Migraine prevalence, socioeconomic status, and social causation

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ABSTRACT

Objective: To determine whether the known higher prevalence of migraine in lower household (HH) income groups is explained by a higher incidence rate or a lower remission rate.

Methods: We used data from the American Migraine Prevalence and Prevention Study, a US national sample of 132,674 females (with a 64.3% response rate) and 124,665 males (with a 62.0% response rate) 12 years of age and older. Data were previously collected on migraine symptoms, onset age, and demographics. Previously validated methods applied to the American Migraine Prevalence and Prevention Study data were used to simulate a cohort study. Incidence and remission rates were estimated within 3 sex-specific HH income groups (<\$22,500, \$22,500–\$59,999, and ≥\$60,000). The χ^2 test was used to determine whether the incidence or remission rates differed by HH income group as an explanation for differences in migraine prevalence by HH income.

Results: Migraine prevalence increased as HH income decreased for females (χ^2 , $p < 0.01$) and males (χ^2 , $p < 0.01$). Differences were not explained by race and other known confounders. Variation in prevalence was explained, in large part, by a higher incidence rate in the lower HH income groups for both females (χ^2 , $p < 0.01$) and males (χ^2 , $p < 0.01$). Migraine remission rates did not differ by HH income.

Conclusions: The higher incidence of migraine in lower HH income groups is compatible with the social causation hypothesis. Once initiated, migraine remission is independent of HH income. Onset and remission may have etiologically distinct causes. *Neurology*® 2013;81:948–955

GLOSSARY

AMPP = American Migraine Prevalence and Prevention; **HH** = household; **MIDAS** = Migraine Disability Assessment; **SES** = socioeconomic status.

In US studies, migraine prevalence increases as socioeconomic status (SES) decreases,¹ a pattern common to many diseases.^{2–12} Two general hypotheses¹³ have been posited. The social selection hypothesis postulates that a disabling disease can cause a decline in social status because an individual may underperform in educational or occupational achievement. For the social causation hypothesis, low SES is associated with stress and other disease mediators that increase the incidence or duration (i.e., lower remission rate) of illness. Differentiating the relative contribution of each of these causal models is important to understanding disease etiology.

In 1992, we judged that both social selection and social causation were plausible explanations for the fact that migraine prevalence was substantially higher in lower income groups.¹ Migraine usually begins in adolescence or early adult life and is sufficiently disabling³ to interfere with educational or occupational achievement and cause economic downward drift. Alternatively, more abundant exposure to stressors in lower income groups could cause either a higher incidence or a longer disease duration (i.e., lower remission rate), plausibly supporting the social causation hypothesis.

To test these alternative hypotheses, we developed distinct predictions (table 1). For social selection, we predicted that neither disease incidence nor remission is related to SES because the

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Table 1 Expected impact of low household (HH) income on migraine incidence and remission rates by causal explanation

Causal explanation	Expected impact of low HH income	
	Incidence	Remission
Social selection	None	None
Social causation (increased incidence)	Higher	None
Social causation (lower remission or prolonged disease duration)	None	Lower

association is attributable to individuals with migraine moving from a higher income to a lower income group. Under the social causation hypothesis, low SES may be associated with an increased incidence rate of migraine. Additionally, Low SES could increase disease duration as measured by a lower remission rate.

METHODS American Migraine Prevalence and Prevention (AMPP) Study data were used to determine whether migraine incidence and migraine remission rates differ by household (HH) income. The source population and survey data and statistical methods are described in detail elsewhere,³ and summarized in brief herein.

Standard protocol approvals, registrations, and patient consents. No patient identifiers were available to the study team. This study was approved by the Geisinger and Einstein Institutional Review Boards and was supported by the National Headache Foundation.

Source population and data. The AMPP Study used the same methods as the American Migraine Studies 1 and 2.^{1,3} A screening questionnaire was mailed in 2004 to a stratified random sample of 120,000 US HHs from a panel maintained by the Taylor Nelson Sofres/National Family Opinion, Inc.³ The survey was completed by the designated head of the HH who reported the HH census and on those having at least occasional self-defined severe headache. Of 257,339 eligible HH members, data were obtained on 162,705 individuals aged 12 years and older, including 85,373 females (64.3% response rate) and 77,332 males (62.0% response rate).³ HH members with severe headaches completed a questionnaire that included the age at first severe headache, 21 questions to assign clinically validated headache diagnoses,^{2,14} and the Migraine Disability Assessment (MIDAS) questionnaire. The latter included a question on the total number of headache days in the past 3 months that is specific to migraine headache days.

An individual was designated as having active migraine if they reported at least one severe headache in the previous year. Chronic migraine was defined as more than 15 severe headaches per month that met established criteria¹⁴ for migraine. A total of 4,364 (5.6%) males and 14,604 (17.1%) females met criteria for active migraine.

Confounding of migraine prevalence by HH income. In order of magnitude, migraine prevalence varies by sex, age, SES, and race and to a lesser extent by other potential confounders (e.g., marital status). In preliminary linear regression analysis, neither race nor marital status was statistically significant in explaining variance in age-specific migraine prevalence by HH income for either females or males. As such, we did not consider either of these variables in our estimates of incidence or remission

rates. We did not include education as a confounder because it is strongly related to and represents the same socioeconomic domain as HH income.

Statistical methods for incidence rate estimates. We previously demonstrated how survey data can be used to simulate the longitudinal experience of a cohort as an efficient method for estimating incidence rates. The current age of all respondents and the age at onset of migraine are required.^{15–17} A cohort structure is simulated by first organizing data on those with migraine by their current age and age at onset (see example in table e-1 on the *Neurology*[®] Web site at www.neurology.org) and then accounting for person-time from birth for all respondents. In the simulation, migraine cases are censored at the age of onset and all other respondents are censored at the age of interview. A variable called LAG is calculated as the current age minus the age at onset of migraine (see table e-1). The age-specific incidence rate is highest for recent onset cases (i.e., LAG is zero or close to zero) because few or no cases have remitted. As LAG increases, the age-specific incidence rate appears to decrease—this is largely explained by remission that naturally increases as time from onset increases. The previously validated estimating methods use information in this array to simultaneously estimate age-specific incidence and remission rates.

In our original estimating method,¹⁶ incidence was modeled as a smooth parametric function of age and LAG. Age-specific incidence rates were then estimated from the prediction curve where LAG was set to zero. This approach assumes that if onset was in the recent past, then recall is probably less error prone. A drawback to this approach is that a fully parametric model was assumed for the bivariate rate function of age and LAG. In addition, it is unclear what effect recall error and/or bias has on the estimated incidence rates. We addressed both of these issues in our more recent method, where the form of the dependency between rate and age and LAG is assumed to be unknown, and could have a complex nonlinear form.¹⁷ We explored semiparametric models (penalized splines) for this purpose. Our simulation studies showed that this approach is quite robust to a variety of types of recall error.

Statistical methods for remission rate estimates. We previously described methods for estimating remission rates from the same type of survey data, where age at onset is reported.¹⁸ We define remission as having had no attacks in the past year. As noted and described in table e-1, remission is apparent by examining how an age-specific incidence rate declines (table e-1 columns) as LAG increases. This method^{15–18} assumes that there are no major secular changes in incidence, a finding that is supported by AMPP studies completed over the past 2 decades.

Prevalence, incidence, and remission were all estimated simultaneously. Sex- and age-specific prevalence were estimated directly from the AMPP data, incidence rates were estimated as described below, and remission rates were estimated using equation 1 of Roy and Stewart¹⁸ and as detailed below.

Estimating sex-, age-, and HH income-specific incidence and remission rates. HH income groups were defined a priori and based, in part, on previous work.¹ To increase statistical power and to reduce computational demand in estimating incidence and remission rates, we reduced the number of income categories from 5 strata, as previously described,¹ to 3 income strata. The lowest income tertile was defined for females (<\$22,500) and the remainder were approximately equally divided into 2 HH income groups (i.e., \$22,500–\$59,999 and ≥\$60,000). The HH income categories for males were defined using these same

categories. We estimated sex- and age-specific prevalence, incidence, and remission rates using the methodology as described and formally tested for differences in each rate by HH income as follows. We specified a single model for each sex that included interaction terms by income category (income by age interaction in the spline model). For the interaction terms, we specified spike and slab priors for the coefficients, with a common spike.¹⁹ Essentially, there is an unknown probability that all of the coefficients of the interaction terms are zero. The posterior mean of the spike can be thought of as the posterior probability of no interaction between income and the outcome. This posterior mean is what we use to “test” for an interaction.

RESULTS Baseline survey participants were between 12 and 100 years of age; 52.5% were female (table 2). A total of 23.1% of females and 16.9% of males were in the lowest income tertile. HH income tertiles

significantly differed by age and race for both females and males. The lowest income group was older and less likely to be Caucasian.

Of the 85,373 females, 17.1% had current or active migraine; 27.3% were in the lowest income tertile. Of the 77,332 males, 5.6% had current migraine; 26.7% were in the lowest income tertile. Both female and male migraine cases in each HH income group significantly differed in their distribution by average pain intensity, headache frequency, MIDAS grade, and use of prescription medications for headache in the past year. Females and males in the lowest income group were more likely to have extremely severe pain and grade IV MIDAS impact, less likely to have infrequent headache days (i.e., 0–3 per month), and more likely to have used prescription medications in the past year (table 3).

Prevalence of migraine peaked at age 38 in females and at age 40 in males. Age-specific prevalence was higher among those in the lowest HH income group (figure, A and B). Female and male age-specific prevalence was significantly different by HH income (table 4). Incidence peaked at age 26 in females and at a younger age than in males. Age-specific incidence was significantly higher among those in the lower HH income groups for both females and males (figure, C and D; table 4). Age-specific remission rates did not differ by HH income in either females or males (figure, E and F; table 4).

DISCUSSION The results from this study are not compatible with the social selection or downward drift hypothesis. Notably, initiation of migraine (incidence) appears to be the dominant mediator of the observed higher prevalence of migraine in lower income groups. Moreover, once initiated, the duration of time that an individual will have migraine does not appear to differ by HH income. The latter is inferred because remission rates do not differ by HH income. In general, these findings strongly support social causation as a mediator of variation in prevalence by HH income, but they do not exclude social selection as a possible explanation of some of the HH income variance. Measures of pain and disability are more severe in lower HH income groups, a finding that is also consistent with the social causation hypothesis.

A higher prevalence of migraine has been consistently reported^{1,2,20–29} among lower income or lower education groups in the United States^{1,2,20,25–27} and elsewhere.^{21–24,28,29} Exceptions^{30–33} are likely explained by small samples, low variability income, or high levels of social support. While there is a direct effect of low income on health, HH income itself is also a surrogate for a number of social, psychological, and physical exposures, some of which may mediate migraine onset. The fact that migraine incidence is related to HH

Table 2 Percent distribution of the original AMPP Study population by sex and HH income, and by established and meaningful confounders of migraine prevalence

Variable	HH income			Total
	<\$22,500	\$22,500–\$59,999	≥\$60,000	
Females	n = 19,767	n = 31,163	n = 34,443	n = 85,373
Age at interview,^a y, %				
<15	3.3	4.0	4.3	4.0
15–24	10.0	11.6	13.4	12.0
25–34	10.2	14.1	10.7	11.8
35–44	11.8	16.1	18.4	16.0
45–54	15.0	17.3	24.0	19.5
55–64	14.5	14.5	17.4	15.7
65+	35.2	22.5	11.8	21.1
Race, %^a				
White	80.5	84.3	89.8	85.6
Black	11.9	9.7	5.0	8.3
Other	7.6	6.0	5.2	6.0
Males	n = 13,057	n = 45,632	n = 18,643	n = 77,332
Age at interview,^a y, %				
<15	4.6	4.7	4.2	4.5
15–24	15.9	13.0	14.4	13.8
25–34	11.4	12.7	8.8	11.5
35–44	12.6	16.5	16.9	15.9
45–54	15.3	17.3	22.3	18.2
55–64	13.8	14.8	20.7	16.0
65+	26.5	21.1	12.8	20.0
Race, %^a				
White	83.4	87.5	91.0	87.7
Black	8.8	6.7	3.5	6.3
Other	7.9	5.8	5.5	6.1

Abbreviations: AMPP = American Migraine Prevalence and Prevention; HH = household.

^a The χ^2 test was statistically significant ($p < 0.01$) for differences in percent distribution of respondents by age and race across sex-specific HH income groups.

Table 3 Percent distribution of the original AMPP Study population, by HH income and by headache features (percentages sum to 100% within columns)

Headache feature	HH income			Total
	<\$22,500	\$22,500–\$59,999	≥\$60,000	
Females	n = 3,992	n = 5,550	n = 5,062	n = 14,604
Average pain of severe headaches, % ^a				
Mild	2.0	0.8	0.5	1.0
Moderately severe	15.5	14.1	14.4	14.6
Severe	41.4	48.7	51.1	47.5
Extremely severe	40.4	36.0	33.6	36.4
Unknown	0.7	0.5	0.4	0.5
Headache days per month, % ^a				
0–3	46.5	61.9	70.0	60.5
4–10	32.1	26.7	22.0	26.6
11–15	5.9	11.4	8.0	13.0
MIDAS grade, %				
I (0–5)	45.4	52.0	57.7	52.2
II (6–10)	14.8	15.8	15.6	15.4
III (11–19)	10.0	10.9	10.0	10.3
IV (20+)	17.0	13.3	10.2	13.2
Unknown	12.9	8.1	6.4	8.8
Prescription use for headache in past year, % ^a				
No	62.4	71.7	73.5	69.8
Yes	34.9	26.3	24.5	28.0
Unknown	2.7	2.0	2.0	2.2
Males	n = 1,165	n = 1,580	n = 1,619	n = 4,364
Average pain of severe headaches, % ^{a,b}				
Mild	2.9	1.5	1.0	1.7
Moderately severe	16.7	14.8	13.3	14.8
Severe	40.3	45.9	47.7	45.1
Extremely severe	39.3	37.2	37.6	37.9
Unknown	0.7	0.6	0.4	0.6
Headache days per month, % ^{a,b}				
0–3	43.7	58.0	68.2	58.0
4–10	32.6	28.8	22.4	27.5
11–15	23.7	13.2	9.4	14.6
MIDAS grade, % ^a				
I (0–5)	53.9	63.5	66.9	62.2
II (6–10)	11.6	10.6	10.0	10.6
III (11–19)	8.7	6.3	6.6	7.0
IV (20+)	12.6	9.2	8.2	9.7
Unknown	13.2	10.4	8.3	10.4

Continued

income may suggest that the etiology of migraine onset is strongly related to exogenous factors. For example, physical and psychological stress may contribute to migraine onset,³⁴ as the frequency of stressful events is known to be more common in lower HH income groups³⁵ and these same factors may mediate variation in attack frequency.³⁶ Exogenous mediators may also differ by sex. For example, women in lower income groups may be more likely to experience abuse or neglect than men, factors known to influence migraine prevalence.³⁷ In contrast, migraine remission may be more strongly related to endogenous or genetic factors. These findings may also be relevant to other chronic episodic disease, most of which are more common among those in lower SES groups.^{4–10}

The cumulative lifetime risk of migraine is 43% in females and 18% in males.¹⁶ In contrast, the 1-year period prevalence of active migraine (i.e., 17% in females, 5% in males) is substantially lower, indicating that remission is common.¹ The lack of variation by HH income may indicate that remission is not strongly related to exogenous factors, including the known better access to health care among higher income groups.¹ Instead, remission may be more strongly mediated by endogenous and genetic factors. More generally, the genetic liability for persistent migraine may primarily be attributable to predisposition to remission.^{38–40,c1}

Previous twin and family aggregation studies have not separately considered genetic liability for migraine onset vs migraine persistence or remission. In a population-based sample of active migraine cases, individuals who are less likely to remit are overrepresented. The limited progress in identifying specific migraine genes may be explained, in part, by the failure to separately search for genes that determine onset and persistence. Notably, because of the high remission rate for migraine, it is likely that individuals who have had migraine and remitted may be included in the control group of a genetic case-control study. More importantly, an active migraine case group sampled from the general population will have a highly heterogeneous prognosis, varying substantially by both past and future duration of disease. The study of migraine genetics may be improved by selecting both remitted and active migraine cases to be representative of the original incidence case pool.

Factors that contribute to migraine onset may also contribute to attack frequency. Low HH income was also associated with increased attack frequency. This would offer a straightforward explanation for why attack frequency varies by HH income. However, changes in attack frequency may not be strongly related to the duration of time that an individual experiences migraines. That is, the factors involved in mediating remission may also differ from those that

Table 3 Continued

Headache feature	HH income			Total
	<\$22,500	\$22,500–\$59,999	≥\$60,000	
Prescription use for headache in past year, % ^{a,b}				
No	63.0	72.9	74.6	70.9
Yes	33.7	24.8	23.5	26.7
Unknown	3.4	2.3	1.9	2.5

Abbreviations: AMPP = American Migraine Prevalence and Prevention; HH = household; MIDAS = Migraine Disability Assessment.

^aThe χ^2 test was statistically significant ($p < 0.01$) for differences in percent distribution of respondents by age and race across sex-specific HH income groups.

^bEvidence indicates that all or most headaches experienced by those with migraine are pathophysiologically migraine headaches. As such, the measures of average pain intensity and headache days refer to migraine headaches. Prescription drug use is also for migraine headaches.

mediate increased attack frequency, and more severe pain from attacks.

The approach we have used to examine HH income and migraine in this study may be applicable to other chronic health problems with episodic manifestations, most of which are more common in lower HH income groups.^{4–10} A higher prevalence of active disease has been reported in lower income groups for epilepsy, asthma, gastroesophageal reflux disease, allergic rhinitis, abdominal pain, low back pain, osteoarthritis, and urinary incontinence, among others. Population-

based studies of these conditions have focused on active prevalent cases for the most part, with only limited attention to incidence and remission. Moreover, psychological factors (e.g., response to stress, anxiety, depression) and related comorbidities may be important in modifying the effect of environmental stressors for chronic episodic conditions.^{c2–c4}

A potential limitation to our findings is that confounders could explain differences in prevalence or incidence by HH income. Because we have accounted for the dominant confounders (i.e., sex, age, HH income), it is not likely that other factors represent a meaningful threat to inference. In order of magnitude, migraine prevalence is known to differ by sex, age, HH income, and race. We completed linear regression analysis adjusting prevalence estimates by HH income for race and marital status. Neither variable was significant. After race, the next most significant consideration is depression and anxiety, factors that are known to be associated with a higher incidence of migraine^{e5,e6} and more prevalent in lower income groups. However, we would not characterize depression and anxiety as possible confounders because they are more likely to be mediators of migraine onset for which adjustment would not be sensible. We did not adjust for education, known to be associated with migraine prevalence, because it is a surrogate for HH income (i.e., our primary stratification variable used to test hypotheses regarding incidence and remission rates). Finally, body

Figure Estimated age-specific prevalence (A and B) and incidence (C and D) and remission (E and F) rates for females and males by household income

For females and males, the black curve is for a household income <\$22,500, the red curve is \$22,500–\$59,999, and the blue curve is ≥\$60,000.

Table 4 Age-specific prevalence and incidence and remission rates by age, sex, and HH income, from the original AMPP Study

	Age group, y							Test of difference by HH income
Measure	<15	15-24	25-34	35-44	45-54	55-64	65+	
Females								
Prevalence (per 100 persons)								
<\$22,500	7.0	19.5	36.5	39.2	31.3	19.6	11.1	<0.01 ^a
\$22,500-\$59,999	4.9	15.2	29.1	29.3	22.1	14.0	8.1	
≥\$60,000	4.2	10.4	20.3	23.1	17.9	10.3	6.0	
Incidence rate (per 1,000 person-years)								
<\$22,500	13.4	22.2	21.2	15.2	8.0	3.7	0.4	<0.01 ^a
\$22,500-\$59,999	8.9	16.1	14.8	8.5	3.7	0.9	0.2	
≥\$60,000	6.4	10.0	9.6	6.3	2.6	0.3	0.0	
Remission rate (per 1,000 person-years)								
<\$22,500	12.5	6.1	5.0	5.3	6.3	8.4	11.1	NS
\$22,500-\$59,999	9.7	3.8	4.3	5.4	5.7	7.3	10.1	
≥\$60,000	15.3	5.3	3.1	4.5	6.3	7.6	9.7	
Males								
Prevalence (per 100 persons)								
<\$22,500	6.5	9.0	13.0	15.0	13.0	7.6	4.2	<0.01
\$22,500-\$59,999	3.9	5.5	7.6	8.3	6.8	4.1	2.3	
≥\$60,000	3.7	4.3	5.2	5.7	4.8	3.2	2.2	
Incidence rate (per 1,000 person-years)								
<\$22,500	8.7	7.9	5.4	5.0	2.6	0.7	0.2	<0.01
\$22,500-\$59,999	5.1	4.7	3.6	2.6	1.5	0.5	0.1	
≥\$60,000	3.7	2.9	1.2	0.8	0.6	0.3	0.1	
Remission rate (per 1,000 person-years)								
<\$22,500	13.9	5.2	2.4	4.2	7.3	9.7	11.6	NS
\$22,500-\$59,999	13.7	7.6	4.1	4.3	6.4	8.9	11.2	
≥\$60,000	14.8	6.7	1.6	2.3	5.9	7.2	5.8	

Abbreviations: AMPP = American Migraine Prevalence and Prevention; HH = household; NS = not significant.

^aThe χ^2 test was statistically significant ($p < 0.01$), indicating differences in the prevalence or incidence rate across sex-specific HH income groups.

mass index, alcohol and tobacco use, and access to health care (e.g., physician diagnosis) may also influence prevalence rates. While these factors are associated with attack frequency and possibly attack severity, the evidence is uncertain for body mass index and it is mixed for a relation to migraine prevalence, especially for alcohol and tobacco use. Alcohol use may be associated with lower migraine prevalence and tobacco use may be associated with higher migraine prevalence²⁸ but evidence is not consistent across studies. More generally, our finding that HH income is not related to remission rates indicates that confounders of migraine prevalence by HH income are inconsequential in explaining migraine remission rates by HH income.

Finally, we consider 3 possible limitations. First, we used HH income at the time of interview, not around the time of onset of migraine. Errors in assigning a respondent to an HH income category will

likely reduce the strength of associations. The significance of our findings indicates that the errors are not likely to be substantial. The median age at onset of migraine is in the third decade of life¹⁶ and HH income at this time is correlated with HH income in the period before and after onset. Moreover, table e-1 shows that more than half of the female migraine incident cases occur within 5 years of the interview, a time that is relatively proximal to the time of reporting HH income. Second, we excluded chronic migraine because of the substantial risk of underemployment and the direct impact on HH income. This form of reverse causation could introduce potentially significant confounding by HH income. We recognize that chronic migraine is more common in lower income groups and as a result we may have overestimated remission in the lower income group. Because there is a 12:1 ratio of

episodic migraine cases to chronic migraine cases, we would not expect much of an effect on remission rates by including chronic migraine cases in the analysis, assuming that chronic migraine is related to duration of disease. Finally, we considered the possibility that the findings could be an artifact of the modeling process that we used. Specifically, if incidence was estimated first within SES strata, it might result in a finding whereby the age-specific incidence estimates mirror the age-specific prevalence estimates and estimates of remission are deterministically estimated from incidence and prevalence. However, prevalence, incidence, and remission were estimated simultaneously to avoid any possible effect that the order of estimating might have on the findings.

AUTHOR CONTRIBUTIONS

Dr. Stewart and Dr. Lipton were responsible for the study concept and design. Dr. Lipton was responsible for the acquisition of the data. Dr. Stewart and Dr. Roy were responsible for the analysis and interpretation of the data. All 3 authors were responsible for the critical revision of the manuscript for important intellectual content. There was no study supervision.

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DISCLOSURE

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