seeds of *Vicia faba* to a stream of air containing 0·4 per cent weight of ozone for 15, 30 or 60 min. and then counted abnormal anaphases in the root tips when the roots had developed to 2 cm. in length. The effect of 30 min. exposure to 0·4 per cent ozone was equal to that of 100 r., and ozone plus irradiation with X-rays showed that their separate effects were fully additive. In this case, the radiomimetic effect of ozone is quite clear.

Combined with the results described earlier in this communication, the probability must be considered that inhalation of ozone, in concentrations which occur in various conditions, may lead to radiomimetic action.

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Rejection of the Peroxide Accumulation Hypothesis of Isoniazid Action

From three quite different lines of investigation it has been postulated that isoniazid kills tubercle bacilli through the intracellular accumulation of hydrogen peroxide[1–3]. In *Micrococcus lysodeikticus*, which contains an exceptionally high concentration of catalase, Chance has been able to demonstrate a peroxidase–catalase complex and to show that the peroxide concentration is controlled by the presence of suitable oxygen acceptors within the cell or added externally[4]. Sensitive though it is, Chance's spectrophotometric method would be difficult to apply to *Mycobacterium tuberculosis*, which has a very low catalase activity[5], and direct chemical determination of intracellular peroxide does not seem possible. It seemed reasonable, however, to test the theory of peroxide accumulation in the presence of isoniazid by growing the bacilli in an excess of a stable exogenous oxygen acceptor capable of penetrating the osmotic barrier of the cell but not inhibiting cell growth. Sodium nitrite appears to fulfil all the requirements in that it reduces free peroxide and reacts rapidly with the catalase–peroxide complex, both *in vitro*[6] and intracellularly[4].

It has now been shown in this laboratory that *M. tuberculosis BCG* grows well in the presence of 500 μgm. per ml. sodium nitrite in a modified Schaefer's medium[7] containing 0·5 per cent glycerol as the sole source of carbon. At the end of 10 days incubation in this medium, 80–90 per cent of the original nitrite was still demonstrable by colorimetric analysis[8]. Thus it appeared that nitrite was not metabolized by *M. tuberculosis*.

It was also possible to show by two different techniques that the nitrite passed through the osmotic barrier of the tubercle bacillus (*BCG* strain). First, using a thick suspension[9] in 0·025 *M tris*-maleate buffer at *p*H 6·8, the nitrite-impermeable volume of the cells was shown to decrease from 2·0 ml. per gm. dry weight to 0·8 ml. per gm. dry weight after 4–5 hr. exposure to 500 μgm. per ml. of sodium nitrite. In contrast, the dextran-impermeable volume of the same cells remained constant at 3·0 ml. per gm. dry weight over the same time. Secondly, the cells were shaken aerobically overnight at 37° C. in the same buffer containing 500 μgm. per ml. sodium nitrite, and were afterwards harvested, rapidly washed in buffer and disrupted in a Hughes press[10]. After centrifugation it was found that, within the limits of experimental error, the concentration of nitrite in the supernatant fraction was the same as that in the buffer to which they had been exposed.

It was then found that the addition of 500 μgm. per ml. of sodium nitrite to modified Schaefer's medium did not alter the bacteriostatic end-point of isoniazid (0·13 μgm. per ml.) for either *M. tuberculosis BCG* or *H37Rv* after 14 days incubation. Since isoniazid will not react with nitrite at *p*H 7·0, it was presumably free to induce intracellular hydrogen peroxide accumulation. Thus, if the inhibition of growth was due to this phenomenon, then the inhibitory action of isoniazid would have been antagonized by the presence of intracellular nitrite. The fact that no such antagonism was found indicates that the peroxide accumulation hypothesis of isoniazid action is untenable.

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Third Allele at the Serum β-Globulin Locus in Humans

Horsfall and Smithies[1] have recently reported evidence for the genetic control of some human serum β-globulins. Two autosomal alleles with no dominance (β^C and β^D) were proposed to account for the variable presence of the β-globulins *C* and *D*, demonstrated by two-dimensional starch-gel electrophoresis[2], in sera from Australian aborigines and Negroes[3]. No evidence was available from previous studies (made for the most part by one-dimensional starch-gel electrophoresis) for the presence of β-globulin *D* in the sera of several hundred Canadians.

Sera from 425 normal blood donors at the Toronto General Hospital have now been examined by two-dimensional starch-gel electrophoresis in order to provide more reliable evidence for the presence or absence of β-globulin *D* in the sera of Whites, since one-dimensional starch-gel electrophoresis is not satisfactory for detecting with certainty the presence of this protein. β-Globulin *D* was not observed in any of these sera, and β-globulin *C* was quantitatively the predominant β-globulin in 420 of them. However,