

Prevalence of eosinophilic esophagitis in a population-based cohort from Southern California

Susan Kim, MD^a, Steven Kim, DO^{b,c}, and

Javed Sheikh, MD^{a,c}

Clinical Implications

- We report a higher proportion of diseased children from racial minority backgrounds compared with adults. This indicates that the inclusion of minorities in studies of eosinophilic esophagitis susceptibility and prognosis will likely reveal important insights into the etiology and pathogenesis of disease.

TO THE EDITOR:

Eosinophilic esophagitis (EoE) has been reported to affect more men than women,¹⁻⁷ more adults than children,^{2,3} and more white individuals than those from racial minority backgrounds.^{1-4,6,7} However, no population-based study has been conducted to evaluate EoE epidemiology using a validated cohort, representative of the community it serves. The objectives of this study were to determine the prevalence of EoE in Southern California using such a cohort, and to examine socio-demographic differences. After institutional review board approval, we reviewed administrative data from the medical records of 3,486,069 members of Kaiser Permanente Southern California (KPSC) enrolled from 2008 to 2013. KPSC comprises a multicenter health plan, inclusive of 14 medical centers and 143 medical offices throughout Los Angeles, Orange, San Bernardino, San Diego, Kern, Riverside, and Ventura counties. This cohort has been shown to be representative of the general population in Southern California for age, sex, race, and socioeconomic status.⁸ *International Classification of Diseases, Ninth Revision (ICD-9)* code 530.13 was used to identify patients diagnosed with EoE. Differences according to sociodemographic factors were tested using χ^2 analysis.

EoE was diagnosed in 1,561 of 3,486,069 patients (prevalence 4.5:10,000). Cases comprised 1344 adults and 217 children. The prevalence of EoE among adults (4.9:10,000) was significantly higher than that among children (2.9:10,000; [Table I](#); $P < .0001$). The prevalence among males (6.8:10,000) was significantly higher than that among females (2.6:10,000; [Table I](#); $P < .0001$); this prevalence ratio did not vary with age.

The racial distribution of our adult KPSC membership overall during the study period differed from that of children ([Table I](#)). Whites made up the majority of adult (76%) and pediatric (52%) cases; however, we found a higher prevalence of disease in children from racial minority groups than in adults ([Table I](#); $P < .0001$). Overall, the prevalence of EoE among whites (11.6:10,000) was significantly higher than that among blacks (3:10,000), Asians (1.6:10,000), and Hispanics (1.4:10,000) ([Table I](#); $P < .0001$).

There was a significant relationship between prevalence and household income. EoE was almost 4 times more common in

those from households of the highest quartile for annual income (8.9:10,000) than in those from the lowest (2.3:10,000), with a clear dose-response relationship noted across the middle quartiles ([Table I](#); $P < .0001$). Among adults, a similar dose-response relationship was observed with regard to education level ([Table I](#); $P < .0001$).

Previous epidemiologic studies report higher prevalence ratios. One study based on national health insurance claims estimated the prevalence to be 5.67:10,000.² A Minnesota investigation found the prevalence to be 5.6:10,000.⁴ Our prevalence estimate, 4.5:10,000, is slightly lower; however, research conducted at tertiary care centers report higher prevalence estimates.^{2-4,7}

We observed that disease is more frequently diagnosed in adults (86%) than in children (14%). Previously, adults comprised 74% to 76% of total EoE cases.^{2,3}

EoE has been reported to be 2 to 4 times more common in men than in women.¹⁻⁷ We found that EoE occurs 2.1 times more frequently in men than in women.

We also report that patients are more likely to be white than from racial minority groups. However, recent studies have shown increased frequency in nonwhite populations.^{5,6} We report a higher proportion of diseased children from racial minority backgrounds compared with adults ($P < .0001$).

Researchers from Children's Hospital of Philadelphia used a previously validated method of block group analysis to confirm that EoE is mainly a white-male-based disease, affecting those of higher socioeconomic strata.⁷ We observed a dose-response relationship between EoE prevalence and household income, as well as between prevalence and education level.

A limitation of this study is reliance on coding data to identify prevalent cases. Although *ICD-9* coding has not been validated to recognize this condition in our particular cohort, a recent study found high specificity (99%) but relatively low (37%-61%) sensitivity of *ICD-9* coding to identify EoE cases.⁹ Our method, however, does not require that clinicians actually be familiar with appropriate coding for EoE because coding is linked automatically to the diagnosis in our electronic health system. It is possible, however, that because of the prevailing belief that EoE is rarer in those from racial minority backgrounds, more patients from racial minority groups are miscoded with esophageal reflux or unspecified esophagitis. Furthermore, race categorization by self-report can be problematic, evidenced by the sizable proportion of patients who identify themselves as "mixed," "other," or "declined to state." With the rise in blending of our multiracial population, future epidemiologic studies will likely continue to encounter difficulties in the analysis of disease by race.

In addition, the use of *ICD-9* coding theoretically might overestimate the prevalence of EoE because it does not take into consideration the diagnosis of proton pump inhibitor (PPI)-responsive esophageal eosinophilia. A representative sample ($n = 75$) was used to validate that the *ICD-9* code appropriately identified cases of EoE (and not PPI-responsive esophageal eosinophilia), with all 75 patients noted to have had a positive esophageal biopsy performed while on high-dose PPI therapy.

Furthermore, we did not ascertain the independent effects of race, sex, and household income on prevalence. Health-seeking and/or provider behaviors may very well explain the racial and

TABLE I. EoE prevalence and sociodemographic characteristics, 2008-2013

Characteristic	Adult, ≥18 y				Pediatric, <18 y				Total				Prevalence per 10,000
	KPSC members	%	EoE cases	%	KPSC members	%	EoE cases	%	KPSC members	%	EoE cases	%	
N	2,735,124	78	1344	86	750,945	22	217	14	3,486,069	100	1561	100	4.5
Sex													
Male	1,171,593	43	903	67	372,323	50	159	73	1,552,966	45	1062	68	6.8
Female	1,563,531	57	441	33	378,622	50	58	27	1,933,103	55	499	32	2.6
Race													
White	836,848	31	1019	76	143,920	19	113	52	976,642	28	1132	73	11.6
Hispanic	911,476	33	130	10	355,449	47	48	22	1,271,981	36	178	11	1.4
Black	216,050	8	54	4	57,378	8	28	13	273,335	8	82	5	3
Asian	269,655	10	40	3	53,920	7	13	6	322,608	10	53	3	1.6
Other*	501,095	18	101	7	140,278	19	15	7	641,503	18	116	8	1.8
HHI†													
>75th	536,605	20	506	38	127,213	17	81	37	662,820	19	587	38	8.9
50-75th	566,423	21	346	26	144,046	19	47	22	710,043	20	393	25	5.5
25-49th	613,318	22	267	20	163,811	22	39	18	776,877	22	306	20	3.9
<25th	606,165	22	146	11	175,237	23	30	14	781,794	22	176	11	2.3
Unknown‡	412,613	15	79	15	140,638	19	20	9	554,535	17	99	6	1.8
Education§													
≥College	1,225,833	45	951	71	—	—	—	—	—	—	—	—	7.8
HS	779,408	28	267	20	—	—	—	—	—	—	—	—	3.4
<HS	317,394	12	47	3	—	—	—	—	—	—	—	—	1.5
Unknown‡	412,489	15	79	6	—	—	—	—	—	—	—	—	1.9

HHI, Household income; HS, high school.

*Other includes Native American, Alaskan, mixed, unknown, other, missing data, declined to state, not reported.

†Annual household income >\$78,043 (>75th percentiles), \$59,108 to \$78,043 (50th-75th percentiles), \$43,431 to \$59,107 (25th-49th percentiles), <\$43,431 (<25th percentiles).

‡Unknown: missing data, declined to state, not reported.

§Education level: ≥College (completed at least some college), HS (completed HS only), <HS (did not complete HS).

socioeconomic disparities observed. And although our cohort is representative of our regional Southern California population, our results do not reflect patients from other geographic regions or patients on a national scale.

The strengths of this study include the use of our large membership at KPSC, a cohort that has been shown to be representative of the geographic region from which membership is drawn. Our investigation is the first population-based study conducted to evaluate the epidemiology of EoE using an appropriately representative cohort. We report a higher proportion of diseased children from racial minority backgrounds compared with adults. These findings indicate that the inclusion of minority groups in studies of EoE susceptibility and prognosis will likely reveal important insights into the etiology and pathogenesis of disease.

^aDepartment of Allergy and Clinical Immunology, Kaiser Permanente Southern California, Los Angeles, Calif

^bDepartment of Pediatric Gastroenterology, Kaiser Permanente Southern California, Los Angeles, Calif

^cSouthern California Permanente Medical Group, Los Angeles, Calif

Conflicts of interest: The authors declare that they have no relevant conflicts of interest.

Received for publication April 2, 2015; revised May 13, 2015; accepted for publication June 12, 2015.

Available online July 9, 2015.

Corresponding author: Susan Kim, MD, Kaiser Permanente Los Angeles Medical Center, Department of Allergy & Clinical Immunology, 1515 North Vermont Ave, 5th Fl, Los Angeles, CA 90027. E-mail: susan.kim@kp.org.

2213-2198

© 2015 American Academy of Allergy, Asthma & Immunology

<http://dx.doi.org/10.1016/j.jaip.2015.06.008>

REFERENCES

1. Liacouras CA, Furuta GT, Hirano I, Atkins D, Attwood SE, Bonis PA, et al. Eosinophilic esophagitis: updated consensus recommendations for children and adults. *J Allergy Clin Immunol* 2011;128:3-20.
2. Dellon ES, Jensen ET, Martin CF, Shaheen NJ, Kappelman MD. Prevalence of eosinophilic esophagitis in the United States. *Clin Gastroenterol Hepatol* 2014;12:589-96.
3. Ally MR, Maydonovitch CL, Betteridge JD, Veerappan GR, Moawad FJ. Prevalence of eosinophilic esophagitis in a United States military healthcare population [published online ahead of print May 15, 2014]. *Dis Esophagus*. <http://dx.doi.org/10.1111/dote.12229>.
4. Prasad GA, Alexander JA, Schleck CD, Zinsmeister AR, Smyrk TC, Elias RM, et al. Epidemiology of eosinophilic esophagitis over three decades in Olmsted County, Minnesota. *Clin Gastroenterol Hepatol* 2009;7:1055-61.
5. Weiler T, Mikhail I, Singal A, Sharma H. Racial differences in the clinical presentation of pediatric eosinophilic esophagitis. *J Allergy Clin Immunol Pract* 2014;2:320-5.
6. Chatham D, Cavender CP, Lieberman JA. Racial disparity in eosinophilic esophagitis from a single, defined population. *Ann Allergy Asthma Immunol* 2014;113:489-91.
7. Franciosi JP, Tam V, Liacouras CA, Spigel JM. A case-control study of sociodemographic and geographic characteristics of 335 children with eosinophilic esophagitis. *Clin Gastroenterol Hepatol* 2009;7:415-9.
8. Koebnick C, Langer-Gould AM, Gould MK, Chao CR, Iyer RL, Smith N, et al. Sociodemographic characteristics of members of a large, integrated health care system: comparison with US census bureau data. *Perm J* 2012;16:37-41.
9. Rybnicek DA, Hathorn KE, Pfaff ER, Bulsiewicz WJ, Shaheen NJ, Dellon ES. Administrative coding is specific, but not sensitive, for identifying eosinophilic esophagitis. *Dis Esophagus* 2014;27:703-8.

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.