As yet we have no data on the exact time of determination of the melanophores in hair follicles, or, which in the end is the same, on the time of differentiation of the Golgi structures characteristic of hair matrices capable of forming melanins. From what has been said above, it is reasonable to expect, however, that this differentiation (after migration, perhaps) will take place by the time that factors *W*, W^v and *f* exert their action. If so, a deficiency in a copper compound, necessary both to hæmoglobin production and to the synthesis of phenolase, will have the effect of causing anæmia and a lack of pigmentogenesis in the hair matrices now in differentiation. The difference between the actions of *W*, W^v and of *f* probably depends upon the time of action and the intensity of the two kinds of factors, the first acting more early and hindering, therefore, all differentiation of melanoblasts (in heterozygotes sometimes only partially), while by the time *f* comes into action only the melanophores of the median belly line would be yet undifferentiated. The flexed tail of the *ff* mice probably is a secondary effect due to the anæmia being at its peak exactly when the fibrous tail structures are differentiating. This seems more probable when it is considered that in mice several factors affect the tail as secondary effects.

The copper compounds upon which the primary action of *W*, W^v and of *f* is exerted will be perhaps related in the two cases. As the steps in the synthesis of copper proteins are not known, we must suppose that the compounds involved in the two anæmias and in depigmentation will be perhaps intermediates between ionic copper and cupro-proteins. The primary action of *W*, W^v and *f* would be a hindering, more transitory in *f*, of the synthesis of these compounds.

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Relation of the Thyroid to Infections

Izzo and Cicardo[1] have reported an inhibiting effect of thyroxine on experimental tuberculosis in guinea pigs. The experiments were suggested by the chemical resemblance of thyroxine to diploicin, which exerts a bacteriostatic action on *Mycobact. tuber.* But the authors envisaged also the possibility of an unspecifically stimulating effect of the thyroid as an explanation of their experimental results. I should like to direct attention to some earlier observations on the influence of the thyroid on infections in general and particularly on tuberculosis.

Kallos[2] reported on human cases of hypothyroidism suffering from severe pulmonary tuberculosis which exhibited a lowered sensitivity to tuberculin. Treatment with thyroid extract improved the condition and raised at the same time the tuberculin sensitivity. Later, Kallos and Müller[3] found that percutaneous tuberculin tests in general are liable to be intensified by the administration of thyroxine. Bettini[4] observed favourable effects of treatment with thyroid extract on laryngeal tuberculosis.

The observations of Kallos and Müller suggest rather a systemic stimulation of the defence mechanisms than a specific bacteriostatic action. An unspecific stimulation in infectious diseases was attributed to the thyroid in 1929 by Guillaume[5]. Lauber[6] achieved an increased resistance against acute infections by administration of thyroid extract, and Weichardt[7] an inhibition of growth of transplanted tumours. In typhoid patients, Reitler[8] observed a marked rise (two- to fourfold) of the O- and H-agglutinin titre after three days administration of small doses of thyroid extract (twice daily 0·025 gm.), while controls in the same stages of disease showed no titre change after so short a time. Larger and smaller doses had a less marked effect. Such an optimum has been described already by Thaddea[9] in respect of the increased erythro- and leucopœsis caused by thyroxine.

An unspecific factor is, therefore, certainly involved in the effect of thyroxine on the course of infections. As Weichardt thought, it is probably due to the increase in cellular metabolism (personal communication). But it may be that in the particular case of tuberculosis this unspecific stimulation combines with an antibiotic action on the causative organism.

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Chromosome Numbers in Cancer Cells

DURING an investigation on the variation of the chromosome system in human cancer of the uterus, we were able to observe frequently (in six epitheliomas of the uterus portio) a mitotic figure with a peculiar aspect of the chromosomes. In such cell divisions (which appear mixed with normal, heteroploid and polyploid ones) 24 chromosomes are present. Their shape resembles the meiotic one, being strongly contracted. The prophase shows thick threads, as in the pachytene stage, and later the synaptic males appear unsplit. No chiasmata are to be seen; the metaphase shows that every element is double.

It seems likely that the result of these divisions may be a reduction of the chromosome number. The cells in which the phenomenon appears are those termed *A* by Caspersson and Santesson[1].

This finding may be connected with the observation in 1906 of Farmer, Moore and Walker[2], and with that by Evans and Swezy[3]. But while the former is generally held to be doubtful, the latter concerns tetraploid cells, where the reduction to haploidy is impossible with a single mitosis. Haploid cells, on the other hand, were actually observed by Koller[4]. Finally, meiosis-like figures were found by Hearne Creech[5] in cultivated mouse fibroblasts previously treated with carcinogenic substances.