Prevalence of Olfactory Impairment in Older Adults

Claire Murphy, PhD
Carla R. Schubert, MS
Karen J. Cruickshanks, PhD
Barbara E. K. Klein, MD, MPH
Ronald Klein, MD, MPH
David M. Nondahl, MS

Context Older adults represent the fastest-growing segment of the US population, and prevalences of vision and hearing impairment have been extensively evaluated. However, despite the importance of sense of smell for nutrition and safety, the prevalence of olfactory impairment in older US adults has not been studied.

Objective To determine the prevalence of olfactory impairment in older adults.

Design, Setting, and Participants A total of 2491 Beaver Dam, Wis, residents aged 53 to 97 years participating in the 5-year follow-up examination (1998-2000) for the Epidemiology of Hearing Loss Study, a population-based, cross-sectional study.

Main Outcome Measures Olfactory impairment, assessed by the San Diego Odor Identification Test and self-report.

Results The mean (SD) prevalence of impaired olfaction was 24.5% (1.7%). The prevalence increased with age; 62.5% (95% confidence interval [CI], 57.4%-67.7%) of 80- to 97-year-olds had olfactory impairment. Olfactory impairment was more prevalent among men (adjusted prevalence ratio, 1.92; 95% CI, 1.65-2.19). Current smoking, stroke, epilepsy, and nasal congestion or upper respiratory tract infection were also associated with increased prevalence of olfactory impairment. Self-reported olfactory impairment was low (9.5%) and this measure became less accurate with age. In the oldest group, aged 80 to 97 years, sensitivity of self-report was 12% for women and 18% for men.

Conclusions This study demonstrates that prevalence of olfactory impairment among older adults is high and increases with age. Self-report significantly underestimated prevalence rates obtained by olfaction testing. Physicians and caregivers should be particularly alert to the potential for olfactory impairment in the elderly population.

JAMA. 2002;288:2307-2312 www.jama.com

THE PREVALENCE OF TASTE AND smell disorders in the United States is unknown. More than 200 000 visits are made to physicians each year for chemosensory complaints,[1] yet most chemosensory problems may be undetected. The prevalence of chronic olfactory problems from the National Health Interview Survey (NHIS) was estimated at 1.42%, or 2.7 million Americans. In the age groups of 55 to 64 years, 65 to 74 years, and 75 years or older, the prevalence rates were 1.99%, 2.65%, and 4.60%, respectively.[2] A National Geographic survey in 1986 estimated prevalence among its readership of 0.8%.[3] These data are informative but are based on self-report and may substantially underestimate prevalence.

Persons with chemosensory problems, particularly those who are elderly or living alone, are at increased risk of both nutritional problems[4,5] and danger from fire or gas explosion.[6] Thus, prevalence of smell impairment and awareness of smell loss are important. A study of 80 healthy elderly persons and 80 patients with Alzheimer disease noted a lack of awareness of decreased ability to detect odor.[7] Laboratory studies involving actual testing of older persons show significant olfactory impairment in the elderly.[8,9]

The current investigation was conducted to determine the prevalence of olfactory impairment in a large population-based study of older adults and to compare the prevalence of olfactory impairment measured by olfaction testing with that measured by self-report.

METHODS

Participants

The Epidemiology of Hearing Loss Study (EHLS) is a population-based study of sensory loss and aging in older adults in Beaver Dam, Wis.[10] The San Diego Odor Identification Test (SDOIT)[11,12] and related olfaction questions were a component of the 5-year follow-up examination (1998-2000) of the EHLS cohort. Eligibility criteria for the baseline EHLS examination were age 43 to 84 years in 1987-1988, residence in the city or township of Beaver Dam in 1987-1988, and participation in the baseline examination (1988-1990) for the Beaver Dam Eye Study.[13] A private census was conducted in 1987-1988 to identify all residents of the city or township of Beaver Dam who were aged 43 to 84 years (n=5924). Of those identified, 83% (n=4926) participated in the eye study. In 1993-1995, the surviving members of this cohort (n=4541) were invited to par-

Author Affiliations: San Diego State University and University of California, San Diego, School of Medicine (Dr Murphy); and the Departments of Ophthalmology and Visual Sciences (Ms Schubert, Drs Cruickshanks, B. Klein, and R. Klein, and Mr Nondahl) and Population Health Sciences (Dr Cruickshanks), University of Wisconsin, Madison.
Corresponding Author and Reprints: Claire Murphy, PhD, San Diego State University/University of California, San Diego, Joint Doctoral Program, 6363 Alvarado Ct, Suite 101, San Diego, CA 92120 (e-mail: cmurphy@sciences.sdsu.edu).

©2002 American Medical Association. All rights reserved.

Downloaded From: http://jama.jamanetwork.com/ on 05/06/2015

ticipate in the baseline EHLS examination. Of these, 82.6% participated (n=3753). There were 3407 participants alive at the beginning of the EHLS 5-year follow-up, of whom 2800 (82.2%) participated in the study; 112 completed only the interview component. Of the 607 who did not participate, 164 (4.8%) died, 436 (12.8%) refused, and 7 (0.2%) were lost to follow-up. Patients with dementia were not excluded, but they were less likely to have complete olfaction data. Complete olfaction data were obtained from 2491 participants (92.7% of in-person examinations). All gave written informed consent according to the protocol approved by the University of Wisconsin–Madison Institutional Review Board.

Comparisons between the participants in the baseline EHLS and 1990 US Census data indicate that the cohort is similar to US non-Hispanic whites in age and sex distributions for people older than 40 years but were slightly less likely to report high annual income (≥$60 000).[10] Comparisons using 2000 census data confirm that the age and sex distributions are similar for participants in the 5-year follow-up and all US persons aged 55 years or older and for the subset of the US non-Hispanic white population.[14] Income data from the 2000 census have not yet been released. The EHLS cohort is predominately non-Hispanic white.

Olfactory Examination

Participants were tested individually by trained personnel with the SDOIT, an 8-item odor identification test that uses common, natural odors typically found in the home (eg, coffee, chocolate).[11,12] Test-retest reliability of this test was r=0.86 when tested with a mean delay of 5 days in a sample of 92 subjects.[11] Odorants were wrapped in gauze and kept in opaque containers to minimize visual clues. Participants were tested with the eyes closed to prevent visual clues, with an interstimulus interval of 45 seconds to minimize adaptation.[15] A picture board with illustrations of the target items as well as distracters was presented to aid in identification. The odorants were presented in random order to the participant. Scores were calculated as the percentage of the odorants correctly identified. To identify a suitable cut point for classification of impairment, scores were examined for the youngest participants in the current study (53-59 years) who reported having no sinus infections/sinus problems in the week prior to examination and did not have nasal congestion on the day of the examination. Ninety-five percent of this group achieved scores of 6, 7, or 8. Therefore, for estimating prevalence, participants identifying fewer than 6 of 8 odorants were considered impaired. This cut point also corresponds to approximately 2 SDs less than the mean score in a group of 75 healthy adults aged 20 to 40 years. Young adults with scores in this range would be considered clinically hyposmic.[16]

Information about self-assessed olfactory loss was obtained from a questionnaire that was administered as an interview. Self-reported smell impairment was determined by the question, "Do you have a normal sense of smell (compared to other people)?" The olfaction questions were asked prior to the administration of the SDOIT.

Data Analysis

Analyses were conducted with SAS software.[17] The relations between impaired olfaction and various potential risk factors were first assessed by means of contingency tables. The χ^2 test for general association was used to assess most relations. For the relations between impaired olfaction and use of labetalol hydrochloride and between impaired olfaction and Parkinson disease, the Fisher exact test was used because of small cell sizes.[18] For the relation between impaired olfaction and age group, the Mantel-Haenszel χ^2 test for overall trend was used.[19] Next, each potential risk factor was assessed in an age- and sex-adjusted logistic regression model with impaired olfaction as the dependent variable. Factors that were at or near statistical significance (χ^2 $P<.05$) were entered together into a multiple logistic regression model, which was then reduced to its most parsimonious form with the backwards-elimination selection method. Potentially important interactions were tested, but none was statistically significant. In addition to odds ratios, prevalence ratios (adjusted for the prevalence of impairment among the unexposed according to the method of Zhang and Yu[20]) are reported.

Potential risk factors examined included age; sex; smoking status; occupation (production, craftsman, repairman, operator, fabricator, or laborer vs other); sinus infection/problems in the past week; nasal congestion on the day of the examination or upper respiratory tract infection within the past week; history of allergies (mold/pollen/animals/food), head injury (skull fracture, concussion, broken nose, loss of consciousness due to a head injury, whiplash, or other serious neck injury), deviated septum, nasal polyps, chemotherapy, stroke, Parkinson disease, diabetes, and epilepsy; and use of selected medications based on past reports[21] (amitriptyline hydrochloride, nifedipine, propranolol hydrochloride, or labetalol).

RESULTS

The mean (SD) age of the participants in the olfactory examination was 68.7 (9.4) years; 58% of the participants were women. The distributions of participant characteristics by olfactory impairment as measured by the SDOIT are shown in TABLE 1. Twenty percent of those considered to have olfactory impairment by testing reported having an abnormal sense of smell, while 6% of those with normal scores on the SDOIT also reported having an abnormal sense of smell.

Overall, the mean (SD) prevalence of olfactory impairment in this population was 24.5% (1.7%). The prevalence of olfactory impairment was greater among men and older age groups (TABLE 2).

The prevalence of olfactory impairment as measured by the SDOIT (24.5%) was much higher than the prevalence of self-reported smell impairment (9.5%). Using the SDOIT test results as the cri-

 ©2002 American Medical Association. All rights reserved.

Downloaded From: http://jama.jamanetwork.com/ on 05/06/2015

terion standard, self-report was a poor measure of olfactory impairment. The overall sensitivity of self-report was 20% and the specificity was 94%. TABLE 3 shows the age- and sex-specific sensitivity (ability of self-report to identify those with impairment) and specificity (ability of self-report to identify those with normal olfaction) for self-reported impairment. Specificity was uniformly high for all groups, indicating that those with normal smell tend to accurately report no abnormalities, but the sensitivity of self-reported impairment was very poor, suggesting that many people with an impaired sense of smell were unaware of the problem. Sensitivity of self-report declined with age.

Next, we evaluated age- and sex-adjusted associations between the potential risk factors presented in Table 1 and the prevalence of olfactory impairment as measured by the SDOIT. As shown in TABLE 4, current smoking, nasal congestion or upper respiratory tract infection, history of stroke, and history of epilepsy were associated with olfactory impairment. When factors that were at or near statistical significance were entered together into a multiple logistic regression model, age, sex, current smoking, nasal congestion or upper respiratory tract infection, stroke, and epilepsy were associated with increased odds of olfactory impairment (TABLE 5). Olfactory impairment did not differ for former smokers compared with those who never smoked cigarettes.

COMMENT

This is the first large population-based study to our knowledge to report the prevalence of olfactory impairment measured by testing. The prevalence of measured olfactory impairment in this study of older persons was quite high (24.5% overall), and impairment increased with advancing age in both men and women. According to the 2000 census, there are approximately 60 million Americans aged 55 years or older.[14] Thus, we estimate that approximately 14 million older adults in the United States have olfactory impairment. The age effects observed in the present study are supported by smaller-scale studies with convenience and laboratory samples.[8,9,22,23] Likewise, in a convenience sample, 80% of the 123 participants aged 80 years or older showed olfactory impairment.[24] In the EHLS population, the prevalence of olfactory impairment was slightly lower among those 80 to 97 years of age (62.5%); both studies indicate a very high prevalence of olfactory impairment among the oldest age group.

This study also found differences in olfactory impairment between men and women. Men consistently demon-

Table 1. Descriptive Characteristics of Study Participants by Olfactory Impairment as Measured by the San Diego Odor Identification Test

Characteristic	Olfactory Impairment, No. (%) Yes (n = 610)	No (n = 1881)	*P* Value
Sex			
Men	318 (52.1)	728 (38.7)	<.001
Women	292 (47.9)	1153 (61.3)	
Age, y			
53-59	34 (5.6)	526 (28.0)	<.001
60-69	147 (24.1)	701 (37.3)	
70-79	217 (35.6)	527 (28.0)	
80-97	212 (34.8)	127 (6.8)	
Manufacturing occupation	218 (38.1)	502 (28.2)	<.001
Smoking			
Never	264 (45.4)	897 (48.5)	.42
Past	258 (44.3)	769 (41.6)	
Current	60 (10.3)	185 (10.0)	
Allergies	207 (34.0)	752 (40.0)	.008
Head injury	153 (25.1)	506 (26.9)	.38
Sinus problems	134 (22.1)	359 (19.2)	.12
Nasal congestion or upper respiratory tract infection	222 (36.4)	513 (27.3)	<.001
Deviated septum	45 (7.6)	128 (6.9)	.56
Nasal polyps	32 (5.3)	69 (3.7)	.08
Chemotherapy	33 (5.4)	53 (2.8)	.002
Stroke	31 (5.2)	29 (1.6)	<.001
Parkinson disease	10 (1.7)	7 (0.4)	.002
Diabetes	80 (13.9)	193 (10.6)	.03
Epilepsy	10 (1.6)	12 (0.6)	.02
Prescription drug use			
Amitriptyline hydrochloride	17 (2.9)	50 (2.7)	.79
Nifedipine	17 (2.9)	43 (2.3)	.43
Propranolol hydrochloride	14 (2.4)	35 (1.9)	.45
Labetalol hydrochloride	2 (0.3)	15 (0.8)	.39
Self-reported abnormal sense of smell	121 (20.2)	114 (6.1)	<.001

Table 2. Prevalence of Olfactory Impairment by Age and Sex*

	Women		Men		Total	
Age, y	No. at Risk	Prevalence, % (95% CI)	No. at Risk	Prevalence, % (95% CI)	No. at Risk	Prevalence, % (95% CI)
53-59	319	3.8 (1.7-5.9)	214	9.1 (5.5-12.8)	560	6.1 (4.1-8.1)
60-69	463	11.2 (8.4-14.1)	385	24.7 (20.4-29.0)	848	17.3 (14.8-19.9)
70-79	429	20.8 (16.9-24.6)	315	40.6 (35.2-46.1)	744	29.2 (25.9-32.5)
80-97	234	59.4 (53.1-65.7)	105	69.5 (60.7-78.3)	339	62.5 (57.4-67.7)
All ages	1445	20.2 (18.1-22.3)	1046	30.4 (27.6-33.2)	2491	24.5 (22.8-26.2)

*CI indicates confidence interval.

©2002 American Medical Association. All rights reserved.

Downloaded From: http://jama.jamanetwork.com/ on 05/06/2015

strated a higher prevalence of impairment compared with women in the same age group. These sex differences have been found in other studies[3,22,25,26] at all ages, even before puberty; similar differences have been observed with other measures of olfactory function (eg, event-related brain potentials[27-29]).

Self-reported abnormal sense of smell was substantially less prevalent than measured smell impairment. The finding is consistent with data from a smaller convenience sample of 80 healthy elderly and 80 Alzheimer disease patients.[7] In that study, both healthy elderly and Alzheimer disease patients showed unawareness of smell loss measured with olfactory thresholds. Hoffman et al[2] found prevalence rates for self-reported olfactory impairment of 1.99%, 2.65%, and 4.6% for age groups of 55 to 64 years, 65 to 74 years, and 75 years or older, respectively, in participants in the NHIS. Overall, the EHLS participants reported a higher prevalence of olfactory impairment than that found in the NHIS, which may reflect methodological differences or the older age distribution of the EHLS cohort compared with the NHIS. The NHIS queried household respondents about chronic (lasting ≥3 months) smell problems experienced by each household member rather than ascertaining self-reported impairment.[2] In contrast, the EHLS participants were asked about their sense of smell on the day of examination, so some transitory losses may have been included. Despite the differences, both the present study and the NHIS had significantly lower self-reported prevalence rates than those obtained by olfaction testing in this study.

Table 3. Sensitivity and Specificity of Self-reported Abnormal Olfaction by Age and Sex Compared With Impairment Detected Using the San Diego Odor Identification Test

Age, y	Sensitivity, %	Specificity, %
Women		
52-59	33	94
60-69	24	95
70-79	16	94
80-97	12	96
Total	16	94
Men		
52-59	36	91
60-69	25	93
70-79	24	95
80-97	18	100
Total	24	93
All	20	94

Table 4. Age- and Sex-Adjusted Odds Ratios (ORs) and Prevalence Ratios (PRs)[20] for Olfactory Impairment*

Exposure	OR (95% CI)	*P* Value	PR (95% CI)
Manufacturing occupation	1.23 (0.98-1.54)	.07	1.17 (0.98-1.38)
Smoking			
Past (vs never)	1.08 (0.85-1.35)	.54	1.06 (0.88-1.25)
Current (vs never)	2.15 (1.49-3.10)	<.001	1.70 (1.34-2.10)
Allergies	0.86 (0.69-1.06)	.16	0.89 (0.75-1.04)
Head injury	0.92 (0.73-1.17)	.49	0.94 (0.78-1.12)
Sinus problems	1.26 (0.98-1.62)	.07	1.19 (0.98-1.42)
Nasal congestion or upper respiratory tract infection	1.61 (1.29-2.00)	<.001	1.42 (1.21-1.65)
Deviated septum	1.16 (0.79-1.72)	.45	1.12 (0.83-1.47)
Nasal polyps	1.22 (0.76-1.97)	.41	1.16 (0.81-1.60)
Chemotherapy	1.44 (0.86-2.41)	.16	1.31 (0.89-1.81)
Stroke	1.99 (1.13-3.51)	.02	1.62 (1.10-2.22)
Parkinson disease	2.37 (0.82-6.81)	.11	1.79 (0.86-2.87)
Diabetes	1.08 (0.79-1.47)	.62	1.06 (0.83-1.33)
Epilepsy	4.12 (1.60-10.62)	.003	2.37 (1.40-3.24)
Prescription drug use			
Amitriptyline hydrochloride	1.18 (0.64-2.16)	.60	1.13 (0.70-1.69)
Nifedipine	1.00 (0.54-1.85)	.99	1.00 (0.61-1.54)
Propranolol hydrochloride	1.11 (0.55-2.25)	.77	1.08 (0.62-1.73)
Labetalol hydrochloride	0.34 (0.07-1.64)	.18	0.40 (0.09-1.42)

*CI indicates confidence interval.

The most common causes of clinically observed olfactory impairment fall into 4 categories: head trauma, inflammatory (including allergic rhinitis and nasal sinus disease), prior upper respiratory tract infection, and neurodegenerative disease (including Alzheimer disease and Parkinson disease).[21] Much less common are exposure to toxic chemicals, congenital anomaly, or other causes, such as craniofacial surgery, seizure disorders, cerebrovascular accidents, olfactory groove or temporal tumors, and endocrine disorders.[21,30,31] Although olfactory impairment has been reported in association with a large number of diseases,[21,30-40] the vast majority of patients presenting for assessment at taste and smell clinics have olfactory impairment as a result of allergic rhinitis, nasal sinus disease, history of prior upper respiratory tract infection, or head trauma.[21] In the present study, olfactory impairment was associated with nasal congestion at examination or having had an upper respiratory tract infection in the past week. In addition, stroke, epilepsy, and current smoking were associated with olfactory impairment. These findings are consistent with previous reports.[21] In some cases, olfactory impairment is reversible; thus, smell impairment is important to identify and treat when possible.[16] Use of antibiotics for bacterial causes of sinusitis and intranasal steroids for allergic sinusitis can reverse olfactory impairment.[16,41]

Schiffman[37] has demonstrated that older adults who take a moderate number of medications but who otherwise live active, healthy lives have poorer taste threshold sensitivity than young adults. The mechanisms for these adverse effects have not been identified but may involve the bitter tastes of many drugs that are present in the saliva or are circulating in the lingual blood supply.[42-45] The extent to which olfactory disorders occur as adverse effects to drugs is not known, but this is under investigation.[38,42] While our assessment of medication effects was limited by the lack of dosage and duration data, the present data suggest that on a population level, medication use

©2002 American Medical Association. All rights reserved.

Downloaded From: http://jama.jamanetwork.com/ on 05/06/2015

may not be a major contributor to the prevalence of olfactory impairment. Well-controlled clinical trials of the adverse chemosensory effects of drugs are needed to identify pharmacological agents that cause taste and smell disorders and to understand the underlying mechanisms for dysfunction.

In addition to known etiologies of olfactory dysfunction, several mechanisms may be involved in age-related olfactory impairment that are not associated with disease states. Both peripheral and central causes of age-related olfactory impairment have been postulated and both may play a role. Insult to the olfactory mucosa as a result of various disease processes and/or exposure to environmental pollutants has been speculated to lead to the replacement of olfactory mucosa with respiratory epithelium.[46,47] Atrophy of the olfactory bulb[48] and tract[49,50] occurs in old age, with numbers of glomeruli and mitral cells declining with age.[51] Structural magnetic resonance imaging shows age-associated volume loss in mesial temporal lobe areas important to olfactory processing.[52] Functional magnetic resonance imaging shows less activation in elderly individuals during olfactory processing, both in mesial temporal lobe areas (C.M., B. Cerf-Ducastel, unpublished data, 2002) and in orbital frontal cortex.[53] Olfactory event-related brain potentials show longer latencies and smaller amplitudes in elderly persons.[27-29]

Nutrition is an important factor to consider in older persons with olfactory deficits. Age-related changes in flavor perception may contribute to changes in food palatability and nutritional intake.[54-56] Since olfactory function is an important component in flavor perception, impaired odor perception, reflected in poor identification of odors, will dull the flavor of foods and beverages. Metabolic rate, energy intake, and macronutrient intake decrease with age, and food selection can become increasingly important in maintaining energy balance.[57] It is well documented that malnutrition and subclinical nutritional deficiencies occur among the elderly population.[4,5] An awareness of olfactory impairment and serious consideration of the issue of meal preparation may facilitate good nutrition in the elderly population.[37,58]

Table 5. Multivariate Model for Olfactory Impairment as Measured by the San Diego Odor Identification Test*

Exposure	OR (95% CI)	PR (95% CI)
Age per 5-year increment	1.82 (1.70-1.94)	1.75 (1.64-1.85)
Sex, male	2.46 (1.96-3.08)	1.92 (1.65-2.19)
Smoking history		
Past vs never	1.05 (0.83-1.33)	1.04 (0.86-1.24)
Current vs never	1.93 (1.33-2.81)	1.59 (1.24-1.99)
Nasal congestion or upper respiratory tract infection	1.60 (1.28-2.01)	1.42 (1.21-1.65)
Stroke	1.99 (1.12-3.53)	1.62 (1.09-2.22)
Epilepsy	4.37 (1.71-11.21)	2.43 (1.46-3.28)

*OR indicates odds ratio; CI, confidence interval; and PR, prevalence ratio.[20] Each exposure variable is adjusted for all other variables in the table.

An important warning system is compromised in older adults with impaired olfactory function, leaving them at increased risk for injury from gas explosion and fire. Indeed, older adults are involved in a disproportionate percentage of home fires,[59] suggesting the importance of heightened awareness of the need for gas and smoke detectors in the homes of impaired older adults.

The present study has some limitations. Olfaction testing was added in 1998-2000 as part of the 5-year follow-up examination for the EHLS. While, as in all studies of aging, mortality was the largest reason for loss to follow-up, the continuing comparability of the age and sex distributions of participants and the US population suggests that these data are reliable point estimates of the prevalence of olfactory impairment among older adults. The participants were primarily of non-Hispanic white race. The lack of racial diversity may limit the generalizability of these data to minority groups. However, others have found no differences in reported prevalence of smell loss by race/ethnicity (white, black, or Hispanic).[2]

Any measure of olfactory impairment, including the SDOIT, uses a limited set of stimulants that may leave some relatively rare specific anosmias undetected. Patients with dementia were not excluded from participation in the present study, but they were less likely to have complete olfaction data. Olfactory impairment is common in dementia. Thus, some cases of olfactory impairment in the cohort may have gone undetected. It is possible that we have underestimated the prevalence of olfactory disorders. However, given the SDOIT's high test-retest performance, low cost, acceptability to participants, and the short testing time required, this test is useful for population-based field studies of health and is clearly less likely to underestimate the prevalence of olfactory impairment than studies that rely on self-report.

CONCLUSIONS

The prevalence of impaired olfactory function found in this study implies that a significant portion of the older population has an impaired ability to identify common household odors and most of them are unaware of the impairment. The oldest participants in this study, those aged 80 to 97 years, demonstrated the highest prevalence of olfactory impairment. Thus, the present findings have important implications for the health and safety of older adults. There is a clear need to educate elderly persons and their caregivers about potential hazards accompanying impairment in olfactory function and how such hazards can be best avoided. Prevalence rates were also significantly greater when determined from actual testing rather than from self-report and the gap between self-reported and tested impairment increased with age. Therefore, administration of smell tests is necessary to obtain accurate prevalence rates of smell impairment, at least in the age

©2002 American Medical Association. All rights reserved.

Downloaded From: http://jama.jamanetwork.com/ on 05/06/2015

groups tested. We estimate that approximately 14 million older US adults have olfactory impairment. Further epidemiological studies are needed to assess prevalence of olfactory loss in racial/ethnic groups other than non-Hispanic whites. Future studies should include measurement of olfactory function.

Author Contributions: *Study concept and design:* Murphy, Schubert, Cruickshanks, B. Klein, Nondahl.
Acquisition of data: Murphy, Schubert, Cruickshanks, B. Klein.
Analysis and interpretation of data: Murphy, Schubert, Cruickshanks, R. Klein, Nondahl.
Drafting of the manuscript: Murphy, Schubert, Cruickshanks, Nondahl.
Critical revision of the manuscript for important intellectual content: Murphy, Schubert, Cruickshanks, B. Klein, R. Klein, Nondahl.
Statistical expertise: Murphy, Cruickshanks, Nondahl.
Obtained funding: Murphy, Cruickshanks.
Administrative, technical, or material support: Schubert, Cruickshanks.
Study supervision: Schubert, Cruickshanks.
Funding/Support: This research was supported by National Institutes of Health grants AG11099 to Dr Cruickshanks and AG04085 to Dr Murphy from the National Institute on Aging and EY06594 to Drs R. and B. Klein from the National Eye Institute.
Acknowledgment: We thank Judith Finkelstein, PhD, for facilitating this collaboration, Terence M. Davidson, MD, for otorhinolaryngological expertise, Michael D. Madowitz for assistance, and the cohort of the Beaver Dam epidemiological studies for participation.

REFERENCES

1. Because you asked about smell and taste disorders. NIH publication No. 95-3231. Available at: http://www.nidcd.nih.gov/health/pubs_st/smltaste.htm. Accessed February 17, 2002.
2. Hoffman HJ, Ishii EK, Macturk RH. Age-related changes in the prevalence of smell/taste problems among the United States adult population: results of the 1994 disability supplement to the National Health Interview Survey (NHIS). *Ann N Y Acad Sci.* 1998;855:716-722.
3. Wysocki CJ, Gilbert A. National Geographic smell survey: effects of age are heterogeneous. *Ann N Y Acad Sci.* 1989;561:12-28.
4. Davis L. Practical aspects of nutrition of the elderly at home. In: Munro H, Schlierf G, eds. *Nutrition in the Elderly.* New York, NY: Raven Press; 1992:203-209. Nestle Nutrition Workshop Series, Vol 29.
5. Vellas B. Effects of the aging process on the nutritional status of elderly persons. In: Munro H, Schlierf G, eds. *Nutrition in the Elderly.* New York, NY: Raven Press; 1992:75-78. Nestle Nutrition Workshop Series, Vol 29.
6. Chalke HD, Dewhurst JR. Accidental coal-gas poisoning. *BMJ*. 1957;2:915-917.
7. Nordin S, Monsch AU, Murphy C. Unawareness of smell loss in normal aging and Alzheimer's disease: discrepancy between self-report and diagnosed smell sensitivity. *J Gerontol B Psychol Sci Soc Sci.* 1995;50:P187-P192.
8. Murphy C. Nutrition and chemosensory perception in the elderly. *Crit Rev Food Sci Nutr.* 1993;33:3-15.
9. Schiffman SS. Perception of taste and smell in elderly persons. *Crit Rev Food Sci Nutr.* 1993;33:17-26.
10. Cruickshanks KJ, Wiley TL, Tweed TS, et al. Prevalence of hearing loss in older adults in Beaver Dam, Wisconsin: the Epidemiology of Hearing Loss Study. *Am J Epidemiol.* 1998;148:879-886.
11. Murphy C, Anderson JA, Markison S. Psychophysical assessment of chemosensory disorders in clinical populations. In: Kurihara K, Suzuki N, Ogawa H, eds. *Olfaction and Taste XI.* Tokyo, Japan: Springer-Verlag Tokyo; 1994:609-613.
12. Morgan CD, Nordin S, Murphy C. Odor identification as an early marker for Alzheimer's disease: impact of lexical functioning and detection sensitivity. *J Clin Exp Neuropsychol.* 1995;17:793-803.
13. Klein R, Klein BE, Lindton KL, De Mets DL. The Beaver Dam Eye Study: visual acuity. *Ophthalmology*. 1991;98:1310-1315.
14. US Census Bureau. Census 2000 summary file 1: tables QT-P1 and P12A. Available at: http://www.census.gov/main/www/cen2000.html. Accessed March 30, 2002.
15. Ekman G, Berglund B, Berglund U, Lindvall T. Perceived intensity of odor as a function of time of adaptation. *Scand J Psychol.* 1967;8:177-186.
16. Davidson TM, Murphy C, Jalowayski AA. Smell impairment: can it be reversed? *Postgrad Med.* 1995;98:107-109, 112-118.
17. SAS Institute Inc. *SAS/STAT User's Guide, Version 8*. Cary, NC: SAS Institute Inc; 1999.
18. Kendall M, Stuart A. *The Advanced Theory of Statistics.* Vol 2. New York, NY: MacMillan; 1979:580-585.
19. Mantel N, Haenszel W. Statistical aspects of the analysis of data from retrospective studies of disease. *J Natl Cancer Inst.* 1959;22:719-748.
20. Zhang J, Yu KF. What's the relative risk? a method of correcting the odds ratio in cohort studies of common outcomes. *JAMA*. 1998;280:1690-1691.
21. Murphy C, Doty RL, Duncan HJ. Clinical disorders of olfaction. In: Doty RL, ed. *Handbook of Olfaction and Gustation*. 2nd ed. New York, NY: Wiley; 2002:461-478.
22. Murphy C. Taste and smell in the elderly. In: Meiselman HL, Rivlin RS, eds. *Clinical Measurement of Taste and Smell*. New York, NY: Macmillan; 1986:343-371.
23. Schiffman SS. Age-related changes in taste and smell and their possible causes. In: Meiselman HL, Rivlin RS, eds. *Clinical Measurement of Taste and Smell*. New York, NY: Macmillan; 1986:326-342.
24. Doty RL, Shaman P, Applebaum SL, Giberson R, Siksorski L, Rosenberg L. Smell identification ability: changes with age. *Science*. 1984;226:1441-1443.
25. Ship JA, Weiffenbach JM. Age, gender, medical treatment, and medication effects on smell identification. *J Gerontol.* 1993;48:M26-M32.
26. Ship JA, Pearson JD, Cruise LJ, Brant LJ, Metter EJ. Longitudinal changes in smell identification. *J Gerontol A Biol Sci Med Sci.* 1996;51:M86-M91.
27. Morgan CD, Covington JW, Geisler MW, Polich J, Murphy C. Olfactory event-related potentials: older males demonstrate the greatest deficits. *Electroencephalogr Clin Neurophysiol.* 1997;104:351-358.
28. Murphy C, Morgan CD, Geisler MW, et al. Olfactory event-related potentials and aging: normative data. *Int J Psychophysiol.* 2000;36:133-145.
29. Murphy C, Nordin S, de Wijk RA, Cain WS, Polich J. Olfactory evoked potentials: assessment of young and elderly, and comparison to psychophysical threshold. *Chem Senses.* 1994;19:47-56.
30. Cain WS, Gent JF, Goodspeed RB, Leonard G. Evaluation of olfactory dysfunction in the Connecticut Chemosensory Clinical Research Center. *Laryngoscope*. 1988;98:83-88.
31. Davidson TM, Jalowayski AA, Murphy C, Jacobs RD. Evaluation and treatment of smell dysfunction. *West J Med*. 1987;146:434-438.
32. Deems DA, Doty RL, Settle RG, et al. Smell and taste disorders: a study of 750 patients from the University of Pennsylvania Smell and Taste Center. *Arch Otolaryngol Head Neck Surg.* 1991;117:519-528.
33. Doty RL, Bartoshuk LM, Snow JB Jr. Causes of olfactory and gustatory disorders. In: Getchell TV, Doty RL, Bartoshuk LM, Snow JB Jr, eds. *Smell and Taste in Health and Disease*. New York, NY: Raven Press; 1991:449-462.
34. Feldman JI, Wright HN, Leopold DA. The initial evaluation of dysosmia. *Am J Otolaryngol*. 1986;7:431-444.
35. Schiffman SS. Taste and smell in disease, I. *N Engl J Med.* 1983;308:1275-1279.
36. Schiffman SS. Taste and smell in disease, II. *N Engl J Med.* 1983;308:1337-1343.
37. Schiffman SS. Olfaction in aging and medical disorders. In: Serby MJ, Chobor KL, eds. *Science of Olfaction.* New York, NY: Springer-Verlag; 1992:500-525.
38. Schiffman SS. Taste and smell losses in normal aging and disease. *JAMA*. 1997;278:1357-1362.
39. Settle RG. The chemical senses in diabetes mellitus. In: Getchell TV, Doty RL, Bartoshuk LM, Snow JB Jr, eds. *Smell and Taste in Health and Disease*. New York, NY: Raven Press; 1991:829-843.
40. Eskenazi B, Cain WS, Novelly RA, Mattson R. Odor perception in temporal lobe epilepsy patients with and without temporal lobectomy. *Neuropsychologia*. 1986;24:553-562.
41. Stevens MH. Steroid-dependent anosmia. *Laryngoscope*. 2001;111:200-203.
42. Schiffman SS. Drugs influencing taste and smell perception. In: Getchell TV, Doty RL, Bartoshuk LM, Snow JB Jr, eds. *Smell and Taste in Health and Disease*. New York, NY: Raven Press; 1991:845-850.
43. Zervakis J, Graham BG, Schiffman SS. Taste effects of lingual application of cardiovascular medications. *Physiol Behav*. 2000;68:405-413.
44. Schiffman SS, Zervakis J, Suggs MS, Shaio E, Sattely-Miller EA. Effect of medications on taste: example of amitriptyline HCL. *Physiol Behav*. 1999;66:183-191.
45. Schiffman SS, Zervakis J, Heffron S, Heald AE. Effect of protease inhibitors on the sense of taste. *Nutrition*. 1999;15:767-772.
46. Nakashima T, Kimmelman CP, Snow JB. Structure of human fetal and adult olfactory neuroepithelium. *Arch Otolaryngol*. 1984;110:641-646.
47. Nakashima T, Kimmelman CP, Snow JB. Immunohistopathology of human olfactory epithelium, nerve, and bulb. *Laryngoscope*. 1985;95:391-396.
48. Smith CG. Incidence of atrophy of the olfactory nerves in man. *Arch Otolaryngol*. 1941;34:533-539.
49. Bhatnagar KP, Kennedy RC, Baron G, Greenberg RA. Number of mitral cells and the bulb volume in the aging human olfactory bulb: a quantitative morphological study. *Anat Rec*. 1987;218:73-87.
50. Jones N, Rog D. Olfaction: a review. *J Laryngol Otol.* 1998;112:11-24.
51. Meisami E, Mikhail L, Baim D, Bhatnagar KP. Human olfactory bulb: aging of glomeruli and mitral cells and a search for the accessory olfactory bulb. *Ann N Y Acad Sci.* 1998;855:708-715.
52. Jernigan TL, Archibald SL, Fennema-Notestine C, et al. Effects of age on tissues and regions of the cerebrum and cerebellum. *Neurobiol Aging*. 2001;22:581-594.
53. Yousem DM, Maldjian JA, Hummel T, et al. The effect of age on odor-stimulated functional MR imaging. *AJNR Am J Neuroradiol.* 1999;20:600-608.
54. Murphy C. Aging and chemosensory perception of and preference for nutritionally-significant stimuli. *Ann N Y Acad Sci.* 1989;561:251-266.
55. Murphy C. Age-associated changes in taste and odor sensation, perception, and preference. In: Munro H, Schlierf G, eds. *Nutrition in the Elderly.* New York, NY: Raven Press; 1992:79-87. Nestle Nutrition Workshop Series, Vol 29.
56. Drewnowski A, Evans W. Nutrition, physical activity, and quality of life in older adults: summary. *J Gerontol A Biol Sci Med Sci.* 2001;56:89-94.
57. Wakimoto P, Block B. Dietary intake, dietary patterns, and changes with age: an epidemiological perspective. *J Gerontol A Biol Sci Med Sci.* 2001;56:65-80.
58. Schiffman SS, Warwick AS. Effect of flavor enhancement of foods for the elderly on nutritional status: food intake, biochemical indices and anthropometric measures. *Physiol Behav.* 1993;53:395-402.
59. Hall JR. *Patterns of Fire Casualties in Home Fires by Age and Sex, 1990-1994*. Quincy, Mass: National Fire Protection Association; 1997:2, 14-15, 52-53.

 ©2002 American Medical Association. All rights reserved.

Downloaded From: http://jama.jamanetwork.com/ on 05/06/2015