theology. Interest in evolution remains high in both popular and scientific circles, and the Lincei was shrewd to catch the mood of the times by attracting a broadly based group of speakers each of whom had something important to contribute towards a better understanding of man's evolutionary history.

CLIMATE

Advance of Sahara

MUCH of the blame for the advance of the northern Sahara has traditionally been laid at the door of man, whose crude farming techniques have, since Roman times at least, created erosion and desert conditions in once fertile regions. A report by the World Meteorological Organization has now placed this belief on a scientific footing and it provides quantitative data on the importance of man's activities to the fertility of the region (H. Flohn and M. Ketata, *WMO Technical Note* 116, WMO, Geneva, 1971).

In addition to the detrimental effect of man, natural climatic changes also produce fluctuations in desert margins. In the Sahara it seems that the overall trend during the past 7,000 years is for the desert to dry up and expand (see, for example, J. L. Cloudsley-Thompson, *Internat. J. Envir. Studies*, **2**, 35; 1971), but this is not to say that farming activity is not important in the short term. During periods of delicate balance between the needs of vegetation and rainfall, overgrazing and the utilization of firewood can have a disastrous and irreversible effect.

The use of artesian wells and the intensive farming of date palms have a dramatic effect on the water balance. Since 1881 the number of date palms in the border region of the Tunisian Sahara has increased by more than 50 per cent. Flohn and Ketata estimate that the effect of this increase has been to reduce the level of subterranean water at a rate of 5 cm per year. Over a period of less than 100 years, it seems likely that effects of this magnitude will dominate over the long term natural climatic variations. As well as the direct effect on evapotranspiration of the economic importance of the palm, other farming techniques encourage the growth of the desert; notably, overgrazing and compacting of the soil by domestic animals such as the goat cause erosion. Even now, however, it may not be too late to take steps to rectify the situation, at least partially. At one farm site north of Nefta, an area of 50 hectares has been protected by barbed wire for the past 60 years. Here, the original vegetation has apparently been restored, with an average soil coverage of more than 80 per cent, a figure which contrasts dramatically with 5 per cent coverage by the degraded vegetation on the other side of the fence.

The report concludes that the historically observed progress of desertic conditions towards the north is caused "not by a natural fluctuation in climatic conditions, but by human interference". Clearly, all activities involving the delicate water balance in the intermediate zone between desert and cultivated land should be carefully planned and coordinated to avoid further adverse effects on the water budget. In particular, caution over the extended use of artesian water and irrigation, encouragement of dry farming (such as olive farming), reafforestation and increase of animal husbandry to allow the return of natural vegetation could do much to redress the balance.

HAEMATOLOGY

Sickle Cell and Flight

from a Correspondent

IT has been known for about twenty years that aircraft flights can cause illness among people with sickle cell disorders—a hereditary disorder of the blood that affects negro populations. But many negroes have only mild forms of the disease and are unaware that they are sufferers. To what extent are these people at risk when they embark on an aircraft flight?

Recent work on this question has been published in the *British Medical Journal* (**4**, 593; 1971) by R. L. Green, R. G. Huntsman and G. R. Serjeant, who suggest that all negroes should be screened for sickle cell disorder before flying. The problem arises because even modern jet aircraft are not fully pressurized as compared with ground level —the cabin pressure in a Boeing 707 at cruising altitude corresponds to an altitude of 5,000 to 7,000 feet—and many scheduled African flights still use unpressurized aircraft.

Sickle cell disease is an abnormality of the haemoglobin molecule. Several forms have been described; for example, substitution on the peptide chain of valine for glutamic acid results in production of haemoglobin S instead of the normal form, haemoglobin A, and another form which Green *et al.* found to be relevant to their studies is haemoglobin C. The abnormal haemoglobin renders the shape of the red blood corpuscles less stable and under conditions of low oxygen tension the

Reading through a UGA Terminator

THE closely related group of RNA phages which include R17, MS2 and f2 possess genomes which specify only three proteins, namely coat and assembly proteins, which form part of the viral capsid, and a subunit of the viral replicase. The phage Qβ, which is not in the same serological subgroup as R17 and its relatives, specifies coat and assembly proteins and a replicase subunit but, in addition, a fourth protein known as A_1 or II*b*, which weighs 36–38,000 daltons, can be recovered from Qβ phage particles.

What is the nature of this fourth protein? According to the experiments of Weiner and Weber and of Moore *et al.* reported in next Wednesday's *Nature New Biology*, it is a larger than life coat protein made when ribosomes fail to terminate synthesis at the end of the coat protein cistron and continue to translate the intercistronic divide and part of the next cistron, which specifies a subunit of the replicase.

Moore *et al.* have shown that the first five amino-acids of A_1 are the same as coat protein and Weiner and Weber have shown that the first eight amino-acids of A_1 (or II*b*) are the same as the coat protein. Furthermore, Weiner and Weber report that A_1 contains tryptophan, histidine and methionine, amino-acids not in the Qβ coat protein, and that A_1 is never synthesized by mutant Qβ phage with an amber mutation in their coat protein cistron.

These data indicate that A_1 is probably produced when ribosomes read through the termination signal at the end of the Qβ coat protein cistron. Because amber (UAG) and ochre (UAA) codons are efficient terminators unlikely to allow read-through, both groups argue that the terminator codon at the end of the Qβ coat cistron is probably a solitary UGA codon, for UGA codons are known to be leaky terminators. Horiuchi *et al.* (*Virology*, **45**, 429; 1971) have also recently reached this conclusion and Weiner and Weber report experiments with a UGA suppressor strain of *Escherichia coli* which confirm it. They find that the molar fraction of A_1 (II*b*) protein in Qβ phage particles increases from 2 per cent to 7 per cent when the phage are grown on a UGA suppressor strain of *E. coli*. Furthermore, introduction of a streptomycin resistance mutation, which reduces the leakiness of UGA termination, reduces by about 30 per cent the yield of A_1.

The A_1 protein is about 2.5 times the size of coat protein, which means it has about 200 amino-acids in excess, and yet it can apparently become incorporated into the phage particles. It will be interesting to learn how this is achieved and how much of the replicase subunit sequence is found in the A_1 chain.