Neurobiology and Human/Animal Behaviour
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problem set #5

THE CHEMICAL SENSES
Relevant reading: Kandel & Schwartz, chapter 34.
S Korsching, 'Olfactory maps and odor images' (pp. 387-392); and JP Montmayeur, & H Matsunami, 'Receptors for bitter and sweet taste' (pp. 366-371); both in *Current Opinion in Neurobiology* **12**(4) (August 2002).

1. Each olfactory receptor molecule, and by extension each olfactory receptor, responds in varying degrees to many different odorants. How then is it possible to recognise so many distinct odours? What does this mechanism have in common with the coding schemes that you've seen in other sensory systems and in the motor system?

2. How does the representation of odours differ between olfactory bulb and pyriform cortex?

3. Patients who've suffered a blow to the back of the head will sometimes complain of anosmia, the inability to smell. What structures may have been damaged in such cases? How is it that these structures can be affected, when the blow is applied to the back of the head? How would the precise site of injury determine whether the anosmia would be permanent or temporary?

4. In the following passage from Marcel Proust's *Du côté de chez Swann* the narrator experiences vivid feelings and memories of his childhood cued by the aroma and taste of tea and madeleine cakes. Relate this phenomenon to what you know about projections from pyriform cortex to structures of the medial temporal lobe important in emotion and memory:

> Et comme dans ce jeu où les Japonais s'amusent à tremper dans un bol de porcelaine rempli d'eau, de petits morceaux de papier jusque-là indistincts qui, à peine y sont-ils plongés s'étirent, se contournent, se colorent, se diffirencient, deviennent des fleurs, des maisons, des personnages consistants et reconnaissables, de même maintenant toutes les fleurs de notre jardin et celles du parc de M. Swann, et les nymphéas de la Vivonne, et les bonnes

> gens du village et leurs petits logis et l'église et tout Combray et ses environs, tout cela qui prend forme et solidité, est sorti, ville et jardins, de ma tasse de thé.
>
> *translation:*
> And just as in the Japanese parlour game where one soaks in a porcelain bowl filled with water many bits of paper which until then are indistinct, but which as soon as they're immersed, stretch and take on shapes and colours, differentiating into flowers or houses or people, definite and recognisable, in the same way now all the flowers of our garden and those of M. Swann's park, and the water-lillies of the Vivonne, and and the good people of the town and their little dwellings and the church and all of Combray and its surroundings, taking on form and solidity, emerged, town and gardens alike, from my cup of tea.

5. In what way are sourness and saltiness simpler than sweetness, bitterness, and umami?

6. In what way do many Chinese take-aways exploit the mechanism of the umami sense?

7. What cranial nerve nucleus in the primary relay for taste? Is it located anteriorly, or posteriorly?

EMOTION

Relevant reading:

Kandel & Schwartz, chapter 48.

Joe LeDoux, 'Emotion Circuits in the Brain', *Annual Review of Neuroscience* **23**:155-184 (2000)

`http://neuro.annualreviews.org/cgi/content/full/23/1/155`

1. What is the somatic marker hypothesis, and how is it related to the earlier James-Lange theory of emotion perception?

2. On the attached midsagittal slice, identify the cingulate gyrus, the posterior limb of the fornix, the mammillary body, and the (general area of the) hypothalamus.

3. On the attached coronal slices, identify the hippocampus and the amygdala. Which of these two slices is the more anterior?

4. What hypothalamic nucleus is smaller in women and homosexual men than in heterosexual men?

5. What two fibre tracts convey the outputs of the amygdala, and to what structures do they project?

6. What experiments established the role of the central nucleus of the amygdala in fear conditioning?

7. What is Klüver-Bucy syndrome, and what lesion produces it? What structures are damaged by such a lesion? What functional aspects of the damaged structures might explain the hyper-orality of Klüver-Bucy syndrome? What disease processes or other conditions can produce Klüver-Bucy syndrome in humans?

NEUROMODULATION, AROUSAL, AND SLEEP

Relevant reading:

Kandel & Schwartz, chapters 51 & 52

EF Pace-Schott & JA Hobson, 'The neurobiology of sleep: genetics, cellular physiology, and subcortical networks', Nature Reviews Neuroscience **3**(8):591-605 (August 2002).

`http://www.nature.com/cgi-taf/DynaPage.taf?file=/nrn/journal/v3/n8/abs/nrn895_fs.html&dynoptions=doi1035414732`

1. Match the following neuromodulators, nuclei, and diseases: acetylcholine, norepinephrine, dopamine, serotonin, substantia nigra, nucleus basalis of Meynert, peribrachial pons, raphé nuclei, locus coeruleus, depression, obsessive-compulsive disorder, Parkinsonism, Alzheimer's disease. Locate each of the nuclei in a neuroanatomical atlas.

2. Serotonin selective reuptake blockers (SSRI's) – a class of drugs including fluoxetine (Prozac), fluvoxamine (Luvox), sertraline (Zoloft), and paroxetine (Paxil) – are so designated because they competitively bind to the serotonin membrane transporter and block it, while having very little affinity for catecholamine transporters. They thus prolong the activity of serotonin in the synapse, while having little or no direct effect on dopamine or norepinephrine. SSRI's are used to treat depression and obsessive-compulsive disorder. Although the pharmacologic effect of SSRI's is fully apparent within half an hour of oral administration, the therapeutic effect – in terms of decreased severity of depressive of obsessive-compulsive symptoms – takes two to four weeks to appear. How might this dissociation of pharmacologic and behavioural effects be explained in terms of interactions between modulatory neurotransmission systems?

3. What nucleus is a circadian clock for sleep? What anatomical properties and experiments support this contention?

4. How does the cellular neurophysiology of the thalamus contribute to the differing levels of cortical excitability in sleep and wakefulness? In what thalamic nucleus does this process occur?

5. From what physiological processes in what microanatomical structures does the scalp-recorded electroencephalogram arise, and how does its detectability depend on the laminar architecture of the cerebral cortex?

6. What is an 'evoked potential' or an 'event-related potential'?

7. Briefly explain the differences between time-domain and frequency-domain analyses of electroencephalographic signals.

8. How do the amplitude and the frequency composition of the electroencephalogram vary during sleep?

9. What is a sleep spindle?

10. What is a PGO spike? Might it have any functional relevance?

11. What early experiments demonstrated the existence of separate centres in the hindbrain for inducing sleep and maintaining wakefulness?

12. A lesion of what neuromodulatory nucleus abolishes sleep paralysis?

13. What is narcolepsy?

ESSAY

Compare and contrast the sensory maps that exist at various levels of subcortical and cortical processing in the various sensory modalities. You should explain the principles by which each of the maps that you discuss is organised, drawing together common elements and pointing out distinctions.