DAMAGE TO TASTE (OTITIS MEDIA) IS ASSOCIATED WITH DYSGEUSIA, INTENSIFIED PAIN EXPERIENCE AND WEIGHT GAIN

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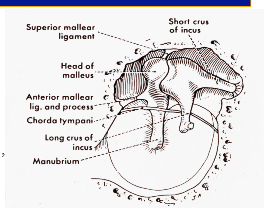
ABSTRACT

The chorda tympani taste nerve traverses the middle ear and may be damaged by infections (otitis media). Lecture attendees (N=6984) answered questions (Have you ever suffered from middle ear infections? Do you ever have persistent salty, sweet, sour, or bitter tastes in your mouth?) and reported height and weight. Analyses were done with multiple regression (controlling for sex and age). Those with histories of otitis media reported more dysgeusia and had higher BMIs (weight corrected for height). A subset (N=1599) rated everyday sensations (eg, strongest pain of any kind; strongest oral pain ever experienced) using the gLMS. Those with a history of otitis media reported the most intense pains. Chorda tympani input may inhibit a variety of central functions. Damage releases that inhibition. Possible consequences: intensification of non-taste oral sensations (eg, creaminess of fats), increased dysgeusia (release of inhibition of one taste nerve on another), increased oral pain (release of inhibition on oral pain). The finding of increased non-oral pain generalizes taste/pain inhibition to non-oral locations. The impact of otitis media on body weight may reflect both the alteration in sensory properties of foods (eg, increased palatability of energy-dense foods) and eating to reduce pain. (Funded by NIDCD).

BACKGROUND



Anatomy. The chorda tympani nerve (branch of CN VII, the facial nerve) leaves the tongue with the lingual branch of the trigeminal nerve (CN V), separates from the lingual nerve and then travels through the middle ear where it is vulnerable to pathogens associated with ear infections (i.e., otitis media). The figure on the right shows the ear drum (from the inside) with the chorda tympani passing behind the malleus bone).

Taste Inhibition and the preservation of eating. Taste input produces central inhibition (e.g., see DiLorenzo & Monroe, 1995; Smith & Li, 1998; Davis, 1993; Wang & Bradley, 1993). This inhibition contributes to taste constancy in the face of localized damage to taste: i.e., damage to one taste nerve releases inhibition on others intensifying their neural responses (Catalanotto et al, 1993; Lehman et al, 1995; Yanagisawa et al, 19989).

This inhibition also reduces oral pain (Bartoshuk et al, 1996; Bartoshuk et al, 1999; Tie et al, 1999; Grushka & Bartoshuk, 2000) and oral touch (preliminary data from Snyder, 2007). Since fats produce oral touch sensations (e.g., oily, viscous, creamy), intensification of oral touch might alter the palatability of high fat foods; this could affect weight.

We have suggested a more general function for taste inhibition: inhibition of a variety of activities incompatible with eating (Bartoshuk & Snyder 2002; Bartoshuk et al, 2005). This led to interest in the possibility that taste inhibition might affect more than just oral pain: pain at other body locations.

METHODS

Data analyzed are from a questionnaire study initiated in 1993. Lecture attendees (cumulative total about 6500) were asked to provide demographic information (age, sex, height, weight) as well as information about histories of otitis media and dysgeusia:

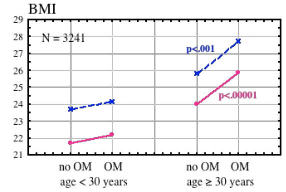
Have you ever suffered from middle ear infections?
(1) no, (2) yes, but not serious, (3) yes, required antibiotics more than once, (4) yes, required tubes in ears
Do you ever have persistent salty, sweet, sour or bitter tastes in your mouth?
(1) never, (2) occasionally, (3) sometimes, (4) always

In addition, attendees rated a variety of sensory experiences on the general Labeled Magnitude Scale (Bartoshuk et al, 2004; 2005); these included "strongest oral pain experienced," and "strongest pain of any kind experienced."

RESULTS

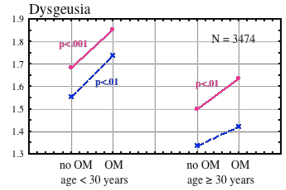
The results of ANOVA are shown below. Note that the Ns vary since some of the measures were not available for all lecture attendees. Each line segment in the graphs below shows the effect of otitis media history (OM). Results from females and males are shown in **pink** and **blue**, respectively. For oral pain, the main effect of sex and the interaction between age, sex, and otitis media history were not significant. Thus the data are plotted for age and OM history only.

BMI



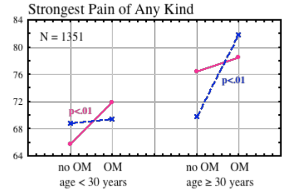
F(sex) = 80.288, p<.0000001
F(age) = 190.596, p<.0000001
F(OM) = 31.157, p<.0000001
F(age x age x OM) = .004, N.S.

Dysgeusia



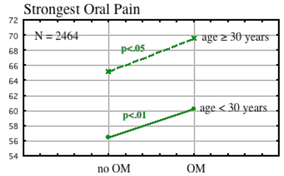
F(sex) = 25.425, p<.000001
F(age) = 58.938, p<.0000001
F(OM) = 22.220, p<.00001
F(age x age x OM) = .319, N.S.

Strongest Pain of Any Kind



F(sex) = .184, N.S.
F(age) = 27.220, p<.000001
F(OM) = 12.769, p<.001
F(age x age x OM) = 6.952, p<.01

Strongest Oral Pain



F(sex) = 1.600, N.S.
F(age) = 52.290, p<.0000001
F(OM) = 10.732, p<.01
F(age x age x OM) = .043, N.S.

DISCUSSION

Dysgeusia and Strongest Oral Pain. Using anesthesia of taste nerves along with study of patients with taste damage, we have hypothesized that loss of taste input releases inhibition producing phantom sensations as well as intensifying sensations evoked by some oral stimuli. The observation that dysgeusia (chronic taste sensations in the absence of stimulation) and intensified oral pain are associated with a history of otitis media is consistent with the damage to taste inflicted by otitis media.

Strongest Pain of Any Kind. The observation that intensified pain on other body loci is also associated with a history of otitis media is consistent with our hypothesis that central inhibition evoked by taste input has more general effects that just those observed in the mouth. Taste inhibition of pain would have the effect of encouraging an organism to eat when injured; such a mechanism has obvious survival value.

BMI. The results in the BMI panel (upper left under RESULTS) show that for both females and males aged thirty and above, a history of otitis media is associated with weight gain. Increased palatability of foods likely contributes to this (Snyder et al, 2003). The finding that pain intensities are higher among those with histories of otitis media might also play a role. Since taste damage is usually only partial with otitis media, eating might produce inhibition that would reduce pain.

The genetic contributions to obesity are well known (e.g., Sorenson et al 1989). Otitis media incidence can be influenced by genetic factors (e.g., see Daly et al, 2004); however, infectious diseases have obvious environmental origin as well.

There are other sources of taste damage. Preliminary data suggest that tonsillectomy (Hoffman et al, 2007; Bartoshuk et al, 2007) and head trauma (Bartoshuk et al, 2007) are also associated with higher BMIs. We suggest that taste damage in general may be a contributor to obesity.

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