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J. Paul Leigh

Juan Du
Old Dominion University, jdu@odu.edu

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Are low wages risk factors for hypertension?

J. Paul Leigh[1], Juan Du[2]

1 Division of Epidemiology, Department of Public Health Sciences, and Center for Healthcare Policy and Research, University of California Davis School of Medicine, California, USA
2 Department Economics, Old Dominion University, Norfolk, VA, USA

Correspondence: Paul Leigh, Division of Epidemiology, Department of Public Health Sciences, UC Davis Medical School, MS1C room 122, CA 95616-8638, USA, tel: +1 530 754 8605, fax: +1 530 752 3239, e-mail: pleigh@ucdavis.edu

Objective: Socio-economic status (SES) is strongly correlated with hypertension. But SES has several components, including income and correlations in cross-sectional data need not imply SES is a risk factor. This study investigates whether wages—the largest category within income—are risk factors. **Methods:** We analysed longitudinal, nationally representative US data from four waves (1999, 2001, 2003 and 2005) of the Panel Study of Income Dynamics. The overall sample was restricted to employed persons age 25–65 years, $n = 17\,295$. Separate subsamples were constructed of persons within two age groups (25–44 and 45–65 years) and genders. Hypertension incidence was self-reported based on physician diagnosis. Our study was prospective since data from three base years (1999, 2001, 2003) were used to predict newly diagnosed hypertension for three subsequent years (2001, 2003, 2005). In separate analyses, data from the first base year were used to predict time-to-reporting hypertension. Logistic regressions with random effects and Cox proportional hazards regressions were run. **Results:** Negative and strongly statistically significant correlations between wages and hypertension were found both in logistic and Cox regressions, especially for subsamples containing the younger age group (25–44 years) and women. Correlations were stronger when three health variables—obesity, subjective measures of health and number of co-morbidities—were excluded from regressions. Doubling the wage was associated with 25–30% lower chances of hypertension for persons aged 25–44 years. **Conclusions:** The strongest evidence for low wages being risk factors for hypertension among working people were for women and persons aged 25–44 years.

A consensus exists that there are strong statistical associations between measures of low socio-economic status (SES) and measures of poor circulatory health, especially hypertension.[1–4] The consensus falls apart, however, when addressing causes of the associations. Since many correlations have been found with cross-sectional data, the most fundamental problem is that poor circulatory health might result in low SES.[1–10] But even assuming that low SES causes circulatory disease, there is controversy surrounding scientific explanations. First, definitions of SES vary and evidence suggests that education, occupation and income each have separate associations.[1–3] Second, even within any one SES category, explanations offered differ. Ragland *et al.*[11] find that male bus drivers experience elevated blood pressures compared to men of the same age and race which they partially attribute to psychosocial factors. One factor involves feelings of self-worth associated with occupational status and incongruity between that status and level of education.[3,4,11] Another factor is 'job strain'—popularized by Karasek *et al.*[12]—which implicates jobs with 'high-demand and low-control' as being injurious to health.[1–4,11,13] Related hypotheses have been advanced for income. Persons with low income, especially in the USA, are less likely to have medical insurance.[14] Another hypothesis is that low income results in low self-esteem and low levels of happiness and the latter two, through neuroendocrine and cardiovascular processes, are associated with hypertension.[15–18]

We extend lines of research on SES and circulatory disease with two contributions. First, we focus on wages—as opposed to simply income—as a key component of SES while simultaneously controlling for measures of education and occupation. We are unaware of any studies that address whether wages are predictors of circulatory disease. This is unfortunate because wages have special characteristics. Wages implicitly capture some aspects of two components of SES: income and occupation. Income is comprised of job earnings, interest, dividends, capital gains, rental income and government transfers, among others. In recent years in the USA, earnings for employees and for sole proprietors has exceeded 61% of income for all persons and 80% for employed persons.[19,20] Wages, by definition, derive from jobs within occupations. According to Kalleberg and Reskin,[21] 'broad consensus exists that wages are a fundamental dimension of job quality'. Moreover, just as there are government policies that can influence education or occupational safety, there are government policies that affect wages such as establishing legal minimum wages, issuing government contracts to unionized, high-wage firms and adjusting pay of government workers. Second, we use longitudinal data widely analysed in social science studies but underutilized in epidemiology and public health. These longitudinal data allow us to minimize bias introduced by possible reverse causality in which disease results in low wages. We investigate associations between wages measured in base years and incidence of disease measured in subsequent years with discrete time survival models and time-varying covariates as well as with Cox proportional hazard models. In base years, we require that no subjects report any hypertension.

Methods

Data

The Panel Study of Income Dynamics (PSID) is a longitudinal, representative US sample of men and women. The PSID contains rich information including, for example, respondents' wages, hypertension status, education and health habits. We combined data on household heads (most frequently men) with what the PSID refers to as 'wives', if any, for four recent waves: 1999, 2001, 2003 and 2005. Since the critical covariate was wages, we limited the sample to adults working at least 500 annual hours whether as employees or self-employed, and between and including ages 25–65 years because many persons are still in college in their early twenties. To analyse the first-dependent variable—incidence of hypertension (yes/no)—we created and combined three cohorts: 1999–2001, 2001–03 and 2003–05. For each cohort, persons with hypertension in the base year were excluded, i.e. for the 1999–2001 cohort, persons with hypertension in 1999 were excluded. To analyse the second-dependent variable—time-to-hypertension—1999 was the single base interview year and 2001, 2003 and 2005 were subsequent years in which hypertension was recorded. Base year values were used to predict subsequent year incident hypertension or time-to-hypertension; the study was therefore prospective. Finally, we excluded persons with missing data.

In our overall sample analysing incident hypertension, the sample sizes contained: 5532 subjects in 1999–2001; 5861 in 2001–03; and 5902 in 2003–05. The overall sample for time-to-hypertension was 5651.

We also analysed four subsamples: persons with ages 25–44 years; ages 45–65 years; women; and men. Age is strongly predictive of hypertension and wages;[1–4,22] more importantly, correlations between morbidity, mortality and SES have been found to differ across age groups.[23,24] Moreover, for our first-dependent variable, persons with hypertension in base years are excluded for subsequent years, thus possibly imparting an age-based bias in the overall sample. For the age groups 25–44 years, there were 1742 (11%) person-years (out of 15 774) that were hypertensive in the first or subsequent base years. For the age groups 45–65 years, there were 2379 (27%) person-years (out of 8844) that were hypertensive in the first or subsequent base years. A higher percentage of the older group was removed from analysis, as would be expected given their increased risk of hypertension. There are also well-known gender differences for hypertension and wages.[1–4,22]

We used the PSID measure for wages which reflected subjects' annual income from work divided by annual work-hours, both measured in the year before the interview. We required that respondents worked ≥ 500 annual hours, or roughly ≥ 10 weekly. The great majority worked ≥ 2000 annual hours, or roughly ≥ 40 weekly hours. This wage-from-previous-year measured all earnings, including 'second jobs', self-employment, bonuses, overtime, tips and commissions. We adjusted wages for inflation using the Consumer Price Index.[25] Our analysis used the log-of-wage since the distribution of wages is skewed.[22] We multiplied log-of-wage by 10 to facilitate interpretations of odds and hazard ratios.

The hypertension variable was created from subjects' responses to this question 'has a doctor ever told you that you have hypertension or high blood pressure?' We coded this as '1' for 'yes' and '0' otherwise.

Independent variables were selected based on earlier studies.[1–13,15,16] Covariates were grouped in five categories: (i) demographic; (ii) other socio-economic (not wages) and geographic; (iii) health behaviours; (iv) other health variables (not hypertension); and (v) years. Age, race and gender are identified in table 1. Non-wage socio-economic and geographic variables included marital status, education, whether respondent had any medical insurance (including employer-provided, private or Medicare), whether self-employed, part-time employment, unemployed, salaried occupation, union member and four US Census regions (table 1). The regions indicated the current residence of the subject. For example, 'southern residence' indicated whether the subject resided in the Southern USA at the time of the interview. Part-time was defined as working < 35 h/week in weeks actually employed.[26] This < 35 h is a standard definition in the USA; in Europe < 30 h is frequently used.[26] Unemployed was defined as working < 45 weeks in the previous year. Again, USA and European standards differ. 'Non-salaried' indicated that subjects reported receiving wages or commissions rather than salaries and generally reflected blue-collar occupation. In additional analysis, we created two occupational indicator variables: one for professional or manager and another for clerical occupations. The results for wages were nearly identical in the additional analyses. Health habits included whether current smoker (≥ 1 cigarettes per day), whether ever smoked and whether heavy drinker (> 2 drinks per day).

We considered three measures of health other than hypertension: obesity (body mass ≥ 30), respondents' subjective evaluation of overall health and number of chronic health conditions (co-morbidities). Subjective health equalled '1' if respondent stated health was 'excellent', 'very good,' or 'good' and '0' otherwise. The number of chronic conditions is diagnosed by a doctor from this list: stroke, diabetes, cancer, lung disease, heart attack, heart disease, emotional problems, arthritis, asthma, mental loss or learning disorders.

But these health variables are problematic as covariates. First, it may be that these measures of health are in the causal pathway from low wages to hypertension. Low wages may lead to high body mass or poor health. Secondly, hypertension is a risk factor for four co-morbidities (stroke, diabetes, heart attack and heart disease) and respondents with hypertension are probably less likely to state that subjective health is 'good.' Any correlation between hypertension and subjective health or co-morbidities may therefore be tautological. If either these variables are in the causal pathway, or they are tautological, then obesity, subjective health and co-morbidities, should not be 'controlled for.' On the other hand, obesity and co-morbidities are risk factors for hypertension and are also believed to be predictors of wages.[22,27] Whereas we prefer arguments suggesting obesity, subjective heath and co-morbidities should be omitted, we nevertheless report regressions that included them.

Statistical method

For the first-dependent variable—hypertension (yes/no)—we used the discrete time event history model that requires logistic regression.[28] For the second-dependent variable—time-to-hypertension—we used the Cox proportional hazard model.[28]

The logistic approach has three advantages. First, it allows for wages to change over time. Secondly, the sample size is roughly triple that for the Cox model. Thirdly, unlike the Cox model, the logistic does not require that the exact time-to-event be known. Within the PSID, the exact time-to-event for hypertension within any given year is unknown. The Cox model has the advantage of allowing for the influence of

Table 1 Means and logistic and proportional hazards regressions for largest samples

Covariates	Means for sample size $n = 17\,295$	Logistic regression without three health variables, odds ratios and (*P*-values)	Cox regression without three health variables, hazard rates and (*P*-values)
Log-of-wages × 10 (continuous)	26.411	0.984* (0.015)	0.987*** (<0.001)
Male	0.490	1.250* (0.013)	1.059 (0.392)
Age (continuous)	40.264	1.077*** (<0.001)	1.076*** (<0.001)
African-American, non-Hispanic	0.252	2.255*** (<0.001)	1.592*** (<0.001)
Years of education (continuous)	13.293	0.956* (0.021)	0.979 (0.124)
Part-time, <35 h per week (yes/no)	0.117	0.902 (0.421)	0.758** (0.009)
Unemployed, <45 weeks (yes/no)	0.175	1.124 (0.230)	1.407*** (<0.001)
Southern residence	0.390	1.364* 0.016)	1.241* (0.030)
Heavy drinker (yes/no)	0.032	1.542* (0.027)	2.234*** (<0.001)

Only statistically significant covariates in one or the other regression enter this table. Both logistic and Cox regressions also included these covariates: Hispanic, all other non-Hispanic races, non-Hispanic white (referent), married, health insurance, self-employed, non-salaried, union member, Northeast, Midwest, other region, West (referent), current smoker, ever smoked and never smoked (referent). In addition, the logistic regression also included two indicator variables for years 2001 and 2003. Sample size for logistic regressions was 17 295 and for Cox regressions was 5651. Mean for hypertension incidence in logistic was 0.077 and for time to hypertension in Cox was 6.117.

***$P \leq 0.001$; **$P = 0.001$–0.01; *$P = 0.01$–0.05, all two-tailed tests. Tests for overall significance of both regressions were statistically significant at the $P < 0.001$ level.

wages to have cumulative effects over many years not just one subsequent year.

Random-effects procedures were used since data on some of the same individuals were combined across 4 years. The Stata 'svy' command (which accounts for geographic clusters) was not available for longitudinal data with random-effects procedures. For the Cox regressions, we used the Breslow method to break ties.[28]

We used Stata. Our sample sizes were 17 295 person-years in the overall sample for the logistic regressions and 5651 for the Cox models.

Results

Table 1 presents data from samples for both the logistic ($n = 17\,295$) and the Cox regressions ($n = 5651$). The first column of numbers presents means for the logistic sample. Hypertension incidence over 2 years averaged roughly 7.7% or 3.9% per-year, a reasonable estimate for aging cohorts. Mean real log-wages per hour was 26.4 ($17.96) for all the years combined. Only results for statistically significant covariates in one or the other regression are reported. The list of additional covariates is in the footnotes.

Table 1, second column of numbers, presents logistic results from regressing hypertension on all covariates except the three health variables: obesity, subjective health and co-morbidities. Log wages was negatively associated with hypertension ($P = 0.015$). The odds ratio (0.984) implied that doubling the wage (100% increase) was associated with a 16% decrease in chances of hypertension. Other notable statistically significant variables in the regression were: male (+, positively associated with hypertension), age (+), African-American (+), education (−), southern residence (+) and heavy drinker (+). In additional regressions, the three health variables were included. *P*-values on the three health variables were exceptionally low, <0.001 for two of them, and had the hypothesized signs: obesity (+), subjective health (−) and co-morbidities (+). The introduction of these three exceptionally powerful covariates increased the *P*-value for education to 0.188, above any significance level. The *P*-value on log wages was 0.090. Assuming the prior belief in the negative effect of low wages was sufficiently strong to warrant one-tailed tests, the corresponding *P*-value would have been one-half of 0.090.

The last column of table 1 presents results for the Cox model in which the three health variables were excluded. Time-to-hypertension averaged roughly 6.11 years (and included censored values). Mean real log wages per hour was 2.7 ($20.20/h) for all years combined. Log wages was negatively associated with hypertension ($P < 0.001$) in both the Cox regression reported in table 1 and an additional one that included the three health variables. The hazard ratio (0.987) in the reported regression implied that doubling the wage (100% increase) was associated with a 13% decrease in time-to-hypertension. The variables that achieved statistical significance were similar to those for the logistic regressions: African-American, southern residence, heavy drinker, obesity, subjective health and co-morbidities. There were also dissimilarities. Education was not significant in either Cox regressions but part-time employment and unemployment were significant in both. The Cox regression results suggested that part-time employment was associated with longer time-to-hypertension but that unemployment was associated with shorter time-to-hypertension.

Table 2 presents logistic regression results on subsamples: ages 25–44 years (column two of numbers), ages 45–65 years (column three), women (column four) and men (column five). In the interest of brevity, only results for log wages and education are presented. Results for education were deemed relevant given that education is frequently viewed as the most important non-income measure of SES in the USA.[2,29,30] Results on other covariates such as African-American, age, heavy drinker, obesity, subjective health and co-morbidities largely mirrored those in table 1. Considering column two, ages 25–44 years subsample, for both logistic regressions, log wage was a strong predictor of incident hypertension with *P*-values at 0.01 or below. Doubling the wage (100% increase) was associated with 25–30% decreases in chances of reporting hypertension. Education generated a *P*-value of 0.042 for the regression that excluded the three health variables, but a *P*-value of 0.170 for the regression that included them. For column three, ages 45–65 subsample, neither log wage nor education generated statistically significant *P*-values in either the regression with or without the three health variables. For columns four and five, gender differences were found. For women, in both regressions, log wage was a strong predictor of hypertension but education was not. For men, in both regressions, log wage did not achieve statistical significance but education did with *P*-values of 0.004 and 0.035 for regressions without and with the three health variables.

Table 3 presents Cox regression results that correspond to the same subsamples and covariate specifications as the logistic results in table 2. For seven of eight regressions, log wages generated *P*-values below 0.01 and in the male regression with three health variables, $P = 0.020$. In only one of eight regressions (male, without the three health variables) did education generate *P*-values below the 0.05 level. Again, results on other covariates such as male, African-American, age, southern residence, heavy drinker, obese, subjective health and co-morbidities were strikingly similar to those in table 1.

Discussion

We estimated the effect of wages on incident hypertension with logistic models and time-to-hypertension with proportional hazards models in longitudinal, nationally representative data on the USA working population for 1999–2005 ($n = 17\,295$ person-years). The design was prospective. For the overall samples and for subsamples of ages 25–44 years and women for both models, we found negatively and strongly statistically significant effects of wages on hypertension. The findings within subsamples differed somewhat between the two models. For the logistic models, whereas statistically significant associations were found for women, insignificant associations were found for the older group (45–65 years) and for men. For the proportional hazards models, statistically significant associations were found for all four subsamples. The stronger findings for younger groups have precedent in the

Table 2 Logistic regressions for dependent variable hypertension for age and gender subsamples

Regression and covariates	Odds ratios and (*P*-values)			
	Subsample for ages 25–44 years, $n = 11\,495$	Subsample for ages 45–65 years, $n = 5800$	Subsample for women, $n = 8814$	Subsample for men, $n = 8481$
Regression without three health variables				
Log-of-wages × 10 (continuous)	0.970** (0.002)	0.997 (0.714)	0.966*** (0.001)	0.997 (0.730)
Years of education (continuous)	0.941* (0.042)	0.972 (0.266)	0.999 (0.963)	0.927** (0.004)
Regression with three health variables				
Log-of-wages × 10 (continuous)	0.976** (0.009)	1.001 (0.890)	0.971** (0.002)	1.002 (0.829)
Years of education (continuous)	0.961 (0.170)	0.991 (0.714)	1.016 (0.585)	0.948* (0.035)

All regressions included covariates listed and footnoted in table 1. Tests for overall significance of all regressions were statistically significant at the $P < 0.001$ level

***$P \leq 0.001$; **$P = 0.001$–0.01; *$P = 0.01$–0.05, all two-tailed tests

Table 3 Proportional hazards regressions for dependent variable time to hypertension for age and gender subsamples

Regression and covariates	Hazard ratios and (*P*-values)			
	Subsample for ages 25–44, *n* = 3841	Subsample for ages 45–65, *n* = 1810	Subsample for women, *n* = 2786	Subsample for men, *n* = 2865
Regression without three health variables				
Log-of-wages × 10 (continuous)	0.984** (0.001)	0.988** (0.002)	0.985*** (<0.001)	0.988** (0.004)
Years of education (continuous)	0.972 (0.231)	0.983 (0.337)	0.997 (0.887)	0.960* (0.040)
Regression with three health variables				
Log-of-wages × 10 (continuous)	0.984*** (<0.001)	0.989** (0.006)	0.985*** (<0.001)	0.991* (0.020)
Years of education (continuous)	1.004 (0.854)	1.005 (0.795)	1.022 (0.302)	0.984 (0.414)

All regressions included covariates listed and footnoted in table 1. Tests for overall significance of all regressions were statistically significant at the $P < 0.001$ level

***$P \leq 0.001$; **$P = 0.001$–0.01; *$P = 0.01$–0.05, all two-tailed tests

literature.[23,24] Shishehbor *et al.*[24] note that there is no consensus regarding why the correlation between SES and circulatory disease should weaken for older age groups, but suggest that 'better coping skills at older ages, perhaps due to having lived longer under unfavourable socio-economic circumstances' may play a role. Attrition could also be a factor, especially in our samples of working people. Persons with low SES are more likely than persons with high SES to drop out of the labour market altogether as they age.[22] The difference in health status between low and high-SES persons in their 20s and 30s who work is likely greater than the difference in health status between low and high-SES persons in their 50s and 60s who continue to work. The findings for women may have resulted from the greater validity of survey blood pressure measurements for women than men.[31] Whereas there appears to be some evidence for low wage and hypertension associations among all four age and gender groups depending on regression models, we prefer the evidence for women and the younger (25–44 years) groups because of the strong statistical significance in every model and because the previous literature supports the hypothesis that SES and disease correlations weaken with age.

A separate issue involves the exclusion of the three health variables—obesity, subjective health and number of co-morbidities—from the models. We believe these three health variables are in the causal pathway running from wages to hypertension and therefore should be excluded. In an initial attempt to test this hypothesis, we regressed the three health variables on log wages and other covariates in all samples and subsamples. In every regression, log wages were predictive ($P < 0.05$ in overall samples) of all three in the expected direction: obesity (−), subjective good health (+), co-morbidities (−). But in additional results in which we switched dependent and independent variables, we found that the three health variables were frequently statistically significant covariates for wages. Given that wages and the three health variables were measured in the same (not subsequent) years, these results were not surprising. We conclude that a proper test of the 'causal pathway' hypothesis must await future research. Nevertheless, it bears mentioning that our results regarding the inclusion and exclusion of other health variables has precedence from the literature. Adams *et al.*[8] use national samples of seniors (aged > 69 years) and find that when measures of morbidity and current health status are included in the analysis, SES variables lose their otherwise strong statistical significance in predicting mortality.

We are not aware of other studies that consider whether union members are more or less likely to report hypertension. Whereas in all 20 regressions we found negative correlations—implying union members were less likely to report hypertension—we found only one $P < 0.05$. Nevertheless, we believe variables measuring unionization ought to receive future public health attention given significant social science research suggesting positive effects of unions on workers independent of wages.[22]

Our study was directed at only one aspect (wages) of one component (income) of SES. We controlled for education with years of schooling and occupation with indicator variables for 'non-salaried', union member and two broad categories (manager or professional and clerical) yet nevertheless found strong wage-hypertension associations. It is therefore useful to consider the literature on SES and hypertension, as well as SES and other measures of health. First, the hypertension literature emphasizes education and occupation rather than family income, and we are unaware of any studies on wages. In the recent Grotto *et al.*[1] review, only one study,[32] gives considerable attention to income. In the older review by Colhoun *et al.*[2] if we count only studies for which direct measures of education, occupation and income are used, we find 31 studies with education, 9 with occupation and 7 with income.

Secondly, for health measures other than hypertension, there is considerable evidence for income. Duleep[33] uses national longitudinal data from the Social Security Administration to demonstrate strong associations between low income and mortality. McDonough *et al.*[34] use PSID data from 1968 to 1989 and find that low income, especially 'persistent' low income, is predictive of mortality. Additional evidence for income links to morbidity and mortality are available in Sullivan and Wachter,[6] Ettner[7] and Frijters *et al.*[35]

Thirdly, our findings are consistent with literature on occupations and hypertension. The general finding is that low-status jobs or jobs with high 'job strain' are associated with hypertension.[12,13,36] Low status and high-job strain are likely associated with low wages.

Any rumination on the biological causes for wages influencing blood pressure is speculative. Marmot[5] cites animal model evidence that changes in social status result in changes in health and a plethora of studies on humans demonstrating associations between social rank and circulatory disease, especially among British civil servants. One hypothesis involves self-esteem. Evidence suggests that low wages are associated with low self-esteem[16] and that low self-esteem and low happiness, through neuroendocrine and cardiovascular processes, are associated with hypertension.[5,15–18] Another hypothesis is that low wages can create stress not due to social rank but due to daily 'hassles' such as insufficient money to buy gas to get to work.[37]

Our data and method have strengths. First, the PSID is highly regarded and widely used, especially by social scientists. From its inception to 2011, roughly 444 papers had been published under the broad category of 'health' and 966 under the category of 'earnings'.[38] Secondly, our results are consistent with the large literature demonstrating links between measures of SES and circulatory disease.[1,2] Thirdly, results on many control variables are consistent with the epidemiologic literature on risk factors for hypertension. [27] These control variables included: male, African-American, age, southern residence, heavy drinking, obesity, subjective health and number of chronic conditions. The fact that so many of our results on control variables were consistent with existing literature augers well for the credibility of our data and methods.

There are limitations. First, hypertension is self-reported. However, considerable evidence suggests that self-reported hypertension is strongly correlated with objectively measured readings of blood pressure and has high validity.[31,39] Secondly, it might be that we under-estimated the effects of wages. Attrition might bias our findings. It is likely that a disproportionate number of persons in low wage jobs or

those who had hypertension died over these years or dropped out of the labour market.[33–35] Had there been no premature death or dropping out, the number of respondents in lower wage jobs would have increased and the estimated incident hypertension within those lower wage jobs would have been higher. Thirdly, it could be that unobserved 'third variables' such as time preference (ability to delay gratification) could be the cause of both hypertension and low wages. But the random-effects model was designed to control for possible spurious correlations that might arise from unobserved individual characteristics that do not change over time. Moreover, smoking prevalence, which we controlled for, is alleged to indirectly measure time preference.[9] Fourthly, we did not have direct measures of occupational risks such as exposure to lead, carbon monoxide, nitroglycerin or 'job strain'.[40] However, we did include four indicator occupation variables. Fifthly, the variable measuring number of chronic conditions is strongly correlated with health-care use and the latter is lower in poorer populations.[14] Finally, our dependent variable did not measure the severity of hypertension.

In conclusion, these nationally representative, longitudinal data provided evidence for correlations between wages and hypertension among working persons. Because of our prospective design and control for numerous possible confounders, we believe the evidence suggests that low wages were risk factors for hypertension, especially for women and persons aged 25–44 years.

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Key points

- SES has three large components: education, occupation and income. Wages are, by far, the largest subcategory of income for working people. Moreover, wages are a critical part of the quality of occupations and can be targeted by government policies such as with minimum wage legislation.
- We are unaware of any studies that address correlations between wages (as opposed to family income) and incidence of hypertension.
- The prospective design allows us to measure wages in base years among persons without hypertension and, in subsequent years, measure whether subjects report hypertension. This design minimizes the possibility of reverse causality whereby hypertension results in lower wages.
- The strongest results suggest that low wages are risk factors for hypertension among women and all persons aged 25–44 years.

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Ethnic differences in self-rated overweight and association with reporting weight loss action: the SUNSET study

Mary Nicolaou[1,2], Irene G. van Valkengoed[1], Colleen M. Doak[2], Rob M. van Dam[3], Karien Stronks[1], Jaap C. Seidell[2]

1 Department of Public Health, Academic Medical Centre, University of Amsterdam, Amsterdam, The Netherlands
2 Department of Health Sciences, Faculty of Earth and Life Sciences, VU University, Amsterdam, The Netherlands
3 Departments of Epidemiology and Public Health and Medicine, Yong Loo Lin School of Medicine, National University of Singapore, Singapore

Correspondence: Mary Nicolaou, Department of Public Health, AMC, PO Box 22660, 1100 DD Amesterdam, The Netherlands. Tel: +31 20 5665033; Fax: +31 20 6972316, e-mail: m.nicolaou@amc.uva.nl

Aim: To investigate ethnic differences in self-rated overweight and self-reported weight loss action. **Methods:** Cross-sectional study (conducted in 2001–03) of 1441 residents (35–60 years) of Amsterdam, The Netherlands: Surinamese of South Asian (SA-Sur) and African (Afr-Sur) origin and ethnic Dutch. Self-rated overweight and self-reported weight loss action assessed by questionnaire. Height, weight and waist circumference (WC) measured in a clinic setting. **Results:** Compared with ethnic Dutch and adjusting for BMI, Afr-Sur men [odds ratio (OR) 0.32; 95% CI 0.19–0.57] and women (OR 0.54; 95% CI 0.34–0.86) were less likely to rate themselves as overweight. However, adjustment for WC reduced differences in self-rated overweight (men: OR 0.79; 95% CI 0.46–1.35; women: OR 0.89; 95% CI 0.59–1.36). SA-Sur participants did not differ significantly from ethnic Dutch when adjusting for either BMI or WC. Surinamese participants were significantly more likely to report weight loss action independent of BMI, WC or self-rated overweight. In Afr-Sur men, elevated WC, not BMI was associated with reported weight loss action (OR 2.31; 95% CI 1.35–3.99 vs. OR 1.52, 95% CI 0.89–2.58, respectively). **Conclusion:** In this population, differences in self-rated overweight were explained by measured weight variables (BMI or WC). Our results do not support the hypothesis that Surinamese migrants would be less likely to be attempting weight loss than their Dutch peers. Further research into the reasons underlying this finding and associated weight loss behaviour seems indicated.

Introduction

The increasing prevalence of overweight and obesity is a major international public health problem.[1] In many Western populations, ethnic minority groups, particularly women, often suffer from higher rates of both overweight and obesity.[2–4,5,6]

African origin and South Asian groups form two of the largest ethnic minority groups in many Western countries, including The Netherlands.[7] Both groups experience high risk of obesity-related lifestyle diseases such as type 2 diabetes, hypertension, coronary heart disease and stroke,[8–10] making them important to consider when planning weight loss interventions.

Possible explanations for ethnic differences in the prevalence of overweight and obesity are numerous[11] and there is evidence that how individuals rate their own body weight, i.e. whether they perceive themselves as overweight or not, may be an important underlying determinant. Reports in the literature indicate that self-rated body weight is likely to influence motivation to avoid weight gain[12] and that incorrect evaluation of one's body weight may be a barrier to healthy lifestyles.[13] Previous research has shown that African Americans are less likely to describe themselves as overweight than White Americans.[14–16] Similarly, UK South Asian women are less likely to describe themselves as overweight compared with White English women.[17,18]

Although there have been many studies of self-rated weight among different ethnic groups,[14,15,19] few have looked at ethnic differences in the relationship between this and reported weight loss action[12,17,20] in both men and women.[21,22] In this study, we investigated ethnic differences in the self-rated body weight and the associations with self-reported weight loss action in three ethnic groups residing in Amsterdam, The Netherlands: Surinamese first-generation migrants of South Asian or African origin (hereafter referred to as South Asian Surinamese or African Surinamese) and ethnic Dutch. We hypothesise that:

(i) South Asian and African Surinamese migrants are less likely rate themselves as overweight than ethnic Dutch.
(ii) Given lower self-rated overweight, South Asian and African persons will be less likely than ethnic Dutch to report trying to lose weight.

Methods

Survey design

Data were obtained from the SUNSET study (acronym for: **SU**rinamese in The **N**etherlands: **S**tudy on **ET**hnicity and health), a cross-sectional study that aimed to assess the cardiovascular risk profile of three ethnic groups in Amsterdam, The Netherlands: Surinamese South Asians, Surinamese of African origin and ethnic Dutch. The SUNSET study is based on a stratified random sample of 2975 individuals, aged 35–60 years, drawn from the population register of two neighbourhoods in Amsterdam. These neighbourhoods were selected due to their large concentration of Surinamese-origin residents. For the sampling, persons who were born in The Netherlands and whose parents were both born in The Netherlands were presumed to be ethnic Dutch. Persons of whom both parents were born in Surinam or persons who were born in The Netherlands and who had at least one parent who was born in Surinam were presumed to be Surinamese.