized CRP to indicate values greater than or equal to 3 m/L, the cutoff associated with elevated risk for major coronary events (Pearson et al., 2003).[2]

Education

Education was measured as the number of completed years in CRELES, SEBAS, and SAHR, while ELSA and MIDUS measured education based on qualifications (ELSA) or degree completion (MIDUS). In several of these countries, particularly Taiwan, the level of education increased dramatically over time. In order to avoid having a disproportionate share of the oldest respondents assigned to the "low" education group and the youngest respondents to the "high" education group, we categorized education relative to other respondents of the same sex, country, and cohort group. Within each sex–cohort–country subgroup, we categorized respondents at the median into low versus high education. Thus, we compared individuals in the top half of the education distribution with those in the bottom half within a given sex, country, and age group. (Note: If instead we had used a median split based on the education distribution of the entire sample in a given country [not sex or cohort specific], then in the case of the oldest cohort of Taiwanese women [born in 1909–1920, aged 85–97 in 2006], we would have ended up comparing 16% of those with the most education [7+ years] with the remaining 84% with the least education [≤ 6 years]. In contrast, among the youngest cohort of Taiwanese women [born in 1950–1953, aged 52–56 in 2006], we would have been comparing 54% with the most education with the remaining 46% who were least educated.) The country–sex–cohort specific cutoffs are given in Table 34.1. The exception is the United States, for which there was virtually no correlation between age and education; thus, for the United States, we used sex-specific (but not cohort-specific) cutoffs.

Analytical Strategy

All models were fit separately by sex, country, and in the United States, by race. In each model, we tested whether physiological dysregulation scores differed by education (high vs. low) controlling for age. We also tested for interactions between age and education in order to explore whether the effects of education on physiological dysregulation increased (the accumulated advantage hypothesis) or decreased (selective attrition) with age. The results of these interaction models are not further reported; we note here that only 3 of the 32 interactions were statistically discernible and that they did not improve model fit (based on BIC)[3] in any model. We used ordinary least squares regression to model the continuous measures of overall PD and CV/metabolic subscore. For high CRP (≥ 3 mL), we used a logit model.[4]

Results

Results of our analyses are shown in Table 34.2. We begin with the overall summary of physiological dysregulation. Men in all countries showed a decline in the summary score by age, with the steepest declines visible in Costa Rica, Russia, and Taiwan (Figure 34.1). The summary score among US men remained fairly flat with increasing age; a slightly steeper slope is visible in England (whites). The age patterns for women

p. 486 were substantially ↳

p. 487 ↳

different from those for men. Most notably, with the exception of Costa Rica, which, consistent with the results for men, displays a decline in dysregulation with age, women in the other four countries have scores that increased or remained relatively flat (in the United States and Russia) with age (Figure 34.2).

Table 34.1 Cutoffs for "High" Education by Sex, Cohort Group, and Country

	Costa Rica (CRELES)	Taiwan (SEBAS)	United States (MIDUS)	England (ELSA)	Moscow (SAHR)
Education Measure	Years	Years	Degree completion	Qualification level	Years
Cutoffs for Men			≥BA/BS[a]		
Born < 1910	3+ years[b]	7+ years[c]		≥NVQ1/CSE[d]	N/A
Born 1910–1919	3+ years	7+ years		≥NVQ1/CSE	14+ years[e]
Born 1920–1929	4+ years	7+ years		≥NVQ2/GCE O level/foreign/other	14+ years
Born 1930–1939	4+ years	7+ years		≥NVQ2/GCE O level/foreign/other	15+ years
Born 1940–1949	6+ years[f]	7+ years		≥NVQ3/A level	15+ years
Born 1950–1959	N/A	12+ years[g]		Any higher education (>NVQ3/A level)[h]	14+ years[h]
Born 1960–1970	N/A	N/A		N/A	N/A
Cutoffs for Women			≥AA/AS/VoTech/3 years college (no degree)[a]		
Born < 1910	3+ years[b]	1+ years[c]		≥NVQ1/CSE[d]	N/A
Born 1910–1919	4+ years	1+ years		≥NVQ1/CSE	11+ years[e]
Born 1920–1929	4+ years	1+ years		≥NVQ1/CSE	11+ years
Born 1930-39	4+ years	1+ years		≥NVQ2/GCE O level/foreign/other	15+ years
Born 1940–1949	5+ years[f]	7+ years		≥NVQ2/GCE O level	16+ years
Born 1950–1959	N/A	7+ years[g]		≥NVQ3/A level[h]	14+ years[h]
Born 1960–1970	N/A	N/A		N/A	N/A

AA/AS/VoTech = associates degree (2-year college) or vocational school; BA/BS = bachelor's degree (4- to 5-year college); N/A = not applicable.

Note: In CRELES, SEBAS, and SAHR, education is measured in terms of completed years (0–17 in CRELES and SEBAS; 2–27 in SAHR). MIDUS measures education in terms of degree completion: 1 = no school/some grade school (1–6 years); 2 = junior high

school (7–8 years); 3 = some high school (9–12 years no diploma/no general equivalency diploma [GED]); 4 = GED; 5 = high school graduate; 6 = 1–2 years college but no degree; 7 = 3+ years college but no degree; 8 = associate's degree/graduated 2-year college or vocational school; 9 = bachelor's degree/graduated from 4- to 5-year college; 10 = some graduate school; 11 = master's degree; 12 = graduate/professional degree (e.g., PhD, MD, JD, etc.). ELSA measures educational qualifications, which we have recoded as follows: 0 = no qualifications; 1 = National Vocational Qualification [NVQ] 1 or Certificate of Secondary Education [CSE]; 2 = foreign/other; 3 = NVQ2 or General Certificate of Education [GCE] O level; 4 = NVQ3/GCE A level; 5 = higher education but no degree; 6 = NVQ4/NVQ5/degree or equivalent.[5]

a For the United States, we did not use cohort-specific cutoffs for education because there is virtually no correlation between age and education.

b Oldest cohort was born in 1896 (CRELES).

c Oldest cohort was born in 1909 (SEBAS).

d Oldest cohort was born in 1907 or earlier for ELSA (they do not provide exact age/cohort for the oldest respondents).

e Oldest cohort was born in 1914 (SAHR).

f Youngest cohort was born in 1946 (CRELES).

g Youngest cohort was born in 1953 (SEBAS).

h Youngest cohort was born in 1952 (ELSA and SAHR).

Table 34.2 Coefficients for Age and Education (High vs. Low) From Models Predicting Overall Physiological Dysregulation (PD), Cardiovascular/Metabolic Subscore, and High CRP (≥3 m/L) by sex and country

Males						
Outcome	**Covariate**	**Costa Rica**	**England Whites**	**Russia**	**Taiwan**	**US Whites**
Overall PD[a]	Age	-0.0152***	-0.0061**	-0.0146***	-0.0105**	-0.0015
	High education	-0.0057	-0.1621***	-0.3508***	-0.0763	-0.3209***
CV/metabolic subscore[a]	Age	-0.0211***	-0.0089***	-0.0166***	-0.0137***	-0.0025
	High education	0.0051	-0.1381***	-0.3222***	-0.0391	-0.3084**
High CRP[b]	Age	0.0292***	0.0230***	0.0124	0.0425***	0.0122
	High education	-0.1192	-0.2831**	-0.6248***	-0.6594**	-0.5421*
N		1,047	2,126	813	547	438

Females							
Outcome	**Covariate**	**Costa Rica**	**England Whites**	**Russia**	**Taiwan**	**US Whites**	**US Blacks**
Overall PD[a]	Age	-0.0226***	0.0064***	0.0036	0.0221***	0.0051	0.0012
	High education	-0.1095*	-0.2484***	-0.2936***	-0.1900*	-0.1440	-0.5631***
CV/metabolic subscore[a]	Age	-0.0244***	0.0048**	0.0040	0.0234***	0.0064	0.0025
	High education	-0.1217*	-0.2270***	-0.2835***	-0.1727*	-0.1603	-0.5293**
High CRP[b]	Age	-0.0102	0.0154***	0.0136	-0.0078	-0.0093	-0.0170
	High education	0.0795	-0.3828***	-0.1628	-0.5817*	-0.1478	-0.6541
N		1250	2603	931	470	522	144

* $p < 0.05$;

** $p < 0.01$;

*** $p < 0.001$

a Based on ordinary least squares regression models fit separately by subgroup (sex, country, and race).

b Based on logistic regression models fit separately by subgroup (sex, country, and race).

In auxiliary analyses (not shown), we examined the age patterns for the individual biomarkers that underlie the summary score in order to identify whether particular markers were driving the negative association with age observed among men (especially in Costa Rica, Russia, and Taiwan) and among Costa Rican women (but not women ↳ in other countries). We found that, among men, markers of obesity (BMI, waist

circumference); lipids (TC/HDL, triglycerides); and DBP were negatively correlated with age in most countries, but especially so in Costa Rica, Taiwan, and Russia.

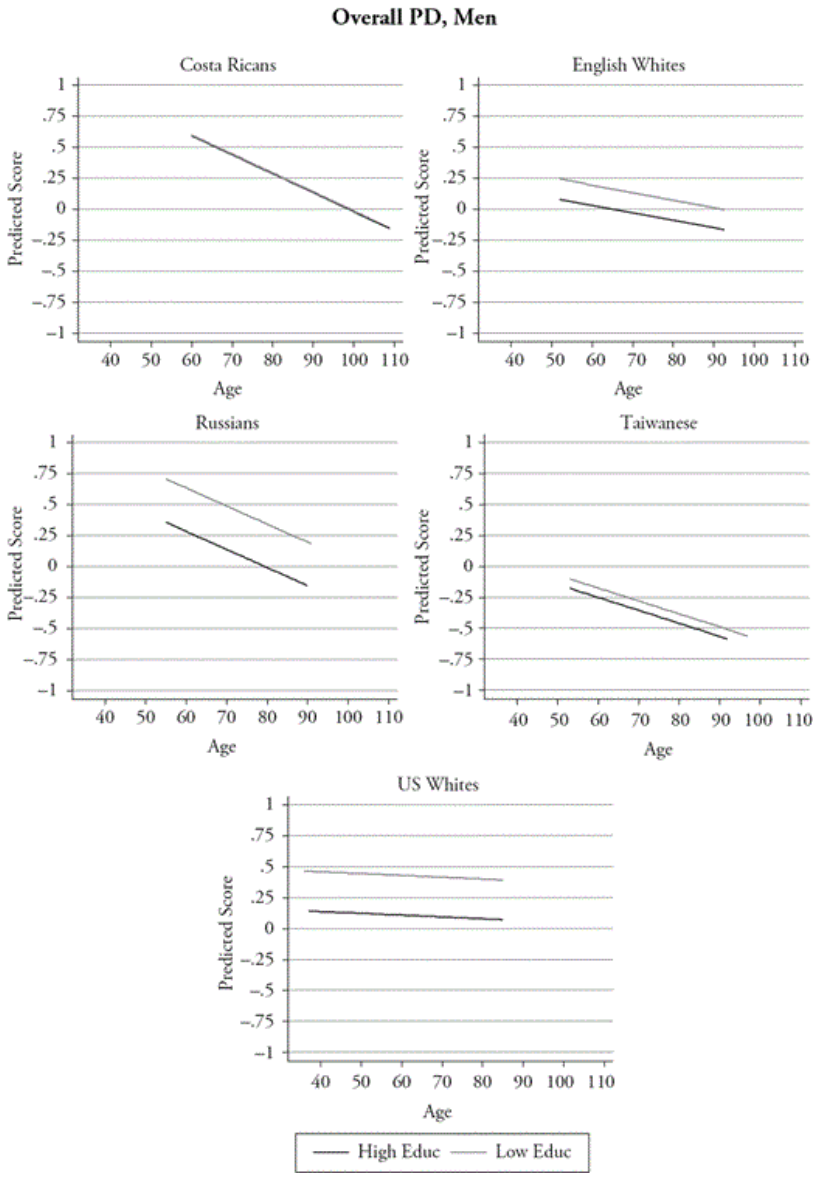

Figure 34.1 Predicted overall dysregulation score by age, education, and country: men.

Among women in Costa Rica, these same markers as well as HbA_{1c} were negatively correlated with age. The age correlations with those markers were generally weaker or positive among women, except for diastolic blood pressure (which was negatively correlated with age among both sexes in all countries). For example, there was a moderate, negative correlation between age and BMI among Costa Rican women (r = -.33), while
p. 489 the correlations (among women) were only weakly ↳ negative in English whites (r = -.08), Russia (r = -.05), and US whites (r = -.05), and they were small but positive in Taiwan (r = .07) and US blacks (r = .01).

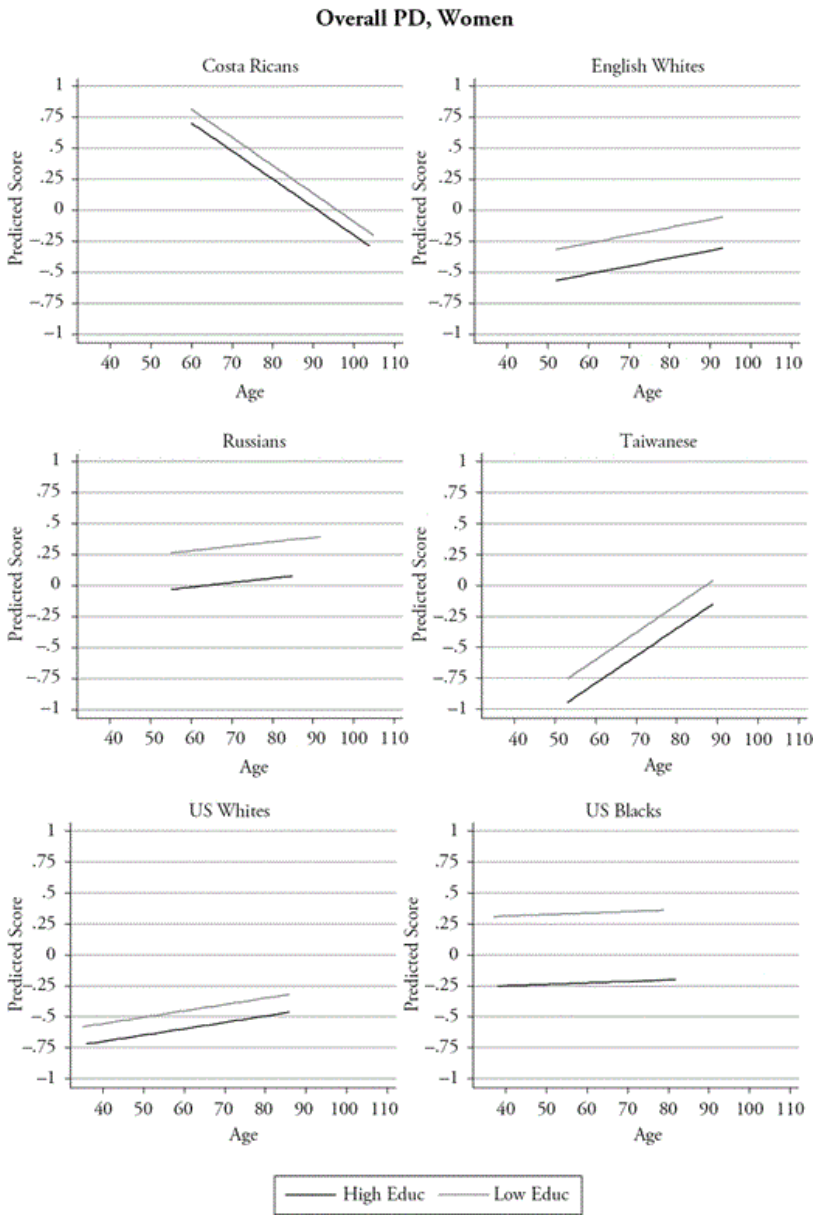

Figure 34.2 Predicted overall dysregulation score by age, education, and country: women.

Only one marker, SBP, exhibited a consistent positive correlation with age (among both sexes in all countries). HbA_{1c} was positively correlated with age in England and the United States (in both sexes), but the correlation was negligible in the other countries (except for Costa Rican women, where there was a notable negative correlation, as noted). CRP was also positively correlated with age among men in all countries and (white) women in England.

p. 490 Among men, Russians exhibited the widest educational gap in overall physiological dysregulation, followed by the United States and England; very small—and nonsignificant—educational disparities were evident in Taiwan and Costa Rica. In women, the largest educational gaps were seen among black US women, followed by Russian and English women; Costa Rican women exhibited the smallest educational gap.

There was no evidence that the sex differences in the education gap varied significantly by country (tested in models fitted to the pooled data). Results for US black women were notably different from those for white women: Black women showed a significant educational disparity, whereas white women did not. Indeed, black women in the United States exhibited the widest educational disparity of any subpopulation, male or female.

Results for the CV and metabolic markers subscore are also shown in Table 34.2. Not surprisingly—the overall score was heavily weighted by the subscore—the results for the two are similar. Men in all five countries had scores that declined with increasing age (flat in the United States; Figure 34.3); women's scores rose or remained flat with age, except in Costa Rica, where the subscore decreased over age (Figure 34.4). Again, among men, the largest differences in education were observed in Russia and the United States (white men). For women, the largest differential was observed among US blacks, followed by sizable differentials in Russia and England. As for CRP, the educational disparities in Taiwan were as large (or larger) than among Russians (Figures 34.5 and 34.6). The relative disparities were largest in Taiwanese men and smallest in Costa Rican women (Table 34.2). Among women, CRP was not significantly associated with age except in England, where the probability of high CRP was greater at older ages. In contrast, the prevalence of high CRP increased significantly with age among men in Costa Rica, England, and Taiwan.

Conclusion

In summary, then, our analyses based on comparable methods and measures across countries confirmed some of the previous literature but revealed some discrepancies. Where we found statistically discernible educational differentials, those differentials were all in the expected direction: Higher education was associated with lower dysregulation or inflammation. We observed the "classic" relationship among men and women in England and Russia except for CRP among Russian women (which was not significant). In the United States, the inverse relationship held among white men but was not observed among white women; indeed, among US white women, neither age nor education was significant. Educational differentials among black women in the United States were significant (except for inflammation) and were substantially greater than the differentials among white women. For Costa Rica, our results for CRP were consistent with those reported by Rosero-Bixby and Dow (2009), showing no significant association with education. However, we found small, but significant, inverse education differentials in CV/metabolic scores for women, but not for men. By contrast, Rosero-Bixby and Dow (2009) found that among Costa Ricans as a whole (both sexes combined), those with no education were the *least* likely to exhibit metabolic syndrome.

Our finding that the educational differential among US women was larger for blacks than for whites appeared to be sensitive to how one defined "high" education. In our analysis, we used the same cutoff (2-year college degree or at least 3 years of college) for white and black women in United States. Yet, because educational attainment tends to be lower for black women compared with their white counterparts, black women were much less likely to fall in the high education group (31%) than white women (53%). If we instead use race-specific cutoffs (splitting each subgroup at the median: 2-year college degree or higher for whites; at least 1 year of college for blacks), the education differential appears similar for black and white women (data not shown). That is, if we compare women in the top versus bottom half of the education distribution *within their racial subgroup*, the disparities look similar. But, if we compare women at the same absolute levels of education, then the difference between women with 3+ years of college (or a 2-year college degree) and those with less than 3 years of college (and no degree) are much greater for blacks than for whites.

Age generally had different effects for men and for women. Among men, CV/metabolic dysregulation was negatively associated with age, while the association with inflammation was positive, although not all coefficients were statistically significant. Among women, the picture was mixed: CV/metabolic dysregulation was negatively associated with age among Costa Rican women, while it rose or remained flat elsewhere; inflammation was significantly—and positively—associated with age only in England. The
p. 491 relationship between ↳ age and physiological dysregulation is complex. Prior studies indicated that some CV/metabolic markers (e.g., DBP, TC, and BMI) decreased at the oldest ages, while others may exhibit little age-related change above age 50 (e.g., HDL). Only one of the CV/metabolic markers included in our analysis

consistently showed a positive correlation with age in prior studies: HbA_{1c} (Glei, Goldman, & Weinstein, 2011, Table A-3). Although many of these markers increase during middle-age years, we may not observe that increase among individuals aged 50 and older (as are all of the samples analyzed here except for MIDUS). Decline in these biomarkers at the oldest ages could be partly a result of mortality selection; increased prevalence of serious illness at the oldest ages may also contribute (e.g., lower BMI because of wasting, lower TC resulting from underlying disease).

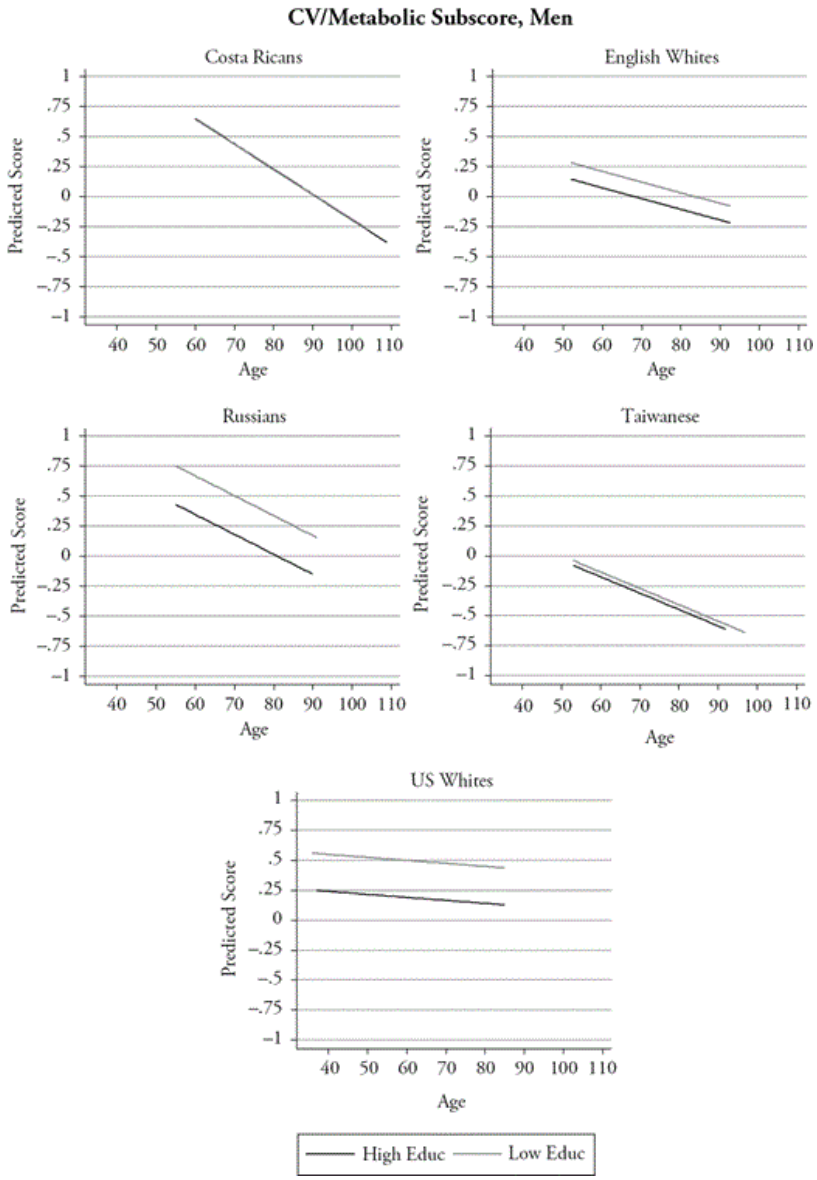

Figure 34.3 Predicted CV/metabolic subscore by age, education, and country: men.

p. 492 Because we are looking at cross-sectional data, there is also the possibility that the older cohorts are healthier than the younger cohorts. For example, the age-associated decline in CV/metabolic dysregulation and CRP among Costa Rican women appears to be linked with obesity, which is higher in younger cohorts. In the model predicting high CRP among Costa Rican women, we added an additional control for BMI (which is strongly and positively associated with CRP) and found that the age coefficient switched sign (from negative to positive) and the positive coefficient for high education weakened (results not shown). Thus, one reason that younger and better educated women in Costa Rica appear to have higher CRP (albeit not significantly so) may be because they are more likely to be obese than their older or less educated counterparts.

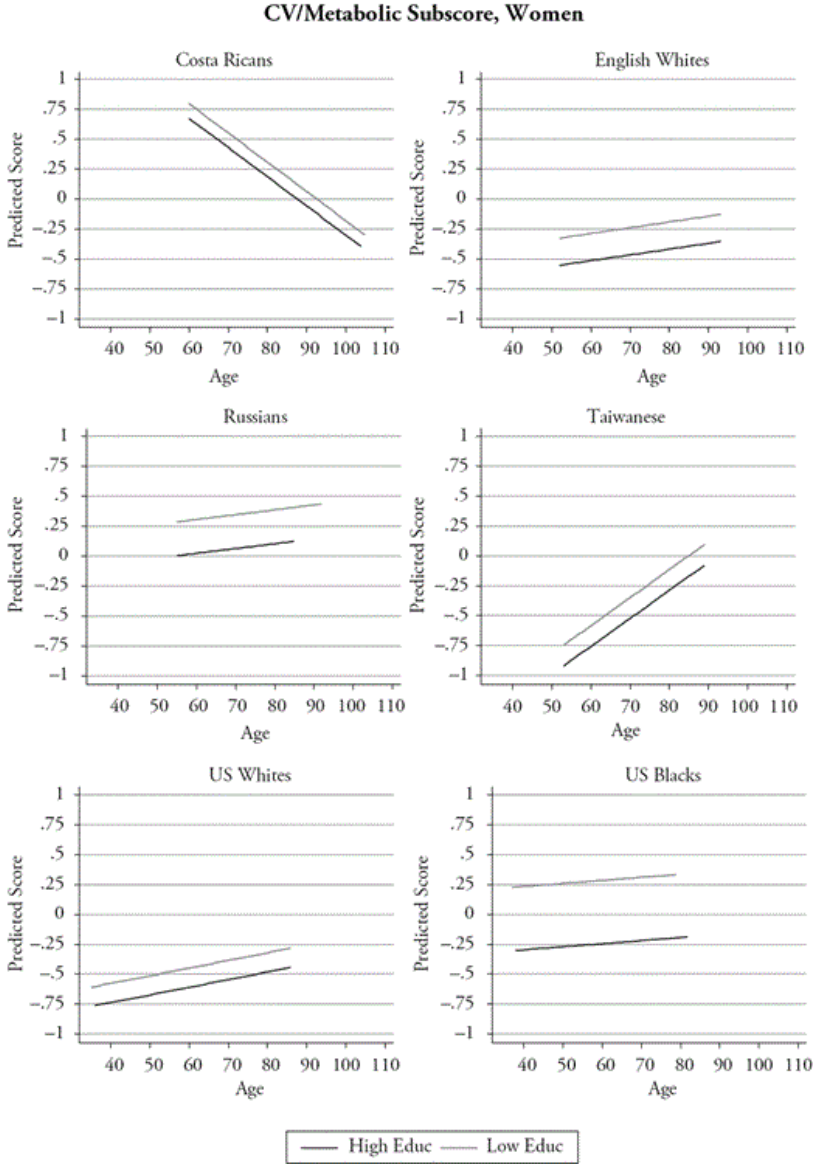

Figure 34.4 Predicted CV/metabolic subscore by age, education, and country: women.

p. 493

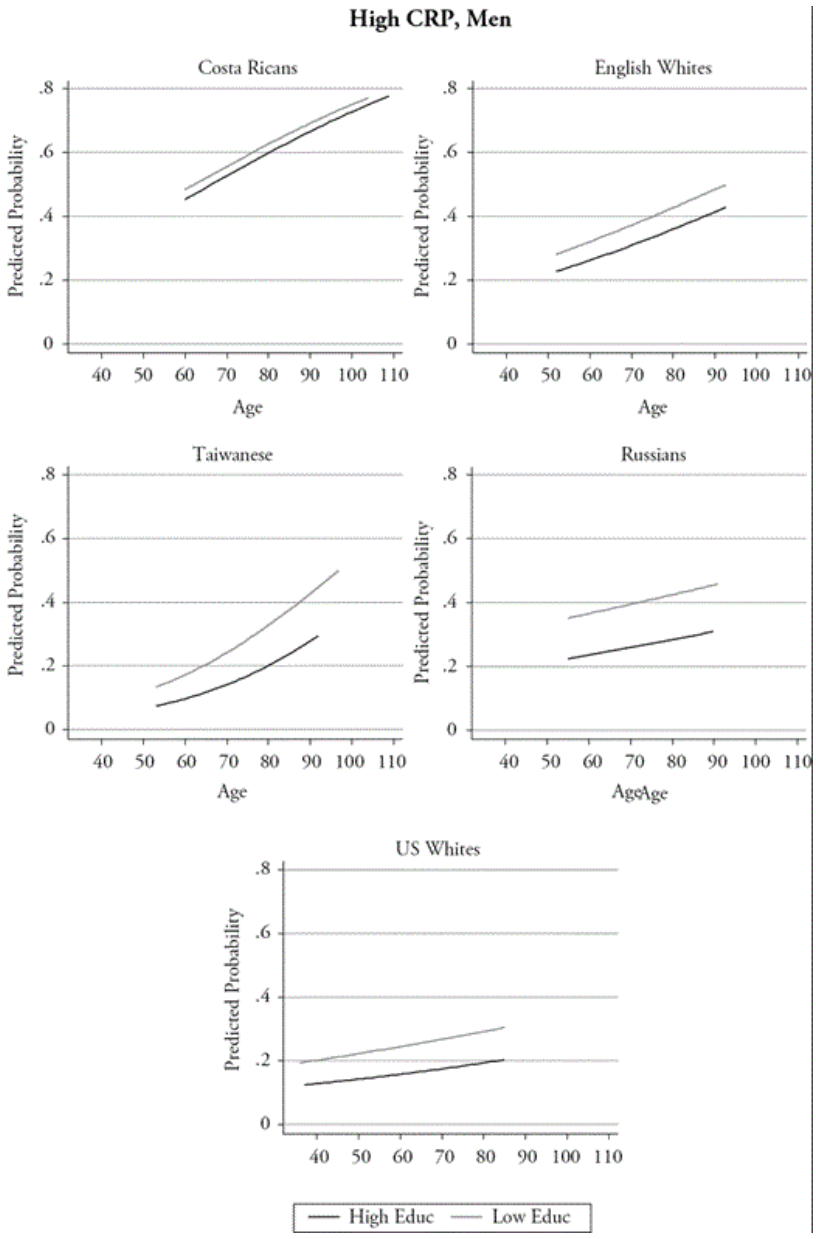

Figure 34.5 Predicted probability of CRP ≥ 3 m/L by age, education, and country: men.

Future Directions

Our review and analyses raise a number of questions for further investigation that lie beyond the scope of this chapter. First, we wonder about the factors that might influence the relation between education and dysregulation. Do income and wealth have effects beyond that of education? What about access to healthcare? The effects of psychological mechanisms are also intriguing: for example, the results of Kubzansky et al. (1999) suggest that the relationship between allostatic load and education may be mediated by hostility.

Second, the way we model education matters. We looked at interactions between age and education, and
p. 494 while the results were generally not significant, we did not have life course data that would ↳ have allowed us to tease out the effects of education across the life span. We adjusted for changes in educational levels across cohorts by using cohort-specific cut points, but without longitudinal data, we cannot assess the extent to which an educational advantage changes throughout life.

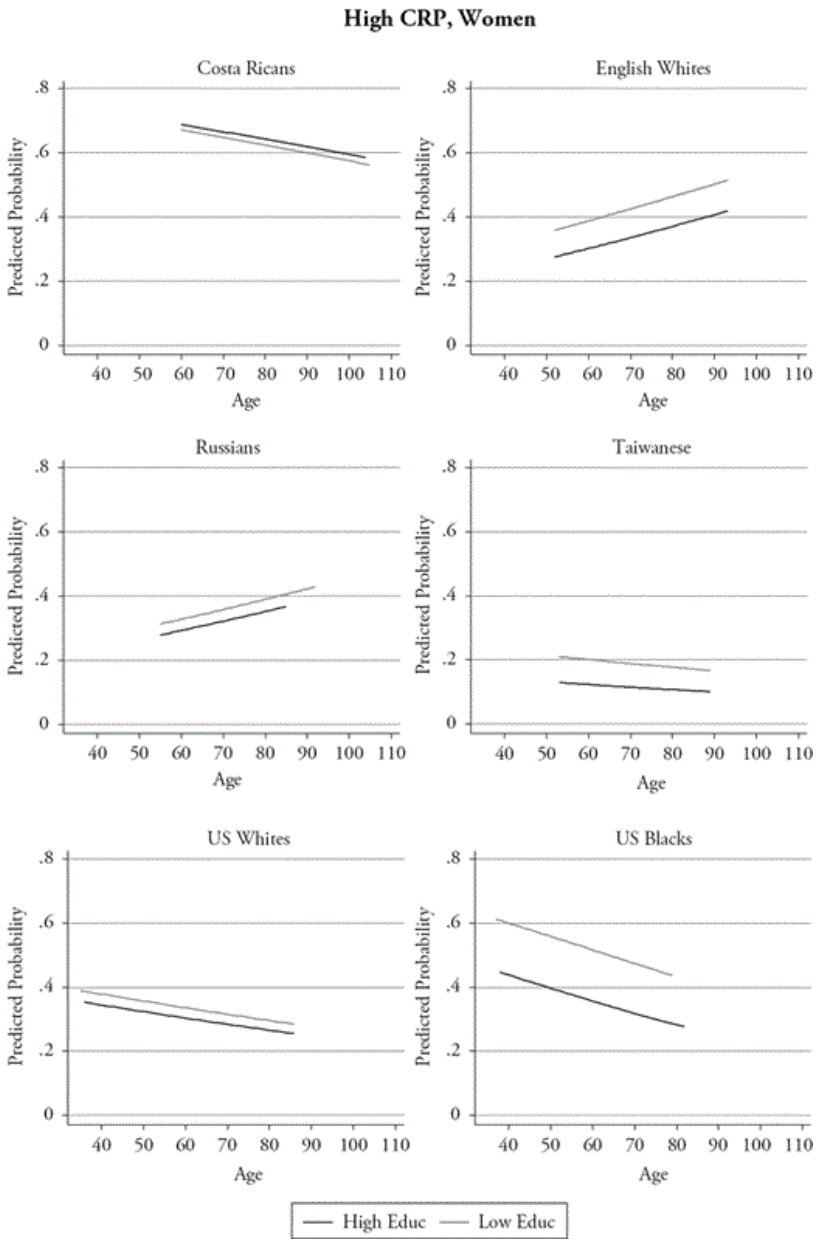

Figure 34.6 Predicted probability of CRP ≥ 3 m/L by age, education, and country: women.

Finally, we find the results for US women intriguing. The educational differentials for white women are in the anticipated direction, but none is statistically discernible. While education appears to have little
p. 495 association with dysregulation among white women, educational differentials among ↳ black women are approximately four times as large. An exploration of the correlates of these differentials should be an important next step in understanding black–white health disparities.

Notes

1. In order to protect confidentiality, respondents aged 90 and older were top coded in the ELSA public use dataset. We have coded age for these respondents to the sex-specific mean age (92.7 for men, 93.2 for women) among the population of England/Wales aged 90 and older on January 1, 2005, based on population estimates from the Human Mortality Database (University of California, Berkeley [USA] and Max Planck Institute for Demographic Research [Germany], 2016).

2. An added advantage of dichotomizing CRP is that it solves the problem of differences between datasets in the lower

detection limit.

3. The Bayesian information criterion (BIC) compares the fit of one model relative to another (whether nested or not) based on the maximized log likelihood, with a penalty for the number of parameters estimated (Schwarz, 1978; StataCorp, 2011).

4. We explored a linear model using logged CRP (in spite of the heavily skewed distribution); the results (not shown) were very similar to those reported here. The only notable difference was that in the model for logged CRP, the education differential was significant among Russian women, whereas in the logit model, the corresponding coefficient was not significant.

5. The "foreign/other" category comprises 5% of men and 12% of women. We compared this group with other educational qualifications in terms of the average age at which they ended full-time education. Those with NVQ1/CSE left school between ages 14 and 15; those with foreign/other qualifications left school between ages 15 and 16; and those with NVQ2/O level left school at age 16. Thus, we coded foreign/other (= 2) between NVQ1/CSE (= 1) and NVQ2/O level (= 3).

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