during this period. Between 1957 and 1960 there is a rapid rise of total column ozone by about 13 DU. Both this abnormal low and the rapid rise thereafter seems to be a unique feature of column ozone over Mount Abu. No significant trend was observed between 1960 and 1965, a rise again of about 10 DU from 1965 to 1974, and a slow decrease thereafter by about 9 DU from 1974 to 1983. Before revision, Ahmedabad/Mount Abu total ozone data showed a marked rise in the year 1976–84 (ref. 1) which was very different from the decreasing trends of other Indian stations, but after the revision, it became in unison with the others. This is also in agreement with the overall down trend in total ozone observed over the latitude zone 26–60° N during 1970 to 1986 using revised Dobson total ozone data[3].

The analysis presented here indicates the presence of inconsistency in the unrevised Ahmedabad/Mount Abu column ozone data. It is hoped that this paper will serve as a reference for the analysis of other Indian ozone data sets in future.

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Comments on 'A computer simulation model for action potential in an excitable membrane'

[M. P. Mujumdar and C. K. Mitra, *Current Science*, 1990, **59**, 920–925]

We would like to raise the following points about the above paper:

1. On page 3 it is mentioned that the authors 'have found GHK equation to represent satisfactorily the potential of the membrane' under non-steady conditions'. But the basic assumption in deriving the GHK equation is that the membrane is in the *steady state*, since in the derivation of the equation total transmembrane ionic flux is set to zero[1]. This is a very basic tenet of electrophysiology, and in view of this, the authors should have at least given a more detailed justification for the use of their modified equation on page 3, which they have failed to do.

2. Figure 2, on page 3, shows that the permeability of potassium (P_K) *decreases* during the action potential. But it is well known that P_K also increases during the action potential, although with a time lag compared to P_{Na} (ref 2). We are not aware of reports in the literature of nerve action potentials in which P_K *falls* during the depolarization phase (although this may occur in cardiac action potentials; see ref. 3).

3. In Figure 3, on page 4, it seems that the rates of depolarization and repolarization of the action potential are almost identical. However, it is experimentally found that the rate of depolarization is considerably faster than that of repolarization. The authors have not commented on this important lack of agreement between experiment and theory.

4. On page 4, at the end of para 1, the authors state that 'the threshold range for this particular system is 55–59 mV'. However the thresholds of experimentally observed action potentials lie in the range – 50 to – 35 mV. If one needs to depolarize a membrane to + 55 to + 59 mV to generate an action potential peaking at + 50 mV (as apparent from Figure 3), then it is unclear as to how this 'threshold' is defined, since the stimulus itself is bringing the membrane potential to its value at the peak of the action potential!

5. The most glaring error in the paper, which seems to contradict all known physiological facts, seems to be the following. In the authors' simulation the action potential, and the ionic permeability changes underlying it, last for only about 5 nanoseconds. This does not seem to be a printing error as it appears in no less than three figures (Figures 2, 3 & 4). However, it is well known from repeated experimental observations that the nerve action potential lasts for at least a *millisecond*, if not more (for original observations see refs. 2 and 4). The discrepancy between the simulated time course and the observed time course is therefore no less than 1,000-fold, and it makes us wonder how far we can trust the validity of the simulation.

The authors have considered the ionic concentrations of squid axon, on which a lot of research was done. Therefore we have also referred to the above observations on the same axon. It is clear that the simulation is at odds with several physiological features of the action potential of the unmyelinated neuron. But in the Discussion the authors have ignored these issues, and have paid addressed any of the discrepancies.

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