In contrast to their altered physical behavior, the blood platelets during menstruation exhibit no deficiency in their lipid composition. In fact, a greater proportion of the clot-aiding type of phospholipid, cephalin, appears to be present. No anomaly could be detected in the lipid composition of platelets from the blood of hemophiliacs; a discussion of other factors which must be investigated in relation to the physical behavior of blood platelets has been given.[7]

Summary. A definitely delayed platelet disintegration rate in menstruation is demonstrated by these studies. Hemophilia also shows similar changes. Lipid analysis of the platelets demonstrates no deficiency of cephalin in either menstruation or hemophilia.

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Time of Death of Lethal Homozygotes in the *T* (Brachyury) Series of the Mouse.*

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Dunn[1] reported the third lethal that was found in the *T* (Brachy) series in the house mouse. This lethal (t^1) was determined by Dunn in a tailless line, which had been isolated by Dobrovolskaia-Zavadskaia and Kobozieff.[2] Tailless mice of this line (29) are Tt^1. Results of matings of tailless mice of line 29 and tests of their descendants showed that both homozygous combinations TT and t^1t^1 are lethal, but the combination Tt^1 is viable.

In order to learn more about the lethal embryos t^1t^1, uteri of pregnant females were examined in different stages. The females used were all heterozygous normal-tailed mice ($+t^1$) and they were mated to heterozygous normal-tailed males ($+t^1$).

Table I shows the results of dissections of pregnant females at the age of 7, 8, 9, 10, and 11 days after fertilization. A total of 40 litters were dissected out, yielding 294 embryos. Of these, 275 were normal and 19 abnormal. Of the abnormal embryos, 13 were resorbed and 6 showed different kinds of abnormalities, but were not resorbed. If the t^1t^1 homozygotes died after implantation,

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1 Dunn, L. C., *Proc. Nat. Ac. Sc.*, 1937, **23**, 474.

2 Dobrovolskaia-Zavadskaia, N., and Kobozieff, N., *C. R. Soc. Biol.*, 1932, **110**, 782.

TABLE I.

Exp.	Age of embryos	No. of litters	No. of embryos	Normals	Resorbed
	days				
1	7	8	51	49	2
2	8	13	101	97	4 (2?)†
3	9	6	40	38	2
4	10	8	59	54	5
5	11	5	43	37	6 (4?)
Total		40	294	275	19 (6?)

† Figures in parentheses represent abnormal, but not resorbed embryos.

25% of the implanted embryos would be expected to be of the lethal type. Since the number of resorbed embryos found at dissection is far below expectation, it has to be assumed that death of the homozygous lethal takes place before implantation, and that the death of the resorbed and abnormal embryos found was due to accidental causes. It is highly improbable that death of the t^1t^1 takes place after the 11th day. All embryos examined at 11 days were entirely normal and no indications were found for later death of t^1t^1.

The litter size in the matings is another factor indicating that death of t^1t^1 occurs before implantation. The average litter size in matings of heterozygous normal-tailed mice of the tailless A line *inter se*—where all embryos are implanted—was found to be 9.8 (taken from 14 litters), the average litter size in matings of $+t^1$ normals *inter se* was 7.4 (taken from 40 litters). If the t^1t^1 homozygotes died before implantation one would expect the number of implanted embryos to be 25% smaller than it would be if *all* embryos were implanted. The figures found correspond very well to expectation.

The study of litters from matings of heterozygous normal-tailed mice in the A line *inter se* ($+t^0 \times +t^0$) confirmed Chesley's observation that death of the homozygous lethal in the A line occurs after implantation (Chesley and Dunn).[3] Of 170 embryos at ages from 6-8 days that were examined in dissections and histologically so far, 115 were found to be normal, the rest abnormal or resorbed. Death of t^0t^0 apparently takes place on the 6th day of development. The details of this investigation on the t^0-homozygote will be described in a later publication.

This investigation was undertaken at the suggestion of Dr. L. C. Dunn, whom I should like to thank for his help during the course of the work.

3 Chesley, P., and Dunn, L. C., *Genetics*, 1936, **21**, 525.