



Chemo sense

Editorial

By Graham Bell
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"No-one ever died from smelling"

Too often, when monetary values are placed on injuries, for the purpose of compensation or legal redress for victims of accidents or crime, the chemical senses take a poor third place to vision and audition. I have heard the argument put, in the field of air pollution, to justify the relative unimportance of olfactory experience to a person, that "no-one ever died from smelling". Despite the flawed logic (people don't die from seeing or hearing either, but rather from what it is they are seeing, hearing, smelling, or tasting) society (courts, EPAs) all too often are swayed by it. Nevertheless, many a lethal substance has a distinctive smell, so to not smell it while breathing its associated molecules, could, clearly, prove fatal. This might happen

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A case of Dysosmia and Dysgeusia: A patient's nightmare

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Recently I saw a patient in my clinic that had the most impaired quality of life that I have encountered since my Taste and smell disorders clinic started in 1996.

Before I begin I would like to define the terms parosmia, phantosmia, parageusia and phantageusia as I use them.

Parosmia is a form of dysosmia that refers to a usually very unpleasant odor triggered by any or specific environmental odor. Phantosmia is a form of dysosmia that is usually unpleasant and occurs spontaneously without a trigger. Parageusia is a form of dysgeusia that is usually unpleasant and triggered by any or specific tastes. Phantageusia is a form of dysgeusia that is usually unpleasant and occurs spontaneously without a trigger.

INSIDE:

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Editorial continued

when smell is totally lost. But what happens when the chemical senses become only partly dysfunctional? Can the act of smelling and tasting become so unbalanced as to threaten one's life? The answer is given in our lead article by clinician Ronald Devere.

ChemoSensory Science is developing a greater appreciation of the value of the chemical senses to life and health. In a short review, Tom Scott brings us up to date on the literature supporting the brain mechanisms underlying the role of taste in matching our bodily biochemical needs to what chemicals the environment makes available. These mechanisms underpin our likes and dislikes for the taste of what we eat and drink, and why they change ■

A case of Dysosmia and Dysgeusia: A patient's nightmare continued

My patient is a 73 year old male who about a year ago developed parosmia that could be triggered by any odor. He said the triggered smell was like faeces and would last 5-10 minutes and recur frequently.

About the same time he developed parageusia whenever he put any food or drink in his mouth and began to chew. The taste he described was a "horrible" sour metallic taste. Over the next 3 months this horrible smell and taste continued to be more frequent and he lost 80 pounds. He required a feeding gastrostomy tube because he couldn't even sip water without getting these symptoms. All food and medications were given through his gastrostomy tube.

His past history revealed excellent health without any recent history of head trauma, sinusitis or upper respiratory tract infection. He was treated for long-standing hypertension taking Lisinopril.

He was extensively tested by his family physician and otorhinolaryngologist to include nasal endoscopy, MRI of the brain including medial temporal lobes, CAT scan of sinuses, Upper GI and standard lab tests including thyroid function, Vitamin B12 and folate level, sed rate, ANA, protein electrophoresis and zinc blood level. Every one of these tests were normal or nonspecific in results.

He went through a hip replacement surgery 3 month after his smell and taste problems. He had no appetite and lost all interest in eating. He became very depressed and didn't know how long he could live in this condition with a feeding tube and no enjoyment in life.

His general medical and neurological exam was completely normal, except he looked emaciated and was very depressed. His

mouth, tongue, gums and palate all looked normal and he had adequate saliva.

I tested his smell using the University of Pennsylvania Smell Identification Test. He scored 18/40 which placed him in the moderate microsmia range. He was administered the Taste Strip Test, which evaluates sweet, sour, bitter and salt at different concentrations. He scored 4/16 which is moderately abnormal. A normal score was 9/16. I was surprised to find that none of the odors in the UPSIT smell test or the tastes in the taste strip test triggered any bad tastes or odours. During my evaluation, I gave him samples of msg, spicy salts and chili powder to see if he could "taste" them. He actually thought they were "tasty" without triggering any of his symptoms. I believed he had moderate anosmia, parosmia, hypogeusia and parageusia of undetermined cause. I decided to try to treat his parosmia and parageusia which was potentially treatable and was the main reason for his inability to eat, depression, Peg tube and horrible quality of life. I decided to try everything I could think of to relieve his symptoms.

I put him on zinc gluconate tablets via gastrostomy tube 40mg TID to see if this could improve his parageusia as described by Heckmann et al 2005. He previously was on a short course of zinc sulphate early on in his disorder which was not effective. I am not sure from Heckman's study why they didn't choose zinc gluconate instead. To help his parosmia, I told him to put in 5-10cc of normal saline in a syringe and in the head down position gently drop this amount in each nostril. When he raises up he is not to sniff so that the saline will stay in the high nasal cavity to try to block any outside odor. He was to do this 4-5 times per day for a week to see if it helps. I also gave him a prescription of gabapentin to try

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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.

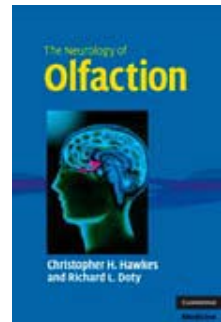
Doty systematically debunks the claims and conclusions of studies that purport to reveal the existence of mammalian pheromones. He demonstrates that there is no generally accepted scientific definition of what constitutes a mammalian pheromone and that attempts to divide stimuli and complex behaviors 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



\$89.00

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.
- Clinically oriented focuses on loss of smell in neurological disease, with illustrative case-histories.
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A case of Dysosmia and Dysgeusia: A patient's nightmare

continued

to reduce his bad smells and tastes. This has been successful in 6 of my past cases of dysosmia and 2 cases of dysgeusia. He started at 300mg at bedtime and over the next 4 days to increase the dose to three times a day by gastrostomy tube.

The patient called me one week later and he noted that his parosmia was decreasing (shorter duration and less intense and not triggered by all environmental smells) His parageusia was unchanged and he still required all feeding by tube. Three weeks later his parosmia was 90% gone and the parageusia was reduced by 50% (less intense and shorter duration). He was now taking his pills by mouth and was able to eat vegetables, some soups and fruit. Chicken or beef still triggered parageusia. I increased his neurontin to 1200mg per day. He also discovered that if he ate very spicy French fries before he ate his regular food his symptoms were much less. In the last 30 days he has gained 10 pounds and no longer has a feeding tube. His depression markedly improved. He is still on neurontin and zinc tablets. He no longer uses saline nasal drops.

DISCUSSION

This case was unusual in my experience because of the occurrence of parosmia and parageusia at the same time both contributing to weight loss, inability to eat and requiring the need of a feeding gastrostomy tube for survival. The exact cause is unclear.

Reviewing some of the literature on the subject of causes, natural history and treatment of dysosmia and dysgeusia, there are very few "large" studies. Most are case reports and many of the treatments are anecdotal. Bonfils, 2005 studied 56 patients with parosmia. The duration of their parosmia ranged from 3 months to

22yrs with an average of 55 months. All patients reported olfactory dysfunction. Seventy five percent had diminished smell and 25 percent had total smell loss. All cases described their parosmia as fowl, rotten, sewage or burnt smell. Eighteen percent of the patients were unable to identify an odor that triggered the parosmia. My patient did not have a trigger. Eighty two percent were able to identify the trigger which included gasoline (30%), tobacco (28%), coffee (28%), perfumes (22%), fruits (mainly citrus 15%) and chocolate (14%). 90% of the patients had trouble identifying flavors.

The causes of parosmia in this large series was upper respiratory tract infection(43%), chronic paranasal sinus disease (12%), head trauma(10%), toxic chemical exposure (7%),nasal surgery 2%),and idiopathic (26%). The temporal relationship between olfactory dysfunction and development of parosmia is not simple. In 57% of cases they occurred simultaneously. In the remainder 43%, parosmia developed after olfactory loss. This ranged from 3 months (34%), and longer than 3 months (9%). The mean time was 1.5 months after olfactory loss.

What Causes Parosmia?

There are 2 theories: Peripheral and Central. In the peripheral theory evidence suggests that abnormal olfactory neurons are unable to form a complete picture of the odorant. This goes along with the clinical feature in this study that all the parosmic patients have an intensity odor loss.

Leopold (2002) states that the peripheral theory is supported by the histology of the olfactory organ in individual patients which show decreased number of neurons, more immature than mature neurons and

distorted growth of olfactory axons.

For patients who develop immediate parosmia with olfactory loss, ephaptic transmission between disconnected axons and others that innervate the olfactory bulb might result in a distorted signal in response to an odorant.

A central theory of parosmia is still viable that states that the integrative or interpretive centers in the brain form parosmia. Leopold(2002) stated in his paper that the support for a central theory of parosmia development is that olfactory auras can accompany seizures and that excising the olfactory epithelium in some of his patients still leaves a feeling of the "bad" smell coming but never occurs.

The fact that gabapentin or other antiseizure medications can improve parosmia, and that they act peripherally and centrally, supports both of these theories.

Treatment of Dysosmia

Patients need to be reassured that their condition does not represent a progressive disorder and in time will eventually disappear. Since the majority of dysosmia patients have a smell loss, they need to be counseled about safety issues like smoke and carbon-monoxide detectors, not to eat open foods or those not date labeled, and have family members monitor their perfume and deodorant use.

There is no particular reference I could find about using normal saline in the nose for parosmia. Leopold (2002) mentions this in his article and states it is effective in 50% of his patients. I find a similar experience. The treatment is done by taking 10cc of normal saline and put it in each nostril in the head down position. After 20 seconds the person is to sit up and let the saline

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continued

block the nasal upper passage where the olfactory organ resides. This is recommended to do 3-4 times a day. Its main purpose is for the saline to block odors from coming in contact with the olfactory organ.

The use of anticonvulsants in dysosmia is mostly anecdotal without a published series. Dr Leopold mentions its use but does not describe any details. I have used gabapentin to treat 8 patients with dysosmia including this case. Six had parosmia and 2 had phantosmia. There was a 90% improvement in 5/6 parosmia patients and one with phantosmia with doses of 900mg to 2000mg gabapentin daily in three divided doses. I only use gabapentin in cases that do not or incompletely respond to the normal saline nasal drops. The majority received gabapentin for 6 months because when the dose was reduced the symptoms returned. Only 2 of my patients are completely off gabapentin without symptom recurrence, probably due to the spontaneous recovery of their symptoms. I have tried Zonisamide, another anticonvulsant in one case of parosmia at 100mg/day with 75% improvement. None of these patients had any significant side effects from these medications. It is important that the doses of each drug be increased slowly every week to get to the dose levels I mentioned. Because this case being reported was so severe, I elected to increase his dose much faster in less than a week.

Leopold 2002 described his first experience in 1988 of excising the olfactory epithelium by nasal endoscopy in intractable phantosmia. His patient recovered completely from dysosmia (phantosmia) and had some residual smell loss. He has

described 18 examples of this procedure in 10 cases over a 13 year period. His criteria for surgery was intractable phantosmia preferably in a unilateral nostril and eliminated temporarily preop with intranasal cocaine. All cases except one made a complete recovery from their phantosmia. The intent of the surgery was to cut all the olfactory axons and destroy all connections between the nasal cavity nerves and olfactory bulb. I am not clear why he only chose phantosmia cases and not parosmia. Despite this, I was contemplating this surgery for my patient if he didn't improve, although he may have still been plagued by his severe parosmia. Follow-up smell tests in his patients over 11 yrs showed no change in 5/10, improved in 2/10 and decreased in 3/10, compared to pre-operative level. Histological changes as previously mentioned in his cases showed peripheral nerve damage with large fascicles lacking neurons. The big puzzle in this treatment is why olfactory function returns after cutting all the olfactory nerves.

Treatment of Dysgeusia

You may be asking yourself if my patient really has dysgeusia. Couldn't putting food in his mouth, allowing the food molecules to travel retronasally to the olfactory organ, produce a very altered flavor? The patient told me that when food entered the mouth and just touched his tongue, he developed the parosmia and his taste testing was very abnormal leading me to believe he had primary parosmia.

In my review of dysgeusia, I couldn't find any reported large studies other than by Heckmann (2005).

In 116 of their cases of dysgeusia, 50 were idiopathic and the remainder were due to allergy of dental material, poor oral and

dental hygiene, poorly controlled diabetes, decreased saliva due to some medication or diseases of the salivary gland, low zinc, low thyroid and side effects from many medications (Doty 2008).

There are many anecdotal reports of treatments of dysgeusia suggesting improvement and being worth a try. I have used these treatments in some of my patients with varied success:

1. Cepacol Lozenges with Benzocaine. Have patient take them before meal time. May help parosmia.
2. Xylocaine, 0.5-1% mouth gel. Apply twice a day.
3. Gabapentin (Neurontin), Anticonvulsant: These category of medication likely work by altering or blocking abnormal electrical discharges arising from the peripherally damaged smell or taste organs as well as altered central brain connections. Begin 300mg at bedtime and increase slowly over 7-10 days to 900-1200 mg in divided doses. I have had success in 4 patients when one and two have failed. I believe this was successful in the current reported case.
4. Zonisamide (Zonegran): an anticonvulsant. (Start at 50mg in am daily and after one week increase to 100mg per day) Helpful in some of my cases of Dysosmia or Dysgeusia.
5. Zinc Gluconate 140mg/day, moderately effective. Improved taste, mood and dysgeusia in 50% of patients. (Heckman 2005).
6. Ice cube stimulation (Fujiyama 2010). Put one small ice cube in mouth for 1 minute just before meals. See below.
7. If insufficient saliva, try artificial saliva before each meal.

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8. Mirtazapine, 15mg at bedtime (Kalpana, 2006). See below.

Heckman (2005) randomized 50 patients with idiopathic dysgeusia to 140mg zinc gluconate and placebo. They rated response to a taste test and self rating the dysgeusia. No side effects were reported. No significant increase in zinc was found. This is probably because zinc is a trace element and is rapidly transferred into cells. Higher doses above 140mg/day has been known to cause anemia, leukopenia and GI symptoms (Salzman 2002). Zinc's value has been reported to help regenerate taste bud cells and influence the activity of carbonic anhydrase in saliva, which is important in breaking down foods in our mouth.

Fujiyama (2010) described an elderly patient who lost the ability to sense sweetness. Whenever she ate foods that were very sweet, she experienced a bad sour taste. Her taste test showed high threshold for saltiness. The author decided to put an ice cube in her mouth for one minute to lower the oral temperature by 5 degrees. They retested her taste capabilities and her saltiness recognition improved. She was told to place an ice cube in her mouth before every meal. After a month the patient reported to her physicians that she could recognize sweet again and lowered her threshold for all other tastants. There has been evidence in the literature that the gustatory nerve fibres are sensitive to temperature changes, due to action of thermosensitive ion channels. A thermosensitive channel, called TRPM5, is present in taste bud cells and can confer a steep temperature dependence on the processing of taste perception. Therefore, the authors say, the

recovery shown in this patient of her taste sensitivity may be caused by interaction between taste and cold signals. The patient reported complete recovery of her sweet taste after the cold treatment and previous sour taste (dysgeusia) was eliminated. The authors speculate that cold treatment may improve blood circulation in the tongue and taste sensitivity recovers. More studies are needed and this should be tried in some of our patients.

Kalpana et al (2006) reported a case of an elderly woman who developed otitis media. She was given an antibiotic, levofloxacin, 500mg a day. After 10 days she developed a spontaneous metallic taste. Her food tasted like bile causing loss of appetite and weight loss. This dysgeusia continued for 3 weeks after her antibiotic was stopped. She didn't report smell problems and smell was not tested. She had a long history of depression and was on fluoxetine for many years. A psychiatrist saw her and changed fluoxetine to Mirtazapine. The patient reported complete resolution of her dysgeusia in 4-5 days after starting Mirtazapine, which is a noradrenergic and specific serotonergic antidepressant. How and why it worked in this case is not clear. More studies need to be done.

Most of the treatments mentioned for dysosmia and dysgeusia have not been scientifically studied to show their benefits. However, the symptoms and impaired quality of life these disorders produce in our patients should prompt us to try these treatments singly or in combination. The majority are very safe and, like my patient, those successfully treated are very grateful ■

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Taste and Reward in Ventral Forebrain of the Rodent

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Taste is the mechanism by which the body's biochemical needs are matched to chemicals the environment offers. Animals inherit a taste system shaped by evolution to encourage consumption of nutrients and rejection of toxins. Naïve rats permitted brief exposures to a wide range of chemicals drink them with uncanny accuracy according to their nutritional value and lack of toxicity.

By what route is the connection established between gustatory appeal and nutritional adequacy?

Anatomical tracing studies from Norgren's lab identified the medial parabrachial nucleus (PBN) as an obligatory gustatory relay in rats (Norgren and Leonard, 1971), and subsequently traced PBN projections both to the thalamocortical axis and to ventral forebrain sites (Norgren, 1974, 1976). These included the lateral hypothalamus (LH), central nucleus of the amygdala (CNA) and bed nucleus of the stria terminalis (BNST), among others.

The functions with which these various targets had been associated, invited the inference that quality and intensity evaluations were made via the thalamocortical path, and hedonic judgments in ventral forebrain.

My purpose here is to review the degree to which studies in the intervening decades have affirmed this inference in ventral forebrain.

Ventral forebrain nuclei reciprocate the projections they receive from the PBN (Li et al., 2005). Their centrifugal fibers emerge from particular subsets of neurons, primarily in the central nucleus of the amygdala. The role of these projections is inhibitory, presumably to sharpen afferent activity from the PBN and to associate the gustatory code more specifically with the subset of neurons most tuned to that basic taste (Lundy and Norgren, 2004).

The sharpened signal that arrives to ventral forebrain brings with it a hedonic valence. The neurochemical index of reward in rodents is typically an elevated dopamine release in the nucleus accumbens shell. Such an elevation is seen upon gustatory stimulation with an appetitive chemical, particularly a sweet stimulus (Grigson and Hajnal, 2007). Had that sweet stimulus previously been paired with nausea to create a conditioned taste aversion, however, the dopamine increase would be blocked (Mark et al., 1991).

These dependent and independent variables can be reversed. Manipulations that reduce dopamine availability in accumbens blunt the rat's avidity for sucrose (Shimura et al., 2002); those that exaggerate normal dopamine levels increase avidity (Hajnal and Norgren, 2001). So nucleus accumbens dopamine levels are directly related to positive gustatory hedonics.

Taste and Reward

Ventral Forebrain of the Rodent

continued

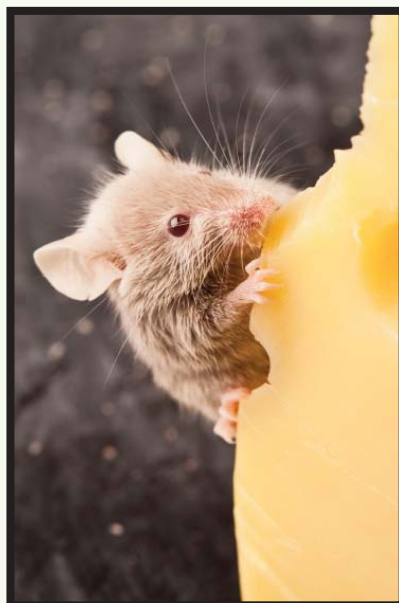
The DA release is mediated by PBN projections to ventral forebrain (Hajnal et al., 2004). Lesions that block this pathway prevent DA increases in response to sweet tastes; lesions to the pathway from PBN to thalamus do not (Norgren and Hajnal, 2005; Norgren et al., 2006).

Yet it is a measure of the PBN's sensitivity to both external (taste) and visceral signals that either is sufficient to drive the DA response in nucleus accumbens. Taste may be isolated from nutritional value by stimulating with non-nutritive sweeteners or by evacuating consumed sugar from the stomach through a gastric fistula. The accumbens DA increase persists in both cases (Hajnal et al., 2004).

Nutrition is isolated from taste by using TRPM5 knock-out mice which lack sweet receptors, yet nonetheless show the accumbens DA elevation to sweet stimuli. Only by eliminating both sensory and nutritional components by using non-nutritive sweeteners in knock-out mice is the DA elevation blocked.

There is a wealth of information from anatomical, electrophysiological, immunohistochemical, and microdialysis studies in the late 20th century that taste cells in the rodent's

hindbrain monitor both gustatory and visceral inputs. Their responses to gustatory stimuli are subject to modification based on the animal's history of conditioning and its physiological condition. Data from this decade demonstrate that rostral projections, especially from the PBN, are adequate to involve ventral forebrain areas associated with motivation and hedonics, empirically establishing the link that had long been assumed between taste and nutrition ■



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