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Chemo sense

EDITORIAL

Mechanisms of taste perception

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A central concern for health is the part played by fat in what we eat. Fat tends to make food more delicious. Too much fat consumption has serious consequences for individuals. In the less-affluent world too little dietary fat is a concern, but cutting down on fat intake, while preserving enjoyment of food is a major concern in the developed world. Is dietary fat a hidden flavour booster, which is perceived only by its action on other tastants, or is it a taste in its own right, a sixth "basic taste", after sweet, sour, salty, bitter and umami? Russell Keast addresses this question and returns answers leaning toward, but not categorically in favour of fatty acids having a primary or basic

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Emerging evidence supporting fatty acid taste in humans

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The sense of taste presumably evolved to inform us about the nutritious or toxic value of potential foods. The primary organ responsible for the sense of taste is the tongue, which contains the biological machinery (taste receptors) to identify non-volatile chemicals in foods and non-foods we place in our mouth. Once a food enters the mouth, the tongue aids manipulation of the food, assisting breakdown and distribution throughout the mouth before swallowing the food. During this critical period of food manipulation the tongue is sampling chemicals in the food, and when food chemicals activate taste receptors, signals are sent from the taste receptors to processing regions of the brain. The signals are decoded by the brain and we perceive the taste of the food, which could be

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taste. The part played by fat in texture and mouthfeel is difficult to isolate in experiments on the taste of fatty acids. Fat eludes capture as a primary taste by virtue of its collaboration with so many players in the game of flavour. Nevertheless, creating fat substitutes, which allow foods to be as enjoyable as if they contained dietary fat, remains a major goal of food manufacturers. The science reviewed here makes a major contribution to attaining this goal, by understanding mechanisms of taste perception.

In John Prescott's new book, *Taste Matters*, reviewed in this Issue, the entire gamut of questions of why we like or are repulsed by what we eat and drink, is treated in flowing style that will enlighten and entertain most if not all readers ■

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one of five distinct qualities: sweet, sour, salty, bitter, and umami, each linked with a appetitive or aversive response, depending on the perceived intensity.

It seems appropriate to classify the sense of taste as a nutrient or toxin detection system, with the qualities (sweet etc..) informing us of suitability to swallow. For example: sweet elicited by sugars reflecting carbohydrate; sour elicited by free hydrogen ions (H+) reflecting acidity; umami elicited by glutamic and other amino acids reflecting protein content; salt elicited by sodium and other ions (Na+) reflecting mineral content, and bitter reflecting potential toxins in foods. Excessive bitterness or sourness are aversive and informs whether the food in the oral cavity may cause harm and the best action is to expectorate rather than swallow. Whereas the qualities sweet, umami and salty are all appetitive and inform us the food contains essential nutrients such as carbohydrate, protein and minerals respectively. In the paradigm of taste as a nutrient detection system that has been critical in species survival, it appears logical that fats, an essential energy-dense macronutrient required in limited amounts for energy and nutritional needs, would be detected through the sense of taste as other macronutrients namely carbohydrates and proteins are detected via the tastes of sweet and umami.

FAT OR FATTY ACID TASTE

Recently, it has been questioned as to whether or not fat or fatty acid should perhaps constitute an independent taste modality(Mattes 2009a). Similar to sweet and umami tastes, whereby the digestive products, sugars and amino acids respectively, are the stimuli required for interactions with cellular receptors, it would also be appropriate for fatty acids to be the chemical moiety that elicits fat taste. Fatty acids as taste stimuli are controversial as it is known that oxidized or reverted fatty acids, or fatty acids at high concentrations have an unpleasant flavour, primarily from activation of chemesthesis and the sense of smell, not taste. In this discussion it is important to note the perceived experience from any chemical stimuli will vary according to their concentration in a food as the perception of the taste runs along a sensory concentration continuum. At very low concentrations fatty acids may be detected, albeit with no taste quality attached, *i.e.*, the concentration is too low to be recognized as a taste (Keast, R & Roper 2007). As the concentration increases, e.g. as a result of fat hydrolysis within a food, fatty acids may then be detected, and if the hydrolysis has been extensive and the concentration of fatty acid in the food is high enough, the flavour will be unpleasant and act as a warning system against ingestion. The levels of fatty acids believed to activate the sense of taste are below the level considered unpleasant, yet sufficient to activate putative oral receptors. For example, the concentrations of fatty acids required for detection are within ranges which could be inherently present in edible fresh and processed foods (0.1–3% *w/v*)(Che Man, Moh & Van der Voort 1999). The level of fatty acid in food also helps answer the other main question of fatty acid taste in humans, whether lingual lipase is required for taste perception. While research has reported some oral lipase activity in humans, any activity reported is low and potentially from oral

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bacteria rather than secreted by humans (Stewart et al. 2010). Nevertheless, as the levels of naturally present fatty acids in foods are high enough to activate fatty acid taste, this becomes an academic rather than practical debate.

Whilst unconfirmed at this stage, the emerging evidence surrounding fatty acid taste meets many of the basic criteria required for taste classification, for example, fatty acids, both saturated and unsaturated appear to activate fatty acid specific receptors located on the apical portion of taste cells, where stimulation of these receptors induces cell depolarization and increases in intracellular $Ca2^{+}$ followed by the secretion of neurotransmitter (noradrenaline and serotonin) from the taste cell. Further supportive evidence comes from fatty acid receptor deletion experiments in rodents that reveal attenuated neural responses from the glossopharangeal and chorda tympani taste nerves specifically impairing fatty acid taste (Cartoni et al. 2010; Khan & Besnard 2009; Laugerette et al. 2005; Matsumura et al. 2007). What follows is a summary of evidence supporting fatty acid as the sixth taste.

ORAL FATTY ACID DETECTION MECHANISMS

Recent evidence has discovered a variety of putative fatty acid receptors on taste cells in the oral cavity.

Delayed rectifying potassium channels

Delayed rectifying potassium channels (DKR) are a group of slow opening and closing voltage-gated potassium channels. Research proposed that DRK played a role in the initial transduction of fatty acid taste (Gilbertson et al. 1997). Studies demonstrated the primary messaging function of fatty acids and site of action at the level of DRK channels (Honore et al. 1994). Fatty acids bind to the DRK, depolarizing the taste receptor cell resulting in the release of neurotransmitter onto the nerve fibre (Gilbertson et al. 1997).

G-coupled protein channels

G-coupled protein receptors (GPR) involve a large family of proteins that transduce extracellular stimuli into intracellular signals. Fatty acid activated G coupled proteins are distributed throughout the body where they play a role in diverse cellular processes related to fat signalling (Gilbertson, Yu & Shah 2010). GPR40 and GPR120 are the two receptors that respond specifically to medium and long chain fatty acids in vitro (Briscoe et al. 2003; Hirasawa et al. 2005).

A rodent study identified GPR40 as active at the back of the tongue and results of the study showed a decreased preference for linoleic acid and oleic acid in GPR40 knock out mice (Cartoni et al. 2010). Due to its specificity to FFA as exhibited in other organs in humans, GPR40 on taste cells may play a role in fatty acid taste in humans(Covington et al. 2006).

In contrast to the location of GPR40, GPR120 is found at the front and back of the tongue (Cartoni et al. 2010). In a recent study using both rodent and human models, results indicated that a mutation in the GPR120 coding sequence that inhibits signalling activity is associated with obesity (Ichimura et al. 2012).

CD36

CD36 is a protein that plays a role in fatty acid transport and was recently located in the taste cells of humans. CD36 knock out mice display a lack of preference for long chain fatty acids, but maintain a preference for sucrose and aversion to quinine suggesting that CD36 is lipid specific (Fushiki & Kawai 2005; Laugerette et al. 2005). The role of CD36 in human taste remains inconclusive, but the evidence from recent studies suggests that CD36 may act as a putative taste receptor for fatty acids and CD36 expression is present in circumvallate and foliate papillae in humans (Simons et al. 2011). Recent findings provide preliminary evidence of CD36 involvement in fat perception. The results of this study defined a CD36 genotype that was associated with an increased liking of added fats and oils and that a variant of CD36 was associated with adiposity (Keller et al. 2012).

Putative receptors for fatty acids appear to be predominantly expressed in type II (sensory receptor) taste cells, which also house the G-protein receptors and a number of taste-signalling molecules (α gustducin, PLCβ2, TRPM5), involved in the perception of sweet, bitter and umami taste (Roper 2007). Whilst still under investigation, the transduction mechanisms involved in the perception of fatty acid taste are believed to involve the binding of fatty acids to membrane bound receptors which initiate a cascade of intracellular events, leading to the secretion of $Ca2^{+}$ and / or the blockage of membrane bound K^{+} channels, followed by cell depolarization and the secretion of neurotransmitter onto afferent nerve fibres, which travel to the CNS **(Figure 1)**. The identification of numerous receptors / mechanisms which may facilitate fatty acid taste seems appropriate, given the diverse range fatty acids present within the food supply which may differ in chain length, saturation or configuration. The identification of multiple receptor

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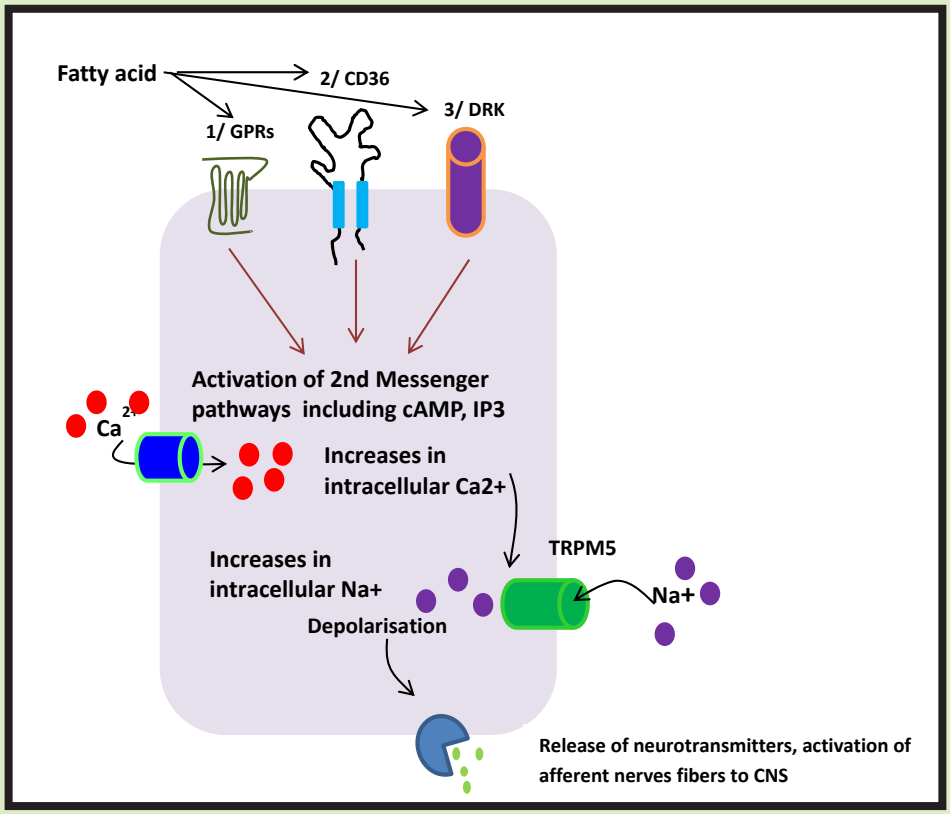

Figure 1: *Schematic representation of putative mechanisms of fatty acid taste transduction on taste cell. 1/ Membrane bound G Protein Receptors (GPR), specifically GPR120 (a receptor for unsaturated and saturated fatty acids with carbon chains between C14-C22) and GPR40 (a receptor for short, medium and long chain FFA, with carbon chains greater than six), are expressed in the circumvallate, foliate and fungiform papillae. 2/ Lipid receptor / transporter CD36 is expressed in taste cells of the circumvallate and foliate papillae, where it binds long chain (> 16 carbons) saturated and unsaturated fatty acids. 3/ Delayed rectifying potassium (DRK) embedded within the apical membrane of lingual taste cells of the fungiform, foliate and circumvallate papillae where long chain polyunsaturated and monounsaturated fatty acids (carbon chains >16 carbons) inhibit DRK channels.*

systems is not unique for fats, and also apparent within other taste modalities, *i.e.*, the T2R family account for 25 GPR bitter taste receptors (Chandrashekar et al. 2006).

PHYSIOLOGICAL EFFECTS FOLLOWING ORAL STIMULATION WITH FATTY ACIDS

Further evidence for fatty acid taste in humans comes from well-controlled studies that evaluate physiological responses to oral fat. For example, a 2.8-fold increase in plasma triglyceride concentrations in response to oral fat loads, or sham-feedings using fat, has been demonstrated (Mattes 2001a, 2001b). Additional investigations have also reported fat-specific cephalic phase responses following oral stimulation with fats that include increases in lipase secretion (Wojdemann et al. 1997), transient stimulation of GI hormones, including CCK, pancreatic polypeptide and PYY (Robertson et al. 2001; Wisen et al. 1992), as well as decreases in postprandial glucose (Robertson et al. 2001), although others have reported increases in postprandial glucose and insulin (Jauregui, Mattes & Parks 2010). These effects are not observed with sensory-matched fat mimetics, textural cues or smell (Mattes 2001a, 2001b), supporting the view that fatty acids activate putative taste receptors within the oral cavity that generate an immediate signal which is transmitted to other parts of the periphery, preparing the body for fat digestion and absorption.

While the evidence supports a form of oral fatty acid chemoreception, taste, in the traditional paradigm, must have a perception in the suprathreshold region, yet this appears to be missing as fatty acid taste is perceived by detection threshold only, with higher concentrations activating the sense of smell and chemesthesis rather than a perceived taste quality.

MEASUREMENT OF FATTY ACID TASTE FUNCTION

Taste function, typically determined via indirect measurements, such as detection, recognition or difference thresholds are common in the majority of taste research, with each different threshold indicating a limit in individual taste perception (Meilgaard, Civille & Carr 2007). Detection thresholds are considered the lowest amount of a tastant for it to be detected, for example the ability to detect that there is something in a sample other than water, but insufficient concentrations of tastant for its quality to be recognized, *i.e.* to recognise sweetness in the sample. A low detection threshold indicates high sensitivity to the tastant under investigation. As the concentration of a tastant in solution increases its quality will be readily detected, *i.e.*, individuals can identify that the sample tastes sweet and this is termed the recognition

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threshold. Concentrations above the recognition threshold are termed 'suprathreshold'.

In humans, well controlled psychophysical investigations have reported taste detection thresholds for un-oxidized saturated and unsaturated fatty acids (Chale-Rush, Burgess & Mattes 2007; Stewart et al. 2010; Stewart & Keast 2011; Stewart, Newman & Keast 2011). In these studies, antioxidants were used to reduce confounding from oxidation products of unsaturated fatty acids. Taste detection thresholds for long-chain fatty acids (oleic (C18:1), linoleic (C18:2) and linolenic (C18:0)), given as stable water emulsions, have been established, with mean thresholds reported as 0.022% (*w/v*), 0.032% (*w/v*), and 0.032% (*w/v*), respectively. Further studies reported taste detection thresholds for long-chain (C18:2: 0.007% (*w/v*) and stearic (C18:0): 0.04% (w/v)), medium-chain (lauric (C12:0): 0.06% (*w/v*)), and short-chain (caproic (C6:0): 0.004% (*w/v*)) fatty acids (Mattes 2009b). Data from our lab reported mean thresholds for C12:0, C18:1 and C18:2 suspended in non-fat milk at 0.062, 0.054 and 0.042% *w/v*, respectively, (Stewart et al. 2010). All these studies controlled for inputs from non-taste sensory cues including texture, specifically lubricity and viscosity, which are common textural mouth-feel cues used to identify the fat content of foods, through the use of gums and mineral oil, added to all samples at 5% *w/v*, which abolishes the difference in these attributes at up to 5% *w/v* fatty acid (Chale-Rush, Burgess & Mattes 2007). Odor through the use of nose clips, and visual cues by conducting all testing under red lights were also minimised (Chale-Rush, Burgess & Mattes 2007).

Additional studies have reported detection, although possibly not taste, thresholds following stimulation with fatty acids, however, these experiments have been less controlled. Nasser et al (Nasser et al. 2001) reported taste effects for conjugated linoleic acid at 0.05% (*w/v*), however, this study did not control for non-taste sensory inputs, such as odour and texture, rendering demonstration of a true 'taste' effect questionable. Kamphuis and colleagues (Kamphuis 2003) proposed the existence of a dichotomy of 'fat-tasters' and 'fat-non tasters' similar to the differences observed within other taste modalities, where some people are ageusic to certain bitter (Keller et al. 2002; Mela 1989) and umami tastants (Lugaz, Pillias & Faurion 2002), however, this study provided subjects with a single, low concentration of linoleic acid (0.00056% (*w/v*)), suggesting that apparent non-tasters may well have detected linoleic acid had they been offered higher concentrations. One aspect of all studies investigating fatty acid taste in humans has been the large individual variation in ability to identify fatty acids.

INDIVIDUAL DIFFERENCES IN FATTY ACID TASTE SENSITIVITY AND POTENTAIL FUNCTIONAL SIGNIFICANCE

Our ability to detect, recognise and rate intensity of various tastants is subject to change and variability. First, individuals differ in their absolute sensitivity (detection threshold) to a number of tastants, that is, the detection threshold for fatty acid will differ between individuals (Garcia-Bailo et al. 2009; Stewart et al. 2010). Second, even within a single taste modality, for example fatty acid, differences in the perceived intensity of different fatty acids C12:0 and C18:1 (Keast, RS, Bournazel & Breslin 2003; Stewart et al. 2010). Third, these differences may be due to genetic predispositions *i.e.*, taste receptor density and expression, differences in physiological state (hunger or satiety, health or disease), or environmental stress, for example changes in diet, health status or with age (Kim et al. 2004; Yensen 1954; Zverev 2004).

Fatty acid taste sensitivity has been defined as an individual's ability to detect fatty acids in solution. Subjects who can detect low concentrations of fatty acid in solution have been termed hypersensitive, while those who require significantly higher concentrations of fatty acid to reach detection threshold have been termed hyposensitive. In rodent models where oral fatty acid sensitivity was measured by electrophysiological activity of taste cells in response to fatty acids, it was found that fatty acid hypersensitive animals preferred less dietary fat and were more resistant to weight gain, while fatty acid hyposensitive animals preferred a diet high in fats and gained weight when exposed to a high fat diet (Gilbertson et al. 2005; Gilbertson et al. 1998). Studies from our laboratory investigated a similar link between oral fatty acid sensitivity, BMI and dietary fat intake and found that subjects who displayed greater sensitivity to oleic acid were lower consumers of dietary fat and had lower energy intakes (Stewart et al. 2010; Stewart, Newman & Keast 2011). It is also suggested that C18:1 hypersensitivity was linked to dietary behaviours such as trimming fat off meat and consuming low fat food options (Stewart, Newman & Keast 2011).

Sensitivity to fatty acids in both the oral cavity and GI tract appears associated, with impaired or attenuated response associated with overweight and obesity. Data from animal investigations provide strong evidence in support of this proposition, with impaired fatty acid

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chemoreception throughout the alimentary canal associated with overweight and obesity. Presumably, impaired fatty acid sensitivity promotes increased consumption of fat due to a reduced satiety response (Gilbertson et al. 2005; Gilbertson et al. 1998). Recent data from studies in humans also support this proposition; for example, individuals with impaired responses to fatty acids both in the oral cavity and the GI tract consumed greater amounts of dietary fat and had higher BMI (Stewart et al. 2010; Stewart et al. 2011).

Determinants of fatty acid taste sensitivity are unclear and it is not yet determined if diet affects taste or if taste affects diet, however the evidence suggests that habitual diet may influence fatty acid taste sensitivity. Rats fed high fat diets display a greater acceptance to fats and an increased capacity to absorb and oxidise fats indicating that exposure to high fat diets may alter mechanisms responsible for fatty acid detection and metabolism (Reed, Tordoff & Friedman 1991). Similarly the fatty acid detection threshold of healthy weight humans fed a high fat diet for 4 weeks was significantly decreased, meaning they were less sensitive to the taste of fatty acids, and this was reversed on a low fat diet (Stewart & Keast 2012).

SUMMARY

The emerging evidence in support of a sixth, fatty acid taste modality is compelling, with the majority of criteria required for a 'taste', well established, especially in animal models. For example, fatty acids have been identified as the chemical moiety responsible for fatty taste perception via interactions with fatty-acid specific receptors (GPR's and CD36) located within taste receptor cells, their interactions at these receptors appear to induce electrophysiological activity within taste receptor cells, and specific losses in fatty acid taste have been reported following the removal of such receptors. Whilst the evidence seems convincing, the complete removal of somatosensory (textural) cues is yet to be confirmed, and this will be required for ultimate verification of the existence of a separate fatty acid 'taste'. The evidence to date regarding fatty acid sensitivity and associations with fat intake and dietary behaviour appear consistent and intriguing. However, many gaps in our knowledge remain and further well-designed and controlled studies addressing determinants of oral and GI sensitivity to fatty acids, how these factors are associated with dietary behaviour, satiety and energy regulation, as well as the overall health implications, are warranted ■

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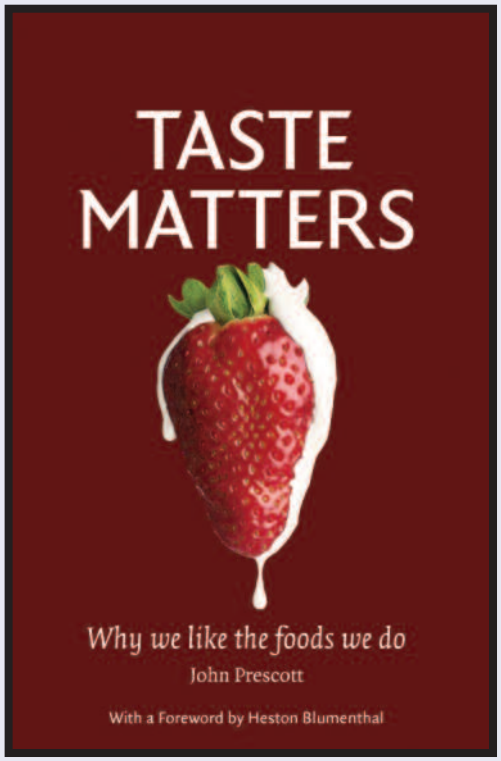
BOOK REVIEW *by Graham Bell*

Taste Matters



By John Prescott

www.taste-matters.org
Published by Reaktion Books, London
www.reaktionbooks.co.uk

This might be the first book on the scientific basis for understanding taste hedonics to have rated a foreword by a chef, and certainly the first to have gained the approval of world-famous personality and molecular gastronomist, Heston Blumenthal. Perhaps this is the book's great achievement: that it brings together science and the pleasure of eating, with the greatest of ease for the reader.

Prescott has had made a scientific contribution of nearly 25 years to food science, which reveal themselves in every page. Every thought and statement is backed by scholarship, experience and careful consideration. It is a book so well-crafted that it is the intellectual equivalent of several epicurean feasts.

Why do we like the foods we do? Why do some people relish morsels that others are disgusted by? How do such food preferences originate? What goes wrong when our food choices lead to extreme over- or under-weight: health problems that threaten our social relationships, our happiness and can shorten our lives? Twelve chapters, each sturdily referenced, expand on these questions and address a range of explanations and considerations.

Taste Matters is a book made available though its interesting and engaging content, its gentle writing style and reasonable price, to be accessible to the widest readership. It is highly recommended to all students of food, be it in science, technology, food service or creative food art ■

Upcoming Events

13-17 October 2012	**Neuroscience 2012** New Orleans USA www.sfn.org
3-7 February 2013	**Australian Neuroscience Society** Melbourne, Victoria, Australia www.ans.org/ans-annual-conference/
7-21 April 2013	**AChemS** Huntington Beach, California, USA www.achems.org

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