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Research Article

The Rate of Age-Related Olfactory Decline Among the General Population of Older U.S. Adults

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Abstract

Background. Age-related olfactory loss (presbyosmia) is a prevalent sensory impairment with a large public health impact. In cross-sectional analyses, we found striking health disparities in olfactory function among older U.S. adults. Here, we report a 5-year follow-up to determine the magnitude of within-person olfactory decline.

Methods. The National Social Life, Health, and Aging Project (NSHAP) interviewed a probability sample of home-dwelling older U.S. adults (57–85 years) in 2005–2006 (Wave 1) and reinterviewed them in 2010–2011 (Wave 2), assessing demographics, social life, and health, including olfaction. Odor identification was measured with a 5-item version of the Sniffin' Sticks (0–5 correct). Fourteen hundred and thirty-six respondents provided olfaction data in both waves. Multivariate linear and logistic regression were used to model the association between change in olfactory performance and demographic, health, and psychosocial factors.

Results. Odor identification declined most rapidly among older individuals (0.25 additional errors per 5 years for each decade of age, $p < .001$) and in men (0.17 additional errors per 5 years compared to women, $p = .005$). Among those with perfect scores in Wave 1, African Americans declined more rapidly than Whites ($p = .04$). Neither socioeconomic status, health conditions, cognition, mental health, alcohol use nor smoking was associated with change in olfaction ($p > .05$, all).

Conclusions. The rate of olfactory decline increases with age and is greater among men than women despite adjusting for differences in psychosocial and health conditions, indicating physiologic factors as drivers. African Americans are more likely to experience initial olfactory decline, consistent with an earlier onset of aging among this subgroup.

Key Words: Epidemiology—Health disparities—Minority aging—Sensory—Neurological

Age-related olfactory impairment (presbyosmia) is an important public health problem, affecting the well-being, quality of life, and health of millions of older adults and predictive of 5-year mortality (1–8). We demonstrated that men had poorer olfactory function

compared to women and African Americans performed worse than Whites—cross-sectional differences that were constant across ages 57–85— in a nationally representative sample of older U.S. adults using cross-sectional data, leading to us to question whether men and African Americans have a more rapid age-related decline (9). Given these differences, an important question is whether the *rate of change* in olfactory function among older adults differs according to gender and/or race/ethnicity. There are few well-powered studies of olfactory function over time (10–12) and no data are available on disparities in rates of change in olfaction. We therefore addressed these questions using 5-year follow-up data from the National Social Life, Health, and Aging Project (NSHAP), the first study to collect detailed data on both social relationships and health (including biomeasures) from a sample of older adults representative of the home-dwelling U.S. population.

If differences in olfactory function between groups are due to accelerated aging or progressive damage, we would expect lower performing groups (eg, men and African Americans) to decline more rapidly over time. In contrast, if these differences have already been established prior to or during middle age, the rate of decline at older ages might be expected to be the same. Because both olfactory function and age-related health decline are associated with several health conditions, behaviors, and social factors, it is important to adjust for these factors when examining differences in the rates of olfactory change.

Methods

Respondents

In the first wave of NSHAP fielded in 2005–2006, interviewers from the National Opinion Research Center (NORC) conducted in-home interviews with 3,005 community-dwelling adults (1,454 men and 1,551 women) selected from the U.S. population of adults born between 1920 and 1947 (age: 57–85) (13,14). Five years later, data were collected again from all Wave 1 respondents who remained alive (2010–2011, Wave 2). Interviews included assessment of demographic, social, psychological, and biological measures, including olfaction as described below. Further details regarding the design, data collection, and baseline characteristics of NSHAP respondents are available elsewhere (13–15). The Institutional Review Boards of The University of Chicago and NORC approved the study; all respondents provided written, informed consent.

NSHAP had a 75.5% weighted response rate in Wave 1 and an 88% conditional response rate in Wave 2 (among those interviewed in Wave 1 who were still alive), both excellent for longitudinal studies using probability samples (14). Among the 3,005 Wave 1 respondents, 2,928 had complete data for the key variables considered here (olfactory identification score, age, gender, and race). By design, in Wave 2, 1,944 of these respondents were randomized to receive repeat olfactory testing. Of these, 458 were deceased, too ill to participate, could not be located or refused to be interviewed. Only 49 respondents (2.5%) refused olfactory testing, while one respondent discontinued the interview prior to the olfaction module. This yielded 1,436 respondents with olfaction data from both waves, and all analyses presented here are based on this subsample (73.9%; 694 males and 742 females; ages 57–85 at Wave 1). Respondents who died or dropped out (n = 458) were significantly different from the analytic sample in that they were less educated, older, and in poorer physical health in Wave 1 (16). The small number of interviewed respondents who refused olfactory testing had similar olfactory ability, based on Wave 1 testing, compared with those in the analytic sample (mean number of correctly identified odors: 3.9 vs 4.2). The flow diagram of the study is presented in Supplementary Figure 1.

Olfactory Identification

All respondents in Wave 1 and a randomly selected two-thirds of the sample in Wave 2 were asked to complete a 5-item, validated odor identification test presented using felt-tipped pens (17,18) (Burghart Messtechnik, Wedel, Germany). Briefly, five odorants were presented one at a time. Respondents were asked to identify each by choosing from a set of four picture/word prompts in a forced choice protocol (19); refusals were coded as incorrect (for test details, see (9,20)). The target odors were rose, leather, orange, fish, and peppermint. The number of correctly identified odors out of five was used as a score at each wave; for context, severity of olfactory dysfunction is categorized as anosmic = 0–1, hyposmic = 2–3, and normosmic = 4–5. Analyses presented here are based on the change in odor identification score, as well as the likelihood of misidentifying individual odors in Wave 2 among those who identified them correctly in Wave 1. Distributions of the score at each wave and of the change in score are shown in Table 1.

Demographic Measures

Race (an established olfactory risk factor (9)) and Hispanic ethnicity were measured via self-report according to standard NIH questions, and respondents were then classified as White, African American, or Hispanic (those who reported their race as "Black/African

Table 1. Distributions of Key Variables (N = 1,436)

Variable	Weighted (%)	N
Odor identification (# correct), Wave 1		
0	0.5	6
1	0.9	24
2	3.2	68
3	12.9	211
4	28.7	425
5	53.8	702
Odor identification (# correct), Wave 2		
0	2.6	41
1	2.5	43
2	6.3	106
3	10.3	171
4	31.5	452
5	46.9	623
Change in odor id (Wave 2–Wave 1)		
≤−3	3.9	58
−2	6.9	110
−1	20.4	300
0	49.0	656
1	16.2	241
2	3.1	57
≥3	0.5	14
Age groups (y, at Wave 1)		
57–64	44.4	535
65–74	35.7	538
75–85	19.9	363
Gender (% men)	47.5	694
Race/ethnicity		
White	81.1	1,017
African American (AA)	10.2	242
Hispanic (non-AA)	6.7	147
Other	2.0	30

American" and answered "Yes" to Hispanic ethnicity were classified as African American). Those reporting their race as "American Indian or Alaskan Native," "Asian," or "Other" were combined into a single other category. Socioeconomic status was measured by highest educational degree or certification earned and net household assets (including houses, cars, or rental properties/businesses owned, plus financial assets including savings accounts, stocks, and pensions minus outstanding debt). Distributions of age, gender, and race/ethnicity for the analytic sample are shown in Table 1.

Factors Associated With Olfactory Dysfunction

We included in our analysis several risk factors for olfactory dysfunction, all of which were measured at Wave 1. Comorbid diseases were measured with the Charlson Index modified for NSHAP (21). Self-rated physical health was measured by a standard 5-point scale (excellent, very good, good, fair, or poor). Current smoking, based on either salivary cotinine level or self-report, and problem drinking were also measured (9,22). These were included to adjust for illness and physical function, which might affect chemosensation secondarily; smoking causes nasal inflammation and thereby may affect olfaction, while alcohol use can cause liver dysfunction which can be associated with smell problems.

Frequency of depressive symptoms, anxiety symptoms, and perceived stressors was measured with standard scales modified for survey use: the 11-item Center for Epidemiologic Studies Depression (CES-D) scale, the Hospital Anxiety Scale (HADS), and the Perceived Stress Scale (PSS) (23). For all three measures, respondents were asked to select the most accurate category of symptom frequency: (a) rarely or none of the time, (b) some of the time, (c) occasionally, or (d) most of the time; the average score among all items in a scale was used in the analysis to adjust for mental health which may affect neurosensory function. Cognitive function (memory and mental arithmetic), which is closely associated with olfaction and involved in the task of matching an odor with a name/picture, was measured with a modified version of the Short Portable Mental Status Questionnaire (SPMSQ) (24).

Statistical Analysis

To estimate the longitudinal effect of age on olfactory function, we used an approach based on that of the standard period life table, in which we used the age-specific 5-year rates of acquiring a deficit to project the experience of a hypothetical cohort, assuming that the current age-specific rates of decline remain constant (25). Let Y_a represent the response to a specific odor identification item for a respondent a years old, with $Y_a = 1$ indicating a correct response and $Y_a = 0$ an incorrect response. Starting with $P(Y_{57} = 0)$ estimated from Wave 1, we calculated,

$$P(Y_a = 0) = P(Y_a = 0 \mid Y_{a-5} = 1) \times P(Y_{a-5} = 1) + P(Y_a = 0 \mid Y_{a-5} = 0) \times P(Y_{a-5}) = 0,$$

for $a = 62, 67, 72, 77, 82$, where $P(Y_a = 0 \mid Y_{a-5} = 1)$ is the probability of acquiring a deficit during the 5-year period from age $a - 5$ to age a, and $1 - P(Y_a = 0 \mid Y_{a-5} = 0)$ is the probability of "recovering" from a deficit. All probabilities were estimated using logistic regression with a linear term for age (a quadratic term was tested but was not statistically significant), stratified by gender. The resulting age-specific probabilities of failing to identify each odor correctly were then plotted.

A series of linear regression models were fit to the change in the number of correctly identified odors (Wave 2–Wave 1, so that negative values signify decline). Covariates included age, gender, race, cognition, education/household assets, self-rated physical health, comorbidity index, depressive and anxiety symptoms, perceived stress, smoking, and problem drinking status (all measured at Wave 1). Sensitivity analyses were performed by refitting the models excluding both respondents who had experienced head injury or nasal surgery (which were rare) and those reporting a cold in Wave 2, neither of which affected the results.

As we have shown previously, baseline (ie, Wave 1) olfaction differs according to age, gender, and race/ethnicity, and this may therefore confound subgroup differences in the rate of change if that change is correlated with the baseline value. However, simply including the baseline value as a fixed covariate is not appropriate due to the presence of measurement error and the resulting regression towards the mean; while it is possible to account for this, doing so requires an estimate of the measurement error which is not available with only two waves of data. Moreover, in cases where previous differences in the rate of change may already be manifest at baseline, adjusting for differences in the baseline value risks masking true differences in the rate of change (26). In addition to these issues, the relatively limited range of the olfactory identification score may itself create problems, as for example, those with a perfect score in Wave 1 cannot improve further, while those with only 0–1 correct responses in Wave 1 have no additional room to decline. In order to address these issues and facilitate interpretation, we thus augmented our analysis by fitting a series of logistic regression models to the likelihood of scoring worse in Wave 2 than in Wave 1, conditional on the Wave 1 score. Models are presented for those scoring 3, 4, and 5 in Wave 1, which together account for 95% of the sample. These models included age, gender, and race/ethnicity as covariates.

Probability weights accounting for differential probabilities of selection and nonresponse were used in all analyses. Design-based *SE*s were calculated using the linearization method (27) together with the strata and Primary Sampling Unit (PSU) indicators provided with the dataset. Multiple imputation of missing data was performed as described previously (9,28,29). Statistical analyses were conducted with Stata (Software release 13. StataCorp LP, College Station, TX).

Results

During the 5 years from Waves 1 to 2, 31% of respondents showed a decline in odor identification score while only 20% showed an increase—an overall decline in olfactory function that was evident in each age group (Figure 1A). Respondents aged 75–85 showed the greatest change (half a point lower on average) while those aged 57–64 showed the least (0.1 points lower on average) (Figure 1B). These age-related functional declines were observed equally among all five odors. Figure 2 plots the probability of acquiring a deficit for each odor (ie, misidentifying the odor in Wave 2 after correctly identifying it in Wave 1) by age; all curves are greater than zero and increase with age by similar rates. By age 85, the probability of acquiring a deficit over the next 5 years (among those able to identify the odor at Wave 1) ranges from 0.29 to 0.45 across the five odors.

The longitudinal effect of age from 57 to 82 on the likelihood of being unable to identify orange (chosen here as being representative of the other odors) is shown in Figure 1D, separately for men and women, together with a plot of the cross-sectional association with age for comparison. Not only is the rate of increase higher with

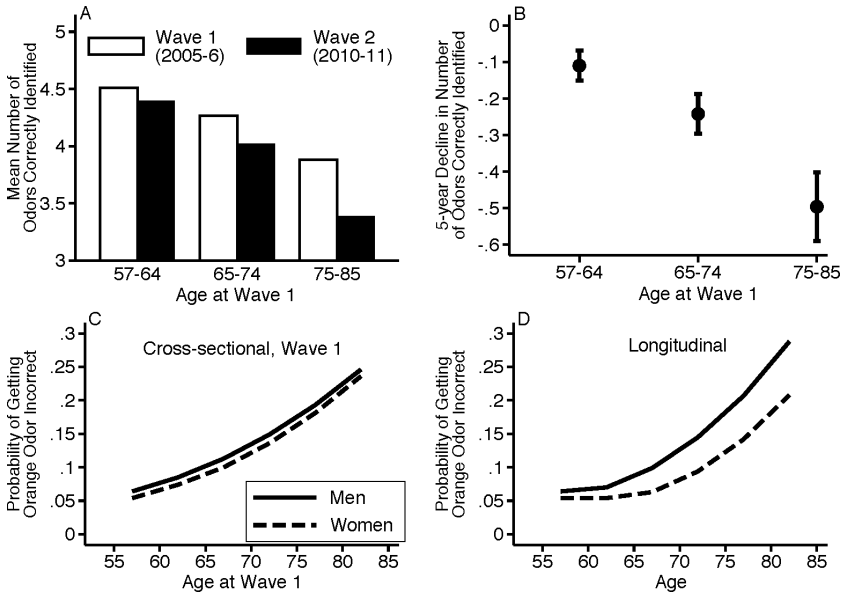

Figure 1. (**A**) Mean number of correctly identified odors at each wave by age group. (**B**) Older respondents had greater olfactory decline than younger respondents. The mean change (*SE*) between waves for those 57–64 years of age was –0.11 (0.04), $p = .009$; for those 65–74 was –0.24 (0.05), $p < .001$; and for those 75–85 was –0.50 (0.09), $p < .001$. Mean values are plotted with error bars representing ± 1 *SE*. (**C**) Wave 1 cross-sectional analysis examining the probability of getting orange incorrect by age and gender. Orange was used as an exemplary odor due to its moderate difficulty. (**D**) Effect of age on the probability of getting orange incorrect, estimated by starting with the probability at 57 (from panel C) and applying the age-specific 5-year rates of change (analyses performed separately by gender).

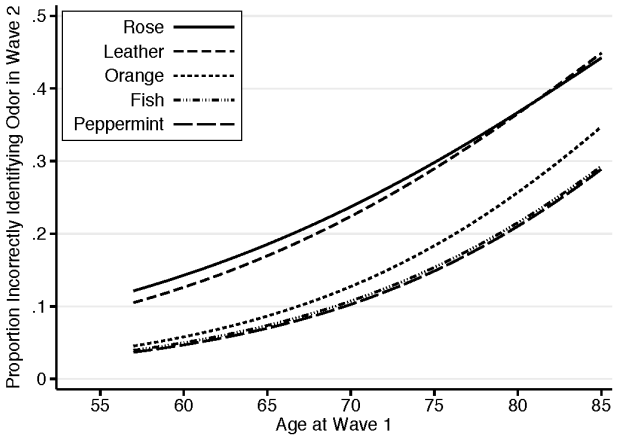

Figure 2. For those who had correctly identified an odor in Wave 1, probability of getting individual odors incorrect in Wave 2 by age at Wave 1.

increasing age, but is also higher for men than for women, with the probability of misidentifying the odor increasing from 0.06 to 0.29 for men and from 0.05 to 0.21 for women. Similar patterns were observed for the other four odors, though with differing overall levels depending on their relative difficulty (Supplementary Figure 2). Thus, the longitudinal analysis reveals a more marked effect of increasing age and a gender difference in the rate of change that are not evident in the cross-sectional association.

Table 2 shows the results of regressing the 5-year change in olfactory identification score on age, gender, and race/ethnicity (Model 1). Consistent with Figure 1B and D, the average decline was larger for older respondents (an additional decline of –0.24 points per decade, 95% CI: –0.34, –0.14, $p < .001$) and for men (an additional decline of –0.15 points relative to women, 95% CI: –0.25, –0.04, $p = .007$). In contrast, despite the cross-sectional disparity found among African Americans in Wave 1, there was no evidence of a larger 5-year decline among African Americans (an estimated *increase* relative to Whites of 0.01 points, 95% CI: –0.16, 0.17, $p = .92$) or Hispanics relative to Whites.

To determine if these differences (or lack thereof) were confounded by differences in socioeconomic factors, health conditions, or health behaviors, we reestimated the model adding education, household assets, cognition, self-rated physical health, comorbidity, depressive and anxiety symptoms, perceived stress, and smoking and alcohol use (Model 2). Adjusting for these covariates had almost no effect on the estimated coefficients for age, gender, and race/ethnicity. Perhaps surprisingly, none of these socioeconomic or health factors—several of which exhibit a cross-sectional association with olfactory function—was significantly associated with changes in olfaction (all $p > .05$).

Finally, to determine whether differences in baseline olfactory function may be spuriously causing or masking differences according to age, gender, and/or race/ethnicity, we estimated a series of logistic regression models of the probability of scoring lower in Wave 2 than in Wave 1, conditional on the Wave 1 score (Table 3). Results were similar to the previous analysis for age and gender, with the odds of scoring worse in Wave 2 higher for both men and older respondents, regardless of whether they scored a five (no errors), four, or three in Wave 1. For example, among those with no errors in Wave 1, the odds ratio for a decade increase in age was 1.82 (95% CI: 1.40, 2.38, $p < .001$) and for men relative to women was 1.80 (95% CI: 1.27, 2.55, $p = .001$); corresponding odds ratios for those with one and two errors in Wave 1 were slightly higher and also statistically significant. In contrast, results for race/ethnicity differed from the previous analysis, with African Americans who had no errors in Wave 1 having greater odds of a decline in Wave 2 than Whites (odds ratio 1.80, 95% CI: 1.03, 3.16, $p = .04$); the same odds ratio was observed among those with one error in Wave 1, although it was not quite statistically significant. Among those who had two errors in Wave

Table 2. Results From Linear Regression Models Fit to the Change in Number of Correctly Identified Odors (Wave 2–Wave 1)

	Model 1			Model 2*		
	Coefficient	95% CI	*p* Value	Coefficient	95% CI	*p* Value
Age (in decades)	−0.24	−0.34, −0.14	<.001	−0.25	−0.36, −0.14	<.001
Men (vs women)	−0.15	−0.25, −0.04	.007	−0.17	−0.29, −0.05	.005
Race (vs White)						
African American	0.01	−0.16, 0.17	.92	0.01	−0.16, 0.18	.93
Hispanic	0.03	−0.23, 0.29	.82	0.01	−0.29, 0.30	.97
Other	0.30	−0.10, 0.70	.14	0.31	−0.11, 0.73	.14

Notes: CI = confidence interval.

*Model also includes education, household assets, cognition, self-rated physical health, comorbidity index, depressive and anxiety symptoms, perceived stress, smoking, and problem drinking.

Table 3. Results From Logistic Regression Models Fit to the Probability of Scoring Lower in Wave 2 Than in Wave 1, Separately by Wave 1 Score

	No Errors in Wave 1 (*n* = 702)			One Error in Wave 1 (*n* = 419)			Two Errors in Wave 1 (*n* = 211)		
Covariate	Odds Ratio	95% CI	*p* Value	Odds Ratio	95% CI	*p* Value	Odds Ratio	95% CI	*p* Value
Age (in decades)	1.82	1.40, 2.38	<.001	2.36	1.72, 3.24	<.001	2.13	1.28, 3.57	.005
Men (vs women)	1.80	1.27, 2.55	.001	2.45	1.56, 3.84	<.001	2.06	1.06, 4.02	.03
Race (vs White)									
African American	1.80	1.03, 3.16	.04	1.80	0.87, 3.76	.11	0.72	0.31, 1.63	.42
Hispanic	1.07	0.57, 2.01	.84	1.85	0.77, 4.41	.16	1.17	0.34, 3.97	.80
Other	0.92	0.20, 4.26	.91	*	*	*	0.48	0.02, 12.26	.65

Notes: CI = Confidence Interval.

*Perfect prediction among the six coded as "Other" who had one error in Wave 1 and these six were dropped from the analysis.

1, African Americans did not differ from Whites in the likelihood of experiencing a decline over 5 years, although the number of African Americans in this group was only 54.

Discussion

To our knowledge, there are three prior longitudinal studies of olfactory function (10–12). Ship and coworkers studied 161 healthy participants from the oral physiology component of the Baltimore Longitudinal Study of Aging, which utilized a 40-item odor identification test administered twice approximately 3 years apart. Schubert and coworkers studied 1,556 residents of Beaver Dam, Wisconsin participating in the Epidemiology of Hearing Loss Study (EHLS), with data collected using an 8-item identification test administered twice 5 years apart. Finally, Hedner and coworkers studied 836 individuals from the Swedish city of Umeå participating in the Betula project, using a 13-item odor identification test administered twice over a 5-year period. Our work builds on these prior studies by using data collected from a probability sample of the U.S. population of older, community-dwelling adults, including oversamples of African Americans and Hispanics. Thus, our results permit inferences about the entire U.S. population of older adults, as well as provide important information about differences between racial/ethnic subgroups.

Consistent with all the three prior studies, we found that the rate of olfactory decline increases markedly with increasing age, such that by age 85, the probability of losing the ability to identify a specific odor over the next 5 years is between 0.29 and 0.45 (based on the five odors studied). This fact is less evident in the cross-sectional association between olfactory identification score and age, likely due in part to a disproportionate number of healthier individuals surviving to older ages. In fact, even our longitudinal results may underestimate the full effect of age, due to possible nonresponse bias incurred by the 18% of respondents who had died or were too ill to interview in Wave 2 (if these individuals experienced greater olfactory decline than the general population). Future work utilizing at least three waves of data is required to determine how the rate of change in olfactory function is related to the likelihood of death or infirmity.

We also found that the rate of olfactory decline is greater among men, as did two of the three prior studies. Physiological differences between men and women may explain these results. Hormonal differences may play a neuroprotective role earlier in life, which may then remain evident even after menopause. Estrogen and progesterone may have beneficial effects on olfactory stem cells in the periphery or the central nervous system, which might retard subsequent losses after their levels decline (30,31). Additionally, nerve function may decline more rapidly in men; for example, cognitive function declines faster in men than women (32) (we adjusted for gross differences in cognitive function in our analysis).

Results concerning racial/ethnic differences were less clear. Although racial/ethnic differences in the mean change were not evident when averaged across the entire sample, there was some evidence that African Americans with a perfect (or near-perfect) score in Wave 1 were more likely to suffer a subsequent decline than their White counterparts. This is consistent with the possibility that the *onset* of aging and its consequences is accelerated among African Americans. However, additional waves of data are required to distinguish this possibility from other potential explanations.

Age and gender differences in the rate of decline were unaffected by adjusting for potential socioeconomic and health confounders, suggesting that presbyosmia is an independent phenomenon not

driven by other health or social factors. This is consistent with several potential mechanisms for presbyosmia discussed in the literature. For example, this may be a natural phenomenon of age-related decline related to decreased stem cell turnover (33), especially since the olfactory bulb is one of the key areas of adult neurogenesis (34). Alternatively, degeneration of the peripheral olfactory system could be affected by changes in mucosal immunity or structural changes in the nose that are known to occur (35).

Interestingly, although not the focus of our study, comorbidity, physical and mental health, tobacco use, alcohol, and cognitive function did not affect the rate of change in olfactory function over 5 years. This may reflect the hardiness of the olfactory system and its ancient evolutionary importance in human physiology. Indeed, our findings provide support for prior work in model systems suggesting that innate features of olfactory physiology such as decline of stem cell turnover in the olfactory epithelium, degeneration of the connections of it to the central olfactory regions, neurosenecence of those central regions themselves, or environmental influences that affect these functions may underlie the decline of the sense of smell with time. Recent experiments in mice suggest plasticity in these processes and implicate specific molecules in this resilience. Although, there are not yet definitive data in humans (36,37), if true, it would offer the possibility that we might develop interventions to mitigate or reverse these changes to improve olfactory performance (38).

Although this study extends prior work by providing nationwide estimates of the rate of age-related olfactory decline and its correlates, more work remains to be done. For example, our study relied on odor identification as the sole measure of olfactory function, however it will be important to determine if the effect of age on other measures, such as olfactory threshold, is similar. In addition, as noted above, further work with additional waves of data is necessary to determine how the rate of change in olfactory function is related to the likelihood of death or infirmity and to model the dynamics of age-related change more thoroughly. Finally, future work should investigate the antecedents of presbyosmia, such as pollution exposure, early life experiences, and detailed information on nasal function. Identifying such factors will help to determine who is at greatest risk of olfactory decline during older age. Future work involving longe follow-up should also be done to determine the consequences—both health related and social—of this condition.

In summary, among older adults, olfactory function declines faster with increasing age and among men. Understanding what specific factors underlie these findings will provide fundamental insight into mechanisms of chemosensory aging, with broad implications for other senses.

Supplementary Material

Supplementary material can be found at: http://biomedgerontology.oxfordjournals.org/

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Conflict of Interest

The authors declare no conflicts of interest.

References

1. Mattes RD, Cowart BJ, Schiavo MA, et al. Dietary evaluation of patients with smell and/or taste disorders. *Am J Clin Nutr*. 1990;51:233–240. PMID:2305710
2. Miwa T, Furukawa M, Tsukatani T, Costanzo RM, DiNardo LJ, Reiter ER. Impact of olfactory impairment on quality of life and disability. *Arch Otolaryngol Head Neck Surg*. 2001;127:497–503. doi:10.1001/archotol.127.5.497
3. Murphy C, Doty R, Duncan H. Clinical disorders of olfaction. In: Doty R, ed. *Handbook of Olfaction and Gustation*. New York: Marcel Dekker; 2003. doi:10.1201/97802033911457.ch22
4. Neuland C, Bitter T, Marschner H, Gudziol H, Guntinas-Lichius O. Health-related and specific olfaction-related quality of life in patients with chronic functional anosmia or severe hyposmia. *Laryngoscope*. 2011;121:867–872. doi:10.1002/lary.21387
5. Nguyen-Khoa BA, Goehring EL Jr, Vendiola RM, Pezzullo JC, Jones JK. Epidemiologic study of smell disturbance in 2 medical insurance claims populations. *Arch Otolaryngol Head Neck Surg*. 2007;133:748–757. doi:10.1001/archotol.133.8.748
6. Pinto JM. Olfaction. *Proc Am Thorac Soc*. 2011;8:46–52. doi:10.1513/pats.201005-035RN
7. Rawson NE. Olfactory loss in aging. *Sci Aging Knowledge Environ*. 2006;2006:pe6. doi:10.1126/sageke.2006.5.pe6
8. Santos DV, Reiter ER, DiNardo LJ, Costanzo RM. Hazardous events associated with impaired olfactory function. *Arch Otolaryngol Head Neck Surg*. 2004;130:317–319. doi:10.1001/archotol.130.3.317
9. Pinto JM, Schumm LP, Wroblewski KE, Kern DW, McClintock MK. Racial disparities in olfactory loss among older adults in the United States. *J Gerontol A Biol Sci Med Sci*. 2014;69:323–329. doi:10.1093/gerona/glt063
10. Hedner M, Nilsson LG, Olofsson JK, et al. Age-related olfactory decline is associated with the BDNF Val66met polymorphism: evidence from a population-based study. *Front Aging Neurosci*. 2010;2:24. doi:10.3389/fnagi.2010.00024
11. Schubert CR, Cruickshanks KJ, Klein BE, Klein R, Nondahl DM. Olfactory impairment in older adults: five-year incidence and risk factors. *Laryngoscope*. 2011;121:873–878. doi:10.1002/lary.21416
12. 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. doi:10.1093/gerona/51A.2.M86
13. Suzman R. The National Social Life, Health, and Aging Project: an introduction. *J Gerontol B Psychol Sci Soc Sci*. 2009;64(suppl 1):i5–11. doi:10.1093/geronb/gbp078
14. O'Muircheartaigh C, Eckman S, Smith S. Statistical design and estimation for the National Social Life, Health, and Aging Project. *J Gerontol B Psychol Sci Soc Sci*. 2009;64(suppl 1):i12–i19. doi:10.1093/geronb/gbp045
15. Lindau ST, Schumm LP, Laumann EO, Levinson W, O'Muircheartaigh CA, Waite LJ. A study of sexuality and health among older adults in the United States. *N Engl J Med*. 2007;357:762–774. doi:10.1056/NEJMoa067423
16. Pinto JM, Wroblewski KE, Kern DW, Schumm LP, McClintock MK. Olfactory dysfunction predicts 5-year mortality in older adults. *PLoS One*. 2014;9:e107541. doi:10.1371/journal.pone.0107541

17. Mueller C, Renner B. A new procedure for the short screening of olfactory function using five items from the "Sniffin' Sticks" identification test kit. *Am J Rhinol*. 2006;20:113–116. PMID:16539306
18. Denzer MY, Gailer S, Kern DW, et al. Quantitative validation of the *n*-Butanol Sniffin' Sticks Threshold Pens. *Chemosens Percept*. 2014;7:91–101. doi:10.1007/s12078-014-9168-1
19. Schumm LP, McClintock M, Williams S, et al. Assessment of sensory function in the National Social Life, Health, and Aging Project. *J Gerontol B Psychol Sci Soc Sci*. 2009;64(suppl 1):i76–i85. doi:10.1093/geronb/gbp048
20. Kern DW, Wroblewski KE, Schumm LP, Pinto JM, McClintock MK. Field survey measures of olfaction: the Olfactory Function Field Exam (OFFE). *Field Methods*. 2014;26:421–434. doi:10.1177/1525822X14547499
21. Katz JN CL, Sangha O, Fossel AH, Bates DW. Can comorbidity be measured by questionnaire rather than medical record review? *Med Care*. 1996;34:73–84. doi:10.1097/00005650-199601000-00006
22. Ewing JA. Detecting alcoholism. The CAGE questionnaire. *JAMA*. 1984;252:1905–1907. doi:10.1001/jama.1984.03350140051025
23. Shiovitz-Ezra S, Leitsch S, Graber J, Karraker A. Quality of life and psychological health indicators in the National Social Life, Health, and Aging Project. *J Gerontol B Psychol Sci Soc Sci*. 2009;64(suppl 1):i30–i37. doi:10.1093/geronb/gbn020
24. Pfeiffer E. A short portable mental status questionnaire for the assessment of organic brain deficit in elderly patients. *J Am Geriatr Soc*. 1975;23:433–441. doi:10.1111/j.1532-5415.1975.tb00927
25. Newman SC. *Biostatistical Methods in Epidemiology*. New York: John Wiley & Sons, Inc.; 2001. doi:10.1002/0471272612
26. Vollmer WM. Comparing change in longitudinal studies: adjusting for initial value. *J Clin Epidemiol*. 1988;41:651–657. doi:10.1016/0895-4356(88)90117-5
27. Binder DA. On the variances of asymptotically normal estimators from complex surveys. *Int Stat Rev*. 1983;51:279–292. doi:10.2307/1402588
28. White IR, Royston P, Wood AM. Multiple imputation using chained equations: Issues and guidance for practice. *Stat Med*. 2011;30:377–399. doi:10.1002/sim.4067
29. Sterne JA, White IR, Carlin JB, et al. Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. *BMJ*. 2009;338:b2393. doi:10.1136/bmj.b2393
30. Pawluski JL, Brummelte S, Barha CK, Crozier TM, Galea LA. Effects of steroid hormones on neurogenesis in the hippocampus of the adult female rodent during the estrous cycle, pregnancy, lactation and aging. *Front Neuroendocrinol*. 2009;30:343–357. doi:10.1016/j.yfrne.2009.03.007
31. Lennington JB, Yang Z, Conover JC. Neural stem cells and the regulation of adult neurogenesis. *Reprod Biol Endocrinol*. 2003;1:99. doi:10.1186/1477-7827-1-99
32. Lipnicki DM, Sachdev PS, Crawford J, et al. Risk factors for late-life cognitive decline and variation with age and sex in the Sydney Memory and Ageing Study. *PLoS One*. 2013;8:e65841. doi:10.1371/journal.pone.0065841
33. Rawson NE, LaMantia AS. A speculative essay on retinoic acid regulation of neural stem cells in the developing and aging olfactory system. *Exp Gerontol*. 2007;42:46–53. doi:10.1016/j.exger.2006.05.021
34. Gould E. How widespread is adult neurogenesis in mammals? *Nat Rev Neurosci*. 2007;8:481–488. doi:10.1038/nrn2147
35. Pinto JM, Jeswani S. Rhinitis in the geriatric population. *Allergy Asthma Clin Immunol*. 2010;6:10. doi:10.1186/1710-1492-6-10
36. Bergmann O, Liebl J, Bernard S, et al. The age of olfactory bulb neurons in humans. *Neuron*. 2012;74:634–639. doi:10.1016/j.neuron.2012.03.030
37. Macklis JD. Human adult olfactory bulb neurogenesis? Novelty is the best policy. *Neuron*. 2012;74:595–596. doi:10.1016/j.neuron.2012.05.005
38. Katsimpardi L, Litterman NK, Schein PA, et al. Vascular and neurogenic rejuvenation of the aging mouse brain by young systemic factors. *Science*. 2014;344:630–634. doi: 10.1126/science.1251141