

Chemo sense

EDITORIAL

Don't over-excite Granny

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In this issue, David Coppola challenges the conventional view of how the nervous system, and specifically olfaction, changes in the absence of stimulation: that crucial influence on nervous tissue to perform normally, to grow and be enhanced when increased, and to atrophy when decreased. The old adage is: "Use it or lose it".

Coppola comes to a surprising conclusion: closing a nostril produces a number of changes consistent with "use it or lose it" but also a number of compensatory mechanisms manifest themselves, such as increase in dopamine D2 and norepinephrine receptors. Despite depletion of bulbar granule cells, the remaining cells increase in excitability.

At the front of the system, the deprived sensory neurones show many signs of doing more than compensation, with changes to olfactory transduction enzymes that can be described confidently as up-regulation of the cells' ability to fire at lower thresholds. This has now been borne out by electrophysiological experiments.

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The effect of stimulus deprivation on the olfactory system: A case of 'use it or find it'!

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The hackneyed aphorism 'use it or lose it' remains a succinct, if oversimplified, statement of the relationship that is generally agreed to exist between a biological system's experience and its functional maintenance. After all, isn't it common knowledge that unused muscles atrophy and exercised muscles grow? Who hasn't heard that impoverished environments, especially early in life, can cause permanent impairment? Then too, enriching one's mental life is touted as a way to postpone the ravages of Alzheimer's in the aged and improve intelligence in the young (Katz & Rubin, 1999). Indeed, the role of experience in forming and maintaining our bodies has been a dominant theme throughout the history of science dating back, at least, to the 17th century debate between Molyneux and Locke concerning vision without experience.

One of the first experimental attempts to determine the effects of experience on neural

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These compensatory mechanisms oppose Donald Hebb's notion of pruning of synapses after sensory deprivation. Instead, the sensory system contains a high degree of plasticity allowing adjustment under extremes of high and low stimulation. Coppola cites recent evidence that odour rich environments have the opposite effect to deprived ones: sensory cells are lost when the system is rendered hyperexcitable in transgenic mice.

The wider implications of this work reflect upon the current fad for mentally stimulating aging people to stave off brain deterioration. Giving Granny too much sensory stimulation (and sudoku) may have an effect on her brain cells opposite to that desired. The use of compensation during deprived times may be crucial in maintaining a normal, surviving organism.

New Chemosensory Mechanism Discovered

We report news of the discovery of a mechanism for the relaying of chemical pain, heat and cold, to the free nerve endings of the trigeminal nerve, by isolated solitary olfactory cells inside the entrance to the nasal cavity.

ISOT 2008 Deadlines

Deadlines for this important international chemosensory scientific meeting can be found on our usual Upcoming Events page.

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continued

development was performed on the olfactory system by the eminent 19th century psychiatrist and neuroanatomist Bernhard von Gudden (1870). He pioneered unilateral sensory deprivation, a technique used to Nobel Prize-winning effect in the visual system nearly a century later (Wiesel, 1982). As the first neuroanatomist to study the effects of early lesions on development

to mean eliminating or reducing sensory experience) should be obvious: in bilateral sensory systems, like olfaction, vision, and audition, unilateral deprivation allows, at least in theory, the unmanipulated side to serve as a control in each subject. Also, with regard to the sense of smell, it is very difficult to achieve deprivation by any other means owing to the ubiquity of odors in the

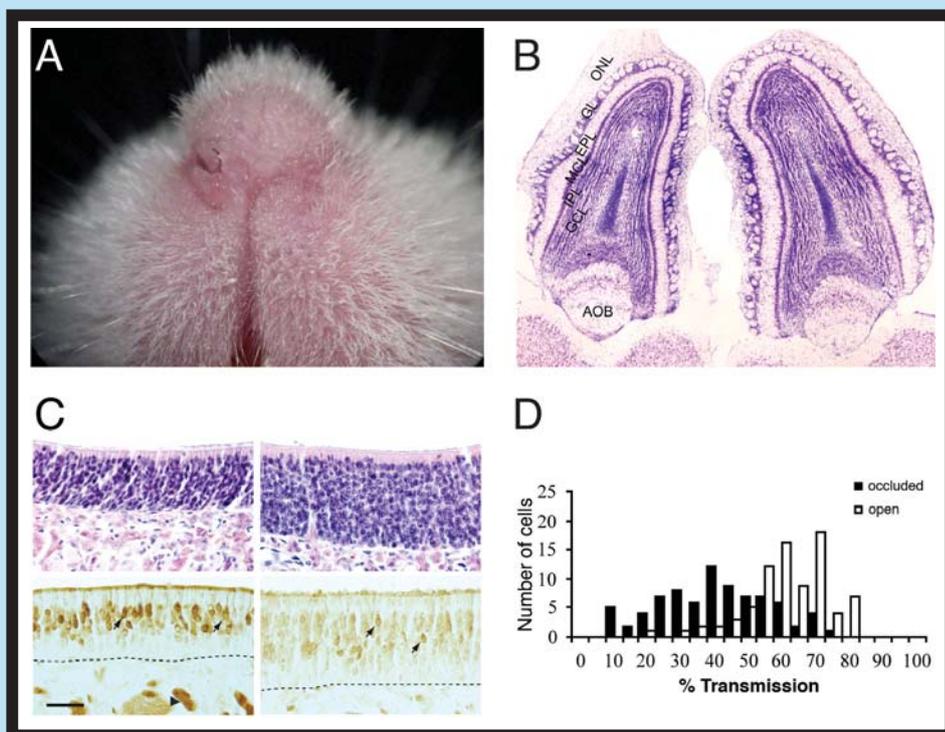


Figure 1. Effects of unilateral nostril occlusion on the olfactory bulb and mucosa of the mouse are shown. [A] This is a close-up photograph of the snout of an adult mouse whose left nostril had been occluded on the day after birth. [B] Horizontal section of Nissl stained bulbs in adult mouse whose left nostril had been occluded on the day of birth. [C] H&E stained sections (top row) or OMP labeled sections (bottom row) through the olfactory mucosa of 18 day old mouse that had been nostril occluded on the day after birth. Arrows point to olfactory sensory neurons; arrowhead points to axon bundles below basement membrane of mucosa illustrated by dashed line (scale bar = 50 μ m; redrawn from *Waguespack et al., 2007*). [D] Optical density measures of a random sample of 80 olfactory receptor neurons from open and occluded mucosa.

of the nervous system (Sarikioglu, 2007), von Gudden showed that occluding one nostril of newborn rabbits caused a pronounced reduction in the size of the olfactory bulb on the same side after 30 days. This finding has been replicated many times in several species (e.g. Meisami, 1976, Meisamin & Safari, 1981; Benson et al., 1984; Brunjes, 1985; see Fig. 1). The advantage of this technique for studying the effects of 'deprivation' (taken in this context

environment, including those produced by the subject's own body. Nevertheless, more than four generations after Gudden we still are not sure what his findings, and those of his many intellectual heirs, mean for the olfactory system.

Use it or lose it

Some of the experimental results and nearly all of the early interpretations, during the modern rediscovery of the unilateral nostril

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continued

occlusion technique, were consistent with the 'use it or lose it' idea. Studies in the developing rat, mouse, rabbit, and other species confirmed that perinatal nostril occlusion leads to the development of an ipsilateral olfactory bulb that is undersized by approximately one quarter (e.g. Meisami, 1976; Benson, et al., 1984; Gudden, 1870; reviewed by Brunjes 1994). While changes have been noted in the external plexiform and glomerular layers of the olfactory bulb following nostril occlusion, by far the most dramatic deficit is in the granule cell layer (Frazier & Brunjes, 1988). Earlier studies with tritiated thymidine and recent studies with bromodeoxyuridine establish that this effect on the granular layer of the occluded side bulb is predominantly due to decreased cell survival and not a difference in neurogenesis (Frazier-Cierpial & Brunjes, 1989; Saghatelian et al., 2005).

In contrast to these anatomical sequelae of nostril occlusion, which take weeks to detect, metabolic defects, as measured by 2-deoxyglucose uptake and Krebs cycle enzyme histochemistry, are apparent in a matter of days (Korol & Brunjes, 1990; Pedersen et al., 1987). Rounding out this picture of activity-dependent bulb development were findings that protein synthesis and gene expression also rapidly decline in the ipsilateral bulb as a function of nostril occlusion (see Brunjes, 1994).

Concerning bulb function, dopamine levels, as measured by quantification of this neurotransmitter's rate-limiting enzyme, tyrosine hydroxylase, are diminished within days of nostril occlusion, an effect that can be reversed by reopening the nostril of experimental animals (see Brunjes, 1994). However, electrophysiological studies have, for the most part, failed to show significant differences in the circuit properties of the ipsilateral bulb or its central targets in nostril occluded subjects (Guthrie et al., 1990).

Studies of the effects of nostril occlusion, as in the case with visual and auditory deprivation, have tended to focus on central effects such that relatively less is known about the changes in the periphery resulting from this procedure. In the mouse, rat, and rabbit, nostril occlusion leads to a decrease in the thickness of the respiratory and olfactory mucosa on the

occluded side (Benson et al., 1984; Farbman et al., 1988; Stahl et al., 1990; see Fig. 1). In the rat, a decline in the rate of mitosis after nostril occlusion occurs both in the respiratory and olfactory receptor epithelium (Farbman et al., 1988). Despite the difference in mucosal thickness, the numbers of mature olfactory sensory neurons is apparently unaffected by nostril occlusion in the mouse and rat, though there appears to be a decrease in the rabbit, a difference which has been attributed to the lack of a nasopharyngeal canal in the latter species (Stahl et al., 1990).

Importantly, the findings reviewed so far are consistent with the 'use it or lose it' view, but detailed developmental studies of nostril occlusion effects go further by making a case for activity-dependent development as well: what could be called 'use it or fail to develop it.' In fact, nostril occlusion performed during an early sensitive period, or "critical period," has more profound effects on the olfactory bulb than similar durations of nostril occlusion in adulthood (Brunjes & Borror, 1983; Henegar & Maruniak, 1991).

Thus, taken together, the findings reviewed so far on the effects of nostril occlusion in the olfactory system are consistent with a massive corpus, based on different sensory modalities, establishing the indispensable role of activity in normal development. Neural activity is believed, through Hebbian mechanisms ('cells that fire together wire together'), to strengthen appropriate functional connections and weaken and ultimately weed out inappropriate connections (Katz & Shatz, 1996).

Use it or find it

In marked contrast to the Hebbian worldview of olfactory development, which I have characterized as 'use it or lose it,' evidence has also accumulated for the opposite proposition. Indeed many of the changes in the olfactory system following nostril occlusion appear to be 'compensatory' in nature in that they are in the direction of preserving olfactory function in the face of sensory deprivation (Leon, 1998). For example, olfactory bulb neurotransmitter systems seem to follow this pattern in that the decrease in bulb

dopamine, which is a consequence of nostril occlusion, is compensated for by a >30% increase in dopamine D2 receptors that cannot be ascribed to shrinkage of lamina (Guthrie et al, 1991). Analogously, the increase in norepinephrine caused by nostril occlusion is compensated, in part, by a decrease in norepinephrine receptors (see Leon, 1998). Also, while nostril occlusion may cause a decrease in the extent of glomerular neuropil and the dendritic arbor of mitral cells, it also causes a more uniform distribution of a synaptic protein synaptophysin, a response that may be viewed as compensatory (see Leon, 1998). The increase in cell death that has been repeatedly demonstrated in the bulb ipsilateral to nostril occlusion is at least partly compensated for by an increased expression of the protooncogene bcl-2, which may prevent cell death (Najbauer et al., 1995). Finally, in a recent study, the depletion of ipsilateral granule cells following nostril occlusion appears to be compensated for by an increased excitability among the remnant granule cell population (Saghatelian et al., 2005). All of these examples may explain how the ipsilateral olfactory bulb of nostril-occluded animals appears to function normally, as far as we know, despite its abnormal development.

Perhaps more provocative are recent findings that the olfactory system does not just partially 'compensate' for the deleterious down-stream effects of the deprivation brought on by nostril occlusion but actually tries to compensate at the 'front end,' and throughout the system, for the sensory loss. These results and their implications form the bulk of my remaining comments and will explain my proposition that the effects of deprivation on the olfactory system are a case of 'use it or find it'!

First, let us consider the olfactory bulb. Late last year, Tyler and his colleagues (2007) published among the first detailed studies of the effect of nostril occlusion on primary and secondary synapses in the olfactory system. Using the whole-cell voltage-clamp technique in a rat slice preparation, they showed that two weeks of olfactory deprivation, beginning on the second day after birth, increases the probability and quantal content of neurotransmitter release

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continued

at primary olfactory synapses. This effect of nostril occlusion could even be demonstrated after three days of deprivation in young adult rats. Further, their immunohistochemistry, directed at a vesicular glutamate transporter and two different glutamate receptor subunits, demonstrated that nostril occlusion caused an up-regulation of these components of primary olfactory synapses. Their voltage-clamp recordings of spontaneous and olfactory-nerve-evoked activity in the predominant second-order neurons of the bulb, including mitral cells, establish that nostril occlusion strengthens synapses in down-stream components of the olfactory circuit, as well. This latter finding may partially explain earlier observations that both the size and intensity of odor-induced 2-deoxyglucose foci, in the glomerular layer of the bulb, are increased by nostril occlusion and that more ipsilateral mitral cells respond to a given odorant (Guthrie et al., 1990). Collectively, the elegant results of Tyler and colleagues (2007) reveal another heretofore-unknown compensatory response, namely that primary and secondary olfactory synapses are strengthened following olfactory deprivation.

But what about the most peripheral components of the sense of smell, the transducing pathways in olfactory sensory neurons? Evidence for a compensatory response to deprivation at this level has only recently emerged and awaits further confirmation. However, the story began more than 10 years ago when, a then-undergraduate student, Mike Reems and I observed that olfactory marker protein (OMP) immunolabeling was more intense in olfactory sensory neurons from the ipsilateral mucosa of nostril occluded mice (see Fig. 1). Given that *less* stimulation led to *more* of this protein we immediately thought of some sort of compensatory response, but since nobody knew the function of OMP at the time, this observation went nowhere. Recently, ten years after our initial observation, with OMP's function more squarely established as a modulator of olfactory transduction (Youngentob & Margolis, 1999), another student, Amy Waguespack and I replicated and finally published the OMP result

(Waguespack et al., 2005). In a series of follow-up experiments we showed that adenylate cyclase type III, a major component of the olfactory transducing cascade, and a non-ciliary phosphodiesterase, which has been shown to be involved in transducing modulation, were also up-regulated in olfactory sensory neurons in response to nostril occlusion (Coppola et al., 2006). At least for adenylate cyclase the implication is clear: decreased olfactory stimulation leads to an increase in this enzyme whose product cyclic AMP ultimately causes olfactory sensory neurons to reach threshold for action potential initiation. Thus, stimulus deprivation could be causing yet another compensatory response by increasing 'gain' in the transducing cascade of olfactory sensory neurons. More recently we have begun to analyze the effects of nostril occlusion on upstream and downstream components of the olfactory transducing cascade using RNA microarrays in the hope this will provide a more complete picture of olfactory sensory neuron responses to deprivation. For example, consistent with the compensation theme discussed here, we predict that olfactory sensory neurons may increase their complement of olfactory receptor proteins and ion channels.

Of course, if the changes we have reported in olfactory sensory neurons after nostril occlusion have any functional significance this should be measurable electrophysiologically. To address this issue, my colleague Chris Waggner and I recently recorded electro-olfactograms (EOG) from matched locations on the olfactory mucosa from the ipsilateral and contralateral nasal cavity of nostril occluded mice (Waggner & Coppola, 2007). Our stimulus set included log-dilutions of a small series of odors common in olfactory research. Consistent with our immunohistochemistry results and the compensation idea, EOG amplitudes from recording sites on the deprived side of nostril-occluded animals were greater for a given odor and concentration of stimulus than those from the open side. For some subjects the magnitude of the EOG on the occluded side was as much as double that on the open side. Given that the EOG is thought to be derived from the summed generator potential of olfactory sensory

neurons in the vicinity of the recording electrode, our results imply that sensory neurons on the occluded side have greater generator potentials or that more of them are recruited by a given odor, or both. Interestingly, while the EOG amplitudes were greater on the deprived side, onset and offset kinetics were both significantly slowed compared to the open side (Fig. 2). These electrophysiological results provide the first direct evidence that the front line of smell, olfactory sensory neuron cilia and dendrites, respond to deprivation in a compensatory manner.

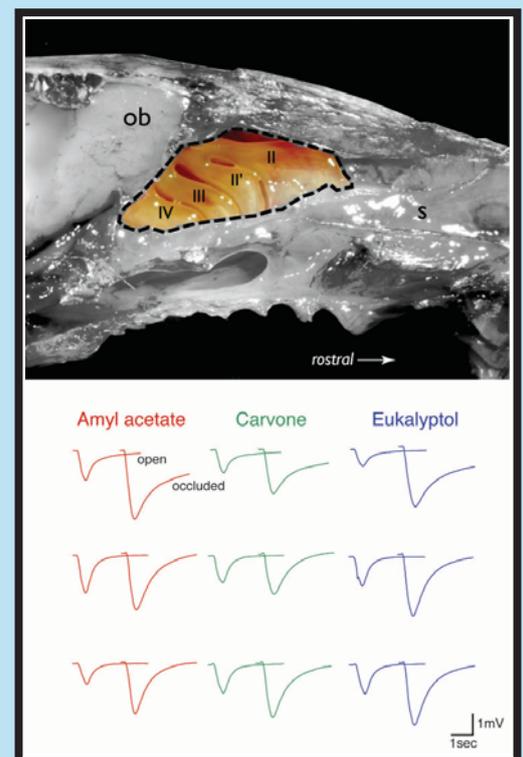


Figure 2. [Top] Medial view of a midsagittal section of adult mouse head that illustrates method of endoturbinate access for electrophysiological recording (ob = olfactory bulb; s = nasal septum). [Bottom] EOG traces from three adult mice (rows) in response to three odorants (columns). Subjects had one nostril occluded on the day after birth. Adjacent traces show responses at matched locations on endoturbinate II from the open side (left trace) and occluded side (right trace). The stimulus was the headspace above a 0.1% concentration of odorant mixed v/v with mineral oil delivered in a 500 ms pulse (redrawn from Waggner & Coppola, 2007).

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Down with Hebb; up with homeostasis

Any notion that sensory experience in the olfactory system might play an 'instructive' role in the layout of the olfactory map in the bulb has crashed on the rocks of experimental evidence. Modern genetic approaches, which have allowed the creation of mouse strains lacking essential components of the transducing cascade of olfactory sensory neurons, establish that the proper guidance of sensory cell axons to glomerular targets does not require sensory activity or even functional synaptic release (reviewed by Yu et al., 2004). While spontaneous activity (i.e. non-sensory driven and presumably uncorrelated) seems to be needed, this situation hardly pertains to the effects of nostril occlusion in which spontaneous activity is presumably normal or even accentuated. Given the problem that developmental linguists have called the "poverty of the stimulus" it was probably never a tenable proposition that >1000 types of olfactory sensory neurons could sort their axons in a matter of a few days, based on sensory driven correlated activity, even in lab-reared animals (Yu et al., 2004). Thus, Hebb's postulate finds little succor in what we have recently learned about the development of the bulb odor map.

The previously discussed strengthening of primary and secondary synapses in the bulb following deprivation is also hard to square with a Hebbian process being more consistent with the notion of homeostatic plasticity (Tyler et al., 2007; Turrigiano & Nelson, 2004). Then too, the apparent compensatory processes we have seen in olfactory sensory neurons would seem to oppose a Hebbian pruning of synaptic connections (Waguespack et al., 2005; Coppola et al., 2006; Waggenger & Coppola, 2007).

However, the fact still remains that nostril occlusion causes proliferating granule cells and other interneurons (Hamilton & Coppola, 2003) in the bulb to die! Given that granule cells are inhibitory on mitral cells, the major output neurons in the bulb, and are thought to participate in lateral inhibition that may sharpen odor discrimination, it is tempting to suggest that a homeostatic process in the olfactory circuit underlies this response. Perhaps in the face of sensory loss or reduction the system is

increasing odor detection at the price of odor discrimination. It would be interesting to know, in this regard, what the actual functional abilities are of an olfactory system that developed under the deprivation regime created by nostril occlusion. However, 137 years after the first nostril occlusion experiment and with the benefit of a spate of recent articles, no report on the psychophysical abilities of adult animals that had been nostril occluded perinatally can be found. It is known from earlier work in the author's lab that 10 day-old mice, nostril occluded at birth, can detect nipple pheromone with their ipsilateral bulb, but this is hardly a thorough test of olfactory abilities (Coppola et al., 1994). This work is continuing, using olfactometry combined with standard operant techniques.

From an evolutionary perspective the compensatory processes, which appear to be implemented at various levels of the olfactory system from olfactory receptor neurons to central circuits, seem to make sense (and scents!). Given their finite dynamic range, nature has designed sufficient plasticity in sensory systems to continuously adjust their response to maximize the useful information transferred about the environment (Stemmler & Koch, 1999). This is why sensory systems adjust themselves to report changes in the environment rather than static levels of the stimulus (see Coppola & Purves, 1996 for an example in vision). Adaptation is a short-term example of this mechanism that has been examined extensively, both empirically and theoretically, in many sensory systems (e.g. Laughlin, 1989). The effects of longer-term deprivation on the olfactory system, such as that seen following nostril occlusion, can be understood in the same light, though their cellular mechanisms, time course, and reversibility may be quite different. From this viewpoint, animals exposed to 'noisy' or 'enriched' odor environments might be expected to show changes opposite to those reported here for the deprived state. In fact, this kind of 'push and pull' has been seen in the OMP content of olfactory receptor neurons exposed to enriched versus deprived odor environments, though this result, like the precise function of OMP itself, remains enigmatic (Waguespack et al., 2006). Odor

rich environments, also, have opposite effects to odor deprivation in the bulb, leading to enhanced granule cell survival (Rocheffort et al., 2002). Finally, a recent study has shown that transgenic mice with a gene-targeted deletion of a potassium channel that renders mitral cells hyperexcitable, actually lose many olfactory sensory neurons consonant with the compensation theme of this review (Biju et al., 2008).

Gudden revisited

von Gudden, mentor to the likes of Emil Kraepelin (of schizophrenia fame) and Franz Nissl (of stain fame, see Fig. 1), was, among his many other professional duties, appointed psychiatrist to the Bavarian Royal Family. On July 13, 1886, while attending to mad King Ludwig II, Gudden was found dead in Starnberg Lake near Munich, presumably at the hands of the Disney-inspiring King who was also found dead in the Lake (Sarikcioglu, 2007). If von Gudden were alive today he might be surprised that so little has been resolved despite the intervening century (and more) since his observations on the effects of nostril occlusion in olfactory pathways. Readers of this review can decide whether that the situation is closer to 'use it or find it' than 'use it or lose it,' though neither of these simplistic aphorisms is terribly helpful. The role of sensory activity in the development and maintenance of the nervous system turns out to be incredibly complex and, as they say, the 'devil is in the details'. For example, some complex 'computed' sensory modules, like orientation maps in visual cortex, develop normally without the benefit of sensory input, while others, present in the same cortical area, such as visual direction sensitivity, require visual experience (cf. Coppola & White, 2004; White & Fitzpatrick, 2007). Further progress in understanding the role of sensory experience in the development and maintenance of the olfactory system is going to require not only fresh experimental approaches, but also, more nuanced theoretical frameworks than have been used up until now. Then perhaps it won't take another century before the effects of nostril occlusion on the olfactory system are understood ■

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NEWS

Onion Tears: New Mechanism Discovered

The burning and tearful experience that anyone knows who has chopped onions, has been explained recently by a new mechanism discovered for solitary olfactory cells found near the entrance of many animal noses, including (probably) humans.

In an article published in the March edition of *J Neurophysiol*, a team including Weihong Lin, Tom Finger and Diego Restrepo, found that these cells respond to many types of odorants and in turn communicate with the free nerve endings of the trigeminal nerve in the deeper tissue layers of the nasal cavity. Previously it was thought that the trigeminal nerve endings alone gave the sensation of pain in the presence of irritating airborne chemicals. Their finding shows that the solitary cells pass a message to the trigeminal nerve which then relay messages to the brain, which are perceived as pain, heat or cooling.

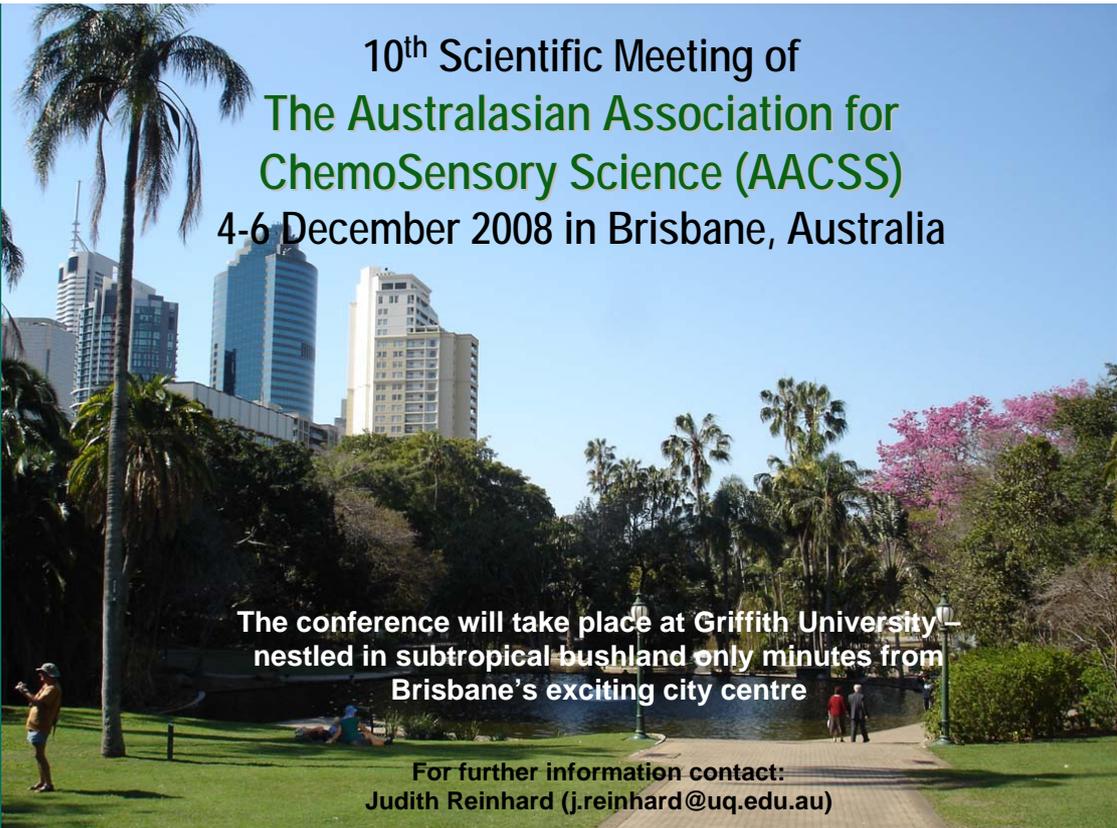
This new mechanism explains why so many odorants have an irritating effect, since they are likely to be specialized for odorant reception to a greater degree than the trigeminal's free nerve endings. The odorant vanillin is believed to be one of the few odorants that, with increasing concentration, does not stimulate the trigeminal system. Most other odorants have no irritating

effect in very low concentration but develop either a cooling or burning sensation with increasing concentration as the trigeminal nerve "kicks in". Presumably the solitary olfactory cells do not recognize vanillin, but do detect sufficient quantities of most other odorants, for reasons that remain to be elucidated.

The discovery was made using mice, in research following up an earlier discovery that these cells contain bitterness receptors. The discovery of the role for these special solitary olfactory cells in oral pain may make it easier to understand the basic relationship between the sensation of chemical "burn" in foods such as onions, and if, how and why "burn" enhances perception of flavour. But first, the solitary cells will need to be confirmed in humans, and ways achieved to study them. Once again, animal research proves its worth in creating knowledge.

The finding also raises the question of whether there are other specialist cells that act as diminutive "end organs" for the widely distributed free nerve endings of the trigeminal nerve.

Graham Bell ■



10th Scientific Meeting of
The Australasian Association for
ChemoSensory Science (AACSS)
4-6 December 2008 in Brisbane, Australia

The conference will take place at Griffith University
nestled in subtropical bushland only minutes from
Brisbane's exciting city centre

For further information contact:
Judith Reinhard (j.reinhard@uq.edu.au)

NEWS

ADVERTORIAL

E-Nose Africa Team Grows

E-nose Africa is proud to welcome Bashan Naidoo as a Member of the Team. His expertise will add significantly to our capacity to service the South African market. He has already been addressing the challenges posed to the industry's odour monitoring projects by the current electricity supply environment.

Bashan has been involved with E-nose for several years. His MSc thesis (2003) is entitled "Towards the Development of an Electronic Nose". In completing this, he worked alongside the E-Nose - Australia team and has jointly published journal articles, conference papers and book chapters in conjunction with them, as well as in his own right.

Bashan holds a BSc(Eng) and an MSc in Electronic Engineering (both from the University of Natal) and is currently working on his PhD through UCT, in the field of image processing and computer vision. A Senior Lecturer at the University of KwaZulu-Natal's School of Electrical, Electronic and Computer Engineering, he lectures in the fields of computer methods, artificial intelligence as well as electronic and computer design.

Bashan and the rest of the E-Nose Africa Team look forward to helping you solve your challenges in odour monitoring and management.

News from Australia

The E-Nose Pty Ltd team in Sydney, currently have two interesting monitoring jobs going on: one at a petrochemical tank farm and one involving dredging polluted sludge from a river bed. Both aim to measure environmental impact of smells on residential neighbours. The latter application involves using two parallel E-noses synchronised to cover a large area and capture odours,

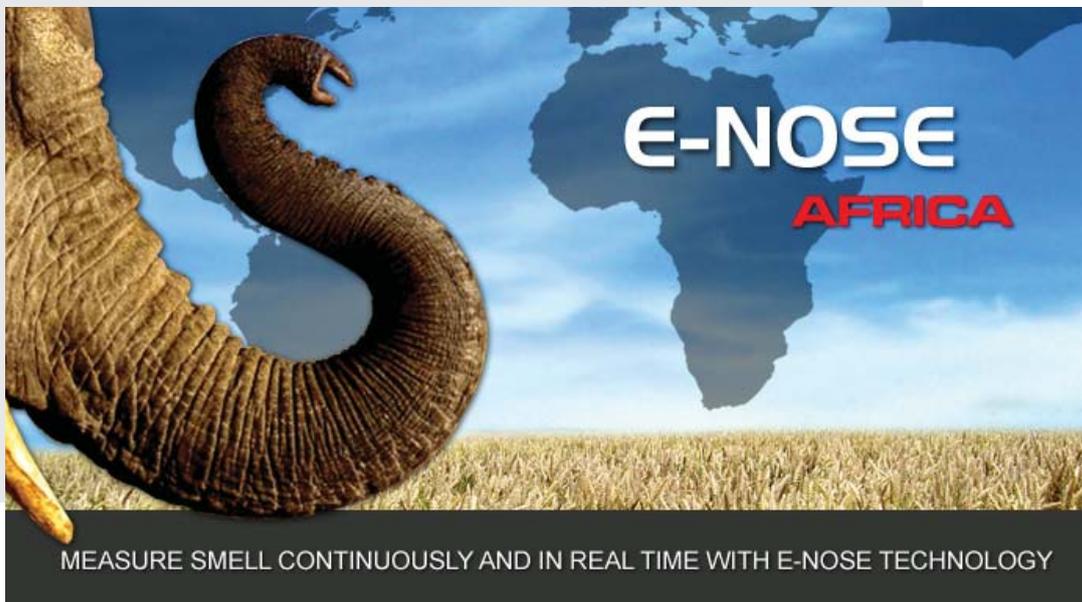
from different sources, when the wind changes direction.

They have recently carried out a trial of their anti-graffiti E-Nose at a railway company. The "Graffiti-E-Nose" was able to detect a four second squirt of spray paint from a distance of 45 metres. Cleaning train carriages of graffiti paint is costing Australia's train companies tens of millions of dollars annually. The "Graffiti-E-Nose" can discriminate cleaning solvent from paint: a necessary task because the vandals are often at work at one end of a train while cleaners are removing paint at the other! The spray-paint-detecting E-Nose will add a new level of deterrence against the vandals and minimize false alarms produced by cameras and movement detectors in this security context. The benefit will be reduced costs to the rail companies, the traveling public and tax payers. "Aerosol artists" and "teenage ninja taggers" will find that squirting chemicals on railway property has some new surprises in store for them.

The latest investor in a Mk 3.3 E-Nose is a large water and sewage treatment company in Brisbane. E-Nose Pty Ltd also recently exhibited at Ecoforum, on the Queensland Gold Coast, where a lot of interest was shown by companies involved in site remediation.

Lessons learned from these projects will be passed on for the benefit of E-Nose Africa and its customers. In return, E-Nose Africa is learning lessons to pass on to the air monitoring community, in its work with providing continuity of measurement through periods of power outage.

18/3/08



E-NOSE
AFRICA

MEASURE SMELL CONTINUOUSLY AND IN REAL TIME WITH E-NOSE TECHNOLOGY

Upcoming Events

6-8 July 2008

International Conference on Environmental Odour Monitoring and Control – NOSE2008

Rome, Italy

<http://www.aidic.it/nose2008/>

21-25 July 2008

International Symposium on Olfaction and Taste (ISOT) and AChemS Meeting

Hyatt Regency Hotel at the Embarcadero

San Francisco, California, USA

Abstract Deadline: 2 April, 2008

Early Bird Registration deadline: 15 April, 2008

Registration deadline 20 June, 2008

<http://www.ISOT2008.org>

17-21 August 2008

The Chemical Senses and Health

Symposium Commemorating 100th Anniversary of ACS-AGFD and 40th Anniversary of Monell Chemical Senses Center. To be held during the ACS National Meeting, Philadelphia, USA

Contacts: bachmanov@monell.org;

beauchamp@monell.org; Lleland@kraft.com

3-7 September 2008

18th European Chemoreception Research Conference (ECRO-2008)

Portoroz, Slovenia.

<http://www.ecro-2008.si>

8-10 October 2008

The 3rd IWA Specialist Conference on Odours and VOC
Barcelona, Spain

Contact: r.steutz@unsw.edu.au

4-6 December 2008

Australasian Association for ChemoSensory Science (AACSS)

Annual Scientific Meeting

Griffith University, Brisbane

Contact: j.reinhard@uq.edu.au ■

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