the atmospheric structure of Uranus would result from its large polar inclination of ~98°, since each hemisphere will spend a substantial period without sunlight during each Uranian year. The north pole of Uranus is just turning towards us, having been turned away from the Sun for more than 40 years. The atmosphere will have cooled substantially in the absence of meridional heat transport. Perhaps at this time the cold hemisphere of Uranus is efficiently absorbing the available solar radiation and therefore radiating less energy than it receives from the Sun.

Certainly, therefore, there are apparently major differences in the structures of the upper atmospheres of these planets. The variations in their individual stratospheric temperature gradients will influence the detectability of minor constituents and account for the observations of Macy and Sinton (*op. cit.*). Furthermore, the absence of a Uranian internal heat source will require any motions in the planetary atmosphere to result from differential solar heating. However, at this distance, the invariant solar radiation is comparatively weak, and we may not anticipate any rapid, large scale, dynamical variations in the atmosphere of Uranus. Neptune, with an internal heat source, could possess some large scale motions. This may then account for the recent perplexing observations of Joyce *et al.* (*Astrophys. J.* **214,** 657; 1977), which indicated a factor of four change in the reflectance of Neptune between 1 and 4 μm during 1975–76, while the simultaneous measurements of Uranus showed negligible change (Hunt *Nature* **269,** 10; 1977). □

Mystery of foetal growth

by Mary Lindley

DURING the past twenty years it has become accepted that some babies are born small not because they are premature but because their growth has been retarded in the uterus. These so-called small-for-dates babies, which are smaller than would be expected for their gestational age, are at greater than normal risk at birth, particularly from asphyxia and hypoglycaemia. They can be treated only by giving them special attention after birth. Earlier intervention is precluded because the mechanisms responsible for intrauterine growth retardation are poorly understood and it is not even possible to identify a foetus that is growing too slowly. A brave attempt to shed new light on this situation at a recent Dahlem Workshop* seemed to show that the clinical problem remains difficult to approach.

Attempts to define the problem and its causes immediately face the difficulty that although small-for-dates babies generally weigh less than 2.5 kg, they are not easy to identifiy within the natural variation of a population. As M. Ounsted (John Radcliffe Hospital, Oxford) pointed out, some small babies are perfectly healthy. Her studies of the birthweights of mothers, their infants and relatives have indicated that a woman exerts a characteristic constraint on the foetal growth of her offspring. This property of maternal constraint is passed on to her daughters. The same phenomenon is known in other mammals.

Other factors that can influence total growth are both genetic and environmental. The former include the absence of an X chromosome in females with Turner's syndrome and the presence of a third copy of certain chromosomes, notably numbers 13 and 18. Environmental factors that restrict foetal growth include metabolic maternal diseases and possibly infections. There was some feeling that more attention should be paid to the effects of parasites such as those of malaria, schistosomiasis and trypanosomiasis.

Ionising radiation has restricted foetal growth among Japanese survivors of the atomic bombs. Thus it belongs in the list that includes smoking, alcohol, drugs and other chemicals in food and in the atmosphere, all of which may restrict foetal growth. Participants felt that effects on birthweight may prove to be a sensitive measure of drug toxicity and should perhaps be included in the evaluation of new drugs.

In spite of years of work on the effects of maternal nutrition on the foetus there seems to be no clear picture of this relationship during human pregnancy. Two well-quoted studies suggest that maternal nutrition can limit foetal growth. In the Netherlands during the famine of 1943-44 the birthweight declined 9%, and in a poor rural community in Guatemala supplementary feeding during pregnancy enhanced the birthweight. But such studies are difficult to interpret because of complicating factors, such as disease, which also effect foetal growth. The group at the workshop added no new wisdom and doubtless arguments will continue to rage. The role of maternal toxaemia and hypertension during pregnancy was also disputed, with agreement that severe forms are associated with retarded foetal growth, but less confidence that any effect is evident with milder forms of the disorders.

How and when all these factors exert their effects on foetal growth remains largely a mystery, although a few weak pointers emerged from the discussion. There was much modesty about the dangers of extrapolating from mice, sheep or monkeys to man, with G. S. Dawes (University of Oxford) anxious to outlaw all reference to animal models in this context. Most participants preferred to retain the familiar terminology. One of those was A. Gropp (Medizinischen Hochschule Lübeck) who described his own mouse model. From crosses between *Mus poschiavinus,* the tobacco mouse which has 26 chromosomes, and *Mus musculus,* the house mouse which has 40 chromosomes, he obtains a range of embryos with a single chromosome missing (monosomics) or with an extra chromosome (trisomics). He has found that monosomic embryos show a deficiency of cells before implantation, while after implantation both they and the trisomic embryos are gradually eliminated.

This hint that the effects of chromosome abnormalities on growth can be manifest very early in gestation was one of the few positive answers to emerge from a discussion of whether early development is involved in abnormal foetal growth. For example, an attempt by V. Sara (Karolinska Hospital, Stockholm) to link the current crop of polypeptide growth factors to foetal growth was unsuccessful. She pointed out that as there is no clear evidence that growth factors are involved in post-natal growth it is unlikely that anything could be said about a role for them in the foetus. Similarly no clues about what happens in the growing foetus could be gleaned from the store of knowledge of cell recognition and adhesion.

Prompted by the embryological contributions of R. L. Gardner (University of Oxford) and H. M. Beier (University of Aachen), aficionados of the mouse and rabbit respectively, participants decided that synchronisation between embryo and mother at implantation should be a focus for further studies. Embryo transfer experiments have shown that asynchrony can influence embryonic growth, but it remains to be seen whether the effects continue to be manifest in the growing foetus or whether they simply result in early death. □

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*The workshop was held on 20–24 February in Berlin; proceedings will be published by Dahlem Konferenzen.