The negative air ion system used by Rosenthal *et al.*[4] to test for O_2^- production, by looking for nitroblue tetrazolium (NBT) reduction, or H_2O_2 accumulation, had several gross inadequacies. They neither grounded, nor even stirred, their target solutions and could not in fact even demonstrate a bactericidal effect of negative air ions, an effect repeatedly confirmed in numerous published reports. Our own work has shown that the effect of negative ions on NBT gives results that seem neither simple nor straightforward. We found that although negative ion treatment did not cause reduction of NBT to the formazan, it did cause the NBT solution to change spectrally, while concomitantly losing its susceptibility to reduction to the formazan by dithionite. Although SOD completely prevented this effect, we also found that denatured SOD, and even bovine serum albumin, gave complete protection. Our results with NBT, although unexplained, offer no evidence either for or against superoxide involvement in negative air ion effects.

Similarly, the failure of Rosenthal *et al.* to find evidence for H_2O_2 production in solutions exposed to negative ions does not in any way argue against O_2^- involvement. They failed to understand that one cannot simply 'add' negative charges indefinitely to a target solution, and as their system consisted simply of H_2O exposed to an ion generator without grounding, or even stirring, one would not expect to find sufficient H_2O_2 accumulation for measurement by even the most sensitive methods, let alone less sensitive chemical methods such as the iodometric that they used.

We apologize that we did inadvertently give an incorrect value for the maximal possible flux of O_2^- in our system—0.18 $\mu M\,h^{-1}$ gives a correct estimation of this flux. Over a 5-h period, this gives a ratio of $\cong 1.4\times10^{10}$ negative ions per bacterium. Although the mechanism of bacterial kill remains obscure, it does not seem to occur by agglutination due to electrostatic effects, because early in our experimental series we observed no agglutination of samples in direct microscopic examination. Furthermore, previous studies using polonium-210 or tritium as ion sources have also demonstrated bacterial kill, despite having a field substantially lower than that seen in corona discharge generators[5]. Earlier work using corona discharge generators[6] found a reversal of damage to cells on exposure to intense visible light, an effect which should not occur with agglutination. Finally, the time required for cell death was much less than the minimal time for agglutination predicted by the von Smoluchowski equation[7]. Many other studies have shown the varied biological activity of negative air ions[8–13], so the suggestion by Rosenthal and Ben-Hur that all negative ion effects are derived from electric field or electrostatic precipitation effects appears at odds with the experimental facts of the literature.

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Role of PGE_2 in anion exchange in gastric mucosa

IT has been suggested by Schiessel *et al.*[1] that a prostaglandin (PGE_2) increases Cl^-–HCO_3^- exchange in gastric mucosa, thereby protecting surface cells against excessive back-diffusion of H^+ from the lumen. Their evidence is necessarily indirect, because it is not known whether such exchange occurs at the nutrient membrane of the surface cell (as demanded by their hypothesis) or, indeed, whether exchange diffusion is a characteristic of this cell type. The authors do show an increase in Cl^- fluxes across the isolated mucosa due to PGE_2, and because anion exchange is rather nonspecific in this preparation[2], as in red blood cells[3,4], it is reasonable to suppose that exchange of Cl^- for HCO_3^- is also increased.

The authors, however, are guilty of a logical *non sequitur*: they deduce that PGE_2 increases exchange of cell Cl^- for nutrient HCO_3^- from a negative finding, that PGE_2 does not affect the depression in nutrient to secretory Cl^- flux due to the removal of secretory Cl^- (trans-concentration effect[5]). Exchange of Cl^- across the epithelium requires a suitable partner anion, yielding a null electrical current. In the secretory solution, the authors use replacement anions (isethionate, sulphate) which do not exchange for Cl^-, and this fact cannot be altered by a drug such as PGE_2.

The criticism here raises a pertinent question: does a modest level of HCO_3^- in the secretory solution reduce the trans-concentration effect, and does PGE_2 then further reduce the effect? Evidence on this question would bear more directly on the authors' hypothesis.

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SCHIESSEL *ET AL.* REPLY—We acknowledge the validity of the argument that PGE_2 cannot alter the trans-concentration effect because replacement anions which do not exchange for Cl^-, were used in the secretory solution, and we were aware of that fact. In these experimental conditions, however, failure of PGE_2 to affect $J_{Cl_{ns}}$ (the flux of $^{36}Cl^-$ measured from nutrient to secretory solutions) more than about the 50% usually found in control experiments in our laboratory suggests that the PGE_2 does not affect either of the remaining components of Cl^- flux—the active or diffusional components. An unfortunate phrasing of the sentence describing our findings may have led to the misunderstanding that we believed these experiments rigorously excluded the possibility that PGE_2 affects exchange diffusion of Cl^-.

We agree that much of the evidence for our hypothesis is necessarily indirect. The finding that PGE_2 did not sustain Cl^- flux in metiamide-inhibited tissues when HCO_3^- was absent from the nutrient solution supports this hypothesis, however. It is also possible that PGE_2 causes an increase in Cl^- permeability without change in exchange diffusion of HCO_3^-/Cl^- exchange.

The suggested experiments would be interesting, but in our opinion difficult to interpret and not definitive in excluding an effect of PGE_2 on exchange diffusion of Cl^-. If all three components of the Cl^- flux are operative when a suitable exchangeable anion (not Cl^-) is in the secretory solution, stimulation of Cl^- flux by PGE_2 would not specifically define the effect of PGE_2 on any of the three components of Cl^- flux. Conversely, failure of PGE_2 to stimulate Cl^- flux in these conditions might be interpreted as evidence that either a direct Cl^-–Cl^- exchange at the secretory membrane is required for the PGE_2 effect or that the non-Cl^- anion inhibited the effect of PGE_2.

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