been chosen because they are known from radio observations to contain complex H II regions.

These ionised gas domains surrounding a recently formed, high luminosity star are most intriguing astronomical objects. The unravelling of the detailed mechanisms of these star formation centres occupies a considerable fraction of astronomical effort and the problems are many and complex. The cores of these regions are thick with dust, often rendering optical identification of the ionising star or protostar extremely difficult, if not impossible. Near-infrared observations however do often reveal stars and protostars surrounded by very hot dust shells whose signals can be the free–free radio continuum emission of the H II region. These regions are also rich sites of molecular clouds, and emission maps of many and various molecules have been obtained at wavelengths of a few millimetres or centimetres for the most intense sources. To attempt to piece together the energetics of the emission nebulae, one must determine the total luminosities of the gas, dust and molecules and so derive the luminosity of the exciting star or stars, which must be the powerhouse of the whole region.

The key to understanding these regions is the presence of dust. The dust grains may be associated with the Stromgren sphere of the H II region and in this case are being heated by the direct flux of stellar ultraviolet radiation or by Lyman α radiation of the hydrogen gas surrounding the star. Alternatively, the grains could be associated with the dense molecular clouds found in and around H II regions, the grain heating being caused by stellar or protostellar objects within these molecular clouds. In the former case, the dust emission, peaking at say 100 μm, should be centred on the position of free–free continuum emission, whereas in the latter, the far-infrared emission should follow the contours of the molecular emission.

Using the UCL balloon system, operating in the wavelength range 40 μm to 350 μm, Furniss *et al.* (*Astrophys. J.*, **202**, 400; 1975) have produced the most detailed far-infrared map of the complex region W3 currently available. Their map reveals that the far-infrared emission seems to follow the radio emission in general, although there does appear to be a long spur projecting westwards from the main source. The authors claim that recent radio free–free emission has also been reported to possess this spur feature and so the tie-in with the radio emission rather than the molecular cloud emission would seem to be more favoured for this source at least.

This, however, is only one source and there are many others; the Great Nebula region of M42 in Orion, for instance, one of the most exciting and complex regions in the sky, shows many differences to W3. It is also found that as one observes at longer wavelengths ($\sim$ 1 mm) one is looking at the cooler dust and this component does appear to follow the molecular cloud emission rather than the H II continuum free–free emission. Once again the complexities of the regions are daunting and challenging. Theoretically one is not at all sure what size are the dust grains in the regions, or of what material; silicates or graphites are the usual choice. Laboratory spectral analysis of selected grain compositions is urgently required by the grain theoretician. Observationally one requires better high resolution, far-infrared and molecular maps and a near-infrared search for the dust-enshrouded protostars in the H II regions before the vastly complex physics and mechanics of these birthplaces of stars can be understood fully. In the far-infrared, one looks to the future for balloon and aircraft-based spectrometers and interferometers. The larger diameter balloon-based telescopes and the first non-military, infrared satellite, scheduled for launch in 1981, will open up a new era of observing in this wavelength range. □

System for steroids

from Robert Shields

An understanding of the way in which steroid hormones regulate gene expression has been frustrated continuously by the lack of suitable model systems in which hormone action can be studied *in vitro*. Ideally what is required are hormonally responsive cell lines where mutant cells can be isolated and the mechanism of hormone action can then be dissected in the way that proved so successful in prokaryotes. Several steroid responsive cell lines are now available, including some potentially useful mutants (Sibley and Tomkins, *Cell* **2**, 213; 1974) but they all have a number of drawbacks. Now it looks as if the endocrinologists prayer is about to be answered, for not only is an almost ideal system available, but combining as it does both endocrinology and tumour virology it should hit the double grant jackpot.

Recently it has been shown that physiological concentrations of glucocorticoids can stimulate the production of mouse mammary tumour virus (MMTV) in a number of cell lines established from mouse mammary adenocarcinomas (Parks *et al.*, *Science*, **184**, 158; 1974). This increase in virus production results from increases in virus specific RNA transcribed from the DNA provirus integrated in the host cell genome (Ringold *et al.*, *Virology*, **65**, 135; 1975; Parks *et al.*, *J. biol. Chem.*, **250**, 3330; 1975). Induction of this viral RNA is mediated through the classical steroid–receptor mechanism since binding of various glucocorticoids to the receptor and transfer of the receptor-bound steroid to the cell nucleus are correlated with the increase in virus production (Young *et al.*, *J. biol. Chem.*, **250**, 3337; 1975; Ringold *et al.*, *Cell*, **6**, 299; 1975). Whether the increases in virus specific RNA produced by glucocorticoids involves a decrease in RNA degradation or an increase in transcription (which seems more likely) is not yet known. This is a vital point since transcriptional compared with post-transcriptional control has been a contentious point in steroid action for some time (see News and Views, *Nature*, **258**, 477; 1975). In any event the action of the steroid appears to be direct since inhibitors of protein and DNA synthesis do not block increases in viral RNA, no general increase in RNA synthesis is seen, and the concentration of the virus specific RNA doubles within 30 min of steroid addition. This effect of glucocorticoids is not confined to B-type RNA tumour viruses (such as MMTV). Glucocorticoids also increase C-type RNA virus produced in cells treated with halogenated pyrimidines (Paran *et al.*, *Proc. natn. Acad. Sci. U.S.A.* **70**, 2391; 1973) and enhance the production of polyoma (DNA) virus from productively infected mouse embryo cells (Morhenn *et al.*, *Proc. natn. Acad. Sci. U.S.A.*, **70**, 1088; 1973).

What is particularly interesting about these systems is that steroids appear to be unable to initiate the transcription of virus sequences, and merely enhance the production of viral RNAs that are produced constitutively. Hence synthesis of C-type virus in KA31 non-producer cells is not induced by glucocorticoids but if virus is first induced with IUdr, virus production is increased many-fold by the hormone. Nor is constitutive production of viral RNA sufficient for a hormone effect since in murine epithelial cell lines constitutively transcribing both B-type and C-type virus sequences only the B-type transcripts are increased (Parks *loc.cit*). Thus it may generally prove to be that steroids are only gene amplifiers and not true inducers.

Glucocorticoid control over virus production is a particularly good model system because viral RNA can be quantitated and tumour virologists have all the tools necessary to characterise mutant cells defective in various stages of viral production. In the not too distant future it may prove possible to remove the DNA provirus from the cell, together with its steroid responsive control elements. □