Star counts ; rel. absorption		No. of stars with colour excess	Mean dist. (parsecs)	A	E	A/E	$\dfrac{A'/E}{A'=}$ $A+0\cdot4$ mag.	$\dfrac{A''/E}{A''=}$ $A+0\cdot7$ mag.	$\dfrac{A''/E}{A''=}$ $A+1\cdot0$ mag.
in log N	in m								
<0·09	<0·25	15	1004	0·15	0·14	1·1	3·9	6·1	8·2
0·09–0·18	0·25–0·50	12	982	0·39	0·23	1·7	3·4	4·7	6·0
0·18–0·36	0·50–1·00	38	1021	0·78	0·22	3·5	5·4	6·7	8·1
0·36–0·54	1·00–1·50	13	966	1·36	0·33	4·1	5·3	6·2	7·1
>0·54	>1·50	8	926	2·31	0·50	4·6	5·4	5·9	6·5
		Mean	995	0·85	0·25	3·4	5·0	6·2	7·4

electric colour excesses of stars between 800 and 1,200 parsecs were used, the distance-moduli being those of Stebbins, Huffer and Whitford[4]. Their colour excesses (E_1) were increased by 50 per cent to reduce them to the International System (E). The stars were grouped according to the degree of obscuration, as shown in the accompanying table.

Column 7 gives the first approximation of A/E. Its systematic dependence on A suggests a zero-point correction to the latter. This is not surprising, since the values of A were derived by assuming the absence of obscuring clouds for certain of the brightest Milky Way regions, and making no allowance for general absorption in these directions. The last three columns of the table give values of A/E on the assumption of a general absorption coefficient of 0·4, 0·7 and 1·0 mag. per kiloparsec respectively.

With an absorption coefficient of 0·7 mag./k.parsec, the systematic dependence of A/E on A disappears. A value of $A/E = 6$ would therefore be indicated from the present data.

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[2] van Rhijn, Groningen Pub. 51 (1946).
[3] Stebbins, *Harvard Obs. Mon.*, No. 7, 6 (1948).
[4] Stebbins, Huffer and Whitford, *Astrophys. J.*, **91**, 20 (1940).

Effect of E-Avitaminosis on the Histiotrophic Nutrition of the Mouse Embryo

A MAMMALIAN embryo obtains its nourishment in two ways, hæmotrophically and histiotrophically. By the former is meant feeding through the placenta, the previously broken-down foodstuffs being transferred osmotically from the maternal blood-system to the embryo ; the latter, on the other hand, means that nutriment is transferred, after breakdown by certain embryonic organs, to the metabolism of the embryo, by the secreting activity of the mucous membrane of the uterus.

Histiotrophic nutrition is more primitive than hæmotrophic. In the simpler placental types the nutrition of the embryo is entirely histiotrophic. As one approaches more developed placental types, hæmotrophic nutrition becomes more and more important. Histiotrophic feeding occurs, however, in combination with hæmotrophic, in all higher placental types also, at least in the earlier period of development of the embryo.

S. Pesonen showed in 1942[1] that histiotrophic nutrition plays a great part in the embryonic development of the hedgehog, and that progesterone from the corpus luteum causes the secretion of 'histiotrophe'. The chief sites of formation of histiotrophe are those parts of the mucous membrane of the uterus which lie between the embryos, and remain modified throughout pregnancy, and where thus no decidua tissue is being formed. In collaboration with Miss Kaarina Vaalanto, I have established that the mouse embryo also uses histiotrophe as its nutriment through-

out pregnancy, although this mode of nutrition is not so obvious as in the hedgehog. The histiotrophe is transferred to the embryo through the decidua capsularis and yolk-sac. The secretion of histiotrophe throughout pregnancy is due to the corpus luteum remaining active during this whole period.

E-avitaminosis in a mouse results in the offspring being stillborn, in miscarriages and resorption of the embryos. Changes in the placenta seem not to be so great that they could prevent hæmotrophic feeding and so be the reason for the discontinuance of pregnancy. According to the investigations I have carried out in collaboration with Miss Elli Laine, there seems to be less histiotrophe in the uterus of pregnant E-avitaminotic mice than in that of pregnant healthy ones. It reaches its highest value, both in the E-avitaminotic and in the normal animals, on the eleventh and twelfth days. After the thirteenth day, there is very little or no histiotrophe in E-avitaminotic mice. The modification of the mucous membrane of the uterus in E-avitaminosis is also less marked than that of the endometrium of normal uteri. The epithelium, especially, of the mucous membrane is less altered. In addition, there is little secretion present in the glands.

The investigations of Miss Elli Laine have also shown that the amount of ionized iron (determined histochemically) in the uterus begins to decrease in E-avitaminotic mice before the resorption of the embryo. Just prior to the beginning of the resorption of the embryos and during this process, it appears in especially large quantities in the yolk-sac.

A fuller report of these investigations will be published later.

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Sinus Gland and Tyrosinase Activity in Carcinus mœnas

IT has been shown[1] that the crustacean cuticle undergoes at each moult a tanning process similar to that taking place in insects. Tyrosine and tyrosinase, which are involved in the tanning of the insect cuticle, are known to occur also in the blood of decapod Crustacea[2,3], and it is therefore of interest to discover whether as in insects there exists a neurosecretory regulatory mechanism for the control of tyrosinase activity in relation to moulting. In the blowfly larva, the oxidation of tyrosine under the influence of tyrosinase activity is modified by the low redox potential of the blood resulting from the action of a glucose dehydrogenase, itself controlled by Weismann's ring, a complex gland near the brain[4].

A study of tyrosinase activity in *Carcinus mœnas* has shown that marked variations occur in the course of the moult cycle, although the blood tyrosine remains more or less constant (0·003 per cent) at all