One day clinical neurologists will wake up to the fact that within the special senses of smell and taste lays an ocean of undiscovered information much of which will be relevant to our understanding of common neurological illness, especially of the degenerative variety. Inevitably the majority of writing on smell and taste is in the non-human domain and this book is no exception. Of the 24 contributors, I would estimate only a handful see patients, so should a clinician read this book? Unquestionably yes.

There is a very readable and authoritative chapter on human olfaction by Nancy Rawson. She deals with current theories on how individual odours are identified and how poorly the process is understood in the human. There is a useful review of olfactory measurement techniques and the nature of smell dysfunction in various diseases. Of major relevance to the neurologist is the virtual wipe-out of olfaction in Parkinson’s disease and the possibility that this abnormality might predate the motor features. I liked the chapter on human gustation by Paul Breslin. Of particular interest is the discovery of a new primary taste ‘umami’—the Japanese word meaning delicious or savoury. This is detected by glutamate receptors in the taste buds and responds to monosodium glutamate. It raises the fascinating possibility that there may be a link between deficiency of umami taste perception and a central lack of glutamate transmitter. Taste quality can be blocked by a variety of oral agents such as amiloride, chlorhexidine, gymnema or lactisole. Amiloride is a sodium channel blocker which alters the saltiness of sodium chloride. This fits in with the suspected receptor mechanism for sodium transduction in the taste buds at the tip of the tongue. Chlorhexidine, the antiseptic, reduces saltiness but the mechanism is not clarified. Gymnema tea reduces the sweetness of sugar and has been used for centuries as a folk remedy for diabetes in India. It also blocks the absorption of glucose from the gut. Lactisole also blocks sweet tastes but the mechanism is not clear for this or for gymnema. Bitter taste blockers are rare but a mixture of phosphatidic acid and lactoglobulin works on some compounds. Blockers of soursness and umami are not yet known. Of major interest is the autosomal recessive condition of taste-blindness to the bitter compounds phenylthiocarbamide and propylthiouracil which affects 30% of otherwise healthy, white people. This might open the way for identifying a gene coding for bitter taste. These observations clearly have implications for the palatability and safety of the food we eat.

On a more molecular front, the work on gene knockout mice is of particular interest and is nicely reviewed by Linda Barlow. The development of taste buds is controlled by brain-derived neurotrophic factor (BDNF) and their innervation by neurotrophin 3 (NT3). If BDNF is not expressed, there is a reduction in taste bud number and impaired taste perception. If NT3 is missing, there is severe loss of somatosensory innervation to the mouth and tongue. It is probable that a taste bud will not develop until it has a nerve supply, so these two growth factors are interdependent. Deficiency of BDNF may have relevance to familial dysautonomia (Riley–Day syndrome) where there is loss of taste and marked reduction of taste bud number. A third growth factor (essential, at least in rodents) mischievously known as sonic hedgehog is probably important for the development of taste buds and their pattern. So far no human disease equivalent is described.

The neural representation of taste is covered by David Smith and Barry Davis. A relatively new fact is that, in
There is a monosynaptic ipsilateral projection to the thalamus (VPMpc) from the nucleus of the solitary tract in the medulla. In non-primates the pathway has to relay in the pons. At cortical level the representation of taste is in the frontal operculum and insular regions with a projection to the orbitofrontal cortex and on to the limbic system. The cortical areas are therefore in close proximity to olfactory areas. Central gustatory neurones are typically broadly tuned in comparison with their peripheral counterparts.

There is a good review of the vomeronasal organ (VNO) and pheromones by Robert Johnston. While the existence of the vomeronasal organ in animals is undisputed, there is doubt regarding the human counterpart. In the developing human foetus there appears to be a VNO which stains for luteinizing hormone releasing hormone (LHRH), but its connections with the olfactory bulb disappear at about 19 weeks of age. In adults a pocket exists on either side of the nasal septum which is said to correspond to the VNO. Application of steroids to this region has been claimed to produce changes in mood, autonomic function and hormone levels but the results are disputed.

The chapter by Piali Sengupta and John Carlson on genetic models of chemoreception probably represents the leading edge of chemoreception research. They discuss the value of genetic manipulation in the fruit fly drosophila, the nematode Caenorhabditis elegans and the mouse. The complete genome sequence is known for C. elegans and it is hoped that reverse genetic techniques can determine gene function, particularly for the large family of guanylyl cyclases and olfactory receptors. Drosophila has several naturally occurring mutant strains which are insensitive to various smells or tastes. Research in this sphere could lead to a better understanding of their controlling genes and application to human chemoreception if their genes are conserved across species. Some mouse strains are anosmic to androstenone or isovaleric acid (sweaty odour). This appears to be caused by a lack of responsiveness of the olfactory receptor neurone in the presence of an otherwise normal olfactory system. The gene responsible for isovaleric anosmia in the mouse has been linked to chromosome 4. Mice rendered deficient in subunit 1 of the cyclic nucleotide-gated channel or to G-protein G-olf are deficient in all their olfactory responses, implying that odour transduction is mediated by a single pathway involving cAMP.

There are many other excellent review chapters covering animal and invertebrate chemoreception, olfactory transduction, coding and signalling which provide an ideal basis for updating ones knowledge of the world of chemoreception.

Christopher Hawkes
Essex Centre for Neurology and Neurosurgery,
Oldchurch Hospital,
Romford, UK