

Socioeconomic Status and Ulcer

A Prospective Study of Contributory Risk Factors

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Peptic ulcer is associated with low socioeconomic status. In this study we used longitudinal population-based data to investigate factors other than *Helicobacter pylori* that might contribute to this association.

Of 4597 Alameda County Study participants, 104 developed ulcers between 1965 and 1974. We examined the impact of baseline risk factors on the association between education and incident ulcer. Among women, high school dropouts had a higher risk of incident ulcer than those who attended college (age-adjusted odds ratio [OR], 3.3; 95% confidence interval [CI], 1.5, 7.3). Adjustment for smoking, alcohol, lack of sleep, skipping breakfast, chronic pain, and liver disease eliminated 21.7% of this excess risk, whereas adjustment for psychological characteristics and life stress eliminated 56.5% of the risk; adjusted for all risk factors, the OR was 1.9. Among men, the risk associated with low education was weaker (OR, 1.9; 95% CI, 0.9, 3.9). Health risk behaviors and poor health had a greater impact (55.5% drop in excess risk with adjustment) and psychosocial factors a lesser impact (33.3% drop) in men than in women. Adjustment for heavy on-the-job labor decreased the risk by 77.8%, whereas the fully adjusted OR was 1.0.

We conclude that psychological stress, health risk behaviors, analgesic use, and hard physical labor may contribute to the increased risk of ulcer in low socioeconomic populations.

Key Words: Cohort study—Educational level—Peptic ulcer—Socioeconomic status—*Helicobacter pylori*—Psychosocial factors—Behavioral factors—Stress—Etiology—Gender—Psychological characteristics.

In epidemiologic studies, peptic ulcer generally occurs in inverse proportion to socioeconomic status (SES) (1,2). The explanation currently given by many gastroenterologists is the corresponding pattern of *Helicobacter pylori* infection (3,4), ascribed in turn chiefly to rates of contact with the organism during childhood (5), but *H. pylori* may not be the only risk factor. Cigarette smoking, for example, a known ulcer risk factor that is not associ-

ated with *H. pylori* in population studies (3,4), could reasonably represent an independent mediator between SES and ulcer. Other prominent candidate factors include high on-the-job energy expenditure (6) and psychological stress (7-10).

In previous prospective analyses of data from the Alameda County Study (a longitudinal investigation of behavioral, social, psychological, and economic influences on health), subjects with low educational achievement or a blue-collar household had an increased risk of developing peptic ulcer (11). Because the Alameda County Study also gathered information regarding a number of other ulcer risk factors that could be distributed unevenly by SES (12), we investigated their impact on the association between SES and ulcer incidence.

METHODS

At its baseline survey in 1965, the Alameda County Study distributed 8038 questionnaires to a stratified random sample of the adult inhabitants of a single county in California and obtained completed questionnaires from 6928 individuals: 3158 men and 3770 women. In 1973-74, 4864 of these panel members completed a similar second questionnaire (85% of located respondents). Details of sampling and follow-up have been reported elsewhere (13).

Subjects were excluded from the present analyses if they reported ulcer in 1965, or if they reported an ulcer in 1973-74 with an onset before 1966. Among the at-risk population, all reporting "stomach or duodenal ulcer" in 1973-74 were considered to have had new ulcers develop.

The first stage of the present analyses established the strength of the age-adjusted association of incident ulcer and educational achievement, one major measure of SES. Other 1965 risk factors, all of which have been shown to be prospectively associated with ulcer in our population (11,12), were then added to the education model as possible intermediaries.

Several health risk behaviors were included: smoking, heavily using alcohol (≥ 81 drinks/month for men, ≥ 61 drinks/month for women), answering "often" or "always" skipping breakfast and sleeping habitually less than 7 hours a night.

Illness variables included painful medical conditions, a measure we have shown to be an adequate proxy for nonsteroidal

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anti-inflammatory drug use (12), and chronic liver disease, which in men was too rare for analysis.

Psychological vulnerability was assessed using an ulcer risk index combining four psychological characteristics associated with ulcer development in this population (anomy, depression, hostility, and personal uncertainty) (12), divided at tertile splits into low, medium, or high.

A group of several concrete life stressors was also examined: household crowding, any period of unemployment during the 2 years before the 1965 survey, marital strain, and problems with dependent children (11).

On-the-job "hard physical work" could be examined only for men, because the question was not asked of women in 1965.

The statistical technique used was multiple logistic regression (SAS software package), using multivariate odds ratios (ORs) to estimate the relative risks associated with educational level. Dummy variables were used for education (high school diploma or less than high school education versus any college attendance), for age (25–39, 40–49, 50–59, or ≥59 years versus 17–24), for smoking (former, <1 pack, 1 pack, or >1 pack per day versus those who never smoked), and for pain (1 or 2–3 painful conditions versus no painful condition).

Men and women were examined separately, despite the decrease in statistical power, because psychosocial influences on ulcer vary by sex in this population and because data regarding on-the-job physical exertion were available only for men.

RESULTS

There were 104 new ulcers reported in 1973–74: 49 among men and 55 among women. Of the 4597 subjects at risk for incident ulcer, 1371 (29.8%) did not graduate from high school, 1485 (32.3%) had obtained a high school diploma but had not furthered their education, and 1741 (37.9%) had completed at least some college. Considering those who attended college as the reference category, the age-adjusted OR for ulcer development among high school dropouts in the population as a whole was 2.4 (95% confidence interval [CI], 1.4, 4.0), and the corresponding OR for high school graduates was 1.7 (95% CI, 1.0, 2.9).

Among women (Table 1), the age-adjusted OR for incident ulcer among high school dropouts was 3.3 (95% CI, 1.5, 7.3). Adjustment for nonpsychosocial risk factors (health risk behaviors and poor health) lowered the OR only to 2.8, a drop of 21.7% $[(3.3 - 2.8)/(3.3 - 1.0) = 21.7\%]$ in the excess risk attributable to low SES. In a model that instead adjusted for psychosocial risk factors (psychological index and life stressors), the age-adjusted OR decreased to 2.0, a drop of 56.5% in the excess risk; there was little incremental decrease in the OR (to 1.9) when this model was expanded to include the nonpsychosocial risk factors as well.

The association between failure to graduate from high school and incident peptic ulcer was weaker in men (OR, 1.9, 95% CI, 0.9, 3.9) (Table 2). Health risk behaviors and poor health had a relatively greater impact on this association than among women (adjusting for them reduced the excess risk by 55.5%), and psychosocial factors a lesser impact (excess risk reduced by 33.3%). Adjustment for heavy on-the-job labor by itself decreased the excess risk by 77.8% (adjusted OR, 1.2). The OR decreased to 1.0 in a model that incorporated all the risk factors taken into consideration.

DISCUSSION

Socioeconomic status is a major determinant of health status; most health outcomes that have been studied, not only peptic ulcer, are worse among persons with low levels of income, occupational status, and/or educational achievement (14–16). Understanding the mechanisms underlying this source of poor health is vital to devising ways of neutralizing its impact.

Because of the steep socioeconomic gradient of *H. pylori* infection (3–5) in populations similar to the one studied (17), it is tempting to conclude that the parallel gradient in peptic ulcer incidence is entirely due to differ-

TABLE 1. Mediators of the prospective association between education and peptic ulcer development among women

Adjustment variables in addition to age	Less than high school		High school graduate	
	OR	95% CI	OR	95% CI
None	3.3	1.5–7.3	2.0	0.9–4.4
Health risk behaviors*	3.0	1.3–6.7	1.8	0.8–4.1
Poor health (chronic pain, liver disease)	3.0	1.4–6.7	1.9	0.9–4.3
Behaviors and poor health	2.8	1.2–6.3	1.7	0.8–3.9
Psychological index†	2.4	1.1–5.3	1.5	0.7–3.5
Life stressors‡	2.7	1.2–6.0	1.7	0.8–3.9
Psychological index and life stressors	2.0	0.9–4.6	1.4	0.6–3.2
Behaviors, poor health, psychological index, and stress	1.9	0.8–4.4	1.4	0.6–3.1

Age-adjusted odds ratios for lower educational categories versus college attenders, adjusted in turn for other ulcer risk factors. Alameda County Study, 1965–1974.

*Smoking, heavy alcohol use, lack of sleep, skipping breakfast.

†Anomy, depression, hostility, personal uncertainty.

‡Household crowding, unemployment, marital strain, children's problems.

OR: odds ratio; CI, confidence interval.

TABLE 2. Mediators of the prospective association between education and peptic ulcer development among men.

Adjustment variables in addition to age	Less than high school		High school graduate	
	OR	95% CI	OR	95% CI
None	1.9	0.9-3.9	1.8	0.9-3.5
Health risk behaviors*	1.4	0.7-2.9	1.4	0.7-2.9
Poor health (chronic pain)	1.8	0.9-3.8	1.7	0.9-3.5
Behaviors and poor health	1.4	0.6-2.9	1.4	0.7-2.8
Psychological index†	1.8	0.8-3.8	1.7	0.8-3.5
Life stressors‡	1.7	0.8-3.6	1.6	0.8-3.4
Psychological index and life stressors	1.6	0.7-3.5	1.6	0.8-3.3
Behaviors, poor health, psychological index, and stress	1.3	0.6-2.8	1.3	0.6-2.8
Heavy, on-the-job labor	1.2	0.6-2.7	1.3	0.6-2.7
All risk factors, including on-the-job labor	1.0	0.4-2.2	1.1	0.5-2.3

Age-adjusted odds ratios for lower educational categories versus college attenders, adjusted in turn for other ulcer risk factors. Alameda County Study, 1965-1974.

*Smoking, heavy alcohol use, lack of sleep, skipping breakfast.

†Anomy, depression, hostility, personal uncertainty.

‡Household crowding, unemployment, marital strain, children's problems.

OR, odds ratio; CI, confidence interval.

ences in *H. pylori* infection rates. We have attempted to determine whether the differential distribution of other risk factors might contribute to the SES pattern of ulcer. We found that the socioeconomic gradient in ulcer incidence was statistically abolished for men and considerably attenuated for women when several non-*H. pylori* risk factors were taken into account. Psychosocial stress seemed to be a more important mediator in women, whereas health risk behaviors played a greater role in men. On-the-job physical exertion may be a particularly important mediator of the association between low SES and peptic ulcer.

We considered the possibility that what we call mediators are instead merely stand-ins for *H. pylori* infection. This seems unlikely, because no evidence links such infection with any of the risk factors examined. Several groups have failed to find associations of *H. pylori* infection with smoking (3,4,18), alcohol consumption (3,4), or nonsteroidal anti-inflammatory drug use (4,18) in the general population. The few studies that have examined the presence or intensity of *H. pylori* infection in relation to psychological distress have found, if anything, an inverse relation between the two risk factors in gastroenterology patients (19,20).

Indirect support for the concept that noninfectious risk factors could contribute to the SES distribution of peptic ulcer lies in the very breadth of the health effects of SES. Lifestyle aspects that contribute to the wider pattern of poor health among less privileged groups (16,21) could reasonably be expected to play a part in ulcer as well.

The single cause infectious model of peptic ulcer disease has intrinsic limits. Not only do most persons who encounter *H. pylori* live their lives free of ulcer, indicating a role for co-factors in triggering clinical disease, but a few persons manage to form ulcers without exposure either to *H. pylori* or to nonsteroidal anti-inflammatory drugs (22,23). This lack of a one-to-one correspondence

leaves room for a multiplicity of factors in explaining the observed patterns in peptic ulcer occurrence, a conclusion that has also been drawn by at least one student of the secular trends (24) and the co-morbidity (25) of *H. pylori*-related gastrointestinal diseases.

Risk factors mediating between low SES and peptic ulcer could operate through potentiation of *H. pylori* or through independent effects. Smoking seems to be ulcerogenic only among persons with *H. pylori* infection (26), whereas rapid gastric emptying (22) has been reported to have effects on ulcer pathogenesis that are additive to those of *H. pylori* rather than interactive. Nonsteroidal anti-inflammatory drugs not only are pathogenetically independent of *H. pylori* (27) but also are a prime cause of *H. pylori*-negative ulcer (26,28), and therefore represent an important potential contributor to ulcer risk patterns. The known interactions between *H. pylori* and its host suggest routes by which psychological stress could facilitate the evolution of infection into ulceration, by stimulating gastric acid secretion (29).

All efforts aimed at clarifying SES-related health outcomes require handling with care. The risk factors of interest are not independent of each other—for instance, people are likely to smoke more in periods of life stress, and the same painful conditions that lead to taking nonsteroidal anti-inflammatory drugs could well cause psychological distress—and they have complex relations with SES. Furthermore, investigations of the mediators between low SES and ulcer, whether psychological and behavioral or infectious, should not be interpreted as explaining the association by detecting confounders, but rather as exploring the nature of the pathways by which SES affects health (21). Any attempt to act on such findings to decrease excess ulcer risk depends on clarification of why and how low education is associated with the factors we have studied.

Because the Alameda County Study gathered no data regarding *H. pylori*, we are limited in our ability to define the role of noninfectious factors in ulcer epidemiology. However, we continue to believe that psychological stress, health risk behaviors, hard physical labor, and analgesic use may act alongside *H. pylori* infection in producing the increased risk of ulcer attributable to low SES.

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