mation that may then be available. The group believes that the chief value of this reconnaissance work is a quick and economical way of providing an overall picture of the distribution of trace elements, which will point to areas which are worth more detailed investigation. Mineral prospectors already make this sort of reconnaissance when looking for metal ores such as lead, zinc and nickel. When the atlas is available they will be able to go straight to an area that looks hopeful.

In certain areas, excesses or deficiencies of elements such as copper, cobalt, molybdenum and manganese can affect the health of crops and farm animals. Using the geochemical atlas, agricultural advisory services will be able to choose areas for more intense investigation. Public health can also be affected by trace elements in the soil and water. For example, there is a good correlation between cardiovascular mortality and the hardness of water, in which trace elements may be involved; and the ratio of copper to zinc in garden soils in North Wales correlated statistically with the incidence of cancer. Medical workers researching these and similar problems should be grateful for the geochemical atlas.

MICROBIOLOGY

Protection from Oxygen

from a Correspondent

In the past decade, a great deal of effort has been expended on investigating the mechanism by which nitrogen fixing, facultatively anaerobic bacteria, such as species of *Azotobacter*, protect the oxygen sensitive components of their nitrogenase system from oxygen. It has been known at least since 1911 that high oxygen tensions inhibit the growth of many anaerobes, and for that matter many aerobic bacteria, but the mechanism of this toxicity remains obscure. Experiments with batch cultures, which have the disadvantage of being difficult to reproduce because of the continually changing environment, have led to a general consensus that the nitrogen fixing systems in species of *Azotobacter* are protected from oxygen by some steric arrangement. The principal evidence for this idea is that the nitrogen fixing systems can be isolated in a particulate form resistant to oxygen. When, however, the components of the particles are made soluble they become extremely sensitive to oxygen, as are the soluble extracts of *Clostridium pasteurianum*, which apparently lacks conformational protection. It is scarcely necessary to say that it is anybody's guess exactly how conformational protection works, although there are two obvious general possibilities: oxygen sensitive sites could be rendered either inaccessible or insensitive to oxygen.

But that is not the whole story. Several groups have suggested that there is a second protective mechanism—augmented respiration—which scavenges oxygen from the neighbourhood of the nitrogen fixing sites; and Dalton and Postgate (*J. Gen. Microbiol.*, **54**, 463; 1969) have just published some compelling evidence in support of this notion, obtained with continuous cultures of *Azotobacter chroococcum*, in which growth conditions are fairly well defined.

Their evidence is four-fold. First, respiration in nitrogen fixing cultures, in which nitrogen supply is limiting, is adjusted to balance oxygen supply, leaving nitrogen fixation largely unaffected. Second, when the carbon source was limiting and respiration was therefore restricted, but conditions were otherwise identical, nitrogen fixation became extremely sensitive to oxygen. Third, the cultures actively maintained a balance between oxygen consumption and supply; as supply rose, so did consumption, and the converse. Fourth, populations not fixing nitrogen were not hypersensitive to oxygen.

Dalton and Postgate have taken their analysis of respiratory protection one step further by studying oxygen sensitivity in phosphate-limited cultures. If respiratory protection works through the cytochrome respiratory pathway, starvation for phosphate should reduce protection because the balance between ADP and ATP would shift so as to reduce respiration. And as predicted by this argument, phosphate-limited cultures were extremely sensitive to oxygen.

GENETIC CONTROL

Arginine Biosynthetic Pathway

from our Cell Biology Correspondent

The way in which the expression of the eight structural genes specifying the enzymes responsible for arginine biosynthesis in *E. coli* is controlled has been something of a puzzle for the past few years. Unlike the lactose operon of *E. coli*, in which the three structural genes are contiguous with their regulatory genes, the eight structural genes of the arginine pathway are scattered in five regions on the circular *E. coli* chromosome. But in spite of this they are all controlled by a single regulatory gene. The other unusual feature of the arginine pathway is that in *E. coli* strain K, added arginine represses the synthesis of arginine biosynthetic enzymes, whereas in *E. coli* strain B, added arginine slightly stimulates enzyme synthesis, and yet mapping and complementation experiments indicate that the regulatory genes in the two strains are allelic.

Gorini's group at MIT now reports, in two papers in the latest issue of the *Journal of Molecular Biology*, a satisfying unitary scheme which accounts for these disparate features. There is evidence that in both strains an amber mutation in the repressor locus prevents all repression; all eight structural genes are expressed continuously, irrespective of the presence of arginine. This implies first that the repressor is at least in part a protein, and second that the structural genes are under negative control—the repressor prevents their synthesis. Furthermore, the repressor protein in the two strains must be very similar because a single amino-acid substitution in the B strain repressor converts it to a K type repressor.

In the first paper, Jacoby and Gorini (*J. Mol. Biol.*, **39**, 73; 1969) suggest that in both strains arginine, or a derivative of it, activates the repressor, presumably by binding to it. In strain K the activated repressor can then repress enzyme synthesis by binding to the operators of the structural genes. In strain B, however, they suggest that high concentrations of arginine reduce the activity of the repressor and therefore the structural enzymes are expressed. One obvious suggestion is that the B strain repressor protein undergoes a configurational change on binding arginine which reduces instead of enhances the affinity of the repressor for the operator regions of the structural genes.