locytes. For practical purposes discrimination of the two reticulocyte types is unnecessary.

Wintrobe[17] has shown that the increased mean corpuscular volume during liver therapy is due to an outpouring of reticulocytes from the marrow. This marked increase in corpuscular volume can also be more fully appreciated when it is considered that part of the cells are reticulated megalocytes which are larger than normal reticulocytes.[15]

The participation of reticulated megalocytes in the reticulocytosis of pernicious anemia patients following liver therapy makes this response qualitatively distinct and different from that in hypochromic anemias after iron therapy, since it is well established that marrows from the latter do not possess megaloblasts. In like manner, the reticulocyte response in guinea pigs after the administration of liver extract[18] is qualitatively different from that in pernicious anemia patients since these animals do not have megaloblasts in their marrow.[19] Reports to the contrary by Gall[20] are due to the fact that he has adopted Sabin's definition of the term megaloblast which indiscriminately includes primitive erythroblasts, promegaloblasts and pronormoblasts (proerythroblasts).

Conclusion. After liver therapy megaloblasts complete their maturation as reticulated and mature megalocytes. The resultant reticulocytosis is composed of reticulated megalocytes and normal reticulocytes. This not only indicates the reëstablishment of normal erythropoiesis but also, that the marrow is being purged of its pathologic red cell series, the megaloblasts. This response is qualitatively distinct from the reticulocytosis of hypochromic anemias following iron therapy and the one in normal laboratory animals after liver treatment.

9799 P

Gonadotropic Hormones in the Hereditary Dwarf Mouse.

ALFRED MARSHAK. (Introduced by Shields Warren.)

From the Laboratory of Pathology, New England Deaconess Hospital, Boston.

Snell[1] described dwarfism in the mouse which was inherited as a

17 Wintrobe, M. M., *J. Clin. Invest.*, 1934, **13**, 669.

18 Jacobson, B. M., *J. Clin. Invest.*, 1935, **14**, 664.

19 Jones, O. P., *J. Lab. and Clin. Med.*, 1936, **21**, 335.

20 Gall, E. A., *Am. J. Path.*, 1937, **13**, 575.

1 Snell, G. D., *Proc. Nat. Acad. Sci.*, 1929, **15**, 733.

recessive character. Smith and MacDowell[2] later showed that, in these mice, the eosinophiles were missing from the anterior lobe of the pituitary and that the growth hormone was either not present at all, or else was produced in amounts much less than in the normal. Transplantation experiments[3] were interpreted as indicating that pituitaries of the dwarfs contained a higher concentration of gonadotropic hormone than the normal. Nevertheless, sexual development was markedly retarded in both male and female, copulation and reproduction being impossible unless normal pituitary lobe was supplied by implantation or injection. Thus, it was difficult to understand why the gonadotropic hormone of the dwarf did not give the proper stimulus to its own gonads and accessory sex glands unless a synergistic action of the growth hormone was involved. These experiments were performed with relatively young mice and another interpretation seems possible from observations on older dwarfs which are to be described below.

Heterozygous parents were obtained from Snell in 1933 and dwarfs bred from these. In addition 2 dwarf males and 6 dwarf females were obtained from MacDowell in 1936. In all of 6 males observed after they had reached an age of a year or more priapism had developed. One such animal was given 2 injections of a suspension of beef anterior lobe at 2-day intervals and the priapism disappeared within a week after the first injection. In 2 animals which were kept untreated for 17 months, the priapism was maintained constantly for the last 6 months. These untreated dwarfs were then sacrificed and were found to have very strikingly enlarged seminal vesicles. Microscopic examination showed that the enlargement was due to retained secretion. In spite of very marked distension of the glands, their epithelial lining appeared to be of the normal columnar type and showed the usual secretion granules. There was little or no difference between normal and dwarf mice in the number of interstitial cells of the testis or the amount of spermatozoa in the testis and epididymis. In the dwarf the vas deferens contained sperm and had an apparently normal epithelium; the urethra was filled with semen containing occasional spermatozoa.

Female dwarfs of the same age had several follicles in each ovary. However, these became atretic before there was any very marked antrum development.

The experiments of Greep and Fevold[4] show that the luteinizing

2 Smith, P. E., and MacDowell, E. C., *Anat. Rec.*, 1930, **46**, 249.

3 Smith, P. E., and MacDowell, E. C., *Anat. Rec.*, 1931, **50**, 85.

4 Greep, R. O., and Fevold, H. W., *Endocrinol.*, 1937, **21**, 611.

hormone of the pituitary (LH) when injected into hypophysectomized males will produce pronounced enlargement of the seminal vesicles and will stimulate, probably indirectly, gametogenesis; but the follicle stimulating hormone (FSH) will not induce any enlargement of the seminal vesicles. In the female Foster and Hisaw[5] have shown that FSH will stimulate the development of follicles only after they have developed antra. Epithelioid cells atrophy after hypophysectomy and enlarge again after injections of LH.[6] Thus, if the above mentioned conclusions be accepted, the enlarged seminal vesicles and the correction of priapism with anterior lobe injections in the male dwarf mouse may be taken to indicate the presence of LH and a partial or complete deficiency of FSH; and in the female, likewise, the presence of large numbers of epithelioid cells and the development of follicles only to medium size again indicate the presence of LH and a deficiency of FSH. It seems probable, therefore, that the dwarf mouse not only lacks growth hormone, but has a partial or complete deficiency of at least one of the gonadotropic hormones.

9800

Method for Separation of Micro-organisms from Large Quantities of Broth Culture.

JONAS E. SALK. (Introduced by R. Keith Cannan.)

From the Department of Chemistry and the Department of Medicine, New York University College of Medicine.

This paper contains a report of a rapid and simple method for removing bacteria from broth cultures. Large volumes can be handled conveniently. Preliminary tests indicate that the antigenic properties of the bacteria remain unaffected.

The method consists in the formation of a precipitate of calcium phosphate in the broth. This adsorbs the bacteria, settles quickly, and can be removed by siphoning away the supernatant liquid and lightly centrifuging the remaining sediment. The calcium phosphate may then be redissolved by suspending the precipitate in water and acidifying to about pH 5.0. The bacteria can be recovered by centrifugation and then washed.

---

5 Foster, M. A., Foster, R. C., and Hisaw, F. L., *Endocrinol.*, 1937, **21**, 249.

6 Hisaw, F. L., personal communication.