

Olfactory Function in Australian Aboriginal Children and Chronic Otitis Media

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Abstract

Chronic suppurative otitis media (CSOM), a severe form of middle ear infection, affects most Australian Aboriginal children with up to 50% in some communities suffering hearing loss as a consequence. To date, there is no information on whether repeated exposure to the pathogens that characterize CSOM and that are present in the upper respiratory airway affect olfactory function. Accordingly, this study aimed to determine whether 1) there was a high prevalence of olfactory loss in Aboriginal children and 2) hearing loss is a predictor of olfactory loss. Two hundred and sixty one 9- to 12-year-old Aboriginal children from 16 rural communities reported to have high prevalences of CSOM and hearing loss were assessed for olfactory loss using a 16-odor identification test and hearing loss. One child was found to be anosmic, 4 were slightly hyposmic, and 42 had hearing loss. No relationship was found between olfactory loss and hearing loss. The test-retest reliability of the 16-odor identification test was 0.98. It was concluded that CSOM does not appear to affect olfactory function in the long term and that hearing loss in Aboriginal children is not a predictor of olfactory loss.

Key words: Aboriginal children, hearing loss, olfaction, otitis media

Introduction

Aboriginal children in Australia have high levels of chronic suppurative otitis media (CSOM) with reports of 95% of infants being affected within a few months of birth compared with 30% among non-Aboriginal infants (Boswell et al. 1995). In some communities, the incidence of children having CSOM with perforated eardrums is as high as 70% (Gibson et al. 1996). Earlier surveys of more than 4500 Aboriginals between 1964 and 1980 detected CSOM in 10–54%, perforated eardrums in 9–36%, and deafness in 10–41%. The pathogens present during CSOM in addition to causing lesions in the cochlear and auditory nerve (Kenna 1994) have also been reported to result in taste loss in adults as a consequence of damage to the chorda tympani that projects to the brain stem via the middle ear (Landis et al. 2005). Lesions of the auditory and gustatory nerves (Gedikli et al. 2001) and the fact that the infection is commonly accepted as having originated in the nasopharynx, extending to the tympanic cavity via the Eustachian tube (Gibson et al. 1996), suggest that one or more of the pathogens causing CSOM could also affect the olfactory epithelium and receptor cells. Although it

is well documented from adult studies that chronic nasal infections including sinusitis and rhinitis can impair smell permanently or temporarily (Doty and Mishra 2001), there does not appear to be a report on the effects of CSOM on olfactory function. Accordingly, it remains to be determined whether CSOM affects olfaction in Aboriginal children. Should olfactory loss occur, it is possible that the affected children may also have hearing loss. Loss of hearing, therefore, may identify the children most likely to have olfactory loss. A characteristic of the olfactory system that may minimize the effects of CSOM is that olfactory receptor neurons can undergo neurogenesis and regenerate (Mackay-Sim 2003). Importantly, neurogenesis occurs at a higher rate in younger individuals (Loo et al. 1996), suggesting that the incidence of recovery from olfactory loss may be higher the younger the patient. Thus, the potential for the olfactory system to regenerate may result in recovery of olfactory function in part or fully in those suffering loss from CSOM. Children may have permanent loss of hearing but not necessarily permanent loss of the ability to smell.

Currently, there is no information available on olfactory function in Aboriginal children or adults. Given that up to 95% experience CSOM during the first 6 years of life and this disease has the potential to cause olfactory disorders, it is of considerable concern that potentially many Aboriginal children and adults could be affected. Because olfactory disorders can negatively affect diet, nutrition, and development and the quality of life in general, it was important to determine if CSOM had negative olfactory consequences for children. Unfortunately, because of the paucity of clinics for the diagnosis and treatment of CSOM in Aboriginal children and the lack of medical records for them in rural Australia, it is not possible to readily identify which children have had CSOM recently and whether any effects of CSOM in the short term on olfaction can be detected. In the present study, another reason for not investigating the immediate effects of CSOM on olfactory function was that even if children had been located, the presence of increased mucous in the nose and reduced volume of the anterior nasal cavity would most likely have interfered with odor perception. Thus, it would have been impossible to determine whether an olfactory deficit resulted from reduced access of odorants to the olfactory receptors (Hummel et al. 1988) or to CSOM pathogens affecting the responses of olfactory receptor cells. Accordingly, because our overall goal was to determine whether the effects of CSOM on olfaction were long lasting, perhaps permanent, we investigated olfactory function in older children, namely, 9- to 12-year olds. This age group was selected for 2 reasons. First, because CSOM is an early childhood disease, most of the children in this age group would not have experienced the disease for 3–6 years. Second, this time period should have been sufficient for regeneration of affected olfactory receptors and function to recover. Duncan and Seiden (1995), for example, reported evidence of recovery following upper respiratory infection as still in progress in some patients after 3 years. Therefore, the main hypothesis of the study was that a high prevalence of olfactory dysfunction in 9- to 12-year olds would indicate that the most likely cause was CSOM and that any loss detected in this age group is likely to be permanent. Older Aboriginal children and adults were not included because there is a rapid escalation of the use of drugs and alcohol and sniffing petrol during adolescence and a high incidence of renal and respiratory diseases in adults, all of which could confound the results. Finally, because CSOM causes hearing loss in up to 50% of Aboriginal children, a second hypothesis was that there would be a relationship between olfactory loss and hearing loss with the latter being a predictor of olfactory loss. Accordingly, children were assessed for both olfactory and hearing loss in the study.

Materials and methods

A total of 261 Aboriginal children (9–12 years; mean age and standard deviation [SD] 10.05 ± 2 years; 133 females

10.01 ± 2 years and 128 males 10.09 ± 2.4 years) from 16 communities in North, North West, and Southern areas of the state of New South Wales participated. Approval for the study was obtained from the Aboriginal Health Medical Research Council, the Human Research Ethics Committee of the University of New South Wales (Ref. No. 04256) and Katungal and Awabakal Aboriginal Medical Corporations. Olfactory function was assessed using a 3-choice odor identification test that requires children to identify 16 common food and nonfood odors of moderate strength (Table 1) delivered from opaque squeeze bottles (Laing et al. forthcoming). A child sniffed one odorant at a time and chose from 3 photographs which one best described the odor. A different set of photographs was used with each odorant, and the location of the photo of the target odorant was randomized across the sets. Testing was conducted on a 1:1 basis with an adult tester. There was an interval of about 20 s between the presentations of odorants. A variety of rooms were used for testing at schools, and these were generally large, quiet, and ventilated.

Because the odor identification test had not been used to assess olfactory function in 9- to 12-year-old Aboriginal children, a measure of the reliability of the test was conducted using a test–retest procedure. The latter was achieved by obtaining the responses of a separate group of thirty-two 9- to 12-year-old Aboriginal children (19 females 9.8 ± 2 years and

Table 1 Test odorants and their identification

Odorants	Identification (%)	SD
Floral	86	0.35
Orange	95	0.23
Strawberry	97	0.16
Fish	90	0.31
Chocolate	97	0.16
Baby powder	99	0.09
Paint	99	0.11
Grass	89	0.31
Sour	89	0.31
Minty	100	0.00
Onion	97	0.17
Vicks VapoRub	100	0.00
Spicy	95	0.22
Dettol	98	0.14
Cheese	72	0.45
Petrol	99	0.09
Mean	94.6	0.22

13 males 10.3 ± 2 years) at 2 sessions held on consecutive days using the same test procedure as described above.

Most of the children (220) were assessed for otitis media (otoscopy and tympanometry) and hearing loss (audiometry), that is, unable to detect a sound of 1000, 2000, or 4000 Hz at 20 dB, either on the same day as the olfactory tests were conducted or within several weeks. The tests were conducted by an audiologist, a nurse audiometrist, or a trained member of a local Aboriginal Medical Service as part of the annual statewide Otitis Media Screening programme. All the children were also screened for taste loss, and the results are the subject of another manuscript.

Results

The test–retest reliability of the olfactory identification test was very high, with a correlation coefficient of $r = 0.98$ being recorded. The latter value compares well with reported values for olfactory tests used by others with adults, for example, $r = 0.79$ (Nordin et al. 1998) and $r = 0.8$ for 6- to 15-year-old children (Davidson et al. 1998). In the present study, the mean correct identification scores for replicates 1 and 2 were 14.91 ± 2.27 and 14.91 ± 2.23 , respectively, the overall mean being 14.91 (93.2%).

The group of 261 children recorded a high mean correct odor identification score (\pm SD) of 15.13 ± 2.4 (94.6%) (females 15.04 ± 2.4 and males 15.21 ± 2.4) and were classified as normosmic if their score was <2 SDs from the mean, hyposmic if their score was >2 SDs below the mean, and anosmic if they had identification scores that were <2 SDs above the chance level of 5.33 (Nordin et al. 1998). Only one child had a chance score (5/16) and was therefore considered to be anosmic, whereas 4 children obtained a score of 12/16 which was just below the cutoff for normal function (12.73, i.e., $<13/16$) and were classed as slightly hyposmic. In the case of the child with anosmia, the cause could not be determined because of the absence of lifelong medical records for Aboriginal children and the difficulty of arranging a nasal examination by an ENT clinician for children in distant Aboriginal communities. The overall mean correct score of 94.6% is comparable to the data from the test–retest measure of 93.2% and fits well with earlier results from 7-year-old nonindigenous children (91.1%) and adults (97.8%) (Laing et al. forthcoming).

Each odorant was identified by at least 86% of the children, except “cheese” for which the mean identification score was 72% (SD = 0.45) (Table 1). A Cochran’s Q test showed that there was a significant difference in the correct identification scores across the odorants ($Q = 391.18$, degrees of freedom = 15, $P < 0.001$), and post hoc McNemar’s tests (alpha level of 0.01) indicated that the difference was due to a lower score in response to cheese ($\chi^2 = 16.55$, $P < 0.001$). A Mann–Whitney test indicated that there was no significant difference between the correct identification scores of females and males for cheese ($P = 0.441$), and an independent samples t -test

indicated that the overall correct scores of females and males across all the odorants were not significantly different ($t(259) = 1.13$, $P = 0.260$). Because the order of presentation of the odorants was the same for all participants and children were given only one odorant to sniff and identify from a set of 3 photographs as a practice for the test, a comparison of the correct identification scores for the first 8 odorants and last 8 odorants sampled was conducted to determine whether there was a learning effect. A simple regression analysis on split-half reliability (Nordin et al. 1998) produced a value of $r = 0.488$, which was highly significant, $F(1,259) = 80.93$, $P < 0.001$, with means \pm SDs of 7.52 ± 0.77 and 7.50 ± 0.76 for the first and second halves, indicating that there was no learning effect.

As regards hearing loss, 42/220 (19.1%) of the children were found to have mild to severe unilateral or bilateral hearing deficits and 44/220 children had evidence of past or present middle ear pathology. The latter group had either a blocked Eustachian tube and or a fluid in the middle ear, scarring of the tympanic membrane from previous encounters with CSOM, or the membrane was perforated. Thirty of the children with hearing loss exhibited a middle ear pathology. The remaining 12 children with hearing loss had no middle ear pathology and because lifelong medical histories of Aboriginal children are rare, it is impossible to ascertain the cause of their loss. Nevertheless, given the high prevalence of CSOM in Aboriginal communities, it is likely that the latter was the cause. Furthermore, the existence of hearing loss and absence of middle ear pathology suggest a sensorineural loss had occurred due to pathogens damaging the cochlea. Of the 5 children with olfactory loss, 3 had hearing loss and 4 had middle ear pathology. The anosmic child and one hyposmic child had hearing loss and current middle ear infection while a second hyposmic child had a perforated tympanic membrane. A third hyposmic child had a middle ear infection but had normal hearing, and another had normal pathology and hearing. The possibility that the 4 children with middle ear pathology had nasal infections that could have interfered with their perception of the odorants cannot be ruled out. However, there was no obvious evidence of nasal infections in any of the children with olfactory loss, and any child who had a “cold” or “runny” nose was not allowed to participate. Comparison of the mean odor identification scores of children with and without hearing impairment using an independent samples t -test indicated that there was no significant difference between the scores (normal hearing group = 15.21, SD = 0.97; hearing loss group = 14.68, SD = 1.97; $t = 1.66$, $P = 0.104$). The latter result indicates that there was no relationship between hearing ability and olfactory loss.

Discussion

The 2 main findings of this study were that the high prevalence of CSOM reported for Aboriginal children during early

childhood does not appear to have a long-term affect on their olfactory function and that hearing loss does not predict olfactory loss. Only 1/261 (0.38%) children had anosmia and 4 (1.53%) were slightly hyposmic. This compares with 42 (19.1%) children who had hearing loss. As mentioned above, whether the cause of olfactory loss in the 5 children was due to CSOM is not known, but it is doubtful. The finding of 2 anosmics in a recent study with 232 (0.86%) healthy 5- to 7-year-old non-Aboriginal children (Laing et al. forthcoming) also suggests CSOM was not necessarily the cause. Importantly, the fact that 19.1% of the children had hearing loss provides indirect but strong evidence that this was a typical group of Aboriginal children living in areas with high prevalences of CSOM. Furthermore, there was no difference between the olfactory abilities of children with and without hearing loss. Thus, it can be concluded that the hypotheses that the high incidence of CSOM in young Aboriginal children could cause a high prevalence of olfactory loss and that hearing loss could be an indicator of olfactory loss were not supported.

A second important outcome of the study was the validation of the identification results using the test–retest reliability procedure. The very high value of the correlation coefficient r of 0.98 provided strong evidence that the low prevalence of olfactory dysfunction in 9- to 12-year-old Aboriginal children was an accurate estimate for the group of children studied and that CSOM has little or no long-term effect on olfactory function. The r value also confirmed the suitability of the odor identification test for use with children and builds on an earlier study (Laing et al. forthcoming) that showed the that test could be used reliably by 5- to 7-year-old non-Aboriginal children. Importantly, with the exception of one study with 6- to 15-year olds, the children's odor identification test is the only test used with children that has reported the reliability of the procedure. The exception was the threshold test of Davidson et al. (1998), which had an r value of 0.8. The latter result was confirmed recently in an unpublished study with 5- to 7-year olds in this laboratory (Tomarchio, Wilkes and Laing, unpublished work), which found an r value of 0.77. All other olfactory reliability tests have involved adults.

Questions that remain unanswered, however, are whether CSOM causes temporary olfactory loss during the first 6 years of childhood when it is most prevalent and whether any loss recovers during the 3- to 6-year period that would have ensued for the participants in the present study. As regards the first question, if any loss occurred, particularly during early infancy (0–12 months), this has the potential to interfere with mother–infant interactions, particularly in relation to the feeding and social behaviors displayed by infants (Doucet et al. 2007). With the older infants (1–6 years), loss could interfere with eating behaviors, nutrition, and growth. Given the high incidence of poor health in Aboriginal children, establishing whether olfactory loss occurs during early childhood remains an important goal. Second, as regards the question of recovery, although there are no systematic studies of the duration of olfactory recovery in humans, reports of

recovery vary between 3 months and 3 years depending on the cause and damage from disease/head trauma/drugs (Costanzo and Becker 1986). Few reports of longer periods have appeared suggesting that in the present study if any loss had occurred during early childhood, it has been overcome probably via the regenerative capacity of the olfactory receptor cells during the period of lower CSOM prevalence.

The finding that only one child was anosmic provides an interesting comparison with the recent report (Bramerson et al. 2004) that the prevalence of anosmia was 5.8% in a random sample of 1387 20- to 80-year-old adults in Sweden. Although the present investigation did not have an epidemiological design, the children were from a wide cross-section of Aboriginal communities from 16 coastal and inland rural townships. Given, as mentioned above, that the present result is similar to that found with 232 non-Aboriginal children from metropolitan Sydney, the data suggest that the occurrence of anosmia in children is far less than in adults. This is not surprising because adults encounter hostile environments more often than children in the form of diseases, accidents, nasal surgery, and in the workplace, and some of the diseases are more common to adults than children. For example, Parkinson's (Doty et al. 1988) and Alzheimer's diseases (Doty et al. 1987) are associated with olfactory loss (Murphy et al. 2002), as has chronic renal disease (Schiffman et al. 1978). As yet, there does not appear to have been any reports of the prevalence of anosmia in children with which to compare the present value of 0.38%. Whether congenital anosmia accounts for the majority of cases of children with anosmia remains to be determined. However, it is possible that genetic disorders may be a predominant cause. Thus, known genetic causes include Kallman's syndrome (White et al. 1983), velocardial syndrome (Sobin et al. 2006), and idiopathic congenital anosmia that has been associated with an autosomal dominant pattern with female predominance of approximately 2:1 (Leopold et al. 1992).

In summary, the high prevalence of CSOM in Aboriginal children does not appear to result in loss of olfactory function. Whether short-term loss occurs during early childhood when the prevalence of CSOM is highest is not known but should be investigated to determine whether any negative effects as regards eating behaviors, nutrition, and growth occur. Finally, prevalence of anosmia in children appears to be substantially lower than that reported for adults.

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