The anomeric compositions of D-glucose in sample solutions became almost the same as the values (35.6% α, 64.4% β) of equilibrium D-glucose in all cases of tissues, and blood samples after sample solutions were kept for 24 h at room temperature (20 °C) to cause D-glucose to mutarotate completely. This result supports the accuracy of the percentage values in the table obtained on fresh sample solutions.

Our colorimetric method[12] for determining D-glucose anomers was tested instead of the present oxygen electrode method, but it was not utilizable for this study because of the interference of color development by reducing substances contained in sample solutions.

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Effect of pentobarbital on the synaptosomal activity of acetylcholinesterase in Mongolian gerbils

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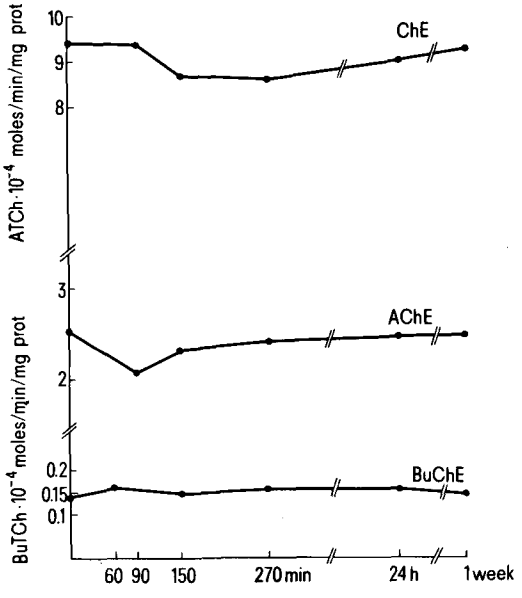
Summary. The influence of Na pentobarbital anesthesia on the activity of specific and nonspecific cholinesterase was studied in the synaptosomal fraction of Mongolian gerbils' brains. These studies have shown that this barbiturate inhibits the specific activity of acetylcholinesterase only.

Barbiturate reduction of either specific or nonspecific cholinesterase was not observed in early postanesthetic period although high concentrations of phenobarbital were described to inhibit in vitro the hydrolysis of acetylcholine in rat brain[1] and in vivo studies to raise the acetylcholine level in rats and cats[2]. In this communication we will describe a transient effect of pentobarbital on the activity of acetylcholinesterase in synaptosomal fraction of Mongolian gerbil's brain.

The gerbils (50–75 g) were anesthetized by i.p. Na pentobarbital injection (20 mg/kg b.wt) and killed at various intervals from 90 min to 1 week following the treatment. Each group consisted of 6–12 experimental and control animals. After decapitation the brains were removed quickly and transferred immediately in 0.32 M sucrose at pH 7.0. The synaptosomal fraction was prepared according to the method of Whittaker and Barker[3].

Acetylcholinesterase (AchE EC 3.1 1.7) activity was assayed in 100 μl aliquots of the fraction by spectrophotometric method of Ellman et al. using acetylcholine as substrate and tetraisopropyl-pyrophosphoramide for inhibition of pseudocholinesterase[4]. Butyrylcholinesterase activity was assayed by the same method but using butyrylcholine (BuTCH) as substrate.

The acetylcholinesterase was the only enzyme affected by the pentobarbital anesthesia. The activity of this enzyme was 27% lower in the experimental than in control brains 90 min after treatment. The reduction was transient since the acetylcholinesterase activity returned to almost normal levels at 150 min. No significant changes of cholinesterase or butyrylcholinesterase activity were seen between the anesthetized and control animals (see figure and table). These findings are different from the ones described by Vernadakis, who observed a markedly



The points represent mean values of 6–12 experiments described in the text. The exact numbers with MEM are illustrated in the table.

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Specific and nonspecific cholinesterase activity

Projection Time	AChE	ChE	BuChE
Control	2.518 ± 0.050++	9.428 ± 0.475	0.141 ± 0.009
90 min	2.090 ± 0.083*	9.432 ± 0.396	0.159 ± 0.006
150 min	2.356 ± 0.080	8.745 ± 0.350	0.144 ± 0.014
270 min	2.398 ± 0.076	8.673 ± 0.180	0.157 ± 0.016
24 h	2.455 ± 0.012	9.150 ± 0.270	0.156 ± 9.011
7 days	2.493 ± 0.050	9.316 ± 0.056	0.145 ± 0.0093

*$p < 0.01$; ++means ± SE of 10^{-4} moles substrate hydrol./min/mg protein.

reduced BuChE activity in whole homogenates of corpus striatum and cerebral cortex of cats at 1–3 weeks post-pentobarbital anesthesia[5]. This discrepancy may be due to the different 1. concentrations of the used anesthetic (50 mg instead of 20 mg/kg) and 2. sampling of brain tissue. Moreover the changes could have been obscured by the values expressed on the basis of wet tissue rather than protein.

Ketamine, another anesthetic agent given in the same concentrations as pentobarbital (20 mg/kg but i.v.), was reported to inhibit reversibly AChE, both the membrane bound and the purified form, as well as to increase the level of acetylcholine in the mammalian brain[6].

A marked rise of free acetylcholine but without reduction of the acetylcholinesterase activity was found in the brains of rats and guinea-pigs exposed to barbiturates by Ksiezak et al.[7]. However, the enzyme was assayed in crude subfractions of the brain and, therefore, the results are not comparable with our model of investigation. As a matter of fact we found little activity of the tested enzymes in the mitochondrial fraction. Furthermore, no significant differences were observed between the experimental and normal brains.

Our findings suggest that the increased brain level of acetylcholine reported to be present after barbiturate anesthesia most likely is due to the inhibition of acetylcholinesterase in the synaptosomes since pentobarbital inhibits the specific activity of acetylcholinesterase.

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A morphological temperature-sensitive mutant of the nematode *Caenorhabditis elegans* var. Bergerac

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Summary. A morphological temperature-sensitive mutant of *Caenorhabditis elegans* displays 2 overlapping temperature-sensitive periods, both occurring during the post-embryonic development. Data prove that these 2 phenotypes are controlled by the same locus and are not inherited as maternal factors.

Previous works[2,3], have shown that in *C. elegans* a similar dumpy phenotype (worms shorter than wild-type, but with the same diameter) may be caused by mutations at many different loci distributed over the karyotype. These mutants generally have a monofactorial determination. Other results[4] led to the idea that the cuticle may be altered by this mutation. To understand how genes control the morphology of the cuticle, we have isolated conditional mutants, since such mutants are generally useful to specify the pattern of gene action during development[5].

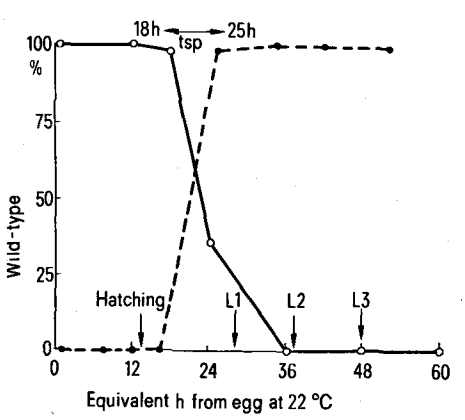

Fig. 1. Determination of the temperature-sensitive period for the roller phenotype of f48ts. During their early embryogenesis fertilized eggs were distributed to drops of fresh medium at the permissive (17 °C) temperature or restrictive (22 °C) temperature. The drops were shifted up from 17 to 22 °C (●---●) or down from 22 to 17 °C (○—○) respectively at the times indicated in the figure. The times on the abscissa have been normalized at the 22 °C growth rate. 80 h after egg deposition, the phenotypes of the adults are determined. The arrows on the figure indicate the times of moulting between larval (L) stages.

The present work reports the study of the temperature-sensitive mutant f48ts obtained in the F_3 progeny of worms of the Bergerac strain[6] mutagenized in the 4th larval stage with ethyl-methane-sulfonate 0.05 M during 5 h in M_9 Buffer[7]. This mutant develops with normal length and crawling behaviour at 17 °C (the permissive temperature) but displays dumpy and roller phenotypes when grown at 22 °C. At this restrictive temperature, movement of f48ts is affected. Worms rotate to the left, along their long axis and do not show, as at 17 °C, the sinusoïdal moving typical of the wild-type.

The temperature sensitive periods. The temperature-sensitive period (TSP) is the developmental period during which the restrictive temperature leads to the expression of the mutant phenotype. The TSPs of the 2 f48ts phenotypes were inferred from shift-up and shift-down experi-

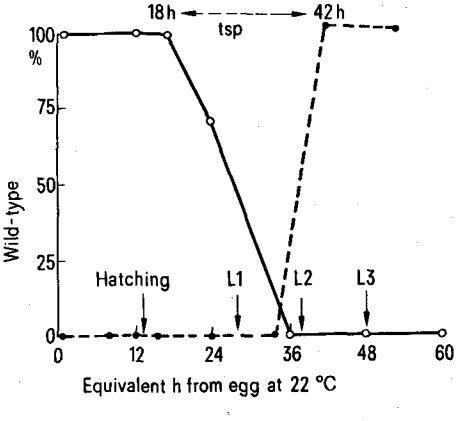

Fig. 2. Determination of the temperature-sensitive period for the f48ts dumpy phenotype. Same method as in figure 1. Shift up (from 17 to 22 °C): ●---●; shift down (from 22 to 17 °C): ○—○.