zyme is not confined to *Lactobacillus* (Hamilton, *J. biol. Chem.*, **249**, 4428; 1974), there is now little doubt that in mammalian tissues it is replaced by a vitamin B_{12}-independent version.

Ethanolamine deaminase is indubitably not a mammalian enzyme. It is found in clostridial bacteria, is vitamin B_{12} dependent and has been studied in great detail by Babior (for example *J. biol. Chem.*, **249**, 4537; 1974). Chang and Chang provide evidence on page 150 of this issue for the presence of the same enzyme in *Salmonella typhimurium* and perhaps also in other enteric bacteria. The enzyme, like the clostridial one, is probably induced as a response to the supply of ethanolamine as the sole source of carbon and nitrogen together with vitamin B_{12}. Exactly why these bacteria should have the potential to grow on such an unnatural duo of nutrients is puzzling.

Hormonal control of spermatogenesis

from R. V. Short

In reviewing the hormonal control of mammalian spermatogenesis, Steinberger commented that "knowledge of the biochemical parameters of the hormonal action on spermatogenesis is too meagre even for a limited attempt to formulate a working hypothesis" (*Physiol. Rev.*, **51**, 1; 1971). Research so often seems to be at a standstill, that it is refreshing to record a number of important advances in our understanding of spermatogenesis in the last four years.

There is now growing evidence from clinical (Franchimont *et al.*, *J. clin. Endocr.*, **34**, 1003; 1972; De Kretser *et al.*, *J. clin. Endocr.*, **38**, 787; 1974) and laboratory studies (Setchell *et al.*, *J. Endocr.*, **62**, 675; 1974) that the pituitary gland is somehow aware of the number of spermatozoa that the testis is producing. "Inhibin", a substance first postulated by McCullogh in 1932 (*Science*, **76**, 19; 1932), really seems to exist. It is apparently produced by the germinal cells of the testis, and acts centrally to suppress the secretion of follicle stimulating hormone by the pituitary gland. A number of laboratories throughout the world are actively working on the purification and identification of this new testicular hormone, and success seems near at hand.

We are also beginning to get a clearer idea of the role of the Sertoli cells in the control of spermatogenesis. These cells, which line the seminiferous tubules of the testis, are in intimate association with the germ cell line at all stages of their development, from spermatogonia to spermatozoa. This close proximity suggests that there could be an important functional interrelationship between germinal and somatic cells, but hitherto we have lacked any biochemical understanding of the nature of this relationship. Thanks to the collaborative work between Drs Hansson (Oslo), Ritzen (Stockholm) and French (Chapel Hill, North Carolina), we can now begin to understand exactly what is happening.

It seems that the Sertoli cell is stimulated by follicle stimulating hormone to secrete an androgen-binding protein with a high affinity for testosterone and its biologically active metabolite, dihydrotestosterone (*Nature new Biol.*, **246**, 56; 1973; *Nature*, **250**, 387; 1974). As Hansson and his colleagues point out in this issue of *Nature* (page 145), testicular androgens are known to be the principal stimulus for spermatogenesis; they seem to be required for almost all the stages of meiotic division (Steinberger, *Physiol. Rev.*, **51**, 1; 1971). In normal circumstances, luteinising hormone secreted by the pituitary gland acts on the interstitial or Leydig cells of the testis to induce testosterone secretion. Though much of this hormone then enters the systemic circulation, the intratubular androgen binding protein may be a vital link in the chain, enabling some hormone to be captured and concentrated within the lumen of the seminiferous tubules, and subsequently transported to the germ cells where it will stimulate meiosis.

It has long been known that it is possible to maintain complete spermatogenesis in hypophysectomised animals if they are placed on testosterone therapy immediately after the operation. But if the testes are allowed to regress following hypophysectomy, testosterone alone will not restore spermatogenesis, and follicle stimulating hormone is required in addition. The present studies suggest that this is because testosterone is only capable of maintaining the secretion of androgen-binding protein by fully developed Sertoli cells.

If the male germ cells make inhibin which regulates follicle stimulating hormone secretion, and if this in turn controls the rate of spermatogenesis by influencing the secretion of androgen-binding protein from the Sertoli cells, we have a subtle biochemical interplay between the germinal and somatic elements of the testis. But what of the ovary? The anatomical relationship between the cumulus cells and the oocyte is reminiscent of that between the Sertoli cells and the spermatogonia. Does the mature oocyte produce the female equivalent of inhibin, and hence regulate gonadotrophin secretion? Do pituitary gonadotrophins act on the granulosa cells to influence secretions which in turn regulate maturation? Little thought has been given to such problems, and yet they might add a new dimension to our understanding of the complex sequence of endocrine events leading up to ovulation.

What are the parents?

from W. T. Toner

New ideas are needed to explain muon to pion production ratios as large as 10^{-4} in high energy proton interactions. Unlike pions, muons and electrons are not strongly interacting and should not be seen in these collisions, except as rare decay products of strongly interacting parents.

Anomalously high production of large transverse momentum muons and electrons was reported last summer by several groups in the USA, at CERN and in the USSR, working with protons at energies above 70 GeV. Roughly equal amounts of μ^+, μ^-, e^+ and e^- are observed. In a recent issue of *Physical Review Letters* (**34**, 103; 1975) Leipuner *et al.* claim a similar result for muons with no transverse momentum, in proton–nucleus collisions at only 28 GeV.

It is ironic that this latest result comes from a re-analysis of a 1969 experiment which showed the absence of one hundred times larger amounts of 'direct' muon production, then claimed by a Utah group to take place in cosmic ray interactions. Those claims found no support and were later withdrawn. At that time, there was no compelling reason to make a painstaking analysis of the small residue, which was thought to come from the rare electromagnetic decays of the ρ^0, ω^0 and φ^0 vector mesons, strongly interacting analogues of the photon. But models based on this idea require that most of the pions observed at the same energies and angles as the muons originate in the other decays of these same three particles: the ρ^0, ω^0 and φ^0 have to be singled out from a hundred and one other ways of generating the ubiquitous pion.

Gordon *et al.* (*Phys. Rev. Lett.*, **34**, 284; 1975) suggest that this may happen at large transverse momentum, if the rapid increase that they observe in the yield of ρ^0 relative to pions continues beyond 1 GeV/c transverse. There is an intuitive appeal to such an idea, since the production of all types of particles at large transverse momentum is unexpectedly high and shows features suggestive