Moth response to climate

SIR — CO_2-sensitive receptor neurons[1] in the labial palp organ[2] of the moth *Helicoverpa armigera*, a major agricultural pest, can detect small fluctuations in CO_2 concentration associated with the metabolic activity of food plants with a sensitivity similar to that of modern technical detectors[3]. As other receptor neurons in insects[4–6] are strongly temperature sensitive, we would expect that temperature fluctuations, common within the microenvironment of insects[7], interfere with the detection of CO_2. Instead, we find that the CO_2-receptor neurons in *Helicoverpa* are temperature compensated, albeit only at the CO_2-background levels characteristic of the pre-industrial world.

When a single receptor neuron is exposed to modulation of CO_2 concentration by a constant percentage for a range of backgrounds (Fig. 1*a*), its response is modulated with a small phase lead[3] in synchrony with the stimulus. Both mean action potential rate and response-modulation depth increase monotonically with background. When modulation of temperature is applied instead (Fig. 1*b*), a different picture emerges: at 100 p.p.m., the response is in phase with the corresponding CO_2 response; at 600 p.p.m. it is of opposite phase. At 250 and 300 p.p.m., the responses are of the same phases as at 100 and 600 p.p.m., respectively, but are much smaller. This indicates that in this particular neuron exact temperature compensation occurred at a single background, between 250 and 300 p.p.m. The present background (350 p.p.m.) is above the compensation point.

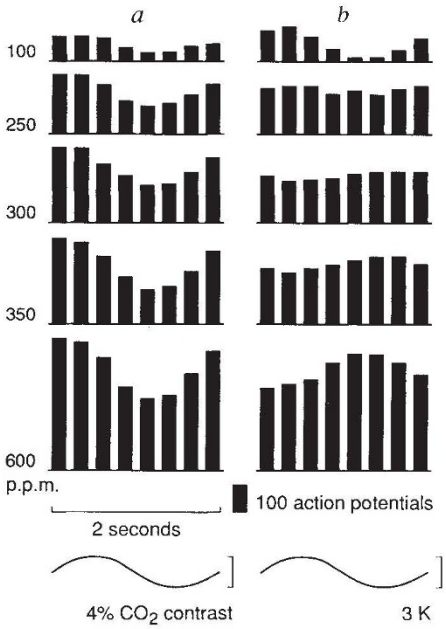

FIG. 1 Pairs of period histograms of action potential probability of a single CO_2-receptor neuron of *Helicoverpa*, logged continuously for 64 periods of a sinusoidal stimulus, in response to modulation of *a*, CO_2 contrast and *b*, temperature, for a range of CO_2 backgrounds (100–600 p.p.m.), at a mean temperature of 298 K. Although the receptor neurons are maximally sensitive at 4 Hz (ref. 3), a stimulus frequency of 0.5 Hz was used because of instrumentation constraints. Measurements conducted at 0.25 Hz gave identical results, indicating independence of stimulus frequency.

For small signals, the responses are proportional to stimulus contrast[3], allowing direct comparison within pairs of measurements at a given background. This enables us to express temperature sensitivity in terms of equivalent CO_2 sensitivity. For example, at 600 p.p.m. (Fig. 1), a CO_2 contrast of 4% causes a response contrast of 59% (gain factor $G_{CO_2} = 14.7$), while 3 K (1% temperature contrast) causes 34% response contrast with opposite phase (gain factor $G_T = -34$). We define the ratio G_T/G_{CO_2} as the temperature sensitivity S, which becomes independent of incidental and often nonlinear properties of individual neurons, such as threshold, adaptation and logarithmic compression.

Figure 2 shows that, with increasing background, S initially decreases steeply from a positive value at low concentrations and becomes negative at higher concentrations, with a decrease in slope. The data closely match the function

$$S = (S_\infty c + c_0)/c$$

where c is the background concentration and $S_\infty = -4.8$, $c_0 = 1{,}190$ p.p.m. Therefore, the temperature sensitivity can be modelled as the sum of two terms with opposing signs. Sign and magnitude of the first, background-dependent, term are consistent with the notion that CO_2 must dissolve in an aqueous medium before reaching the molecular sites of sensory transduction within cell membranes. The solubility of CO_2 in water decreases with temperature, by $-2.8\%\ K^{-1}$ at 300 K. Therefore, if the membrane sites are sensitive to the concentration of CO_2 in the aqueous phase, a value of $S_\infty = -8.4$ ($-2.8\%/0.33\%$) is expected, which is within a factor of two of observation. The positive sign of the second, background-independent, term is consistent with observations on other arthropod receptor neurons where temperature sensitivity is determined by the generation of receptor currents[8]. It appears, therefore, that the occurrence of two opposing effects is an inevitable consequence of basic properties of the chemistry of CO_2 and of the physiology of receptor neurons.

We observe that $S = 0$ at a single background, or within a narrow range of backgrounds if we allow for variability between individual receptor neurons. For

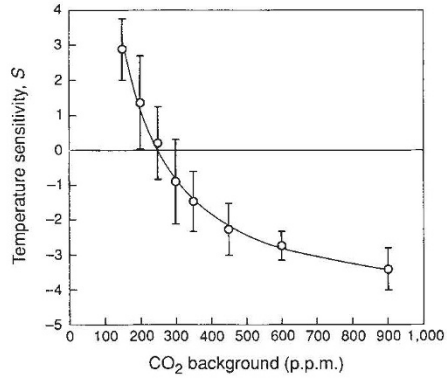

FIG. 2 S as a function of CO_2 background. Circles, means of seven single-cell recordings from different individuals of *Helicoverpa*; error bars, standard deviations; curved line, least-squares fit of the function $S = S_\infty + c_0/c$.

the dataset in Fig. 2 and incomplete datasets from *Helicoverpa* and three other lepidopteran species ($n = 19$), all zero crossings occurred within the range 190–320 p.p.m. The present atmospheric CO_2 background is 350 p.p.m.; it was 300 p.p.m. in 1925 and 280 p.p.m. immediately before the industrial revolution. Fossil records from ice-core samples[9] indicate values of 270–250 p.p.m. between 2,000 and 15,000 years ago; over the 150,000 years before that, the value fluctuated between 190 and 290 p.p.m. Therefore, the moth CO_2-receptor neurons are better adapted to the background levels that prevailed in the recent past than to present levels.

It appears that the unprecedented rate of change in background caused by anthropogenic emissions exceeds the rate at which the moths can genetically adapt. As a result, further increases in CO_2 background will progressively drive the sensory system out of the temperature-compensated range, causing it to confuse fluctuations of temperature with fluctuations of its adequate stimulus, CO_2. There are serious implications of this finding for plant/herbivore interactions in response to future climate change.

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1. Bogner, F., Boppré, M., Ernst, K. D. & Boeckh, J. *J. comp. Physiol.* A**158**, 741–749 (1986).
2. Kent, K. S., Harrow, I. D., Quartararo, P. & Hildebrand, J. G. *Cell Tissue Res.* **245**, 237–245 (1986).
3. Stange, G. *J. comp. Physiol.* A**171**, 317–324 (1992).
4. Waldow, U. *Z. Vergl. Physiol.* **69**, 249–283 (1970).
5. Coro, F. & Perez, M. *Naturwissenschaften* **77**, 445–447 (1990).
6. Miles, C. I. *Physiol. Ent.* **17**, 169–175 (1992).
7. Willmer, P. G. *Adv. Insect Physiol.* **16**, 1–57 (1982).
8. French, A. S. *J. comp. Physiol.* A**156**, 817–821 (1985).
9. Barnola, J. M. *et al. Nature* **329**, 408–414 (1987).