IN THE NEWS

Will you still love me in the morning?

On being accused of drunkenness by fellow politician Bessie Braddock, the British prime minister Winston Churchill is quoted as replying "And you are ugly, but at least I shall be sober in the morning". Clearly, Churchill had not succumbed to the 'beer goggles' effect; otherwise he might have viewed his colleague in a more favourable light.

Many of us are aware of beer goggles — the invisible optical aids that make members of the opposite sex appear more attractive after a few drinks — but until recently, the phenomenon had not been subjected to the rigours of scientific experiment. However, Barry Jones, a psychologist from the University of Glasgow, has set out to change all that ("Beauty is in the eye of the beerholder", *BBC News*, 18 August).

In his experiment, Jones showed students 120 photographs of men and women, and asked them to judge their attractiveness on a scale from one to seven — seven being the most attractive. He discovered that, on average, the students who had drunk a moderate amount of alcohol found the people 25% more attractive than did those in the sober control group.

Jones related his new findings back to one of his previous studies, saying "What we may have here is an explanation for why moderate doses of alcohol increase the chance of unprotected sex. It might be because the alcohol makes the person's partner seem more attractive" (*Scotland on Sunday*, 18 August).

Professor Geoff Palmer, from Heriot-Watt University's International Centre for Brewing and Distilling, had a more straightforward view: "This research confirms what many drinkers already know; the world seems a hell of a lot better after a couple of pints" (*Scotland on Sunday*).

Heather Wood

NEURODEGENERATION

Autophagy lurks around lurcher

Lurcher mice suffer from cerebellar degeneration owing to a mutation in the ionotropic glutamate receptor subunit δ2 (GluRδ2). This mutation leads to constitutive receptor activation, causing persistent cation entry into Purkinje cells. Ion influx is widely believed to cause neuronal death in lurcher mice, but Yue *et al.* now provide compelling evidence that autophagy is responsible for Purkinje cell death in these animals.

Autophagy is a process whereby cells digest their own cytoplasm during starvation. Morphologically, autophagy is characterized by the rearrangement of intracellular membranes and the formation of vacuoles that deliver cytoplasmic elements to lysosomes. Molecularly, autophagy involves proteins such as Apg6 in yeast. Beclin 1 is the vertebrate orthologue of Apg6, and Yue *et al.* found that this protein interacts with a new protein called nPIST, which in turn can bind GluRδ2.

Using a two-hybrid screen to identify binding partners of GluRδ2, the authors isolated nPIST and established that it is expressed in the cerebellum. Moreover, GluRδ2 and nPIST co-fractionated in cerebellar extracts and were enriched in the postsynaptic density. But because the function of nPIST is not known, Yue *et al.* performed a second screen to identify partners of this protein, isolating beclin 1 and subsequently showing that the three proteins form a complex in the cerebellum.

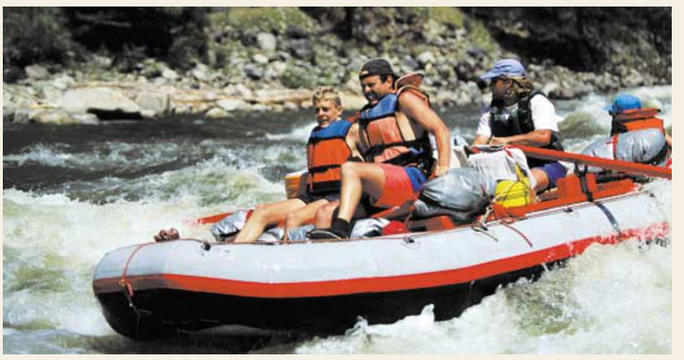
As beclin 1 participates in autophagy, Yue *et al.* explored the question of whether its interaction with nPIST and GluRδ2 modulates its role in the death process. They found that co-expression of nPIST and beclin 1 increased the number of autophagic cells compared with that observed when beclin 1 was expressed alone. Strikingly, this increase was found to be significantly larger if nPIST lacked the domain that interacts with GluRδ2. Similarly, co-expression of beclin 1 with the lurcher mutant, but not with wild-type GluRδ2, increased the number of autophagic cells. Last, the authors found that

CELL BIOLOGY OF THE NEURON

A raft of possibilities

Neurotransmission can be regulated at several levels, including the rate of neurotransmitter release, the availability of receptors on the postsynaptic membrane and the activities of downstream signalling pathways. In a paper published in the *Journal of Neuroscience*, Delling *et al.* now show that an adhesion molecule can regulate the lipid-raft-dependent trafficking of an ion channel, and this might provide a new mechanism for controlling synaptic transmission.

It was previously shown that, in mice deficient in the neural cell-adhesion molecule NCAM, the serotonin 5-HT_{1A} receptor becomes more responsive to certain agonists than that of wild-type littermates. However, there was no evidence that NCAM affects the expression or function of the receptor itself, so Delling *et al.* turned their attention to another component of the signalling pathway.



The signalling activity of the 5-HT_{1A} receptor is mediated through the inwardly rectifying potassium channel Kir3, and the authors showed that Kir3 currents were increased in the absence of NCAM. However, NCAM did not affect the overall expression levels of Kir3, nor did it seem to alter the conductance of individual channels. By transfecting NCAM and Kir3 RNA into *Xenopus* oocytes and neurons, Delling *et al.* showed that in the presence of NCAM, fewer of the Kir3 channels became localized to the cell membrane, indicating that this adhesion molecule blocks the transport of these channels to the cell surface.

So, how does NCAM prevent the channel from reaching its destination? An important clue came from the observation that both NCAM and Kir3 are associated with lipid rafts — cholesterol-rich lipid domains that are used to transport proteins around the cell and to organize signalling complexes on the membrane. NCAM can be modified by the lipid palmitate, and only the palmitoylated