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ChemoSense

Editorial

By Graham Bell, Editor
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Dismal outcomes for many people with head injuries led early physicians to declare that there was no plasticity inside the skull and spinal cord and hence little hope for recovery from damage to the central nervous system. By comparison, peripheral nerves recovered from damage, including transection, and reconnected to their end-organs at rates that could be reliably timed and predicted. Plasticity, we now know, occurs in the CNS but often without completion of functional connections. The olfactory system is rich in on-going cell turnover and reconnections, but these mechanisms remain to be understood. Nevertheless,

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A clinical approach to plasticity of the olfactory bulb

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Among the human sensory systems the olfactory system is unique in that way that olfactory receptor neurons in the olfactory epithelium and neuronal cells in the olfactory bulb (OB) are continuously renewed. Although the cause and consequence for the continuous replacement of neuronal cells in the OB is still under debate there is growing evidence for a close relationship between olfactory function and the volume of the OB. Change of olfactory function in humans is typically associated with an increase or decrease of the OB volume. Based on the review of the literature, the processes that could impact on the plasticity of the OB are discussed. In addition, the clinical use of volumetric measurements of the OB is highlighted.

Olfactory signal pathway

Olfactory receptor neurons

Olfactory receptor neurons (ORN) are the only

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continued

relatively simple ways of observing plasticity in the olfactory system offers the clinician new tools, as is demonstrated by this issue's review by Gadziol and Hummel.

The 2010 AACSS meeting will be held on December 2-4, in the lovely Yarra Valley, home of Australia's earliest vineyards and now a thriving wine production area. Future meetings are anticipated in New Zealand in 2011 and Queensland (Barrier Reef) in 2012 ■

A clinical approach to plasticity of the olfactory bulb
continued

known sensory receptor neurons that regenerate in humans on a regular basis, probably within a few months [1,2]. The ORN that are located in the olfactory epithelium in the nose send their ciliated dendritic knob into the nasal mucous to detect odorants. Thereby ORN are exposed to potentially neurotoxic agents that enter the nose. In turn, this exposure can cause damage to the neurons. Continuous regeneration of the ORN appears to be mandatory to renew damaged or old ORN in order to maintain olfactory function. The axons of the ORN form bundles (olfactory nerve) which pass through the cribriform plate and insert into the olfactory bulb.

Odor quality coding

Humans express approximately 350 different olfactory receptors[3,4]. Importantly, a single ORN expresses only one type of olfactory receptor [3,5]. At least in rodents axons of all ORN expressing the same olfactory receptor converge in a median and in a lateral situated glomerulus in each olfactory bulb (OB)[5]. In the glomerulus the signal arriving from the ORNs is switched over to output neurons like mitral cells and tufted cells. The OB is not only the first relay in the olfactory signal pathway, it is actually also a site of signal modification. Granule cells and periglomerular cells can develop dendrodendritic synapses with the mitral cells, optimizing olfactory function by reducing overlap for odor representation by mitral cells[6,7]. Additionally, each glomerulus projects to its partner glomerulus on the opposite side of the OB via tufted cells which results in a reciprocal connection between the glomeruli that receive input from ORN expressing the same olfactory receptor enabling them to coordinate their responds to ORN excitation[8].

Olfactory bulb

The olfactory system is not only capable to renew on the peripheral level (ORN). Since the work of Altmann in 1969 it is known that the OB receives cells from the subventricular zone (SVZ) throughout adulthood [9]. The progenitor cells originating from the SVZ migrate via the rostral migratory stream (RMS) to the OB. In rodents approximately 30,000 progenitor cells migrate to the OB per day[10]. There they differentiate into granule (approximately 90%) and periglomerular cells[11]. Upon their arrival in the OB the progenitor cell experience maturational changes, develop dendrites, and establish dendrodendritic synapses with the mitral and tufted cells in the OB[12]. Due to the fact that the OB volume does not increase in adulthood and that less than 10% of in young adult mice born granule cells become older than 21 months and apoptosis is found regularly in the granule cell and periglomerular cell layer in the OB, it is believed that newly born granule and periglomerular cells replace older cells in the OB[13-15]. That continuing replacement of neurons in the OB enables the olfactory system to stay functionally plastic such that olfactory experience modulates the neuronal network in the OB[16]. Another hypothesis says that replacement of neurons in the OB is owned to the fact that the OB appears to be relatively unprotected towards viruses, bacteria, or toxins entering the brain through the cribriform plate[17-19]. The damage done by these external toxins may be the reason why

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replacement of neurons in the OB is needed to maintain the sense of smell throughout life.

Impact of olfactory deprivation and odor enrichment

In order to understand the continuing structural changes that take place in the OB it is interesting to focus on the structural changes that are caused by olfactory deprivation. Unilateral olfactory deprivation by means of obstructing of the nasal cavity or destructing of the olfactory epithelium in mammals results in complex changes in the OB cell structure. Although no changes in the mitral cell density is observed after odor deprivation some authors found decreased soma size and dendritic elaboration in the mitral cells[20-22]. The most pronounced changes in cell density were observed in the tufted cells and in the granule cells and their associated glia[20]. These changes may have several causes. First, it appears that odor deprivation results in apoptosis of existing neurons in the OB[15,23]. Second, cell death of precursor cell in the SVZ is increased [24]. Additionally, migration, the survival and differentiation of precursor cells in the OB is reduced[12,15,24,25]. These changes together with a reduced interaction within the neuronal network in the OB due to reduced sensory input result in a 25- 27% decrease of the OB size on a macroscopic view [26,27].

What are the structural changes that are observed in the OB when olfactory function is increased by opening a formerly closed nasal cavity or odor enriching of the environment? The observed changes are reverse to the changes induced by odor deprivation. After opening a closed nasal cavity an increase of tufted cell and granule cell density are observed in the OB[28]. This increase appears to be a result of prolonged cell survival in the OB[15,29]. Additionally, increasing olfactory input boosts survival and differentiation of precursor cells in the OB[30]. In concert with increasing interaction within the bulbar neuronal network these changes result in an increase of the OB volume toward normal size after elimination of odor deprivation[28].

Olfactory function and olfactory bulb volume in humans

Magnet resonance imaging (MRI) was employed first by Yousem and coworkers (1996) to study the relation between olfactory function and OB size in humans in vivo[31,32], albeit in relatively small sample sizes. It was demonstrated that MRI based volumetric measurements of the central olfactory system revealed an 88 to 98% accuracy compared to the true volume (measured by water displacement). The inter-observer difference was between 0.1 and 3.7%. Repeated MRI- scans and volumetric measurements revealed volumetric differences within a range of less than 5%[33]. Thereby, MRI- scan based volumetric measurements appear to be a reliably tool for the morphologic assessment of central olfactory structures.

Subsequently, studies were performed to evaluate the OB volume in relation to different causes of smell loss. It is well known that olfactory function decreases with aging[33]. It could be demonstrated that the OB shrinks parallel to decreasing olfactory function in older individuals[34,35].

Additionally, in patients with post-traumatic and post- infectious olfactory loss smaller OBs were found compared to normosmic individuals[36,37]. Smaller OBs could also be found in patients with chronic rhinosinusitis with polyps who present with olfactory loss[38-40]. In concert with the findings of OB degeneration in odor deprived mammals it appears reasonable to assume that reduced olfactory stimulation of the OB due to damage to the olfactory epithelium by a virus or disruption of the olfactory signal pathway from the ORN to the OB are the cause of smaller OB volumes in patients with post- traumatic and post- infectious olfactory loss[28]. On the other hand it could be possible that neurotropic viruses affect the olfactory system in the nose and migrate centrally. In the OB these viruses could damage interneuronal interactions resulting in a degeneration of the neuronal network which eventually might lead to a reduced OB volume. Additionally, neurotropic viruses can impact on production, migration and maturation of precursor cells originating from the SVZ[18]. Again, the reduction of the influx of precursor cells in the OB could result in a decrease of OB volume. In patients with chronic rhinosinusitis inflammatory mediators that diffuse from the inflamed nasal mucosa into the OB can impact similarly to neurotropic viruses on the OB volume. It was demonstrated that inflammatory mediators can regulate the proliferation of progenitor cells, their ability to differentiate, direct the migration of new neurons and control their survival[41]. Additionally it was shown that drugs, or metal dust may diffuse via the olfactory signaling pathway from the nasal cavity

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continued

into the OB[42,43]. Thereby it could be possible that inflammatory processes in the nasal cavity lead to a reduction of the OB size by a top down regulation of the neuronal regeneration of the OB[44].

On the other hand chronic rhinosinusitis can cause olfactory loss by obstructing the olfactory cleft or due to inflammation of the olfactory epithelium[45]. The latter causes would impact similarly to post-traumatic and post- infectious olfactory loss as a bottom- up regulation on OB regeneration.

Neurodegenerative disease like idiopathic Parkinson´s disease is associated with olfactory loss[46]. Interestingly OB volume in these patients appears to be within the range of normosmic individuals although the investigated patients demonstrated smell loss[47,48]. The olfactory epithelium in patients with Parkinson´s disease presents no immunohistochemical abnormalities[49]. Together with the clinical finding of normal OB size it appears likely that olfactory loss in Parkinson´s disease is not a consequence of damage to the olfactory epithelium but rather results from central-nervous changes[50,51]

Top-down versus bottom-up?

In another study reduced OB volume was found among hyposmic patients with acute major depression[52]. Despite other possible reasons, the authors emphasized that reduced neurogenesis could be related to depression; reduced OB volume, and, consequently, decreased olfactory function. If this hypothesis could be confirmed then evidence would grow for top- down regulation of the OB volume.

Possible prognostic value of OB volumetry

Studies indicating that the OB volume is smaller in patients with olfactory loss do not provide data about the OB´s ability to recover to normal size. From animal studies it is known that the OB volume normalizes within a short period of time after cessation of olfactory deprivation[28]. In humans, using MRI, it could be demonstrated that with spontaneous regeneration and increasing olfactory function in patients with post- traumatic and post- infectious olfactory loss OB volume increased within 15 month[53]. Another study found an average increase of the OB volume by 9% on the left side and 19% on the right side in patients after treatment of chronic rhinosinusitis with polyps[39] . Interestingly studies revealed a significant correlation between the improvement of odor threshold and the change of the OB volume. This fact may additionally emphasize the close relationship between OB volume and ORN function because odor threshold scores are believed to represent peripheral olfactory function[54,55] to a higher degree than more complex tasks, e.g. odor identification[56,57]

Conclusions

Typically, the volume of the human OB changes with changes in olfactory function. Peripheral olfactory loss due to odor deprivation or disruption of the olfactory epithelium may reduce the influx and differentiation of precursor cells originating from the subventricular zone which would result in a decrease of the OB volume. These ideas would argue for a bottom- up regulation of the OB regeneration. On the other hand there is growing evidence that migration of viruses and inflammatory mediators from the nasal cavity centrally along the olfactory signal pathways (like in post-infectious olfactory loss or in chronic rhinosinusitis) may impact on the influx and differentiation of precursor cells which would indicate a top-down regulation. In addition, a decrease of the OB volume is also found in patients with depression, indicating top-down regulation of these processes.

Future investigations will tell whether changes of the OB contain significant information on the cause and prognosis of olfactory loss ■

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AACSS 12th Scientific Meeting
Yarra Valley, December 2-4, 2010

The 12th Scientific Meeting of the Australasian Association for ChemoSensory Science (AACSS) will be held from December 2-4, 2010 in the beautiful Yarra Valley wine region near Melbourne. The meeting will commence Thursday December 2nd in the evening, and conclude Saturday December 4th with lunch. This leaves the weekend free to visit the many nearby wineries if desired. The conference venue is the Balgownie Estate Vineyard Resort and Spa: www.balgownieestate.com.au/yarra-valley/

We have assembled a very exciting line up of international speakers and in particular are privileged to announce that a plenary speaker at the meeting will be renowned chemical senses researcher Professor Richard Doty, author of many seminal books in the chemical senses and inventor of the UPSIT smell identification test.

Plenary speakers:

Professor Richard Doty, Director, University of Pennsylvania Smell and Taste Center

Neurodegenerative Diseases and Olfaction

Associate Professor Giovanni Galizia, University of Konstanz

Olfactory information coding in the insect antennal lobe

Assistant Professor Helen Treloar, Yale University

Axon guidance in the mammalian olfactory system

This is a great opportunity to hear from and talk to these outstanding researchers in a beautiful and relaxed setting, so take advantage of it!

Information regarding the program and invited speakers, registration, abstract submission and accommodation is available on the AACSS website, www.aacss.org

The deadline for registration and abstract submission is 15th October, 2010

Any queries regarding the meeting can be directed to Coral Warr, coral.warr@monash.edu

ecoforum

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Each year EcoForum gives environmental managers and their clients a chance to get together in Sydney at Australia's leading ecoforum. The organisers of EcoForum 2011, to be held from 9–11 March, are calling for papers now and until 30 September on the following broad themes. They would also welcome presentations from Chemosense readers on topics that are not already listed.

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You can see a full list of suggested topics, find out more about EcoForum and submit an abstract at **www.ecoforum.net.au/2011**.

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BOOK REVIEW *by Graham Bell*

Navigating Smell and Taste Disorders

By Ronald Devere and Marjorie Calvert

2011, Soft-cover, 180 pages, US$ 19.95
Published by demosHEALTH, New York.
www.demoshealth.com

This book combines explanation of the physical structures and functions that go wrong when smell and taste loss occurs, the disease states and other causes of such loss and what you might be able to do about it. It informs patient and practitioner of what to expect and most importantly offers a positive outlook to achieving a positive outcome, be it in achieving real recovery or learning to live with the disorder.

The first part of the book is the physiological and medical "navigation" and then we are taken into the important domain of what patients might eat and drink and how foods might be prepared to take pressure off the patient and relieve the agony that often accompanies the dramatically unpleasant experiences of smell and taste disorders. Such a juxtaposition of information is welcome and reveals the authors' mission to help people and provide information by which they can steer themselves toward recovery and restoration of an undisturbed lifestyle.

In this vane it is no surprise that an Appendix provides lists of smell and taste clinic in the USA and in several other countries, as well as much other resource information, an unusual but possible necessary explanation of what neurologists do, so that one might moderate one's expectations of what help they can realistically offer, and an excellent glossary of terms.

All-in-all this adds up to demystifying the problems that people will encounter, and points them in the direction of improvement and recovery.

This timely, accessible and inexpensive volume is a "must read" for anyone who has experienced chemosensory loss, their carers and family, and for anyone who offers them professional, medical and practical help ■

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The Great Pheromone Myth

Richard L. Doty, Ph.D., University of Pennsylvania

$65.00

Mammalian pheromones, audiomones, visuomones, and snarks — Dr. Doty argues that they all belong in the same category: objects of imagination. For more than 50 years, researchers — including many prominent scientists — have identified pheromones as the triggers for a wide range of mammalian behaviors and endocrine responses. In this provocative book, renowned olfaction expert Richard L. Doty, Ph.D., rejects this idea and states bluntly that, in contrast to insects, mammals do not have pheromones.

Doty systematically debunks the claims and conclusions of studies that purport to reveal the existence of mammalian pheromones. He demonstrates that there is no generally accepted scientific definition of what constitutes a mammalian pheromone and that attempts to divide stimuli and complex behaviors into pheromonal and nonpheromonal categories have primarily failed. Doty's controversial assertion belies a continued fascination with the pheromone concept, numerous claims of its chemical isolation, and what he sees as the wasted expenditure of hundreds of millions of dollars by industry and government.

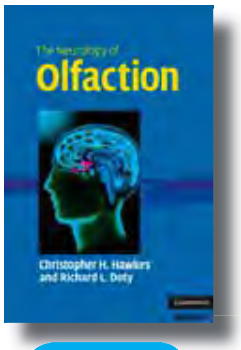
The Great Pheromone Myth directly challenges ideas about the role chemicals play in mammalian behavior and reproductive processes. It is a must-have reference for biologists, psychologists, neuroscientists, and readers interested in animal behavior, ecology, and evolution.

"Simply delightful reading. In a concise but totally convincing manner, Richard Doty sweeps away the pervasive mythology of pheromones." — Floyd E. Bloom, Scripps Research Institute

"The field of mammalian pheromones is a bit sloppy and human pheromones a complete mess. This book will make a major contribution to the field by either galvanizing people to prove Doty wrong or applying brakes to a field that may be fast moving down the wrong track."— Donald A. Wilson, author of *Learning to Smell: Olfactory Perception from Neurobiology to Behavior*

The Neurology of Olfaction

Christopher H. Hawkes, Neuroscience Centre, Barts & The London School of Medicine & Dentistry, London
Richard L. Doty, University of Pennsylvania School of Medicine



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Testing the sense of smell is often omitted or trivialized during neurological examination. This comprehensive review addresses this shortcoming by emphasizing the significance of this important sensory modality. The Neurology of Olfaction describes the anatomy and physiology of human olfaction and how it may be measured. The book covers neurologic disorders in depth and a comprehensive chapter is devoted to neurodegenerative disorders, particularly Alzheimer's disease and Parkinson's disease, where loss of smell is frequent and may be an early preclinical feature that could predict the onset of disease in asymptomatic subjects. Finally, the authors describe methods of treatment for anosmia, evaluate its medicolegal importance, and give guidance for those unfortunate enough to have lost their sense of smell.

Written by two experts in the field, this book provides information useful to physicians for assessing and managing chemosensory disorders and summarizes the current scientific knowledge of human olfaction.

Inside you'll find:

- Comprehensive summary of human olfaction and its disorders – this is the only book you'll need.
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Upcoming Events

13-17 November 2010	**Society for Neuroscience** San Diego, California, USA www.sfn.org
2-4 December 2010	**AACSS Annual Scientific Meeting** Australasian Association for ChemoSensory Science Yarra Valley, Victoria, Australia www.aacss.org
2-4 December 2010	**Chemosensation 2010** Dresden, Germany www.tudresden.de/medkhno/reichen_schmecken/chemosensation.ttm
31 January – 3 February 2011	**Australian Neuroscience Society Annual Conference** Auckland, New Zealand www.ans.org.au
13-17 April 2011	**33rd AChemS** Tradewinds Resort, St Pete Beach Florida USA www.achems.org
3-5 May 2011	**ISOEN 2011** New York, USA www.olfactionsociety.org
4-8 July 2011	**20th International Clean Air and Environment Conference** Christchurch, New Zealand www.casanz.org.au
10-14 July 2011	**9th Pangborn Sensory Science Symposium** Bangkok, Thailand www.pangborn2011.com

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