histones prior to staining decrease uniformly the stainability of the chromatin and nucleolus of both the plants. However, with eosin Y compared to the normal plants, the asynaptic plants show a deeper staining of both chromatin and nucleolus. Moreover, acetylation prior to eosin Y staining removes the stainability of the mutant more strongly. These results indicate that the asynaptic mutant is richer in a labile histone fraction which is not detected by alkaline fast green staing after TCA hydrolysis, which removes labile histones. It is also clear that this fraction is lysine-rich since it is affected by acetylation. The staining reaction of the microsporocytes also lead to the same conclusion. In contrast to the root-tip cells, the microsporocytes show no appreciable coloration of the chromatin material of normal and mutant plants with any of the stains used. However, the nucleoli stain very well and, with bromphenol blue and eosin Y, the nucleoli of the mutant show more intense staining than those of the normal plants. When deamination or acetylation is carried out before staining, the loss of stainability is more from the mutant than from the normal. Similarly, eosin Y stainability of the mutant nucleoli is more susceptible to acetylation. Thus it is concluded that the asynaptic mutant of *Phaseolus mungo* contains an excess amount of histone which is labile and lysine-rich.

One of the possible roles of histones is that they are involved in coiling and condensation of chromosomes[5,6]. Moreover, it is the lysine-rich histone fraction which cross-links DNA-containing fibres to form condensed chromosomes[7]. Normally the pairing of homologous chromosomes takes place when the chromosomes are highly attenuated. Hence an excess of lysine-rich histone in meiotic prophase may induce precocious condensation leading to asynapsis. Alternatively, excess histone may mask the macromolecules which are responsible for pairing and cause asynapsis.

Zusammenfassung. Die Wurzelspitzen und meiotischen Prophasezellen normaler und asynaptischer Pflanzen von *Phaseolus mungo* wurden nach Azetylierung oder Desaminierung der Histone mit alkalischem Fastgreen, Bromphenolblau und Eosin Y gefärbt. Die asynaptischen Mutanten ergaben einen erhöhten Wert an labilen lysinreichen Kernhistonen.

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Additional Studies on the Effect of the Lactate Dehydrogenase Virus on Murine Sex Ratios[1]

Previously, Crispens[2] reported a significant alteration in the sex ratio of weanlings born to C57BL/Fg mice infected with the lactate dehydrogenase (LDH) virus[3] for 10–19 days before conception. The present paper will describe results obtained in further studies of this phenomenon.

Materials and methods. Adult male and female C57BL/Fg mice were given an i.p. injection of 0.1 ml of mouse plasma containing $10^{7.0}$ ID_{50}/ml of the LDH virus. 10 days later, they and the controls, which received an equivalent amount of saline, were mated with nontreated animals (2 males and 5 females per cage). Pregnant females were caged singly on the 17th to the 18th day of gestation; the number of babies born to each was recorded at birth. Dead animals, found during twice-daily observations, were examined for sex as well as gross pathological lesions. When 5 weeks of age, all progeny were weaned and sexed. The weanlings and their parents were then bled, and the units of LDH in plasma samples were determined as described previously[4].

Results. Table I shows that the sex ratio among the offspring of infected females mated to nontreated males was identical to that observed in control mice (51:49). By contrast, in the case of nontreated females mated to infected males, the sex ratio among their progeny was 43:57 (chi square, 7.15; $P < 0.01$). This finding of a significant difference between the 2 types of matings indicates that the alteration in sex ratio stems from a response of the male parent to the LDH virus. As such, the observation that none of the weanlings were infected in the 'male infected' group (Table I) takes on added importance since it both: (1) provides confirmation of results reported in an earlier paper[5]; and (2) suggests that the response occurs prior to fertilization.

In an attempt to learn more about the nature of the response, adult male C57BL/Fg mice received 5 i.p. injections, at 24 h intervals, of 20,000 units of normal mouse liver LDH[6]. Matings with nontreated females were established at 8 rather than 10 days since infected animals do not show the characteristic increase in plasma LDH activity until 36–48 h after virus inoculation[7]. Otherwise, the method of procedure was as described above.

The results are presented in Table II. It can be seen that the sex ratios among the offspring of nontreated females mated to enzyme-injected and saline-injected males were 45:55 and 51:49 respectively. While the difference between the 2 types of matings is not significant (chi square, 1.83; $P > 0.10$), it should be emphasized that

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Table I. Sex ratios derived from maternal and paternal infection with the lactate dehydrogenase (LDH) virus

Items	Experimental mice		Control mice
	Dam infected[a]	Sire infected[a]	
Litters	36	54	50
Babies	239	326	310
Weanlings	198	276	270
Infected weanlings (%)	60	0	0
Male: female offspring (number)	111:105[b]	128:168[c]	148:142[d]
Male:female sex ratio (%)	51:49	43:57[e]	51:49

[a] Intraperitoneal injection of 0.10 ml of plasma containing $10^{7.0}$ ID_{50}/ml of LDH virus at 10–19 days before conception. [b] The sex of 23 offspring could not be determined. [c] The sex of 30 offspring could not be determined. [d] The sex of 20 offspring could not be determined. [e] $P < 0.01$.

it was not possible to maintain a constant enzyme elevation by periodic administration of homologous LDH. Accordingly, this finding should be regarded as partial or inconclusive support for the assumption that the level of plasma LDH in the male parent is not a factor in the alteration of the sex ratio[8].

Résumé. On met en évidence le fait que le virus LDH exerce un effet sur la proportion des sexes chez les souris par l'intermédiaire du père. En ce qui concerne le mécanisme impliqué dans ce phénomène, les résultats obtenus jusqu'à présent semblent insuffisants pour permettre une conclusion.

Table II. Sex ratio derived from paternal administration of lactate dehydrogenase (LDH)

Items	Experimental mice[a]	Control mice
Litters	27	20
Babies	175	124
Weanlings	131	108
Male:female offspring (number)	63:77[b]	59:57[c]
Male:female sex ratio (%)	45:55[d]	51:49

[a] Each sire received 5 i.p. injections of homologous mouse LDH (20,000 U/24 h) beginning at 8–12 days before conception. [b] The sex of 35 offspring could not be determined. [c] The sex of 8 offspring could not be determined. [d] $P > 0.10$.

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Effects of Temperature on the Development of Scutellar Bristles

The scutellum of *Drosophila melanogaster* flies usually has 4 macrochaetes, 1 at each of the 2 anterior corners and 2 close to the posterior margin. In flies with more than 4 scutellar bristles the extra bristles can occur at these posterior and anterior sites or on the dorso-lateral margins of the scutellum between the anterior and posterior sites (interstitial bristles)[1].

The number and positions of extra scutellar bristles can be modified by the temperature at which the flies are cultured[2]. Not all stocks of *D. melanogaster* exhibit these temperature effects but when they are present the mean scutellar bristle number is inversely related to the culture temperature. In addition flies cultured at 29 °C often have a higher proportion of posterior bristles and fewer anterior bristles than flies from the same stock cultured at lower temperatures.

The mean scutellar bristle number of a wild-type stock (Athens), maintained at 25 °C was 4.56, 61.0% of the extra bristles were interstitial, 34.7% anterior and 4.3% posterior. Culturing the Athens stock at 20 °C had no effect on the proportion of flies with extra bristles or on the mean bristle number but the proportion of extra anterior bristles increased to 49% (Table I). At 29 °C the Athens stock had a lower mean scutellar bristle number and 25% of the flies had extra bristles, all in posterior positions. 10% of the flies cultured at 29 °C had less than 4 scutellar bristles and without exception the bristles were missing from anterior sites.

Male flies from the Athens stock were crossed to females carrying the second and third chromosome markers *Pm* and *Mé* respectively. The resulting *Pm*/+, *Mé*/+ flies were intermated and their progeny cultured at 20 °C. Male and female progeny of each of the 4 marker genotypes were assayed to determine the effect on scutellar bristle number of Athens second and third chromosomes.

These data (Table II) show that although the mean scutellar bristle number of the Athens stock is the same

Table I. Effect of culture temperature on the scutellar bristle number of the Athens stock

Culture temperature	Joint mean scutellar bristle numbers of females and males.	% flies with extra bristles	% extra bristles at the 3 sites		
			Posterior P	Anterior A	Interstitial I
20 °C	4.5	46.4	–	49.0	51.0
25 °C	4.5	42.3	4.3	34.7	61.0
29 °C	4.3	25.0	100	–	–

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