Table 3 Results from a pooled analysis (random-effects model) of gastric cancer incidence by green tea consumption in Japanese men, 1984–2004

		Green tea consumption					p For heterogeneity (for the highest category)	p For heterogeneity (for trend)
		<1 cup/day	1–2 cups/day	3–4 cups/day	≥5 cups/day	p For trend		
	Total	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)			
Total								
No of subjects	100479	19877	21355	26369	32878			
Person-years	1035158	205165	219427	271469	339097			
No of cases	2495	420	452	610	1013			
Age-standardised rate (per 100 000) (Random effect model)	241.12	236.20	236.06	222.44	257.18			
Age- and area-adjusted (model 1)		1.00 (Reference)	0.98 (0.85 to 1.14)	0.95 (0.82 to 1.09)	1.10 (0.90 to 1.34)	*0.394*	0.026	0.110
Multivariate-adjusted (model 2)		1.00 (Reference)	0.97 (0.84 to 1.12)	0.94 (0.81 to 1.08)	1.06 (0.86 to 1.29)	*0.792*	0.024	0.132
Multivariate-adjusted (model 3)		1.00 (Reference)	0.97 (0.83 to 1.12)	0.93 (0.81 to 1.08)	1.06 (0.86 to 1.30)	*0.739*	0.025	0.104
Smoking status								
Never smokers								
No of subjects	19334	4257	4176	5229	5672			
Person-years	204380	45197	44025	54939	60219			
No of cases	312	56	60	73	123			
Age-standardised rate (per 100 000) (Random effect model)	157.74	142.01	162.62	135.76	177.75			
Age- and area-adjusted (model 1)		1.00 (Reference)	1.12 (0.73 to 1.72)	0.97 (0.67 to 1.41)	1.28 (0.90 to 1.82)	*0.063*	0.518	0.535
Multivariate-adjusted (model 2)		1.00 (Reference)	1.10 (0.74 to 1.64)	0.96 (0.66 to 1.39)	1.27 (0.89 to 1.81)	*0.337*	0.581	0.730
Multivariate-adjusted (model 3)		1.00 (Reference)	1.15 (0.75 to 1.76)	0.99 (0.68 to 1.45)	1.34 (0.93 to 1.92)	*0.221*	0.552	0.671
Current smokers								
No of subjects	53438	10510	11540	13724	17664			
Person-years	555136	109862	119803	142719	182752			
No of cases	1366	227	254	342	543			
Age-standardised rate (per 100 000) (Random effect model)	265.29	252.94	272.87	256.63	272.79			
Age- and area-adjusted (model 1)		1.00 (Reference)	0.99 (0.83 to 1.19)	1.00 (0.82 to 1.22)	1.05 (0.82 to 1.35)	*0.564*	0.064	0.050
Multivariate-adjusted (model 2)		1.00 (Reference)	0.99 (0.82 to 1.19)	0.99 (0.81 to 1.20)	1.03 (0.81 to 1.31)	*0.817*	0.090	0.107
Multivariate-adjusted (model 3)		1.00 (Reference)	0.98 (0.81 to 1.18)	0.97 (0.80 to 1.19)	1.01 (0.79 to 1.29)	*0.727*	0.086	0.053
Subsite								
Proximal (upper third)								
No of subjects	65321	15019	14943	16517	18842			
Person-years	662495	155665	152476	168202	186152			
No of cases	217	38	41	42	96			
Age-standardised rate (per 100 000) (Random effect model)	36.82	30.61	31.60	26.53	49.10			
Age- and area-adjusted (model 1)		1.00 (Reference)	1.11 (0.71 to 1.74)	0.76 (0.46 to 1.26)	1.43 (0.97 to 2.12)	*0.069*	0.973	0.847
Multivariate-adjusted (model 2)		1.00 (Reference)	1.09 (0.70 to 1.72)	0.77 (0.46 to 1.29)	1.42 (0.96 to 2.11)	*0.080*	0.994	0.785
Multivariate-adjusted (model 3)		1.00 (Reference)	1.10 (0.70 to 1.73)	0.79 (0.46 to 1.35)	1.43 (0.96 to 2.14)	*0.081*	0.919	0.737

Continued

Table 3 Continued

	Total	Green tea consumption				p For trend	p For heterogeneity (for the highest category)	p For heterogeneity (for trend)
		<1 cup/day	1–2 cups/day	3–4 cups/day	≥5 cups/day			
		HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)			
Distal (lower two thirds)								
No of subjects	65321	15019	14943	16517	18842			
Person-years	662495	155665	152476	168202	186152			
No of cases	947	185	185	249	328			
Age-standardised rate (per 100 000) (Random effect model)	151.35	136.73	144.95	154.07	160.99			
Age- and area-adjusted (model 1)		1.00 (Reference)	0.92 (0.74 to 1.13)	0.97 (0.80 to 1.18)	1.02 (0.84 to 1.24)	*0.690*	0.370	0.270
Multivariate-adjusted (model 2)		1.00 (Reference)	0.89 (0.72 to 1.11)	0.93 (0.77 to 1.14)	0.95 (0.78 to 1.15)	*0.746*	0.469	0.299
Multivariate-adjusted (model 3)		1.00 (Reference)	0.91 (0.73 to 1.12)	0.95 (0.77 to 1.16)	0.96 (0.79 to 1.17)	*0.856*	0.481	0.316

Model 2: Adjusted for age (continuous), area (JPHC-I, JPHC-II and JACC only), smoking (never smoker, past smoker, current smoker of 1–19 cigarettes/day, or current smoker of ≥20 cigarettes/day), ethanol intake (never/former drinker, occasional drinker (<once/week), regular drinker (≥once/week: <23 g/day, 23–<46 g/day, ≥46 g/day), rice intake (<4 bowls/day, ≥4 bowls/day), soy bean paste soup (<daily, daily), and coffee intake (<1 cup/day, 1–2 cups/day, ≥3 cups/day).
Model 3: Adjusted for pickled vegetable intake (<weekly, 1–2 times/week, 3–4 times/week, daily) and green–yellow vegetable intake (<weekly, 1–2 times/week, 3–4 times/week, daily) in addition to the variables included in Model 2.

between studies for the highest category of green tea consumption for male total gastric cancer risk showed significant heterogeneity (p = 0.025), and the I^2 statistic suggested that 61% of between-study heterogeneity among the highest category was attributable to variability in the true effect of green tea.

In women (table 4), in contrast, subjects who consumed ≥5 cups of green tea every day had a significantly decreased risk of gastric cancer (HR = 0.79, 95% CI = 0.65 to 0.96). We also observed a significant trend of decreased risk with increasing consumption (p for trend = 0.043). Results did not change for never smokers (HR = 0.79, 95% CI = 0.64 to 0.97 for ≥5 cups of green tea). When outcome was confined to gastric cancer at a distal site, similar decreased risk was observed (HR = 0.70, 95% CI = 0.50 to 0.96 for ≥5 cups of green tea; p for trend = 0.042). Results between studies for female never smokers showed significant heterogeneity (p for heterogeneity <0.001), and the I^2 statistic suggested 85% of between-study heterogeneity for trend association was attributable to variability in the true effect of green tea.

DISCUSSION

Although many experimental studies have indicated a role for green tea in cancer prevention,[2] epidemiological evidence for the effect of green tea consumption on cancer risk is conflicting. To address this discrepancy, we carried out a pooled analysis of major population-based cohort studies in Japan. Results showed a significant decrease in risk only among women in the highest category of green tea consumption. This decrease in risk was similarly observed among never smokers and for distal gastric cancer. We observed no association between green tea consumption and gastric cancer in men.

For the heterogeneity of results among the highest category of total men, two studies which were started in the mid 1980s, in other words earlier than other studies, tended to show an increased risk while the other later studies showed a decreased risk tendency. This heterogeneity may have resulted from a slight difference in the birth cohort due to the earlier starting point. In women, in contrast, heterogeneity was observed only for the trend association among never-smokers, in which one of the two studies started in the mid 1980s showed different results from the other studies. Therefore, these heterogeneities observed in men and women may not be solely attributable to such differences in birth cohort.

Our results raise several noteworthy issues on the association between green tea consumption and gastric cancer risk. First, we observed a clear sex difference in the association between green tea consumption and gastric cancer risk. Although most previous cohort studies in Japan have reported a null association, those which conducted separate analyses by sex[9 12 13] in fact observed a decreased risk tendency in women, whereas those which only reported combined results tended to observe an overall null association.[10 11]

Several possibilities may explain the null association for men. The first is that the highest category in women may have included more subjects with a higher consumption of green tea than the highest category in men, hampering the detection of an effect in men, if any. One of the cohorts, JACC, in which information was obtained on the number of cups consumed per day, showed no such trend.[9] Further, the null association in men may have been partly due to residual confounding effects, especially cigarette smoking. In our previous systematic review, we concluded that there is convincing evidence that cigarette smoking moderately increases the risk of gastric cancer among

Table 4 Results from a pooled analysis (random-effects model) of gastric cancer incidence by green tea consumption in Japanese women, 1984–2004

		Green tea consumption						
		<1 cup/day	1–2 cups/day	3–4 cups/day	≥5 cups/day		p For heterogeneity (for the highest category)	p For heterogeneity (for trend)
	Total	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	p For trend		
Total								
No of subjects	118601	23316	21460	32459	41366			
Person-years	1250810	244097	226618	342038	438057			
No of cases	1082	215	174	303	390			
Age-standardised rate (per 100 000) (Random effect model)	86.50	99.89	87.01	87.88	78.96			
Age- and area-adjusted (model 1)		1.00 (Reference)	0.98 (0.84 to 1.15)	0.92 (0.77 to 1.11)	0.81 (0.67 to 0.97)	0.031	0.416	0.415
Multivariate-adjusted (model 2)		1.00 (Reference)	0.90 (0.73 to 1.10)	0.93 (0.77 to 1.11)	0.80 (0.66 to 0.96)	0.063	0.402	0.265
Multivariate-adjusted (model 3)		1.00 (Reference)	0.90 (0.73 to 1.10)	0.92 (0.76 to 1.11)	0.79 (0.65 to 0.96)	0.043	0.351	0.283
Smoking status								
Never smokers								
No of subjects	95558	18422	17360	26897	32879			
Person-years	1023763	196333	185652	287616	354163			
No of cases	871	171	144	246	310			
Age-standardised rate (per 100 000) (Random effect model)	86.36	100.79	89.07	85.78	78.61			
Age- and area-adjusted (model 1)		1.00 (Reference)	0.90 (0.72 to 1.14)	0.90 (0.73 to 1.11)	0.80 (0.66 to 0.98)	0.692	0.574	<0.001
Multivariate-adjusted (model 2)		1.00 (Reference)	0.91 (0.72 to 1.15)	0.91 (0.74 to 1.12)	0.80 (0.65 to 0.98)	0.770	0.548	<0.001
Multivariate-adjusted (model 3)		1.00 (Reference)	0.91 (0.73 to 1.15)	0.90 (0.73 to 1.11)	0.79 (0.64 to 0.97)	0.780	0.531	<0.001
Current smokers			(≥1 cups/day)					
No of subjects	7694	1636	6058					
Person-years	78141	16561	61580					
No of cases	66	12	54					
Age-standardised rate (per 100 000) (Random effect model)	88.54	74.54	89.21					
Age- and area-adjusted (model 1)		1.00 (Reference)	0.94 (0.48 to 1.82)			0.744	0.715	0.882
Multivariate-adjusted (model 2)		1.00 (Reference)	0.86 (0.44 to 1.68)			0.690	0.488	0.459
Multivariate-adjusted (model 3)		1.00 (Reference)	0.90 (0.41 to 1.97)			0.799	0.299	0.383
Subsite								
Proximal (upper third)			(≥1 cups/day)					
No of subjects	72611	16271	56340					
Person-years	758865	173390	585474					
No of cases	53	8	45					
Age-standardised rate (per 100 000) (Random effect model)	7.60	7.05	7.80					
Age- and area-adjusted (model 1)		1.00 (Reference)	1.23 (0.56 to 2.71)			0.869	0.993	0.828
Multivariate-adjusted (model 2)		1.00 (Reference)	1.17 (0.53 to 2.59)			0.844	0.974	0.834
Multivariate-adjusted (model 3)		1.00 (Reference)	1.17 (0.52 to 2.60)			0.874	0.979	0.850

Continued

Table 4 Continued

	Total	Green tea consumption				p For trend	p For heterogeneity (for the highest category)	p For heterogeneity (for trend)
		<1 cup/day	1–2 cups/day	3–4 cups/day	≥5 cups/day			
		HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)			
Distal (lower two thirds)								
No of subjects	72611	16271	14878	18983	22479			
Person-years	758865	173390	157522	199008	228944			
No of cases	370	83	64	117	106			
Age-standardised rate (per 100 000)	52.10	58.86	45.95	61.30	44.24			
(Random effect model)								
Age- and area-adjusted (model 1)		1.00 (Reference)	0.80 (0.57 to 1.12)	0.97 (0.72 to 1.31)	0.74 (0.53 to 1.03)	0.100	0.221	0.314
Multivariate-adjusted (model 2)		1.00 (Reference)	0.80 (0.57 to 1.12)	0.96 (0.71 to 1.30)	0.70 (0.50 to 0.995)	0.051	0.274	0.889
Multivariate-adjusted (model 3)		1.00 (Reference)	0.80 (0.57 to 1.13)	0.96 (0.71 to 1.30)	0.70 (0.50 to 0.96)	0.042	0.358	0.361

Model 2: Adjusted for age (continuous), area (JPHC-I, JPHC-II and JACC only), smoking (never smoker, past smoker, or current smoker), ethanol intake (never/former drinker, occasional drinker (<once/week), regular drinker (<23 g/day, ≥23 g/day)), rice intake (<4 bowls/day, ≥4 bowls/day), soy bean paste soup (<daily, daily), and coffee intake (<1 cup/day, 1–2 cups/day, ≥3 cups/day).
Model 3: Adjusted for pickled vegetable intake (<weekly, 1–2 times/week, 3–4 times/week, daily) and green–yellow vegetable intake (<weekly, 1–2 times/week, 3–4 times/week, daily) in addition to the variables included in Model 2.

the Japanese population.[27] In the present study, however, adjustment for smoking status did not change the results. Likewise, in stratified analysis by smoking status, we observed no substantial difference in the effect of green tea consumption between never smokers and current smokers. An anti-*Helicobacter pylori* effect by green tea is another possible explanation. A previous nested case–control study in two of the six cohorts[28] reported that *H pylori* did not distribute differentially in relation to tea polyphenol level in men, while positivity of *H pylori* infection was higher among women with lower tea polyphenol levels. This suggests some possibility in the sex difference in relation to the effect of green tea on *H pylori*, although this does not explain directly why green tea is associated with a decreased risk in women only. Further research on this issue is needed.

A difference in the effect of green tea by sex has also been observed for cardiovascular disease,[14 29] for which an oestrogen-related mechanism has been proposed. In support of this, tea flavonoids such as kaempferol have been shown to exhibit oestrogenic activity in vitro.[30] In addition, tea contains lignan polyphenols, such as secoisolaracinol, which are considered phytoestrogenic.[31] The phytoestrogens in tea might also partly account for the stronger protective effect of green tea against cancer in women than in men,[32 33] although an oestrogen-related protective mechanism against gastric cancer, if any, warrants further investigation. The pro-oxidant properties of tea polyphenols[34 35] or other factors related to men may explain the null findings observed in men.[28]

Second, a decreased risk in women was only seen for the distal subsite, and not for the proximal subsite. Only three studies have investigated the association by anatomical subsite,[6 7 12] of which two showed a decreased risk for the distal but not proximal subsite.[7 12] Consumption of tea at scalding temperatures increases the risk of proximal gastric cancer;[7] if present, this practice may have attenuated the risk reduction by green tea itself, confounding the results for the proximal subsite. Although the association with proximal gastric cancer was not clear in women, the risk appeared to be increased in the highest green tea consumption category in men. This may have been partly due to the effect of scalding hot tea. Due to the small number of proximal cancer cases in women, we bundled several frequent consumption categories together, and this may also partly explain the unclear risk trend for proximal cancer in women. Additional factors may include the proposed difference in aetiology between proximal and distal subsites, as well as the influence of *H pylori*. Specifically, *H pylori* may be associated with an increased risk of distal gastric cancer but not of cardia or oesophageal adenocarcinoma, in which eradication of the bacteria rather increases the risk of gastro-oesophageal reflux.[36] Experimental studies support the notion that green tea catechins have an inhibitory effect on *H pylori* infection and suppress *H pylori*-induced gastritis.[37–39] These findings suggest that the protective effect of green tea on gastric cancer may operate by decreasing the effect of this bacterium.

The present study had several strengths. First, we analysed data from cohort studies that used validated questionnaires to collect data on green tea consumption. In particular, the question used to assess green tea consumption was almost identical across the studies. Second, each study controlled for a common set of variables that are known or suggested to cause or prevent gastric cancer. Third, with a large number of habitual consumers of green tea, we were able to examine the effect of green tea with reasonable statistical power, albeit that power appeared insufficient in the sub-analyses in each cohort.

Our study also had several limitations. First, we used only baseline information on green tea consumption, and thus could not assess the effects of lifetime consumption on risk or changes in consumption during follow-up. Non-consumers of green tea are rare in Japan and it is possible that these subjects are a selection of the population that is at increased risk of gastric cancer. Some subjects with gastric cancer might have decreased their consumption before the diagnosis because of their symptoms. Likewise, it is possible that the observed protective effect of green tea among heavy drinkers only might be that the gastrointestinal symptoms associated with *H pylori* infection might force a person to avoid drinking green tea. Such change in practice might have biased their recall of past intake in such a way that they underestimated their true consumption, resulting in spurious inverse association. However, analyses of each cohort which excluded the early cases did not substantially change the results.[9 10 13 14] Second, the proportion of missing values for green tea consumption among the study subjects was 4.2% and excluded from the study. The exclusion of these subjects may have distorted the results, although the proportion was low and any influence may not have been substantial. Third, random variation related to exposure measurement might have attenuated the associations. In addition, we used the indicator terms for missing covariates, and this may have introduced bias. The proportion of missing data was 8.6% for smoking, 8.1% for alcohol intake, 2.7% for rice intake, 2.2% for soy bean paste soup intake, 15.7% for coffee intake, 4.1% for pickled vegetable intake and 4.5% green–yellow vegetable intake, showing variation by covariate, some cases of which were not negligible. We conducted analyses which were restricted to subjects with complete information and obtained closely similar values. Fourth, we are unable to exclude the possibility that our estimates were distorted because of residual confounding. Finally, we did not obtain information on *H pylori* infection status for the whole population, a strong risk factor for gastric cancer. Green tea is suggested to have antibacterial effects,[37–39] and green tea may be associated with gastric cancer risk through the effect of green tea on this infection. It is therefore likely that the failure to adjust for this infection may have resulted in the apparent protective effect of green tea on gastric cancer risk.

Allowing for these methodological issues, this pooled analysis of data from large prospective studies in Japan confirmed a significant decrease in risk of gastric cancer among women with high green tea consumption, especially for the distal subsite. Further investigation of our findings of differences in effect by sex and subsite will help elucidate the mechanism underlying the etiology of gastric cancer.

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Editor's quiz: GI snapshot

Robin Spiller, *editor*

Epigastric pain in a man with previous subtotal gastrectomy

CLINICAL PRESENTATION

A 68-year-old man presented to our hospital with a 2-day history of upper abdominal pain and non-bilious vomiting. Twenty years previously he had undergone a subtotal gastrectomy with Billroth II reconstruction because of a gastric ulcer. He denied alcohol consumption or trauma. Physical examination revealed that his upper abdomen was tender with muscle guarding and rebound tenderness. Laboratory tests showed the following: haemoglobin 11 g/dl (normal, 14–16 g/dl), white blood count 12.9×10^9/l (normal, $4.0–10.0\times10^9$/l), amylase 1744 IU/l (normal, 27–131 IU/l) and lipase 4587 IU/l (normal, 8–58 IU/l). Abdominal CT scan demonstrated a markedly distended, fluid-filled afferent loop crossing the midline (fig 1). Additionally, a 5×3 cm lesion was identified on CT images showing the target sign in the proximal segment of the afferent loop (fig 2). A diagnosis of afferent loop syndrome (ALS) complicated by acute pancreatitis was made based on symptoms, laboratory studies and CT images. The patient underwent an emergency laparotomy.

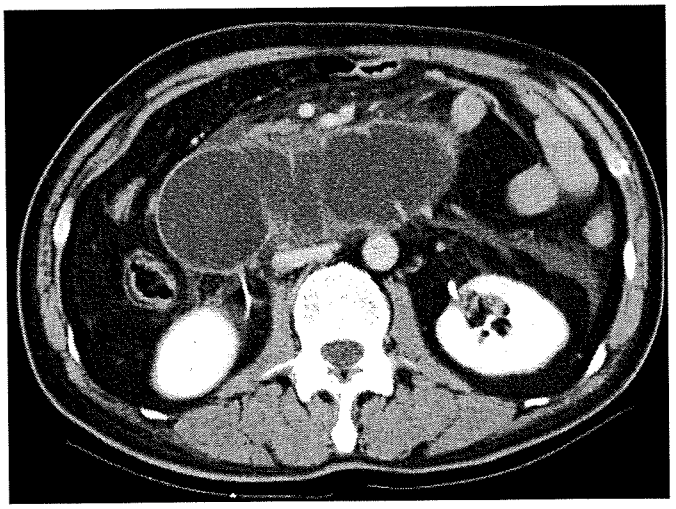

Figure 1 Abdominal CT scan demonstrated a markedly distended, fluid-filled afferent loop crossing the midline.

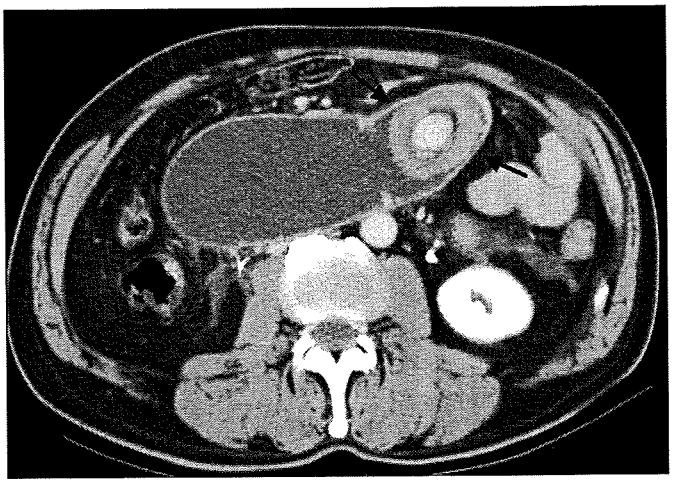

Figure 2 A 5×3 cm lesion with target sign in the proximal segment of afferent loop was identified on CT images (arrows).

QUESTION

What is the cause of afferent loop syndrome?

See page 1436 for the answer.

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