## MATTERS ARISING

## Stability of zeolites under electron irradiation

RECENT electron microscopic studies<sup>1,2</sup> of structural changes in zeolites under electron irradiation have led to the plausible assumption that water, normally contained in zeolites, is linked with the process. It is thought<sup>2</sup> that the highintensity electron bombardment, used for high-resolution electron microscopy, produces significant numbers of OH<sup>-</sup> ions by electron impact. These ions are in turn believed to weaken the bonds of the aluminosilicate framework, causing its eventual collapse into a disordered state.

It seems likely that the effect observed by Bursill *et al.* is related to a type of degradation through electron, ion or photon irradiation, that I previously reported, which appears to occur in all substances containing  $M^+$  cations of the alkali metals or alkaline earths and which is therefore expected to take place also in zeolites. My colleagues and I believe<sup>3</sup> that the primary function of the irradiation is not to produce OH<sup>-</sup> radicals, but both to detach cations from their normal positions according to

$$\mathbf{MX} + \text{radiation} \rightarrow \mathbf{M}^+ + \mathbf{X}^- \qquad (1)$$

and to form catalytically active surface centres where they are converted from  $M^{\rm +}$  to M.

In the presence of H<sub>2</sub>O

$$M + H_2 O \rightarrow MOH + H^{\uparrow}$$
 (2)

and

$$X^- + H_2 O \rightarrow X H + O H^- \qquad (3)$$

At suitable surface sites, the  $OH^-$  produced in step (3) can break the M-X bond so that

$$MX + OH^{-} \rightarrow MOH + X^{-} \qquad (4)$$

following which steps (3) and (4) are cyclically repeated as long as water vapour is available.

Note that  $H_2O$  need not be present during the actual irradiation, but can be admitted later, and once the catalytic centres are formed and reaction step (1) completed, the irradiation may be discontinued, as in each reaction cycle the OH<sup>-</sup> radical necessary for its successor is produced.

This was in fact the experimental situation in our work, but in the investigations of Bursill *et al.*,  $H_2O$  was available throughout, and as their bombardment intensity exceeded ours by several orders of magnitude, impact ionization could have provided a second, and perhaps even dominant, source of OH<sup>-</sup>, thus explaining the very fast reaction rate implied by their observation of significant breakdown within only a few minutes. In contrast to this, although we have not experimented specifically with zeolites, our experience with other substances suggests that in the absence of significant impact ionization, such as when dehydrated materials are exposed to a moist atmosphere after irradiation, some weeks or even months may have to elapse before degradation becomes evident. Experiments along such lines might assist in elucidating the reported instability of zeolites.

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BURSILL ET AL. REPLY—We thank Hirsch for his remarks. In ref. 1, he has proposed a reasonable mechanism for the production of  $OH^-$  in argon ionbombarded bulk specimens in the presence of moisture.

In their normal condition, zeolites are very rich in water content—up to  $\sim 50\%$ by volume in some cases—so that the passage of 200 KeV electrons through

## Mechanisms of slow postsynaptic potentials

THE review article by Hartzell<sup>1</sup> contains factual errors with regard to neuro-glandular synapses. His Table 1 lists information on cat salivary glands and pancreas. The data given in respect of cat pancreatic acinar cells are wrongly cited, misleading and partly non-existent.

To my knowledge, microelectrode measurements on cat pancreas have only been reported once<sup>2</sup>, where it was shown that exogenous acetylcholine (ACh) caused membrane depolarization (not hyperpolarization as claimed in the review article<sup>1</sup>), but no information was given on latencies, duration of responses or ionic mechanism. Acinar membrane potential changes following nerve stimulation were not described for the cat pancreas. such materials is similar to the production of an electrical discharge through water. Under these circumstances, it would not be surprising for the rate of production of  $OH^-$  to be considerably higher and for the rate of destruction of the crystal to be commensurately greater than in the situations cited by Hirsch. One fact stands out: thorough dehydration of zeolites before their examination by electron microscopy is crucial in enhancing the beam stability so as to permit highresolution imaging of the kind we described<sup>2-5</sup>. The mechanism proposed by Hirsch may be more relevant to dehydrated than hydrated zeolites.

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All this information is, however, available for mouse (and rat) pancreatic acinar cells (see Table 1 for values and refs). I have also listed information for mouse parotid acinar cells because ionic mechanisms have been investigated in much greater detail in this species than in the cat. Substance P and adrenaline applied to rat parotid acinar cells by microionophoresis evoke membrane potential changes that have a reversal potential and latencies also characteristic of the action of ACh<sup>3</sup>. Peptides belonging to the cholecystokinin and bombesin groups act on pancreatic acinar cells by evoking potential and conductance changes similar to those evoked by ACh<sup>4</sup>, although these peptides interact with two types of receptor site distinct from the muscarinic receptors<sup>5</sup>. The minimum latencies found for peptidergic cell activation were 500-1,500 ms (ref. 4).

Table 1     Selected examples of slow postsynaptic potentials in mouse gland cells						
Tissue Parotid acinar cells	Response Biphasic: depol.; hyperpol.	∆ Conductance ↑Na †K	Latency (ms) 250	Duration (s) 2-5	Transmitter ACh	Refs 7, 8
Pancreatic acinar cells	Depol.	∱Na ↑Cl ↑K	900	5-20	ACh	9, 10