Smoking, alcohol and gastric cancer risk in Korean men: the National Health Insurance Corporation Study

We investigated the risk of gastric cancer by subsite in relation to cigarette smoking and alcohol in a large population-based cohort of 669,570 Korean men in an insurance plan followed for an average of 6.5 years, yielding 3,452 new cases of gastric cancer, of which 127 were cardia and upper-third gastric cancer, 2,409 were distal gastric cancer and 1,007 were unclassified. A moderate association was found between smoking, cardia and upper-third (adjusted relative risk (aRR) 2.2; 95% confidence interval (CI) 1.4–3.5) and distal cancers (aRR = 1.4; 95% CI = 1.3–1.6). We also found a positive association between alcohol consumption and distal (aRR = 1.3; 95% CI = 1.2–1.5) and total (aRR = 1.2; 95% CI = 1.1–1.4) gastric cancer. Combined exposure to high levels of tobacco and alcohol increased the risk estimates further; cardia and upper-third gastric cancers were more strongly related to smoking status than distal gastric cancer.

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Keywords: cigarette smoking; alcohol intake; gastric cancer; subsite

Until recently, gastric cancer was the second most common cancer worldwide; now, however, with an estimated 934,000 new cases (8.6% of new cancer cases) in 2002 alone, it is in fourth place behind lung, breast and colon and rectum cancers (Parkin et al., 2004). With declining in Korea, gastric cancer is still the commonest cancer (Shin et al., 2005). An extensive review indicates that smoking is a moderate risk factor for gastric cancer (International Agency for Research on Cancer, 2004), but little support exists for an association with alcohol (Gammon et al., 1997; Sjödahl et al., 2006). The possibility of a differential effect of smoking and alcohol consumption on different gastric subsites, however, remains to be clarified.

In a prospective cohort study, we investigated the effects of smoking and alcohol consumption on the risk of gastric cancer by subsite in the National Health Insurance Corporation Study (NHICS). The study participants were derived from 692,108 men aged 30 years or over who participated in the National Health Examination Program in 1996 and were in the NHICS cohort. Of the 692,108 participants, we excluded 27,322 patients who had cancer at enrollment according to the Korea Central Cancer Registry (KCCR). We also excluded the following because of missing information: 214 for weight or height, 9936 for smoking, 3,019 for alcohol intake and 6,675 for dietary preference. Ultimately, 669,634 participants were included.

Based on questionnaire responses at the baseline examination of the NHICS cohort, the participants were classified as ‘current smokers’ if they reported smoking currently for at least 1 year, ‘nonsmokers’ if they never smoked and ‘former smokers’ if they had smoked but quit. Current smokers were further classified by the average number of cigarettes smoked per day (1–19, ≥20) and duration of smoking (1–19, 20–29, or ≥30 years). Alcohol intake per day was classified as follows: no drinking (0 g), light drinking (1–14.9 g), moderate drinking (15.0–24.9 g) and heavy drinking (≥25.0 g). Total daily alcohol intake was expressed as the number of glasses per week of Korea’s most popular alcoholic beverage, ‘Soju’. One glass of Soju contains about 12 g of ethanol. A preference for saltiness in food (low salt, normal and salty) was included because of possible relevance to stomach cancer (Tsugane et al., 2004). We used the World Health Organisation body mass index (BMI) standards for Asians (World Health Organisation, 2000).

MATERIALS AND METHODS

The NHICS is a cohort investigation that was designed to assess the risk factors for the incidence of and mortality from cancer (Yun et al., 2005; Park et al., 2006). In brief, the cohort consisted of government employees, teachers and their dependents who were insured by the Korea National Health Insurance (NHI) Program in 1996, had at least one medical examination, and completed a self-administered questionnaire.

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We used anatomic site and histological classification information from the pathology reports of the KCCR based on the International Classification of Diseases for Oncology. Tumours at the oesophagogastric junction or upper third of the stomach were classified as cardia and upper-third gastric cancer (C16.0–16.1) and those at the lower end of the stomach as distal gastric cancer (C16.2–16.7). Mixed site (C16.8) and site not otherwise specified (C16.9) were regarded as unclassified. Virtually all (>98%) of the gastric cancers were confirmed histologically, 94% being adenocarcinoma (M814–857), and the few cases (n = 212) that were not excluded. During the 6.5-year follow-up period, we included 3452 patients diagnosed with a gastric adenocarcinoma in a final cohort comprising 669,570 participants.

We also gathered 1996–2002 mortality data from the National Statistical Office. Subjects without cancer were followed until 31 December 2002; the follow-up period for each cancer case was defined as the interval between enrollment and diagnosis.

### Statistical analysis

The Cox proportional hazard regression model was used to estimate the relative risk of gastric cancer by subsite according to smoking status and alcohol intake (first adjusted only for age, then adjusted for other potential risk factors). Every model included the length of follow-up as a time-dependent covariate. The proportionality assumption was verified by inspecting hazard plots. The trends were assessed by assigning ordinal values for categorical variables. Non- and former smokers were excluded from the analysis of duration and intensity (cigarettes per day) when calculating the P-value for trend, as suggested by Lefforondré et al (2002). For alcohol consumption, trends were calculated among those who drank at least 1 g per day.

The interaction effects were evaluated by calculating an interaction term, that is, multiplying a dummy variable for smoking (current smoker = 1, nonsmoker or former smoker = 0) by one for alcohol consumption (drinks once per month = 1, never drinks = 0). Interactions between smoking and alcohol drinking were formally tested using the likelihood ratio method, comparing models with and without the interaction terms. We calculated a population-attributable risk (Rothman and Greenland, 1998) to assess the potential public health impact of smoking on gastric cancer by anatomic site, using the smoking prevalence data from the 1998 Korea Health Survey (Ministry of Health and Welfare, 1999). All confidence intervals (CIs) were at 95%, and a P-value of

| Characteristic | Cardia and upper third (PY = 451) | Distal (PY = 8349) | Total (PY = 12242) |
|---------------|----------------------------------|-------------------|-------------------|
| Total         | 669,570                          |                   |                   |
| Age at entry (years) |                   |                   |                   |
| < 40          | 249,932 37.0                     | 12 9.4            | 234 10.1          |
| 40–49         | 229,403 34.3                     | 33 26.0           | 575 24.8          |
| 50–59         | 159,958 23.9                     | 59 46.5           | 1130 48.7         |
| ≥ 60          | 32,277 4.8                       | 23 18.1           | 379 16.4          |
| Body mass index (kg m⁻²) |                   |                   |                   |
| < 22.9        | 274,971 41.1                     | 42 33.1           | 853 36.8          |
| 22.9–24.9     | 197,558 29.5                     | 40 31.5           | 725 31.3          |
| ≥ 25.0        | 197,041 29.4                     | 45 35.4           | 640 27.6          |
| Smoking status |                   |                   |                   |
| Never         | 212,900 31.8                     | 24 18.9           | 616 26.6          |
| Former        | 98,229 14.7                      | 24 18.9           | 405 17.5          |
| Current       | 358,441 53.5                     | 79 62.2           | 1297 56.0         |
| Cigarettes per day |                   |                   |                   |
| < 19          | 281,672 78.8                     | 60 76.9           | 1027 79.7         |
| ≥ 20          | 75,567 21.2                      | 18 23.1           | 261 20.3          |
| Smoking duration (years) |                   |                   |                   |
| < 19          | 196,575 56.5                     | 29 38.2           | 391 31.4          |
| 20–29         | 103,392 29.7                     | 21 27.6           | 431 34.6          |
| ≥ 30          | 47,673 13.7                      | 26 34.2           | 424 34.0          |
| Alcohol intake (g day⁻¹) |                   |                   |                   |
| 0            | 188,830 28.2                     | 29 22.8           | 661 28.5          |
| 1–14.9        | 198,998 29.7                     | 36 28.3           | 633 27.3          |
| 15.0–24.9     | 124,711 18.6                     | 31 24.4           | 430 18.6          |
| ≥ 25          | 157,031 23.5                     | 31 24.4           | 594 25.6          |
| Preference for salty food |                   |                   |                   |
| Low          | 274,971 41.1                     | 12 9.4            | 335 14.5          |
| Normal       | 197,558 29.5                     | 82 64.6           | 1535 66.2         |
| Salty        | 197,041 29.4                     | 33 26.0           | 448 19.3          |

PY = person-year. *Including cardia and upper-third (C16.0–16.1), distal (C16.2–16.7), mixed site (C16.8) and site not otherwise specified (C16.9) gastric cancer. **Data are means ± standard deviation or number of cases and percentage. ***For current smokers.
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RESULTS

The 669,570 study cohort members were followed for an average 6.5 years, contributing a total of 4,353,317 person-years. The population was primarily middle aged (mean of 44 years) and had a low average BMI (23.6 kg m\(^{-2}\)) with 29.4% of men over 25 kg m\(^{-2}\). At baseline, 53.5 and 71.8% of the men were current smokers and drinkers, respectively (Table 1). During follow-up, we identified 3452 new cases of gastric cancer, of which 127 (4%) were cardia and upper-third cancers, 2409 (70%) were distal gastric cancer and 1007 were unclassified. Some characteristics of the participants are presented in Table 1. Almost 90% of cases occurred in persons above 40 years.

Table 2 shows the adjusted relative risk (aRR) for gastric cancer in relationship to smoking and alcohol intake. The risk of cardia and upper-third gastric cancers was doubled or more among current smokers (aRR = 2.2; 95% CI 1.4 – 3.5) when compared to those who had never smoked. For distal and total gastric cancer, the corresponding risks of current and never smokers were 1.4 (95% CI 1.3 – 1.6) and 1.5 (95% CI 1.4 – 1.6), respectively. Relative risks of gastric cancer increased with increasing numbers of cigarettes per day and years of smoking, although the trend was not statistically significant. The age-only adjusted risk estimates for smoking changed only slightly after adjusting for alcohol and other variables (see Materials and Methods), including alcohol intake (data not shown). We estimated the multivariate-adjusted population-attributable risks from cigarette smoking as 36.8% (95% CI 16.2 – 52.4) in cardia and upper-third gastric cancer, and 19.3% (95% CI 13.9 – 24.3) in distal gastric cancer; overall, 22.5% (95% CI 18.3 – 26.5) of gastric cancers were attributable to smoking.

Alcohol consumption was also associated with an increased risk after adjusting for smoking status (Table 3). The risks of distal and total gastric cancers were increased among patients who reported drinking at least 25 g of alcohol per day to 1.3 (95% CI 1.2 – 1.5) and 1.2 (95% CI 1.1 – 1.4), respectively, when compared to nondrinkers, and the P-value for trend was significant when only drinkers were considered. Although the risks of cardia and upper-third gastric cancer were increased among drinkers, these were not significant.

The independent and joint effects of smoking and alcohol intake on risks by gastric subsite are examined in Table 4. Smoking over 20 cigarettes per day combined with alcohol consumption exceeding 25 g per day was associated with a nearly five-fold increased risk of cardia and upper-third gastric cancer (HR = 4.5, 95% CI 1.7 – 11.9), and a two-fold increased risk of distal gastric cancer compared to nonusers. The interaction between smoking and alcohol drinking was not statistically significant for total gastric cancer (P = 0.48), cardia and upper-third cancer (P = 0.68) or distal cancer (P = 0.89).

DISCUSSION

Smoking and alcohol use were associated with gastric cancer risk by anatomic subsite in this large cohort study. Current smokers showed elevated risks, higher in cardia and upper-third than in distal gastric cancer among current smokers. Furthermore, we found that the risk of cancer increased with the number of cigarettes smoked per day and years of smoking. Positive associations were also found with alcohol consumption, though for cardia and upper-third gastric cancers this was not significant. The results of the multinomial logistic analysis were similar to those of a Cox proportional hazard regression: the incidence of cardia and upper-third gastric cancer was 2.2 times higher for current smokers than for never smokers, and 1.4 times for distal gastric cancer (data not shown). Combined exposure to high levels of tobacco and alcohol further increased the risk estimates.

Although smoking is well recognised as a moderate risk factor, few population-based cohort studies have been conducted for gastric subsites (Sasazuki et al, 2002; Koizumi et al, 2004; Sjödahl et al, 2006). In the last 5 years, seven case–control studies have also been reported: five of them (Gammon et al, 1997; Zaridze et al, 2002) observed a higher risk for cardia cancers, while two did not.

Table 2 Multivariate relative risk by smoking habit for gastric cancer according to anatomic subsite

| Smoking status | Cardia and upper third | Distal | Total* |
|----------------|------------------------|--------|--------|
| No. of subjects | n | aRR \(^{b}\) | n | aRR \(^{b}\) | n | aRR \(^{b}\) |
| Smoking status | | | | | | |
| Never | 212,900 | 24 | 1.0 | 616 | 1.0 | 901 | 1.0 |
| Former | 98,229 | 24 | 1.9 (1.1–3.3) | 405 | 1.3 (1.2–1.5) | 593 | 1.3 (1.2–1.5) |
| Current | 358,441 | 79 | 2.2 (1.4–3.5) | 1297 | 1.4 (1.3–1.6) | 1958 | 1.5 (1.4–1.6) |
| Cigarettes per day | | | | | | |
| Never | 212,900 | 24 | 1.0 | 616 | 1.0 | 901 | 1.0 |
| 1–19 | 281,672 | 60 | 2.3 (1.4–3.7) | 1027 | 1.4 (1.3–1.6) | 1535 | 1.5 (1.4–1.6) |
| ≥20 | 75,567 | 18 | 2.5 (1.3–4.7) | 261 | 1.4 (1.2–1.6) | 406 | 1.5 (1.3–1.7) |
| Smoking duration | | | | | | |
| Never | 212,900 | 24 | 1.0 | 616 | 1.0 | 901 | 1.0 |
| ≤19 years | 196,575 | 29 | 2.9 (1.6–5.1) | 391 | 1.4 (1.2–1.6) | 589 | 1.4 (1.3–1.6) |
| 20–29 years | 103,392 | 21 | 2.9 (1.0–3.4) | 431 | 1.4 (1.3–1.6) | 652 | 1.5 (1.3–1.6) |
| ≥30 years | 47,673 | 26 | 2.4 (1.3–4.2) | 424 | 1.5 (1.3–1.7) | 642 | 1.5 (1.4–1.7) |

aRR = adjusted relative risk; *including cardia and upper-third (C16.0–16.1), distal (C16.2–16.7), mixed site (C16.8) and site not otherwise specified (C16.9) gastric cancer; **95% confidence intervals from multivariate Cox proportional models after adjusting for age, body mass index, alcohol intake and preference for saltiness in food. \(^{\top}\)Trend calculated among current smokers.
et al, 1994). We found moderately strong associations between smoking and gastric cancer in both cardia and upper third, and distal locations. Several dose–response associations were also suggested, adding to evidence of a causal association. Although a significant association of cardia cancer with alcohol has been reported (Inoue et al, 1994), most studies have not confirmed this (Okabayashi et al, 2000; Sasazuki et al, 2002; Zardzze et al, 2002). We found that alcohol intake was significantly related to an increased risk both of gastric cancer as a whole and of the distal stomach. By contrast, the positive association for cardia and upper-third cancers was not significant, perhaps because of the relatively small numbers.

Several potential limitations of our study resulted from the use of data collected as part of an insurance plan. First, the self-reported smoking and alcohol details were not validated, and the amount smoked per day was classified only as ‘1–9’ and ‘20 or more’ on the 1996 questionnaire. Therefore, we could not examine, for example, 15–24 cigarettes smoked daily to cover the effect of rounding to a common value.

Second, our study cohort was not representative of all Koreans. Although enrollment in the NHI Program is largely mandatory for Koreans, our study covered only employed persons (government employees and teachers) and their families, and consequently, may have under-represented heavy users of alcohol and tobacco. However, follow-up should be essentially complete because of our using record linkage with unique personal identifiers to national databases.

Third, we lacked information on *Helicobacter pylori*, a strong risk factor for gastric cancer (International Agency for Research on Cancer, 1994). In Korea, the reported prevalence of *H. pylori* IgG antibody among males above 40 years is 77–83% (Shin et al, 2005a; Kim et al, 2006). Moreover, a nonsignificant increased risk for gastric cancer associated with the presence of *H. pylori* was observed among subjects in a longer than 5-year follow-up study in Korea (Shin et al, 2005a). In addition, evidence has shown that the association of smoking and gastric cancer is independent of *H. pylori* infection (Siman et al, 2001; Sasazuki et al, 2002). Given these findings, the lack of *H. pylori* data is unlikely to be an important issue for interpreting our findings.

Fourth, no detailed information on nutritional factors was available, including the intake of antioxidative vitamins, which might have a protective effect against gastric cancer (Kono and Hirohata, 1996).

In our study, cardia and upper-third gastric cancer was more strongly related to smoking status than distal gastric cancer, while alcohol consumption may be associated with an increased risk of distal and total gastric cancer. Larger numbers of cardia gastric cancer, however, would be needed to investigate a dose–response relationship reliably.

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### Table 3 Multivariate relative risk by alcohol consumption for gastric cancer according to anatomic subsite

| Alcohol consumption (g/day⁻¹) | Cardia and upper third | Distal | Total |
|-------------------------------|------------------------|--------|-------|
|                               | n  | aRRb | n  | aRRb | n  | aRRb |
| 0                             | 188830 | 29 | 1.0 | 661 | 1.0 | 999 | 1.0 |
| 1–14.9                        | 198998 | 36 | 1.3 (0.8–2.1) | 633 | 1.0 (0.9–1.2) | 946 | 1.0 (0.9–1.1) |
| 15.0–24.9                     | 12471 | 31 | 1.7 (1.0–2.8) | 430 | 1.2 (1.0–1.3) | 644 | 1.1 (1.0–1.3) |
| ≥25                           | 157031 | 31 | 1.3 (0.8–2.2) | 594 | 1.3 (1.2–1.5) | 863 | 1.2 (1.1–1.4) |

\( P_{\text{test}} = 0.5914 \)

\( 0.0002 \)

\( 0.0001 \)

\( aRR = \) adjusted relative risk. Including cardia and upper-third (C16.0–16.1), distal (C16.2–16.7), mixed site (C16.8) and site not otherwise specified (C16.9) gastric cancer. 95% confidence intervals from multivariate Cox proportional models after adjusting for age, body mass index and smoking status in food. Trend calculated among those who drank at least 1 g day⁻¹.

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### Table 4 Smoking, alcohol and risk of gastric cancer by subsite

| Exposure status | Smoking | Alcohol | No. of subjects | n  | aRRb | n  | aRRb | n  | aRRb |
|-----------------|---------|---------|-----------------|-----|------|-----|------|-----|------|
| Cardia and upper third | Never smoked | Never | 88431 | 8 | 1.0 | 270 | 1.0 | 397 | 1.0 |
|                  | Never smoked | <25 g/day⁻¹ | 96063 | 15 | 2.0 (0.9–4.8) | 255 | 1.0 (0.9–1.2) | 378 | 1.0 (0.9–1.2) |
|                  | Never smoked | ≥25 | 28406 | 1 | 0.4 (0.1–3.4) | 91 | 1.3 (1.0–1.6) | 126 | 1.2 (1.0–1.5) |
|                  | <20 cigarettes per day | Never | 56786 | 11 | 2.5 (1.0–6.2) | 235 | 1.5 (1.3–1.8) | 350 | 1.5 (1.3–1.8) |
|                  | <20 cigarettes per day | <25 g/day⁻¹ | 149534 | 31 | 3.1 (1.4–6.9) | 499 | 1.5 (1.3–1.7) | 758 | 1.5 (1.3–1.7) |
|                  | <20 cigarettes per day | ≥25 | 75352 | 18 | 3.7 (1.6–8.6) | 293 | 1.8 (1.5–2.2) | 427 | 1.8 (1.6–2.1) |
|                  | ≥20 cigarettes per day | Never | 16585 | 2 | 1.5 (0.3–7.3) | 44 | 1.0 (0.8–1.4) | 85 | 1.4 (1.1–1.7) |
|                  | ≥20 cigarettes per day | <25 g/day⁻¹ | 27572 | 7 | 3.9 (1.4–10.8) | 93 | 1.6 (1.6–2.0) | 145 | 1.7 (1.4–2.0) |
|                  | ≥20 cigarettes per day | ≥25 | 31410 | 9 | 4.5 (1.7–11.9) | 124 | 2.0 (1.6–2.5) | 176 | 1.9 (1.6–2.3) |

\( aRR = \) adjusted relative risk. Including cardia and upper-third (C16.0–16.1), distal (C16.2–16.7), mixed site (C16.8) and site not otherwise specified (C16.9) gastric cancer. Adjusted risk ratios and 95% confidence intervals from multivariate Cox proportional models after adjusting for age, body mass index and preference for saltiness in food.

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