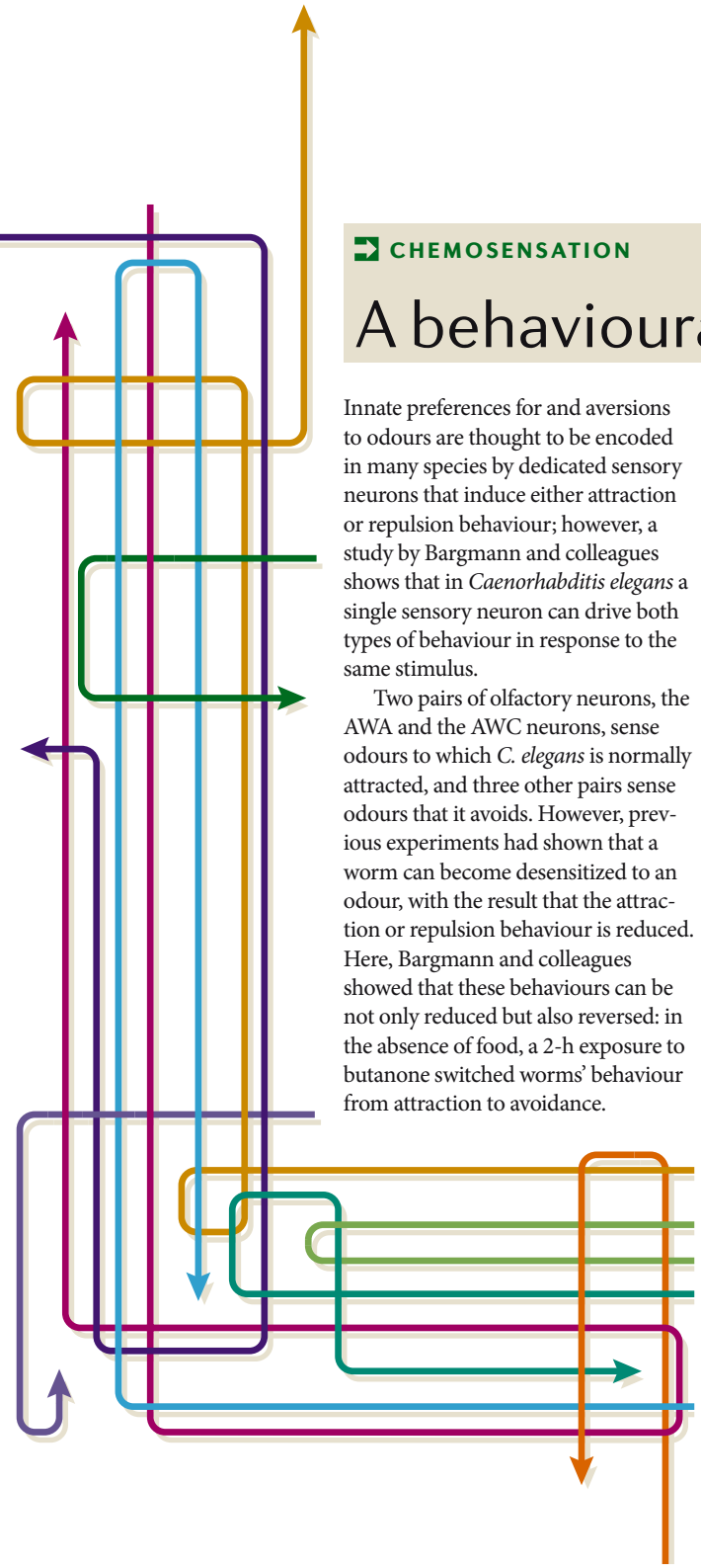

CHEMOSENSATION

A behavioural U-turn

Innate preferences for and aversions to odours are thought to be encoded in many species by dedicated sensory neurons that induce either attraction or repulsion behaviour; however, a study by Bargmann and colleagues shows that in *Caenorhabditis elegans* a single sensory neuron can drive both types of behaviour in response to the same stimulus.

Two pairs of olfactory neurons, the AWA and the AWC neurons, sense odours to which *C. elegans* is normally attracted, and three other pairs sense odours that it avoids. However, previous experiments had shown that a worm can become desensitized to an odour, with the result that the attraction or repulsion behaviour is reduced. Here, Bargmann and colleagues showed that these behaviours can be not only reduced but also reversed: in the absence of food, a 2-h exposure to butanone switched worms' behaviour from attraction to avoidance.

Looking for the mechanism that underlies this behavioural switch, the authors used a genetic screen to identify mutant worms that moved away from a butanone source instead of approaching it. They found worms that had a loss-of-function mutation in the gene that encodes a receptor-type guanylate cyclase (*gcy-28*). The gene is expressed in the AWC^{ON} neuron (the olfactory neuron that senses butanone), and an intact AWC^{ON} neuron was required for the mutation's behavioural effect. The *gcy-28* mutants also avoided other odours that are sensed by the AWC^{ON} neuron, indicating that the mutation causes a systematic change in this neuron.

Wild-type and *gcy-28*-mutant worms exhibited similar AWC^{ON} neuronal activity (as measured by changes in Ca^{2+} levels) in response to a sudden reduction in butanone, indicating that the mutation does not affect activation of the olfactory neuron, the first step in the sensory transduction process. As one of the three *gcy-28* splice variants is expressed mostly in axonal processes, the authors wondered whether the mutation might change synaptic transmission between the AWC^{ON} neuron and its downstream interneurons.

To investigate this possibility, they manipulated levels of diacyl glycerol (DAG) in worms, as alterations in DAG signalling are known to affect neurotransmission. They found that increasing DAG signalling in *gcy-28* mutants normalized their behavioural response to butanone. Worms with a mutation in *pkc-1*, the gene for protein kinase C (a target of DAG signalling), showed the same behavioural switch as *gcy-28* mutants. Experiments with double mutants revealed that mutations in *pkc-1* and *gcy-28* had no additive effect, indicating that GCY-28 and PKC act in the same pathway. Indeed, expressing a constitutively active form of PKC in the AWC^{ON} neuron of *gcy-28* mutants rescued the behavioural abnormality. Together, these findings indicate that reduced DAG signalling leading to decreased PKC activation in the AWC^{ON} neuron might be a mechanism by which the *gcy-28* mutation causes butanone avoidance.

Future experiments might reveal whether a similar mechanism underlies the switch in behavioural response after odour conditioning. Regardless, these findings suggest that, in *C. elegans*, stimulation of a single sensory neuron by one odour can have opposing behavioural effects through changes in intracellular signalling.

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ORIGINAL RESEARCH PAPER Tsunozaki, M., *et al.* A behavioral switch: cGMP and PKC signaling in olfactory neurons reverses odor preference in *C. elegans*. *Neuron* **59**, 959–971 (2008)