FAX FROM SEOUL

by Graham Bell

We streaked down the freeway in our Daewoo sedan, like everything now in Korea, fast and optimistic. Having booked our hotel through the web - the site promising "special opening rates for a brand new hotel" - we were a little surprised to find a vintage hotel that has been having opening specials for forty years. Oh well, that's optimism for you, and obviously a translation error - "new brand hotel", perhaps. Nevertheless, I soon settled in to my ondol, a traditional Korean room, bedecked with mother-of-pearl inlaid furnishings, relaxed on my bed on the floor and dozed off to the strains of John Denver. My white knuckles restored, I found consolation in a round of "eating for Australia" as a diplomat friend once called it...a delightful series of Korean meals of every type. Besides the peculiar obsession of the Koreans (born out of surviving long winters), with ubiquitous fermented bak-choy (cabbage), garlic and chilli, this has to be one of the most eclectic food cultures in the world. Take for instance, the peach-flavoured carbonated milk. I did, hesitantly, and discovered it to be absolutely delicious. And I thought we could teach them about dairy products. Then there were the special restaurants serving a young clientele with just chicken, another with beef, another with pork. Live chickens, roaming free, greeted us on the way into the chook eatery. The food: great, so long as you like it spicy. A dozen shopping districts teem with young people with money to spend. And they do...all night long, in all-night megastores, ten floors high, like Migliore. Some close at 5 a.m. It feels as if Seoul cant wait for the new day to dawn, so it stays up all night. By the way, we have had panels of Korean consumers evaluate 27 Australian products, and we have interviewed 120 people on their food habits. We have also given 70 people the PROP test for supertaster status. So we are earning our keep, while we eat our way through this interesting and lively city. Everywhere the mood is very positive and the outlook is excellent for understanding, and trade between our countries.

Upcoming Events

2000

Sept. 30 – Oct. 6 — **Modern Analytical Methods in Food Microbiology.**
University of New South Wales, Sydney.
Contact:: Dr Julian Cox
E-mail:Julian.Cox@unsw.edu.au

Oct. 10-12 — **Foodtech/ Packtech**
Auckland, New Zealand
Tel: (649) 300 3950
Fax (649)379 3358

Oct. 24th — **Food Allergens Workshop** Issues and Solutions for the Food Industry. Stamford
Sydney Airport Hotel, Sydney.
Contact: the UNSW Dept. of Food Science and Technology
Tel: (02) 9385 5350
Fax: (02) 9313 6635

Oct. 20-31 — **CCR Fieldtrip to JAPAN**
Sensory Assessments of Australian products.
Contact: Marilyn Styles
Tel (02) 9209 4086
Email: m.styles@unsw.edu.au

Dec. 1 — **Australasian Association for Chemosensory Sciences (AACSS) Annual Meeting.**
Swinburne University, Melbourne.
E-mail: John.Prescott@stonebow.otago.ac.nz

2001 Mar. 31 — **Foodex Japan.**
Nippon Convention Centre, Tokyo
Information at www.jma.or.jp/FOODEX
Tel: (81 3) 3434 3453 or sales agent
Robyn Organ (02) 9700 1400

Jul. 22-26 — **Pangborn Sensory Science Symposium**
Dijon, France.
Abstract deadline 31 Jan. 2001
www.dijon.inra.fr/aromes/pangborn

COMING UP in ChemoSense

What is flavour?

Optimum school lunches

Role of fat

*** Visit us at our NEW WEBSITE**
http://www.chemosensory.com

ChemoSense (ISSN: 1442-9098) is produced and published quarterly by the Centre for ChemoSensory Research, University of New South Wales, Australian Technology Park, Sydney, Australia 1430
Production: This issue was edited and produced by Graham Bell, Carol Fleet and Hae-Jin Song
Subscriptions: Annual subscription A$52, postage inclusive.
Fax your order to The Centre for ChemoSensory Research (+61 2) 9209 4081
or email m.styles@unsw.edu.au
Advertising rates available upon request.

Vol.2 No.4 September 2000

ChemoSense

Olympic fever is upon us in Sydney. Soon the "roar of the grease-paint, the smell of the crowd" will be but a happy memory.

In the Olympian spirit, ChemoSense strives to be more pleasing and useful to you, in the world of smell, taste, pungency and chemical communication.

This ChemoSense brings you news of artificial sensing within liquids, which will make our environment and our foods cleaner and safer.

A mini-review by Laurence Dryer, shows how human variations in olfactory capabilities are largely, but not completely explicable in genetic terms.

Christine Broughan argues that cultural influences determine what we smell, and smell of. The constant across time is that people value odour highly and are influenced by it.

For the sensory practitioner, two articles may have special value: Paula Durham's on food product development and Graham Bell's on data snooping.

This issue also brings important news from AACSS, the international chemosensory community, and more ....

New Biosensors for Wine Analysis

by Manihar Situmorang and J.Justin Gooding

School of Chemistry, The University of New South Wales, Sydney, Australia
justin.gooding@unsw.edu.au

There is little doubt most of us enjoy a glass of wine. What we want from our favourite winery is a reasonable quality product, consistent with what is claimed on the label. To achieve consistent quality the wine maker needs to be able to monitor a variety of chemical analytes during the winemaking process: analytes which are indicative of certain flavours and of how far a particular vintage has travelled towards becoming a pleasurable dinner table accompaniment. To ensure the wine complies with a myriad of regulations pertaining to what can and can't be added, regulatory bodies also need to monitor a range of analytes, as has been highlighted by recent scandals related to tampering with wine by wineries. For both the wine maker and the wine regulator, it would be ideal if inexpensive analyses could be performed on site, where the sample is collected, by untrained personnel rather than incurring the time and cost of using an instrumental analytical laboratory. The invention of the biosensors makes this possible.

Biosensors are analytical devices which exploit the extraordinary specificity of certain biological molecules, called biorecognition molecules, for their target substrate. The most frequently used biorecognition molecules are enzymes, antibodies and more recently DNA. Peptides, whole cells and even pieces of plant tissue have also been used. In a biosensor the biological molecule is coupled with a signal transducer. The transducer relays information to the user about the extent of the reaction between the biomolecule and the target analyte. The signal transducer may be an electrode, an optical fibre or a microbalance.

The biosensor that many people may be familiar with is the glucose meter used by many diabetics. The test strips used by

cont. page 2

INSIDE:

Centre for ChemoSensory Research
The University of New South Wales
ISSN 1442 9098

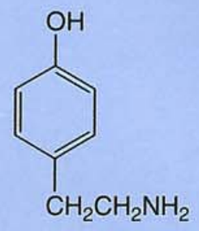
these glucose meters contains the enzyme glucose oxidase immobilised throughout a polymer layer deposited over an electrode. When a blood sample is placed onto the strip, the glucose oxidase reacts with glucose and in the process produces a molecule which can be oxidised at the electrode. Hence a diabetic can perform the analysis of a complex substance, blood, in their home without any particular training in analysis.

The adaptation of the generic principle of electrochemical enzyme biosensors for the detection of analytes in wine is of considerable interest [1]. At The University of New South Wales we are conducting a project for the wine industry, developing electrochemical enzyme biosensors for analytes of importance to the wine industry; particularly sulfite and malic acid.

Hurdles to the development of enzyme biosensors for wine

The success of the glucose meter has not however been translated to other analytes. Apart from the glucose meters there are very few commercially successful biosensing devices and none which monitor wine analytes. That is not to say that biosensors for monitoring wine analytes will not eventually fulfill their promise, just that certain problems need to be overcome. Stumbling blocks to the successful commercialisation of biosensors either relate to inherent properties of the biological molecule, such as stability and the concentration range over which the enzyme will respond to its substrate, or the fabrication process. With regard to biosensor fabrication the majority of the difficulties relate to poor reproducibility between devices and interferences of other chemical species in the wine, not with the enzymes but directly at the electrode. So it is prudent to ask why the UNSW approach will be any more different?

At UNSW a method of immobilising the enzymes over the electrodes has been developed to overcome these two main problems of poor reproducibility and interference. This approach relies on the electropolymerisation of tyramine directly on the surface of the transducing electrode. The enzyme is codeposited onto the electrode with the polymer. This serves to entrap the enzyme within the polymer film. It is still possible for the enzyme to leach out of this polymer film, resulting in a highly irreproducible device. The polymerization however does not affect the free amine on tyramine (see structure). This amine can be used as a linker to covalently attach the enzyme to the polymer backbone, thus overcoming the leaching problem. The real strength of this electropolymerisation approach is that the amount of polymer which is deposited on the electrode, and hence the amount of enzyme, can be precisely controlled. This is in contrast with other methods of depositing enzyme-modified polymers where the control over the thickness of the deposition layer is insufficient to ensure reproducibility between devices [2]. This problem of reproducibility being associated with difficulties in reliably depositing the enzyme layer are highlighted by the commercial glucose meters. In a recent New Zealand study, none of the 13 glucose meters available meet the performance criteria of the American Diabetes Association guidelines [3]. Relative standard deviations for commercial glucose meters can be 10% or more. In contrast enzyme biosensors fabricated using polytyramine, using glucose oxidase as the enzyme for comparison, had a relative standard deviation of less than 1% [4].



Tyramine

The polytyramine method is also effective at tackling the other main stumbling blocks for the commercialization of enzyme biosensors, namely interferences. Although the enzymes used are highly specific for their substrate, in complex samples other species can react directly at the electrode. Polytyramine membranes are effective at providing a barrier between the many interfering agents found in blood and wine, and the electrode [5]. The permeable selectivity of enzyme-polytyramine membrane is related to both size and charge exclusion properties of the polymer. The power of polytyramine-enzyme biosensors to operate in complex samples is demonstrated by a glucose oxidase-polytyramine biosensor operating directly in serum giving the same analytical result as the standard analytical method [4]. Such a result gave us confidence that enzyme biosensors made in this way could be used in wine samples. As a consequence we have been developing enzymes biosensors for monitoring both sulfite and malate in wine.

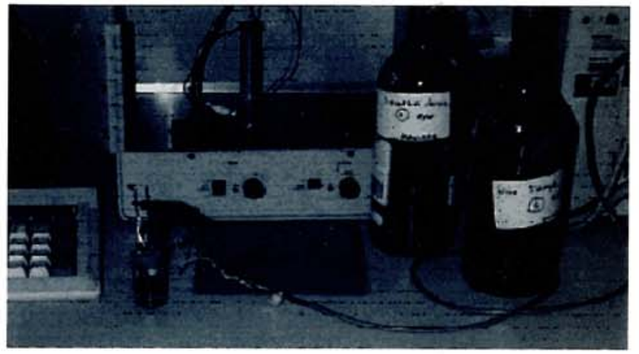
Sulfite biosensor

Sulfite is used extensively as a preservative in both wine and beer because of its ability to act as an antioxidant and as an inhibitor for enzymatic or microbial activity in wine products. The role of sulfite in food has been under legislation since it was known that sulfite at a certain level causes asthmatic attacks and allergic reactions in some individuals [6]. Although many analytical method have been introduced for sulfite, the Monier-William procedure followed by titration is still recommended as an official method for sulfite by the Association of Official Analytical Chemists (AOAC) [7]. Unfortunately this method is complicated, time consuming and insensitive.

The polytyramine based enzyme biosensor for sulfite uses the enzyme sulfite oxidase (SOD). The electrochemical sensing is based on the measurement of hydrogen peroxide, produced by enzymatic reaction (eq. 1), at the transducing electrode.

$$\text{Sulfite} + O_2 + H_2O \xrightarrow[\text{EC.1.8.3.1}]{\text{SOD}} \text{Sulfate} + H_2O_2 \qquad (1)$$

Vol. 2 No. 4 September 2000



Wine biosensor prototypes being tested

The current response for the injection of sulfite increased linearly in proportional to the concentration of sulfite. One of the difficulties experienced by previous work on sulfite biosensors was the direct oxidation of the sulfite and other interferences at the electrode. As with the glucose analysis in serum, the deposited enzyme-polytyramine membrane was effective in eliminating this direct oxidation. The biosensor has been applied for the determination of sulfite in various types of wine samples, and the results obtained are in agreement with that using AOAC method [8].

Malate biosensor

Malate is an important analyte in the winemaking process as its concentration plays a key role in the flavour and nose of a wine. Towards the end of the alcohol fermentation the malolactic fermentation (MLF) begins. In the malolactic fermentation the diprotic acid malic acid is converted to a monoprotic lactic acid. The result is a decrease in acidity of the wine and the loss of a musty smell. Winemakers like to exercise control over the MLF to dictate the level of malic acid and provide the wine with a particular flavour. Hence the need for a biosensor. As with sulfite, the official method of analysis of malate consist of long analytical procedures starting from the separation of malate from the wine before the polarimetric measurement been made [7]. In contrast the Analytical service of the Australian Wine and Research Institute a uses a spectroscopic enzyme assay [9] which also requires specialist equipment and trained personnel. The malate biosensor we have developed uses the enzyme malic dehydrogenase. This enzyme however, did not produce a product which could easily be oxidized or reduced at the electrode. Therefore an alternative approach based on detecting a pH change due to the enzyme reaction was employed, see reaction (2).

$$\text{L-Malate} + NAD^+ \xrightarrow[\text{EC.1.1.1.37}]{\text{MDH}} \text{Oxaloacetate} + NADH + H^+ \qquad (2)$$

The reaction of malate and the enzyme cofactor NAD^+, under the enzymatic catalysis of malic dehydrogenase (MDH), produces hydrogen ions which are detected potentiometrically at a tungsten electrode. The tungsten electrode serves simply as a solid state pH electrode. The beauty of this system is that the malate biosensor could use the same pH meter present in most wine laboratories. We are currently validating this malate pH electrode for the use in real wine samples.

The use of polytyramine for the fabrication of enzyme biosensors, is showing considerable promise for the analysis of wine. The electrodeposition of this polymer directly over an electrode surface provides enhanced reproducibility between devices compared with other methods of forming the sensing membranes. This excellent reproducibility and the biosensor's ability to operate in real samples without suffering from interference, means the wait for commercial biosensors for analysis of wine is not too far away.

Acknowledgement

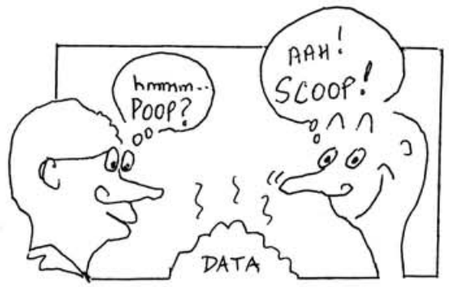
We would like to thank the Grape and Wine Research and Development Corporation for financial support and our colleagues at UNSW Professor D.B. Hibbert and Associate Professor Don Barnett with whom we have conducted this research.

References

1. M. D. Luque de Castro, J.L. Luque-Garcia, Anal. Lett. 33 (2000) 963.
2. Hall, E.A.H.; Gooding, J.J. and Hall, C.E., Mikrochimica Acta, 121 (1995) 119.
3. Henning, T.P. and Cunningham, D.D., (1998), Biosensors for Personal Managements in Commercial Biosensors (ed. G. Ramsay), Wiley, New York p. 3-46.
4. Situmorang, M.; Gooding, J.J. and Hibbert, D.B., Anal. Chim. Acta 394 (1999), 211.
5. Situmorang, M.; Gooding, J.J. and Hibbert, D.B., Electroanalysis 12 (2000), 111.
6. Allen, D. H. , Food Technol. Aust. 37 (1985), 506.
7. Association of Official Analytical Chemists: Official methods of analysis, 12th ed., Arlington, V.A. AOAC; 1975, pp. 406.
8. Situmorang, M.; Hibbert, D.B.; Gooding, J.J. and Barnett, D. Analyst 124 (1999), 177
9. Mollering, H., (1989), L-Malate: In Bergmeyer, H.U. (Ed.), Methods of Enzymatic Analysis, Vol. VII, VCH, Weinheim, p.39-47.

Poop or Scoop?
Are YOU a Data Snoop?

by Graham Bell, Centre for ChemoSensory Research, UNSW, Sydney. g.bell@unsw.edu.au



It's nice to get a quick result, but early peeping at the data, can lead to disaster.
There have been many celebrated announcements of great research discoveries, that were soon after retracted, never heard from again, failed to be replicated, or painfully debunked.. Recent cases you might remember include cold fusion and organisms on Mars.

Although there have been cases where dishonesty was involved (Piltdown Man, fossils in the Himalayas, and lots involving new drugs and wonder cures) many large bloopers have occurred through "premature ejaculation" of research data - i.e. someone getting too excited too quickly and messing up the solution!
These missed research targets are far more common than most realise or admit to. Most researchers can recall a "great" result that set them going on a number of experiments, seeking budgets and otherwise costly time and energy expenditure, only to never see the "great" effect again. Where did it go?

Well, it never existed in the first place. It was the result of a kind of hallucination brought on by the excitement of knowing you could be onto the big one. At the core of most of the problems that follow, is the premature "snoop" of the data. Your attention becomes selective, or what you look at may be a preliminary and inadequate data set. You may also have made inadequate assumptions in the experimental design, such as leaving out crucial controls or replicates, "to do later".

In applied and short-term contract research , the "snoop" can be very expensive. Companies do take gigantic risks and make huge commitments (by the standard of a research project budget) at the first hint of a trend in the data. So-called "top-lines" may be crude means with no statistical tests applied. No problem- spend the next budget item and let the boffins waste their time completing the stats. Result:- wrong decision, costly cover-up.

As universities become increasingly involved in contract research their more naiive researchers, who may previously have done a bit of "harmless" data snooping - usually labeled pilot experiments, will need to become aware of the dangers they may be exposed to. Beware, for example, of the man with the gold chains and a diamond in his tooth, who wants "a quick experiment to prove the principle". Chances are he has already nipped out and patented your idea - disclosed over a "free lunch" and you are about to supply the key "reduction to practice" (scoop) for him. He will never believe a null result (poop) if he can possibly help it. You will be enticed into more and more free lunch experiments until you go mad or decide that science is no longer for you. This is a tragic result of data snooping, but it is not the worst type. Turning poop into gold is another more nasty form.

Data snooping can cost millions (could be you)or earn millions (never you). Usually the bad guys benefit and the good guys lose. For example, "poop into gold" snooping is done inside a research project by a risk-averse manager or perhaps by a downright corporate crook, known as a "rug-puller". This new-age alchemist has a big vested interest in the direction a result takes. He snoops the data as it comes in, sample by sample, and if it doesn't look as if it is going his way, bang!, the rug is pulled and the project is stopped. It is then re-started (with fresh crew and after randomly applied punishment) and the process is repeated until the desired result is obtained. In the worst case, this data is used to defraud an unsuspecting bystander: an investor or client, say for 10 million or so.

Well, excuse me. I must rush off to see how the latest counts look on the machine that goes bing.

Vol.2 No.4 September 2000

Conference Launches Sensometrics Society

by John Best, CSIRO, Sydney

The 5th International Sensometrics Conference was held at the beautiful campus of the University of Missouri-Colombia, USA in July. About 170 attendees. People from a dozen countries gathered in airconditioned comfort to talk about the statistics of sensory science, while outside the tar bubbled.

Among the most interesting of the invited talks were those given by Jingla Tan and Ruben Gabriel. Tan spoke about using neural networks to relate taste test scores to underlying physical and chemical properties of foods. Gabriel, who is now retired, is well known as the inventor of the statistical biplot. He spoke enthusiastically about its applications, but food applications weren't mentioned. We know however, that the biplot is gaining utility in identifying complex product and attribute relationships in food sensory analysis.

Of the contributed papers two of the highlights were the presentations by Ms Giboreau explaining the detailed tactile sensory work which is the basis of the fabric choice for seating in Peugeot cars and Tom Carr's case study relating sensory data to product concept and consumer vocabulary.

The new Sensometric Society was launched at the meeting. A web-site is at www.dina.kvl.dk/SensometricSociety. Membership information can be obtained by emailing gbd@kvl.dk.

Food Product Development: the power and the pitfalls

by Paula Durham,
Concept Product Development, Melbourne
cpd@connect.net.au Ph: +61 3 9510 7250

Success, in many businesses, particularly the food industry, is measured by growth. Developing new products is a common strategy which will contribute to desirable growth.

However, many new products launched into the market place fail, and no company is so big that it can afford new-line failures. A planned and well-executed product development approach will help manufacturers and marketers reduce that risk of failure.

Good product development practice is a systematic and integrated process covering all activities from concept to launch. A products success requires corporate commitment and the integration of product development into business strategy. Success also depends on a coordinated team of specialists who are responsible for the quality and speed of execution of each step in the development process.

Where Do the Ideas Come From?

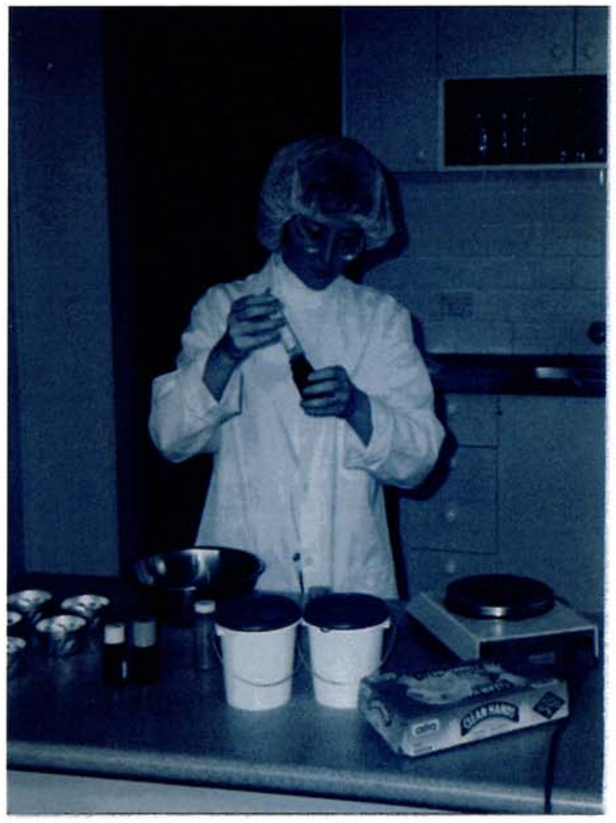
New product development opportunities are created by influences such as other cultures and cuisines, nutritional trends, and changing family routines. If a company fails to respond to these and other indicators, it risks existing products becoming obsolete. Continually updating products and taking up opportunities helps companies protect existing business and grow their share of the market.

To avoid reliance on one or two key lines, companies need to diversify. This means developing different products within the same category or through vertical integration, developing products which will work synergistically with existing lines. Additionally, if a company relies on seasonal lines, new products should be developed to smooth out the effects of the seasonality.

Successful new products may be developed by maximising the use of existing resources and / or reformulating and repacking existing lines to offer the consumer additional benefits. Additionally, companies may look to contract manufacturing to increase their diversity and complement their existing lines.

Early Investment is Repaid

The product development process starts with exploration, screening and business analysis. These three stages form the foundation and direction for development and should include consumer research. Larger companies tend to dedicate significant resources, internally and externally to these activities, while small businesses often rely on gut feeling. Ignoring these 'upfront investments' will increase the risk of abandoning the project or launching a product that fails – both financially devastating.



On completion of the 'upfronts', the target market, consumer needs, product concept and target launch date will be determined. Once a favourable financial analysis is established and the project objectives fit the overall business strategy the development phase can begin with a detailed product brief.

During the development phase a comprehensive technical evaluation is made and a starting formulation established, this may be based on products the company already manufactures or modified versions of selected formulations. When determining the initial formulation the developer must comply with the ANZFA Food Standards codes and pre-empt potential issues with the manufacturing process, shelf life, claims, product safety, quality assurance and availability and suitability of raw materials.

Samples Lead to Prototypes

From the initial formulation, samples are prepared and the formula adjusted using a scientific approach until the desired prototype is achieved. Once a suitable prototype is developed it is tested for consumer acceptance and shelf life performance. Depending on the outcome of this testing, further modification may be required.

The next critical phase is to conduct a pilot production run. Ideally it will be followed by production trials. How this is conducted largely depends on the individual company and available facilities. Where pilot facilities are not available it is commonplace to use a reduced-size production batch, and step this up to a full scale production batch. For reasons of time and budget a successful result in a small batch, it is often assumed that a full batch and subsequent batches will succeed, but this assumption is a dangerous one and often leads to disaster at the first commercial production.

The Decision to Proceed with a Prototype

How a product is 'approved' for launch varies greatly from company to company. Before final approval, numerous criteria should be checked including cost, taste, appearance, performance, shelf life and the overall project objectives. A vulnerable situation occurs when approval is given by either a small group or by one person. To approve a product in this way is a high risk as no individual has a 'golden palate'. A safer approval process is to conduct professionally supervised sensory evaluations. This may be done using some form of taste panelling either internally or by using an external consumer group. Sensory analysis reduces risks by identifying issues before packaging is printed, advertising and promotions are planned and the product is in store.

Packaging concepts and designs should run concurrently with product development, taking into consideration protection and presentation of the product. The labelling on the package must meet legal requirements and successfully attract the consumer's attention. It is not until product approval that the ingredients list, nutritional panel and any health claim can be confirmed, prior to production.

The final phase of development is commercialisation. This phase involves the co-ordination of the logistics of the first production run through to the distribution and delivery to market. Generally Commercialisation involves a contribution from finance, marketing, technical and production personnel.

Develop Today for Tomorrow's Market

The time required to develop a new product depends on the type of product, degree of technical difficulty and availability of resources. Most frequently, the development plan may be written to co-incide with a logical launch period or time frame.

Expediency is very important in developing and launching new products. Excessive time spent during each of product development phases can mean a competitor may launch a similar product first. Conversely, rushing and inadequately attending to detail risks launching a substandard product.

Methodically addressing and monitoring each of the phases of product development greatly improves the chance of launching a successful product. The cost of developing a product is comparatively low when compared with the increased profitability a company will achieve through a successful new product.

The Odorant Receptor Gene Repertoire in Humans

by Laurence Dryer, Department of Biology and Biochemistry, University of Houston, TX 77204-5513, USA ldryer@bayou.uh.edu

Introduction

Humans are supposed to be visual organisms. We are said to respond primarily to visual stimuli, and rely extensively on our visual system to navigate through our everyday sensory world. And indeed, compared to our visual capabilities, and compared to the olfactory capabilities of other species, our olfactory performance seems poor. Specifically, our powers of discrimination are lower, and our detection thresholds are higher. However, the extent to which we are aware of our exposure to specific olfactory stimuli in everyday life is almost certainly underestimated. Some individuals make a living of using their unusually discriminate sense of smell in the wine or fragrance industry. On the other hand, a certain percentage of the population is unable to detect some odorants at a concentration perceived by most people (Amoore, 1977). This phenomenon is called specific anosmia, a term reserved for pure odors, although we all have first-hand knowledge of the variability between individuals in terms of sensitivity to common (i.e., complex) odors in everyday life. So, are our comparatively poor olfactory abilities due to specific molecular features? Are those unusually discriminating "noses" endowed with more receptors for certain odors? Do anosmic individuals lack certain odorant receptors (ORs), or do they possess mutated receptors? And finally, are we all endowed with the same odorant receptor gene repertoire?

The OR gene repertoire in non-human species

The best characterized OR gene repertoire so far is that of the rodent. Mouse ORs are encoded by a large multigene family that belongs to the class of opsin-like G protein-coupled receptors (Buck and Axel, 1991). This family is characterized by high diversity in terms of gene sequences, but its members can be grouped into many subfamilies of genes with high (>80%) sequence similarity. Mouse OR genes are distributed on many chromosomes, arranged in clusters of various sizes on chromosomal domains, known as paralogous domains, that share common ancestry (Sullivan et al., 1996). The OR gene repertoire of several other species has been partially characterized, and their estimated sizes appear much smaller than that of the rodent repertoire (see Mombaerts, 1999a, and Dryer and Berghard, 1999 for reviews). In spite of their smaller size, the OR gene repertoires of lower vertebrates as well as some invertebrates exhibit an increased diversity in OR gene sequences. In other words, these repertoires comprise fewer genes, and those genes are less similar to each other (Clyne et al., 1999; Voshall et al., 1999; Berghard and Dryer, 1998). Furthermore, some vertebrate OR genes share very little similarity with rodent ORs and instead resemble neurotransmitter receptors (Berghard and Dryer, 1998). However, many of the OR gene families isolated so far are known to be incomplete. It is difficult to estimate OR gene repertoire size, especially in species with highly divergent ORs. Thus, at present, all we have are rough estimates.

The human OR gene repertoire

Human OR genes and gene clusters identified to date have been mapped to all but two chromosomes (for review see Mombaerts, 1999b). It appears that, as in rodents, at least some of these are the result of massive gene or gene cluster duplications within the human genome (Trask et al., 1998b). The human OR gene repertoire differs from that of the rodent in two important ways. First, the human repertoire is reportedly smaller than that of the rodent (Dryer and Buck, unpublished data). The reduced size of the repertoire seems intuitive, since we are reputed to be "poor smellers". However, one should keep in mind that repertoire size estimates are entirely methodology-dependent. So far, estimation methods have mostly consisted of screening genomic libraries or intact chromosomes with a probe or a mixture of probes obtained by PCR. This severely restricts the likelihood of finding highly divergent OR genes, as these methods rely on hybridization, i.e., the similarity between the probe and the target. This is comparable to estimating the total fish population in a pond from the "catch of the day", using only one type of bait. What of the fish that do not like that particular bait? Experience in lower vertebrates has shown that assuming sequence similarity when searching for new ORs leads to a biased and therefore incomplete representation of the OR gene repertoire (Berghard and Dryer, 1998). Therefore, the estimated number (about 500) of human OR genes may be inaccurate, and the average Homo sapiens could possess as many OR genes as the average mouse. Therefore, it may be premature to correlate our apparent poor sensory ability with a gross estimate of our OR gene repertoire.

Second, the human OR gene repertoire seems to comprise a rather large number of pseudogenes, i.e., genes that are not normally expressed (Rouquier et al, 1998; Ben-Arie et al., 1994; Glusman et al., 1997; Buettner et al., 1998). The presence of pseudogenes might seem natural in a species that has accumulated several large-scale duplications. Once a gene has been duplicated, the new copies are at first free of evolutionary pressure and can mutate. Some of these mutations will render the gene non-functional, i.e., a pseudogene. Unfortunately, it is difficult to confirm that these pseudogenes are indeed non-functional, because they often are isolated from genomic DNA instead of RNA, owing to the difficulty in obtaining human tissue samples. In other systems, certain gene modifications allow pseudogenes to be expressed (Mc Cormack et al., 1991). Therefore, it is possible that some human OR pseudogenes are expressed and contribute to individual olfactory sensitivity profiles (Crowe et al., 1996).

Interestingly, it was found that large numbers of human OR genes are located at the very tip of chromosomes, the telomere (Trask et al., 1998a). Telomeres are known to be hot spots for recombination that can lead to the creation of multiple new genes or copies of an existing gene. The phenomenon is so striking that these chromosomal regions are sometimes referred to as "gene nurseries". Nevertheless, not all clusters of ORs are located on telomeres, and some ORs, along with unrelated genes, seem to have been duplicated several times as part of large domains onto several different chromosomes.

Diversity of olfactory phenotypes

Besides the anecdotal knowledge of the various olfactory sensitivities in humans, psychophysical studies have characterized a number of olfactory sensitivity anomalies in the human population (Amoore, 1977; Amoore and Steinle, 1991). Most of these are specific anosmias. Depending on the odorant, 3% (isovaleric acid)to 47% (androstenone) of the population cannot detect a specific odorant at a concentration that other people can perceive. There is some evidence that this phenomenon has a genetic basis (Wysocki and Beauchamp, 1984; Gross-Isseroff et al., 1992; Whissell-Buechy and Amoore, 1973), and is unlikely to be the result of a defect in central processing of the sensory information. Thus, the odorant receptor repertoire becomes a prime candidate to be the site of the event(s) responsible for the sensory defects. Are anosmic people missing an OR gene or group of genes? There is at least one precedent for missing genes leading to perceptual defects. In the visual system, an array of genes encode for opsins, the proteins responsible for color vision. Certain visual defects (in particular red/green color blindness) are caused by the absence of some of these opsin genes, the result of a recombination event that deletes one or more opsin genes from the genome (Nathans et al., 1992; Nathans 1994). One could imagine the same phenomenon taking place in the olfactory genome. However, such drastic events require very specific conditions to take place. In particular, the recombination can only take place if the areas surrounding the gene to be deleted are highly similar to another genomic region with which they can recombine. Unfortunately, we have little information so far about these surrounding areas for the olfactory genome.

The epitope model and specific anosmia

In the rodent, ORs are distributed topographically in the olfactory epithelium, an arrangement thought to be functionally relevant for the encoding of sensory information (Ressler et al, 1993; Vassar et al., 1993; Koshimoto et al., 1994; Strotman et al., 1994). Each receptor is expressed in one zone only, but each zone contains many types of receptors that are not necessarily similar in sequence (i.e., that do not belong to the same subfamily). Furthermore, sensory neurons that express the same ORs send their axons to the same targets, a series of spherical structures called glomeruli, in the olfactory bulb (Ressler et al, 1994; Vassar et al, 1994). This segregation of input (in the epithelium) followed by convergence (in the bulb) is thought to be the basis for a complex combinatorial process by which odors are encoded. According to this model, each receptor recognizes an epitope, that is, a small molecular determinant within the odorant, rather than the entire odorant molecule itself. Thus, a given epitope can be present in several different odorants, allowing for the binding of multiple odorants to a given receptor (Fig.1). Note that this does not imply that the receptor binds to all epitopes with the same pharmacological affinity (see below).

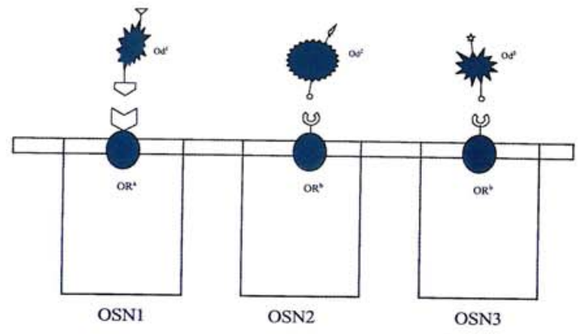


Fig.1: The epitope model for odorant recognition. Each OR recognizes one epitope that can be unique to an odorant (as in Od^1), or common to several odorants (as in Od^2 and Od^3). Odorants can carry several different epitopes. This allows multiple odorants to bind to a given OR, and a single odorant to bind to several ORs.

If the epitope model is true, then anosmic individuals should be missing an epitope-specific receptor in order to lose perception of an odor (Fig.2). However, anosmia is a relative concept, not an absolute one. For example, individuals who are anosmic to isovaleric acid can still perceive fishy smells, although they need the odorant to be presented at a much higher concentration than the rest of the population. Thus, sensitivity to the epitope is not completely lost. How could a partial loss of sensitivity result from complete deletion of a receptor? Studies in rodents (Krautwurst et al., 1998, Zhao et al., 1998) and in humans (Wetzel et al., 1999; Hatt et al., 1999) have shown that single receptors respond preferentially, but not exclusively, to one odorant. Mutational analysis in rodents has also shown that single amino-acid substitutions can cause a shift from one preferred odorant to another (Krautwurst et al, 1998). Therefore, it is possible that in the absence of an epitope-specific receptor, other receptors would still allow detection of the epitope-carrying odorant, but with a lower affinity. . Thus the individual would have a higher detection threshold for the odorant.

A second way to reconcile the epitope model with the existence of specific anosmia is to consider a genomic event less drastic than gene deletion, such as gene mutation (Fig.2). The opsin gene array is also a good example for gene mutations that can cause shifts in sensitivity to certain wavelengths, i.e., colors (Nathans, 1994). This gene polymorphism has generated a host of interindividual differences in light sensitivities in humans. Polymorphism in the human OR gene repertoire is harder to demonstrate, because the difference between a mutated gene and another normal member of its multigene family is sometimes tenuous. When does gene X ceases to be X or X' (its mutated version), and becomes gene Y, in a multigene family that comprises hundreds of similar members? Sensus stricto, gene X and Y are considered different when they are not located at the same locus on the genome. At present, the human OR genome map is incomplete, and we are often unable to rely on genomic location to discriminate one gene from another. One of the techniques that has provided preliminary evidence for polymorphism in the human OR gene repertoire relies on the conformation adopted by a DNA fragment depending on its nucleotide sequence. This method is called Single Strand Conformation Polymorphism (SSCP) and allows detection of single point mutations within OR gene sequences. Because SSCP allows comparison of alleles from multiple individuals in parallel, this method ensures that the same allele is being compared across individuals (instead of similar members of the same gene subfamily). SSCP has revealed several examples of OR gene polymorphism in humans (Dryer and Buck, unpublished data). Interestingly, most mutations resulting in amino-acid changes were located in the presumptive binding pocket of the receptor. Mutations located outside of the presumptive binding pocket tended to be neutral, that is, they didn't result in any significant protein change. The evolutionary implication is that there is a positive selection pressure that favors sequence diversity in the binding pocket of the OR to match the plethora of odorants available. The impact on individuals is that we might all be endowed with a unique set of OR genes, our own olfactory "fingerprint", which confers to each of us a specific profile of sensitivity to odorants.

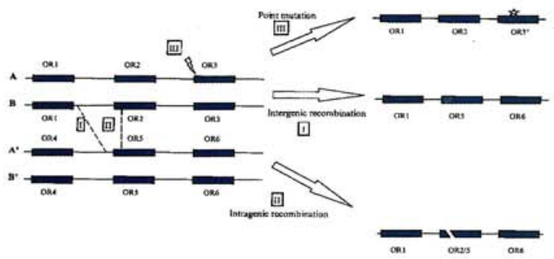


Fig.2: Possible genomic events that lead to polymorphism within the human OR gene repertoire. **I**: An intergenic recombination results from a cross-over that deletes one or several ORs (in this case, OR2 and OR3). **II**: An intragenic recombination results from a cross over that ultimately generates a hybrid OR (in this case, a hybrid of OR2 and OR5). **III**: A point mutation is a simple event that modifies an OR at a specific residue.

Human ORs and other adjacent genes

As human OR genes were mapped, it was found that some OR genes are linked to apparently unrelated genes. For example, some ORs map to the Major Histocompatibility Complex (MHC), a group of genes involved in immune responses (Fan et al., 1995, 1996). Genes in the MHC are the most polymorphic genes known in vertebrates (Apanius et al., 1997). This hypervariability is maintained by positive selection. Interestingly, the MHC loci are also involved in determining "odortypes", that is, the specific odor that an individual emanates through urine (mouse) or sweat (human) (Yamazaki et al., 1999a, 1999b; Apanius et al, 1997). How this is achieved is intriguing, since MHC genes encode proteins and peptides, which are not volatile compounds and therefore do not qualify as odorants per se. Nevertheless, some fascinating data have shown that the MHC influences several aspects of social interactions: female mice show a marked preference for mating with MHC-dissimilar males, which they recognize by olfactory cues (Brown et al., 1987; Apanius et al., 1997). In humans, females appear to find the scent of shirts worn by MHC-dissimilar males more preferable (Wedekind et al., 1995). Given the role of the MHC and the location of some ORs within the MHC loci, it is difficult to think of this as pure coincidence. Why are these OR genes in the MHC loci, and is there an interaction between both types of genes (Fig.3)? Studies in mouse have shown that it is the MHC genes themselves, and not the OR genes, that determine odortypes (Yamazaki et al., 1999). However, since individuals show a marked preference for individuals with an odortype different from their own, one could imagine that the type of MHC allele expressed in an individual could promote the expression of ORs located nearby within the genome. Thus, the expressed MHC allele could confer a specific olfactory sensitivity to an individual, allowing the individual to discriminate MHC-similar from MHC-dissimilar conspecifics. Conversely, the OR gene(s) could drive the expression of specific alleles in the MHC locus. Finally, it is possible that an OR gene could "utilize" its location within an extremely polymorphic locus to generate polymorphism within itself, and that there is no specific interaction between the two gene types. All three MHC loci are known to contain genes that have nothing to do with immune functions. It would be interesting to determine if these other genes are also unusually polymorphic, or if their function is somewhat correlated with some additional non-immune role of the MHC genes.

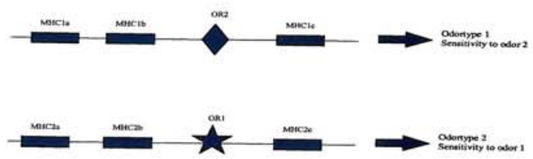


Fig.3: Illustration of the MHC/OR genes co-localization in the human genome. Top, individual is endowed with MHC1 and OR2 genes. The MHC genes confer the odortype 1 and sensitivity to odor 2 to the individual. Conversely, on the bottom, the individual is endowed with MHC2 genes, which confer the odortype 2 to him, but also with OR1, which confers sensitivity to odor 1. Both individuals are MHC-dissimilar and receptive to each other's odortypes.

Conclusion

At this time, ongoing mapping studies have not yet yielded an accurate size estimate of the human OR gene repertoire. However, it is becoming increasingly clear that this repertoire is highly polymorphic. Evolutionarily recent genomic events such as large-scale duplications, point mutations, and partial or complete deletions seem to have resulted in the current variability in the human OR gene complement. This is matched by an extreme diversity in human olfactory thresholds and preferences. Indeed, we might all be endowed with a different olfactory sensitivity profile. It would be unreasonable to attribute all human olfactory sensory ranges to genomic events, especially when considering the accumulation of pseudogenes within the human OR gene repertoire. Some higher brain processing must also account for some of the interindividual differences within the human population. Nevertheless, linking genotypic and phenotypic variabilities could shed some light on the function and specificity of individual receptors, as well as on specific odorant-evoked human behaviors. Therefore, it seems critical at this point to correlate psychophysical data with individual olfactory profiles.

REFERENCES:

- Amoore, J.E. (1977): Specific anosmia and the concept of primary odors. In *Chemical Senses and Flavor* **2**, pp. 267-281.
- *Amoore, J., & Steinle, S. (1991) A graphic history of specific anosmia. In* Chemical Senses, *(Wysocki, C.J., and Kare, M.R., eds.) Vol.3, pp. 331-351.*
- *Apanius, V., Penn, D., Slev, P.R., Ruff, L.R., & Potts, W.K. (1997): The Nature of selection on the major histocompatibility complex.* Critical reviews in Immunology ***17****, 179-224.*
- Ben-Arie, N., Lancet, D., Taylor, C., Khen, M., Walker, N., Ledbetter, D.H., Carrozzo, R., Patel, K., Sheer, D., Lehrach, H., & North, M.A. (1994): Olfactory receptor gene cluster on human chromosome 17: possible duplication of an ancestral receptor repertoire. *Hum. Mol. Genet.* **3**, 229-235.
- Berghard, A., & Dryer, L. (1998): A novel family of ancient vertebrate odorant receptors. *J. Neurobiol.* **37**, 383-392.
- Brown, R.E., Singh, P.B., & Roser, B. (1987): The major histocompatibility complex and chemosensory recognition of individuality in rats. *Physiol. Behav. 40*, 65.
- *Buck, L.B., & Axel, R. (1991): A novel multigene family may encode odorant ereceptors: a molecular basis for odor recognition.* Cell 65, *175-187.*
- *Buettner, J.A., Glusman, G., Ben-Arie, N., Ramos, P., Lancet, D., & Evans, G.A. (1998): Organization and evolution of olfactory receptor genes on human chromosome 11.* Genomics ***53****, 56-68.*
- Clyne, P.J., Warr, C.G., Freeman, M.R., Lessing, D., Kim, J., & Carlson, J.R. (1999): A novel family of divergent seven-transmembrane proteins: candidate odorant receptors in Drosophila. *Neuron* **22**, 327-338.
- Crowe, M.L., Perry, B.N., & Connerton, I.F. (1996): Olfactory receptor-encoding genes and pseudogenes are expressed in humans. *Gene* **169**, 247-249.
- Dryer, L., & Berghard, A. (1999): Odorant receptors: a plethora a G protein-coupled receptors. *T.I.P.S.* October (vol.20). In Press.
- Fan, W., Cai, W., Parimoo, S., Lennon, G.G., & Weissman, S.M. (1996): Identification of seven new human MHC class I region genes around the HLA-F locus. *Immunogenetics* **44**, 97-103.
- *Fan, W., Liu, Y.C., Parimoo, S., & Weissman S.M. (1995): Olfactory receptor-like genes are located in the human major histocompatibility complex.* Genomics ***27****, 119-123.*
- *Glusman, G., Clifton, S., Roe, B., & Lancet, D. (1996): Sequence analysis in the olfactory receptor gene cluster on human chromosome 17: recombinatorial events affecting receptor diversity.* Genomics ***37****, 147-160.*
- *Gross-Isseroff, R., Ophir, D., Bartana, A., Voet, H., & Lancet, D. (1992): Evidence for genetic determination in human twins of olfactory thresholds for a standard odorant.* Neurosci. Lett. ***141****, 115-118.*
- Hatt, H., Gissleman, G., & Wetzel, C.H. (1999): Cloning, functional expression and characterization of a human olfactory receptor. *Cell. Mol. Biol.* **45**, 285-291.
- Koshimoto, H., Katoh, K., Yoshihara, Y., Nemoto, Y., & Mori, K. (1994): Immunohistochemical demonstration of embryonic expression of an odor receptor protein and its zonal distribution in the rat olfactory epithelium. *Neurosci. Lett.* **169**, 73-76.
- Krautwurst, D., Yau, K.W., & Reed, R. (1998): Identification of ligands for olfactory receptors by functional expression of a receptor library. *Cell* **95**, 917-926.
- McCormack W.T., Tjoelker, L.W., & Thompson, C.B. (1991): Avian B-cell development: generation of an immunoglobulin repertoire by gene conversion. *Annu. Rev. Immunol.* **9**, 219-241.
- Mombaerts, P. (1999a): Molecular biology of odorant receptors in vertebrates. *Annu. Rev. Neurosci.* **22**, 487-509.
- *Mombaerts, P. (1999b): Odorant receptor genes in humans.* Curr. Opin. Genet. Dev. ***9****, 315-320.*
- *Nathans, J. (1994): In the eye of the beholder: visual pigments and inherited variation in human vision.* Cell ***78****, 357-360.*
- *Nathans, J., Merbs, S.L., Sung, C.H., Weitz, C.J., & Wang, Y. (1992): Molecular genetics of human visual pigments.* Annu. Rev. Genet. ***26****, 403-424.*
- Ressler, K.J., Sullivan, S.L., & Buck, L.B. (1993): A zonal organization of odorant receptor gene expression in the olfactory epithelium. *Cell* **73**, 597-609.
- Ressler, K.J., Sullivan, S.L., & Buck, L.B. (1994): Information coding in the olfactory system: evidence for a stereotyped and highly organized epitope map in the olfactory bulb. *Cell* **79**, 1245-1255.
- Rouquier, S., Taviaux, S., Trask, B.J., Brand-Arpon, V., van den Engh, G., Demaille, J., & Giorgi, D. (1998); Distribution of olfactory receptor genes in the human genome. *Nat. Genet.* **18**, 243-250.
- Sullivan, S.L., Adamson, M.C., Ressler, K.J., Kozak, C.A., & Buck, L.B. (1996): The chromosomal distribution of mouse odorant receptor genes. *Proc. Natl. Acad. Sci. USA* **93**, 884-888.
- Strotman, J., Wanner, I., Helfrich, T., Beck, A., & Breer, H. (1994): Rostro-caudal patterning of receptor expressing neurons in the rat nasal cavity. *Cell Tissue Res.* **278**, 11-20.
- Trask, B.J., Massa, H., Brand-Apron, V., Chan, K., Friedman, C., Nguyen, O.T., Eichler, E., van den Engh, G., Rouquier, S., Shisuya, H., & Giorgi, D. (1998b): Large multi-chromosomal duplications encompass many members of the olfactory receptor gene family in the human genome. *Hum. Mol. Genet.* **7**, 2007-2020.
- Trask, B.J., Friedman, C., Martin-Gallardo, A., Rowen, L., Akinbami, C., Blankenship, J., Collins, C., Giorgi, D., Iadonato, S., Johnson, F., Kuo, W.L., Massa, H., Morrish, T., Naylor, S., Nguyen, O.T., Rouquier, S., Smith, T., Wong, D.J., Youngbloom, J., & van den Engh, G. (1998a): Members of the olfactory receptor gene family are contained in large blocks of DNA duplicated polymorphically near the ends of human chromosomes. *Hum. Mol. Genet.* **7**, 13-26.
- Vassar, R., Chao, S.K., Sitchern, R., Nunez, J.M., Voshall, L.B., & Axel, R. (1994): Topographic organization of sensory projections to the olfactory bulb. *Cell* **79**, 981-991.
- Vassar, R., Ngai, J., & Axel, R. (1993): Spatial segregation of odorant receptor expression in the mammalian olfactory epithelium. *Cell* **74**, 309-318.
- Vosshall, L.B., Amrein, H., Morozov, P.S., Rzhetsky, A., & Axel, R. (1999): A spatial map of olfactory receptor expression in the drosophila antenna. *Cell* **96**, 725-736.
- Wedekind, C., Seebeck, T., Bettens, F., & Paepke, A.J. (1995): MHC-dependent mate preferences in humans. *Proc. R. Soc. Lond. B. Biol. Sci.* **260**, 245.
- Wetzel, C.H., Oles, M., Wellerdieck, C., Kuczkowiak, M., Gisselman, G., & Hatt, H. (1999): Specificity and sensitivity of a human olfactory receptor functionally expressed in human embryonic kidney 293 cells and Xenopus laevis oocytes. *J. Neurosci.* **19**, 7426-7433.
- Whissell-Buechy, D., & Amoore, J.E. (1973): Odour-blindness to musk: simple recessive inheritance. *Nature* **242**, 271-273.
- Wysocki, C.J., & Beauchamp, G.K. (1984): Ability to smell androstenone is genetically determined. *P.N.A.S.* **81**, 4899-4902.
- *Yamazaki, K., Singer, A., & Beauchamp, G. (1999a): Origin, functions and chemistry of H-2 regulated odorants.* Genetica ***104****, 235-240.*
- *Yamazaki, K., Beauchamp, G.K., Singer, A., Bard, J., & Boyse E.A. (1999b): Odortypes: their origin and composition.* Proc. Natl. Acad. Sci. ***96****, 1522-1525.*
- *Zhao, H., Ivic, L., Otaki, J.M., Hashimoto, M., Mikoshiba, K., & Firestein, S. (1998): Functional expression of a mammalian odorant receptor.* Science ***279****, 237-242.*

Odours in Culture: How our Olfactory World Changes with History and Fashion

by Christine Broughan, Coventry University, U.K.
c.broughan@coventry.ac.uk

"The persuasive power of an odour cannot be fended off, it enters into us like breath into our lungs, it fills us up, imbues us totally. There is no remedy."

Patrick Suskind. Perfume.

As Patrick Suskind so eloquently puts it, there is very little the majority of us can do, consciously or unconsciously, to prevent odours penetrating our sensory system, but how odours actually influence our behaviour is somewhat less clear. Physiological studies have outlined the mechanisms involved in the processing of such sensory stimuli and it has been found that there is reasonable agreement as to how olfactory signals move through the olfactory pathways. However, how odours are interpreted by the higher brain centres appears to be somewhat less clear and a number of theories regarding olfactory discrimination and detection have been put forward by theorists over the years. In this article we will review some evidence to suggest that response to odours can be influenced by an individual's cultural expectations.

Historical View

The influence of culture on our evaluation and response to odours is particularly evident when one takes an historical perspective. Even as far back as the first-century AD evidence exists of the cultural significance of odours, as Petronius wrote;

> Wines are out of fashion
> Mistresses are in
> Rose leaves are dated
> Now cinnamon's the thing!

Corbin's (1986) account of the way in which odours have been regarded over the centuries is a particularly readable text. He suggests that there was little individual response to malodours before the mid-18th century in modern Europe. These malodours formed part of daily life for all members of society - they formed a part of your life regardless of your class or status. In his novel 'Perfume', Patrick Suskind described Eighteenth-century Paris;

> "In the period of which we speak, there reigned in the cities a stench barely conceivable to us modern men and women. The streets stank of manure, the courtyards of urine, the stairwells stank of mouldering wood and rat droppings, the kitchens of spoiled cabbage and mutton fat; the unaired parlours stank of stale dust, the bedrooms of greasy sheets, damp featherbeds, and the pungently sweet aroma of chamber-pots. .... People stank of sweat and unwashed clothes; from their mouths came the stench of rotting teeth, from their bellies that of onions, and from their bodies, if they were no longer very young, came the stench of rancid cheese and sour milk and tumorous disease. .... The peasant stank as did the priest, the apprentice as did his master's wife, the whole of the aristocracy stank, even the King himself stank, stank like a rank lion, and the Queen like an old goat, summer and winter."
>
> Patrick Suskind, p. 3-4

During this time bathing was considered a foolhardy practice as it was believed that the best protection against disease was to smell strongly oneself. The lack of personal hygiene meant that pores were clogged which was believed to add a protective layer to ward off any disease. Furthermore, a final coating of musk and civet would not only protect the individual from disease but it would also increase their sexual attractiveness!

It was only when scientific discoveries associated infections with organic waste (Pringle, 1750) that malodours were regarded with intolerance. During the plague doctors wore large leather coats and nosegays which resembled a bird's beak or bill (hence the term 'quack' used to describe doctors) which they believed would protect them from breathing in the infectious air. This scientific discovery not only led to massive sanitary reforms and the use of fragrances to mask bodily odours, but it also provided a mechanism for the identification of social classes through smell. Under this interpretation, scientific advances initiated a cultural shift in the perception and significance of odours, leading to a society in which both body odour and the sexual significance of animal odours were suppressed. The use of powerful masking perfumes now cast doubt upon a person's cleanliness.

Towards the end of the 18th century, musk and civet were no longer popular. Masking one's own personal odour was to question one's own personal hygiene. Instead then odours such as rose, thyme, violet and rosemary were used to delicately fragrance to emphasis their own odour rather than to mask or obliterate it.

Thus a cultural division emerged between those who could afford sanitation, baths and fine fragrances and those who could not. It also led to odours being used as mechanisms for racial and social inequalities. George Orwell expressed it thus: "The real secret of class distinctions in the West....is summed up in four frightful words...The lower classes smell". In the same way that 'beauty is in the eye of the beholder', odours became more a matter of choice and preference. One could use odours as a cultural signifier of class and social taste.

> In the UK a Wigan teacher is reported to have had a boy sent home because of his smell. The boy's mother sent her this note "Dear Miss, our Johnny smells the same as his Dad and his Dad smells lovely. I should know, I've slept with him for 25 years. The trouble with you, Miss, is that you're an old maid and don't know what a proper man smells like."
>
> (Van Toller, 1986).

So, rather than odours becoming less and less important to us, it would appear that they still have a very important role to play in sensory evaluation. Hall (1969) suggested that people of different cultures "inhabit different sensory worlds". Howes (1991) suggests that the use of odours as cultural signifiers is greater in non-western societies. He suggests that the importance of odours in their cultural and historical context have been weakened in Western society as a result of the declining presence of smells in the wake of the perceptual revolution of the mid-eighteenth century. He further suggests that the representation of smell as more individual or 'private' than social, more animal than human, more biological than historical is a cultural representation, peculiar to the West, rather than an empirical reality. However this notion does not receive validation when one examines how much is spent on the production and elimination of odours in Western society each year. The focus of the odours that we use as cultural signifiers may have changed, but the importance of olfactory cues within our sensory world still appears to have strong support.

However, cultural differences in odours are still apparent in society today. Classen, Howes and Synnott (1994) use the example of the odour of cow manure to demonstrate these differences. Typically, in western Society, the odour of cow manure would have negative associations. However, among certain cattle-herding tribes in Africa it is perceived in a very positive way, often being associated with power and prestige.

Practical Implications

Will certain fragrances from the past return to popularity? Perhaps in ten years' time young people will again discover the beauty of sandalwood and pituli oil that their grandparents did in the 1970s. Food and fragrance companies might benefit from exploring such resurrections in olfactory experience. Will the "smellies" (movies with smell) achieve historical accuracy with the right odours for the period-setting of the movie...and what will they use in futuristic creations? Will fragranced and flavoured products be designed with greater sensitivity to the age and cultural background of the customer? The search for "global" designs for products is likely to prove futile as long as people and cultures keep evolving. Perhaps we should start a new museum to capture the fragrances and flavours of cultures in historical context, so that future generations may properly appreciate the past with all the sense at their disposal. Product designers could then use the olfactory archives to explore creative ways of reintroducing specific odours to the modern world.

References

Classen, C., Howes, D. & Synott, A. (1994). Aroma, the cultural history of smell. Routledge, London.

Corbin, A. (1986). The foul and the fragrant. Berger Publications: Leamington Spa.

Hall, E.T. (1969). *The Hidden Dimension.* Doubleday: New York.

Howes, D. (1991). *The varieties of sensory experience.* University Toronto Press: Toronto.

Suskind, P. (1991). *Perfume: The Story of a Murderer.* Washington Square Press: Washington

Synnott, A. (1991). A Sociology of Smell. Canadian Review of Sociology and Anthropology, 28(4), 437-459.

Van Toller, S. & Dodd, G.H. (eds.)(1988). Perfumery: the psychology and biology of fragrances. Chapman & Hall, London & New York.

Electronic Noses Turn Up in Brighton

Overview of the 7th International Symposium on Olfaction & Electronic Nose (ISOEN 2000: 20-24 July 2000)

by Bashan Naidoo,
University of Natal, Durban, South Africa

Who said e-noses were just a fringe topic of the chemical senses? Certainly no-one attending the combined meetings of the 7th International Symposium on Olfaction & Electronic Nose (ISOEN 2000) and the 13th International Symposium on Olfaction and Taste (ISOT 2000) in Brighton, UK in July. The event, attended by about 400 of the world's keenest chemosensory scientists created a splendid opportunity for the convergence of multi-disciplinary groups with a common interest in taste and smell.

ISOEN 2000 reflected the state of the art in electronic nose research and provided useful discussions between researchers, engineers, manufacturers and entrepreneurs. There were 50 oral presentations, 37 posters and 2 plenary lectures, mostly originating from Europe, with significant contributions from the USA and Russia.

From nose to tongue

Several new commercial electronic nose devices and technologies were presented. For example, a general-purpose hand-held electronic nose device, and a laboratory instrument that combines fingerprint mass spectrometry with traditional sensor arrays. New technologies included the development of electronic tongues for detecting dissolved organic compounds, calorimetric sensors and silica micro spheres coated in fluorescent indicators. The emergence of feasible electronic tongue technology creates exciting new possibilities in sensing applications. This is a field that will certainly receive more attention in future. In some respects commercial groups seemed to define the state of the art by developing advanced technologies. It was good to see commercial players deliver some useful research papers.

Data processing techniques also received significant attention. A discussion on the popular Principal Component Analysis technique exposed differences of opinion regarding its usefulness. A database of olfactory measurements is being compiled to facilitate the development and evaluation of data processing techniques.

What does the future hold? From and engineering perspective the field of chemical sensing is here to stay. The Institute of Electrical and Electronics Engineers (IEEE) is currently establishing an electronic nose committee and a sensor journal. The field is still in the open ended discovery phase with research efforts covering a broad spectrum. It is safe to predict a future convergence of research efforts along more focused paths. For the moment, two broad areas may be defined, sensor technology and data processing methodologies. Issues that are likely to be dealt with in the near future include electronic tongues, biomedical applications, data processing techniques and ongoing sensor development.

Vol.2 No.4 September 2000

IN THE NEWS

AACSS TO MEET, MELBOURNE, 1/12/00

The Australasian Association for ChemoSensory Science (AACSS) has nominated December 1st for the date of its next annual meeting to be hosted by Caroline Owen and John Patterson of the Sensory Neuroscience Laboratory at Swinburne University of Technology in Melbourne.

Abstracts are now called for and should be submitted to John.Prescott@stonebow.otago.ac.nz as email attachments, ideally in MS Word format.

AACSS JOINS ICOT AND GAINS ISOT IN 2008

At the recent International Symposium on Olfaction and Taste (ISOT) in Brighton, U.K., AACSS presented its credentials to the board of the International Commission on Olfaction and Taste (ICOT), under whose auspices the ISOT meeting are run. It was formally accepted, that AACSS be represented on this board along with the US, European and Japanese Chemical Sensory Associations. AACSS now stands in line to host an ISOT meeting in 2008, somewhere in Australasia. Start planning.