SUMMARY
1. Prolactin caused induction of ODC mRNA and enzyme levels in the liver and kidney of adult male rats.

2. The extent to which PRL induced ODC mRNA expression was greater in castrated than in intact rats. However, the reverse was seen in the case of ODC activity. This suggested a possible effect of testosterone on PRL induction of ODC in kidney.

3. The action of prolactin in enhancing renal ODC activity and expression involved the process of transcription.

4. In kidney and liver, treatment with cycloheximide inhibited the action of prolactin in enhancing ODC activity, indicating that de novo protein synthesis is necessary for this action of prolactin.

5. Inhibition of diacylglycerol formation by the phospholipase C inhibitor quinacrine caused an inhibition of prolactin induction of ODC, indicating that this effect of prolactin may be mediated via this pathway.

6. Further confirmation of the above suggestion was made by the observation that the protein kinase C inhibitor quercetin was also able to block the effect of prolactin in inducing ODC. Thus, in the liver and kidney, prolactin seems to act via the inositol phosphate-protein kinase pathway to induce ODC.

7. The effects of growth hormone in the liver and kidney were investigated and this hormone was shown to induce both ODC
activity and expression in these organs of the rat.

8. The effects of growth hormone in inducing ODC mRNA expression were much higher in intact than in castrated rats, indicating an effect of testosterone similar to that seen in the case of prolactin.

9. Ongoing transcription was observed to be necessary for the action of GH on ODC.

10. Cycloheximide treatment confirmed that the effect of GH in enhancing ODC required de novo protein synthesis.

11. Testicular ODC activity was observed to decrease with the age of the rat, whereas ODC mRNA expression showed an opposite trend.

12. Human chorionic gonadotropin (hCG) enhanced ODC activity and mRNA expression in the testis of immature rat.

13. Desensitisation of the ODC response to hCG was seen to occur at the levels of both ODC activity and mRNA expression.

14. The effect of hCG on ODC was not mediated via diacylglycerol formation.

15. Follicle-stimulating hormone was seen to induce both ODC activity and expression in the immature rat testis.

16. The effect of FSH, like that of hCG, was not mediated via
diacylglycerol formation.

17. PRL stimulated ODC in the testis during the period between immaturity to puberty.

18. The effect of PRL on ODC in the testis was mediated via diacylglycerol formation, as indicated by the use of the phospholipase inhibitor, quinacrine.

19. Growth hormone induced ODC in the rat testis between immaturity and puberty.

20. The effect of GH was observed to occur in both the seminiferous tubules and Leydig cells.

21. The effect of GH on ODC in testis is mediated via stimulation of diacylglycerol formation.