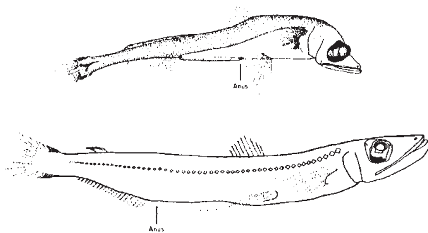

Post-larva (56.0 mm standard length) (above) and adult stage (124.0 mm standard length) (below) of *Benthalbella infans* (from Merrett *et al.*, *J. Zool., Lond.*, **170**, 1; 1973).

nescent organisms, or the silhouettes of prey against the lighted sea surface. It is suggested that during active feeding prey is stalked from below and struck at by a rapid backward arching of the forepart of the body. The head is assumed to be capable of an extra swing on account of the flexibility of the anterior vertebrae, thus widening the gape. The jaws are well equipped with teeth and *Benthalbella* is evidently capable of catching large prey as an adult, even if it does seize its prey by this half back-spring method.

Adult *Benthalbella* have also been found to have four ventral light organs which are lemon yellow in colour, but are not clearly visible, for the melanophores which lie in the epidermis covering them almost occlude the organ when expanded. This ability to mask the ventral lights could clearly be an advantage. The formation of these light organs is of a type rarely observed in deep-sea fishes, for they seem to be derived from muscular tissue and their luminescence is presumably controlled by modified muscle innervation. These light organs do not become fully developed until maturity and Merrett *et al.* suggest that their main function is specific recognition between sexually mature animals, the downward-pointing lights and upward-looking eyes being advantageous in a species with apparently solitary habits.

The discovery that *Benthalbella,* like other iniomous fishes, is hermaphrodite, with simultaneously maturing ovarian and testicular tissue, is perhaps only to be expected in this interesting fish which is so well equipped for productive encounters between specimens and for life in the deep sea.

GENETICS

# Mutagenic Effects

from a Correspondent

THE third meeting of the European Environmental Mutagen Society took place in Uppsala on June 4–7 and was opened by Dr B. A. Bridges (MRC Cell Mutation Unit, Brighton) who suggested some general principles of mutagenicity screening and proposed a three-tier framework for evaluating genetic hazards of new compounds. Compounds would be assayed qualitatively for mutagenicity by *in vitro* tests if consumed in small quantities (tier 1), or *in vivo* in animal systems if consumed in large quantities (tier 2). Food additives, medicines and some pesticides would fall into the second category. Substances found to be mutagenic by these tests, but considered to be of great value and having no known substitutes, would then be subjected to a quantitative risk/benefit analysis (tier 3).

Some of the difficulties likely to be encountered in such a thorough screening procedure became apparent in a contribution by Dr F. J. de Serres (National Institute of Environmental Health Sciences, NIH, Bethesda) who described a case study on hycanthone, a drug used by the World Health Organization for mass chemotherapy of schistosomiasis in the tropics. Hycanthone was found to be mutagenic, teratogenic and carcinogenic in a variety of systems. Attempts to find structural analogues which retain the desirable pharmacological properties but are free of undesirable toxicological properties, however, led to the discovery that the analogues were in some cases more, in others less, mutagenic than hycanthone itself.

Dr J. A. Miller (University of Wisconsin) discussed the relationship between mutagenicity and carcinogenicity. The active forms of carcinogens and mutagens are both electrophilic reactants, suggesting a strong formal relationship between the two processes, even though they may not be causally related. Hence many mutagens are likely to be potential carcinogens. Because most chemical carcinogens require metabolic activation, Dr Miller stressed the importance of including activation enzymes in mutagenicity test systems for detecting potential carcinogens. One way of carrying this out is by using host-mediated assays. An alternative new method, developed by Dr B. Ames, whereby the mutation test system includes a rat liver microsomal fraction to activate the potential mutagen as a substitute for the host-mediated assay, seems very promising. Such a rat liver system has also been used with success by Dr R. Barale (Institute of Genetics, Pisa) who included it in an assay to measure the production of gene conversions in yeast by ethyl methane sulphonate. Dr P. Brookes (Chester Beatty Research Institute, Pollards Wood) discussed the mechanism of the mutagenic

# $\beta_2$ Microglobulin Synthesis and IgG Production

ONE of the most esoteric of biological time perspectives derives from studies of molecular homologies. Histone proteins, for example, which are arguably the most ubiquitous proteins, are nearly completely identical in all species of plants and animals that have been studied. This probably means that the genes which code for the histones have remained largely unchanged since the parting of the plant and animal kingdoms hundreds of millions of years ago.

In *Nature New Biology* next Wednesday (July 11), Nilsson and his colleagues present another example of delving into the past by studying $\beta_2$ microglobulin which is a low molecular weight protein found in human biological fluids and, apparently, the urine of dogs. They point out that this protein is homologous with the constant domains of the light and heavy chains of immunoglobulin (IgG) and has, in addition, an intrachain disulphide loop of the same size and location as those found in the constant domain of IgG.

Nilsson *et al.* sought $\beta_2$ microglobulin on some twenty-nine different tissue culture cell lines—some lymphoid, others non-lymphoid. In carrying out this exercise they were trying to ascertain whether $\beta_2$ microglobulin was only to be found in association with cells of the leukocyte series and whether its synthesis could be in any way linked with the synthesis of IgG.

All the non-lymphoid cell lines produced considerable amounts of $\beta_2$ microglobulin. The lymphoid cells were usually more variable in the amount produced, one out of eight being consistently negative. Some lymphoma lines which did not produce soluble IgG nevertheless synthesized $\beta_2$ microglobulin. Nilsson *et al.* go to considerable trouble to show that the $\beta_2$ microglobulin produced *in vitro* is identical with that found in serum and urine.

Their overall conclusion is that there is no correlation between the capacity to synthesize immunoglobulin and the production of $\beta_2$ microglobulin. And yet the sequence similarity between IgG and $\beta_2$ microglobulin seems to point to an ancestral common gene. The four constant domains of the IgG are thought to demonstrate gene duplication and Nilsson *et al.* suppose that a similar duplication led to the now independent faculty for $\beta_2$ microglobulin synthesis. There is now more understanding of the biological significance of $\beta_2$ microglobulin, but this elegant demonstration that the regulation of its production is now divorced from the production of IgG promotes the biological time-traveller to wonder when the divorce occurred. Perhaps clues will emerge from a study of the sera of living representatives of the primitive vertebrates.

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