HHS Public Access

Author manuscript

Clin Gastroenterol Hepatol. Author manuscript; available in PMC 2024 October 01.

Published in final edited form as:

Clin Gastroenterol Hepatol. 2023 October ; 21(11): 2968–2971.e3. doi:10.1016/j.cgh.2022.10.012.

Socioeconomic status and ethnic enclave as risk factors for gastric adenocarcinoma in Hispanic and Asian Americans, a CCR analysis

Christina P. Wang, MD[1], **Meg McKinley, MPH**[2,3], **Scarlett L. Gomez, MPH, PhD**[2,3,4], **Samir Gupta, MD, MSCS**[5,6], **Steven H. Itzkowitz, MD**[1], **Shailja C. Shah, MD, MPH**[*,5,6], **Gastric Cancer Control and Prevention Collaboration**, **Ji Yoon Yoon, MD**[1], **Annie Vu, MPH**[2,3], **Michelle Kang Kim, MD, PhD**[1]

[1]Dr. Henry D. Janowitz Division of Gastroenterology, Icahn School of Medicine at Mount Sinai, New York, NY

[2]Department of Epidemiology and Biostatistics, University of California San Francisco, San Francisco, California

[3]Greater Bay Area Cancer Registry, UCSF

[4]Helen Diller Family Comprehensive Cancer Center, UCSF

[5]GI Section, VA San Diego Healthcare System, San Diego, CA

[6]Division of Gastroenterology University of California San Diego, San Diego, CA

Gastric cancer (GC) is the fourth leading cause of cancer-related mortality and the fifth most common cancer worldwide.[1] The majority of GC cases are noncardia gastric adenocarcinomas (NCGA), while cardia gastric adenocarcinomas (CGA) account for a small minority. In the United States (US), the frequency of NCGA and CGA among racial and ethnic groups differs[2], with non-White groups having significantly higher NCGA rates and non-Hispanic White groups experiencing significantly higher CGA rates.

[*]**Corresponding Author:** Shailja C. Shah MD, MPH, 3rd Floor, GI Section, Mail Code 111D, 3350 La Jolla Village Drive, La Jolla, CA 92161, Telephone number: 619-854-9550, s6shah@health.ucsd.edu.

Authorship Contributions:

Conceptualization: SCS; Data curation: MM, AV; Formal analysis: MM, AV; Investigation: SCS, MM, AV; Methodology: SCS, MM, AV; Project administration: SCS, CPW; Resources: SCS, MM, AV; Software: MM, AV; Supervision: SCS, CPW; Validation: SCS, CPW, MM, AV; Visualization: SCS, CPW, MM, AV; Writing original draft: SCS, CPW, JYY; Writing review & editing: SCS, CPW, SG, SHI, SLG, MKK, MM, AV

Conflict of Interest:

The authors declare no relevant conflicts of interest related to this article.

The collection of cancer incidence data used in this study was supported by the California Department of Public Health pursuant to California Health and Safety Code Section 103885; Centers for Disease Control and Prevention's (CDC) National Program of Cancer Registries, under cooperative agreement 5NU58DP006344; the National Cancer Institute's Surveillance, Epidemiology and End Results Program under contract HHSN261201800032I awarded to the University of California, San Francisco, contract HHSN261201800015I awarded to the University of Southern California, and contract HHSN261201800009I awarded to the Public Health Institute, Cancer Registry of Greater California. The ideas and opinions expressed herein are those of the author(s) and do not necessarily reflect the opinions of the State of California, Department of Public Health, the National Cancer Institute, and the Centers for Disease Control and Prevention or their Contractors and Subcontractors.

A recent meta-analysis demonstrated that early-generation immigrants from areas of high GC incidence and mortality (e.g. East Asia and Central/Latin America) retain this elevated risk upon moving to lower incidence countries (e.g. US).[3] Generally, this risk appears to decline with each subsequent generation born to and acculturating in the host country, thus implicating non-genetic—and thus, potentially modifiable—factors.

Prior studies have examined GC incidence by neighborhood socioeconomic status (nSES), race, and ethnicity.[2,4] Enclave status is a composite index that reflects immigration and acculturation traits among residents of a neighborhood and encompasses the built environment and social context of a community. We hypothesized that nSES and enclave status are associated with GC risk, given that these social determinants of health (SDOH) are known to impact healthcare access, diet, and physical activity.[5] Herein, we aimed to extend the literature by conducting a population-based analysis to evaluate differences in NCGA and CGA incidence among Hispanic and Asian populations according to neighborhood enclave and nSES.

nSES and neighborhood acculturation indices (enclave status) were derived from 2007–2011 (nSES) and 2008–2012 (acculturation) American Community Survey data using principal component analysis. The final nSES index contained the following variables: 1) education index, 2) percentage of persons above 200% poverty line, 3) percentage of persons with a blue-collar job (working-class job typically involving manual labor), 4) percentage of persons employed, 5) median rental, 6) median value of owner-occupied housing unit, 7) median household income. The final Hispanic- and Asian-specific acculturation indices reflected immigration and acculturation traits of residents at the census block-group and tract level. Variables included the percentage of recent immigrants (i.e., immigrated to the US after the year 2000), percentage of Spanish or Asian language-speaking households that are linguistically isolated, percentage of Spanish or Asian language-speakers with limited English proficiency, and percentage of census block-groups and tracts that are Hispanic or Asian. Detailed methods are provided in the Supplemental Material.

Between 2008–2012, 9009 incident gastric adenocarcinoma (GA) cases were registered in the California Cancer Registry (CCR) among Hispanic and Asian Americans ≥20 years-old (Supplemental Tables 1–2). GA incidence stratified by enclave, nSES, and enclave x nSES are illustrated in Figure 1A–C. NCGA but not CGA incidence varied according to enclave status. Hispanic and Asian Americans living in high enclave (least acculturated) neighborhoods had higher NCGA incidence (7.51 [95% CI: 7.11–7.92]) per 100,000 Hispanic Americans; and 9.68 [95% CI: 9.06–10.33] per 100, 000 Asian Americans) compared to their respective counterparts living in low enclave neighborhoods (5.60 [95% CI: 5.02–6.22]) per 100,000 Hispanic Americans; and 8.22 [95% CI: 7.58–8.90] per 100,000 Asian Americans, Figure 1A). Hispanic and Asian Americans residing in high enclave communities had a greater incidence of intestinal-type NCGA, while there was minimal variation in diffuse-type NCGA based on enclave status (Supplemental Tables 1–2).

NCGA incidence increased as nSES tertile decreased, with a significant difference observed between the lowest and highest nSES tertiles among both Hispanic and Asian Americans (Figure 1B). The same inverse pattern between intestinal-type NCGA and nSES tertile

was observed for Hispanic and Asian Americans, but there was less variation observed for diffuse-type NCGA according to nSES (Supplemental Tables 1–2). Among Asian Americans, CGA incidence was greatest among those in the highest nSES tertile, whereas among Hispanic Americans, variation in CGA incidence was less pronounced according to nSES.

When evaluating enclave and nSES together, Hispanic and Asian Americans in the lowest nSES and least acculturated (high enclave) neighborhoods had the highest NCGA incidence (Figure 1C). Among Hispanic Americans, enclave and nSES status influenced NCGA incidence more so than for CGA. In contrast, among Asian Americans, nSES tertile more so influenced GA (CGA and NCGA) incidence compared to enclave status. Compared to intestinal-type NCGA, there was less variation in diffuse-type NCGA incidence according nSES x enclave. (Supplemental Tables 1–2).

Herein, we extend the current body of literature[4,6] by providing detailed assessments of anatomic site-specific GA incidence according to nSES and acculturation level among Hispanic and Asian Americans, which are the two fastest-growing ethnicities in the US. Similar to Gupta et al,[4] we confirmed that NCGA incidence varies by nSES for both Hispanic and Asian Americans.

Neighborhood and built environment, and social and community context are types of SDOH.[5] Enclave and nSES may be considered midstream SDOH in that they partly result from upstream policies and influence the risk of adverse downstream outcomes, such as GA; such examples include crowded living, water quality, and access to food variety. Low nSES and high enclave (low acculturation) are often associated with overcrowding and poor sanitation that promote *Helicobacter pylori* transmission. *H. pylori*, the strongest modifiable risk factor for intestinal-type NCGA, is most often acquired during childhood and it is the cumulative exposure over time, along with other endogenous and exogenous factors, that drives intestinal-type NCGA risk.[6,7] By contrast, we observed minimal variation in incidence according to nSES and enclave for diffuse-type GA, which develops by different, less defined, pathways where the impact of specific environmental and other factors is not as well understood.

Similarly, enclave and nSES may act, in part, as surrogates for other individual-level risk determinants that are relevant to GA risk, and are potentially modifiable (e.g. smoking, obesity, diet).[7] Tobacco smoking, which is a risk factor for both NCGA and CGA, positively correlates with low nSES but inversely correlates with neighborhood immigrant density.[8] Obesity is a stronger risk factor for CGA than NCGA.[7] Rates of obesity tend to be higher in low nSES neighborhoods, but rates of obesity according to enclave vary depending on the racial or ethnic group.[7,9,10] Diet varies markedly according to nSES and enclave status and the interaction between diet and NCGA or CGA risk are complex.[2] Data regarding these individual-level exposures are not available in the CCR and this certainly merits future investigation.

Apart from the lack of individual exposure data, there are some additional limitations of our study. Use of the CCR may limit the application of our findings to at-risk individuals

residing in different regions of the US. Small case counts limited the ability to draw more extensive conclusions by gender. Finally, enclave and nSES data were available for Hispanic and Asian immigrants but not for other groups for which these same concepts might also be relevant; future studies should investigate this.

In conclusion, the present study identified neighborhood-level metrics that could serve as adjunctive measures for understanding how SDOH influence GA risk. As the US immigrant population expands and diversifies, better understanding enclave and nSES as SDOH as they relate to neighborhood and built environment, as well as social and community context, offers critical opportunities to intervene and promote the GA risk attenuation efforts that are sorely needed.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Funding:

No direct funding was received for this study, nor for writing support. The authors declare the following grant support: CPW receives research salary support from the National Cancer Institute of the National Institutes of Health under Award Number T32CA225617. SCS receives research salary support from an American Gastroenterological Association Research Scholar Award and Veterans Affairs Career Development Award (ICX002027A). The content is solely the responsibility of the listed authors and does not necessarily represent the official views of the funding agencies listed. MM, AV, and SLG receive support from the NCI Surveillance, Epidemiology, End Results (SEER) Program (HHSN261201800032I) and the California Department of Public Health.

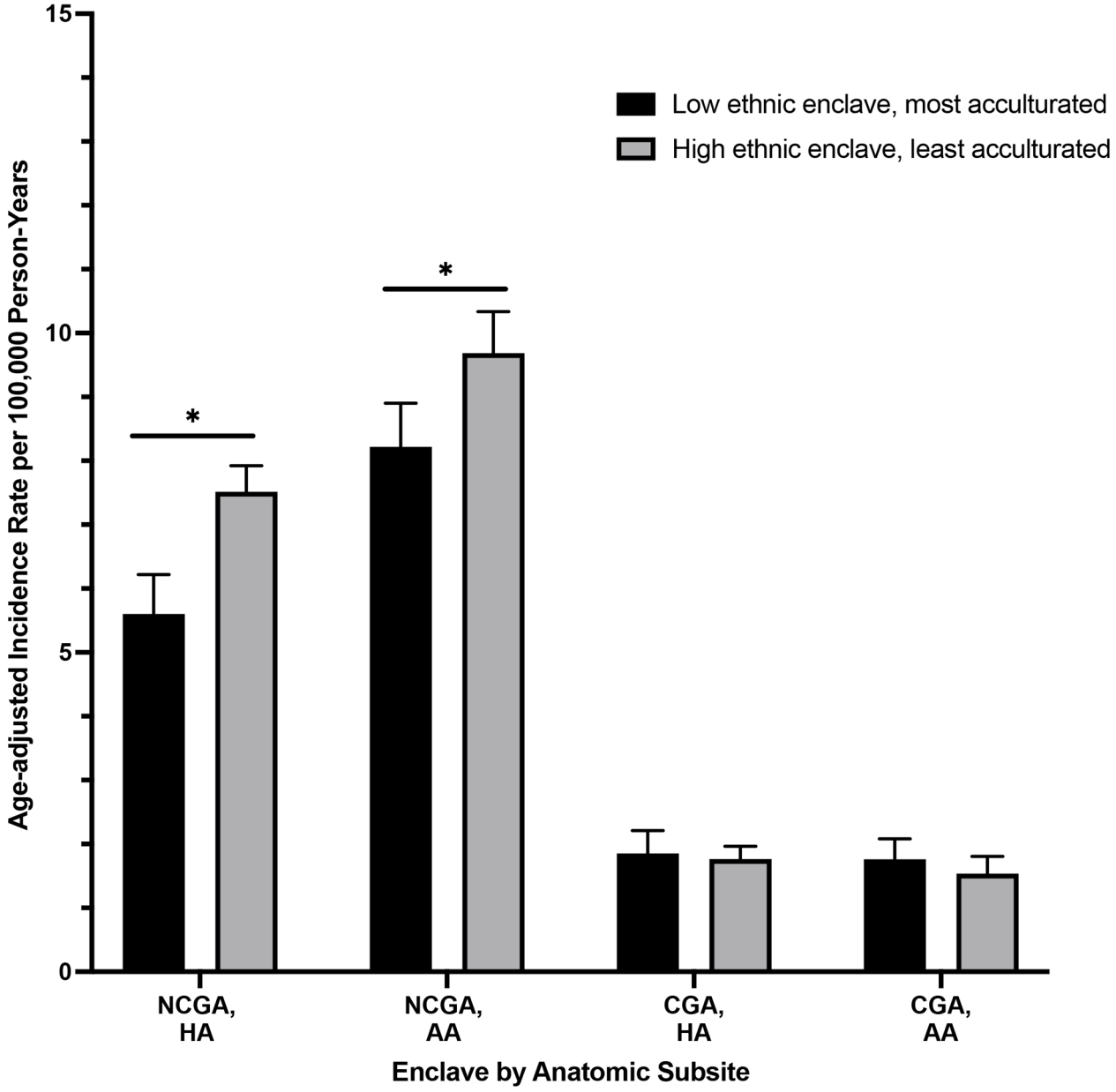
References

1. Sung H, et al. CA Cancer J Clin. May 2021;71(3):209–249. [PubMed: 33538338]
2. Shah SC, et al. Gastroenterology. Nov 2020;159(5):1705–1714 e2. [PubMed: 32771406]
3. Pabla BS, et al. Clin Gastroenterol Hepatol. Feb 2020;18(2):347–359 e5. [PubMed: 31154030]
4. Gupta S, et al. Gastroenterology. Jan 2019;156(1):59–62 e4. [PubMed: 30267713]
5. McLeod MR, et al. Hematol Oncol Clin North Am. Jun 2022;36(3):415–428. [PubMed: 35504786]
6. Chang ET, et al. Cancer Epidemiol Biomarkers Prev. May 2012;21(5):709–19. [PubMed: 22374991]
7. de Martel C, et al. Gastroenterol Clin North Am. Jun 2013;42(2):219–40. [PubMed: 23639638]
8. Karriker-Jaffe KJ, et al. J Ethn Subst Abuse. 2016;15(1):73–91. [PubMed: 26115317]
9. Powell-Wiley TM, et al. Prev Med. Sep 2014;66:22–7. [PubMed: 24875231]
10. Wong MS, et al. Prev Med. Jun 2018;111:371–377. doi:10.1016/j.ypmed.2017.11.029 [PubMed: 29197530]

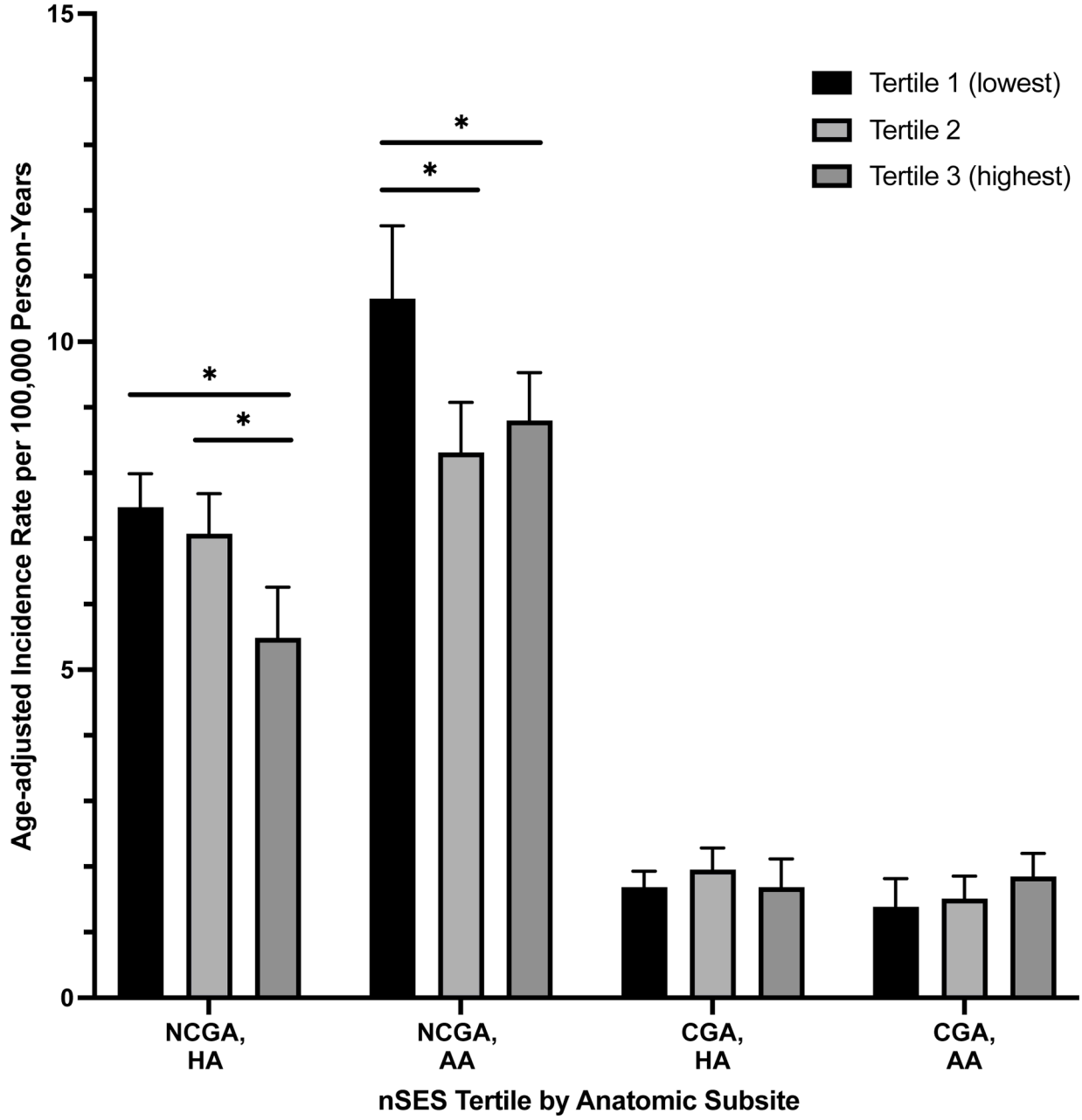
Data Transparency Statement:

The data informing this article were provided by the California Cancer Registry after submitting a protocol and being granted data access permissions. Data will not be shared publicly, but interested parties have the opportunity to contact the California Cancer Registry to request permissions.

A.



B.



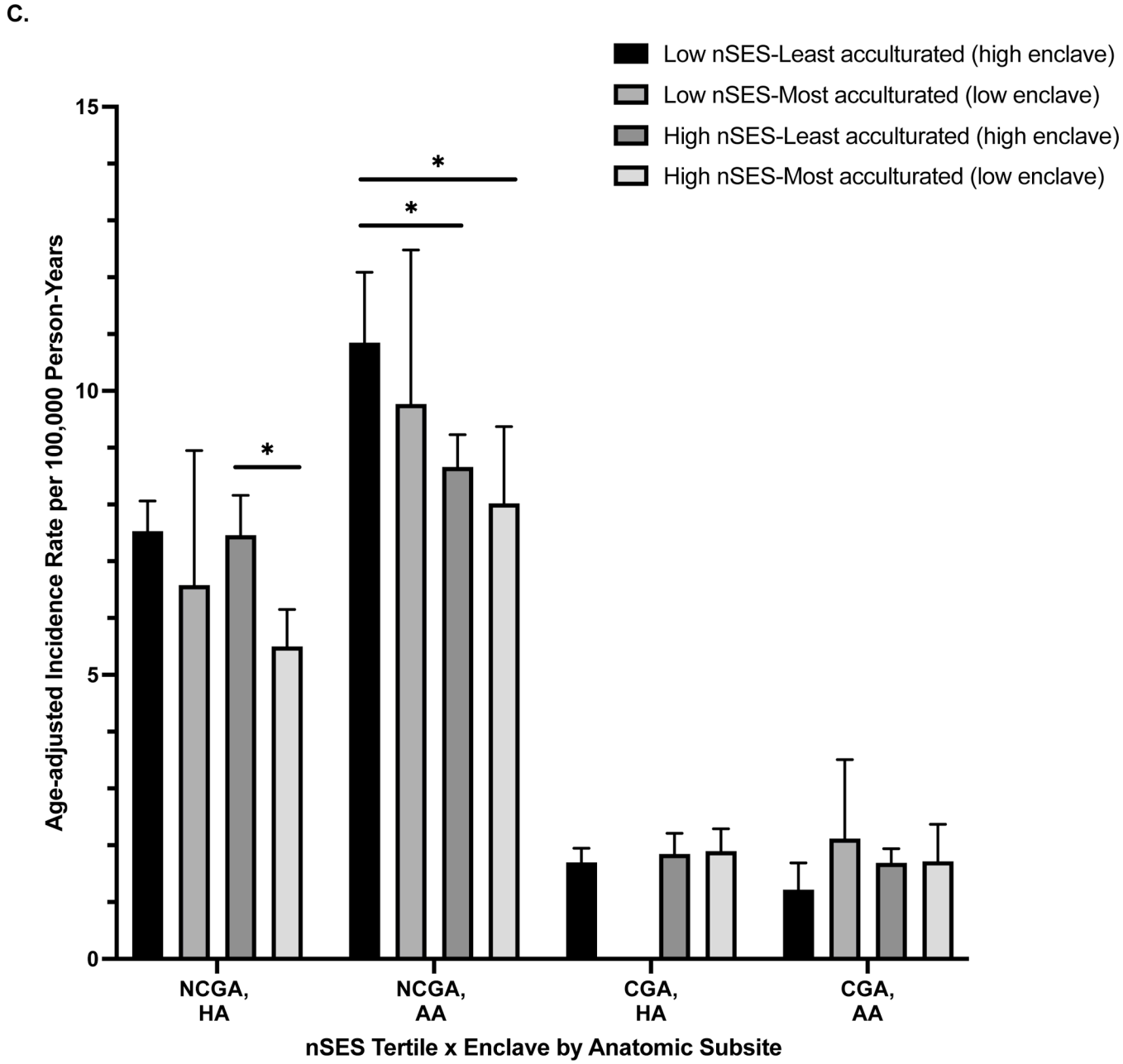


Figure 1.

Gastric adenocarcinoma incidence per 100,000 person-years among individuals aged 20 years or older by anatomic site and (A) enclave, (B) nSES, (C) nSES x enclave in Hispanic and Asian Americans, California Cancer Registry, 2008–2012.

HA = Hispanic Americans; AA = Asian Americans;

NCGA = noncardia gastric adenocarcinoma

CGA = cardia gastric adenocarcinoma

nSES = neighborhood socioeconomic status

*Significant, $p < 0.05$