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

R. Ouazana[1]

Laboratoire de Génétique physiologique et Nématologie, Université Claude Bernard Lyon-I, Laboratoire associé au C.N.R.S. n° 92, 43, Boulevard du 11 Novembre 1918, F–69621 Villeurbanne (France), 17 June 1977

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].

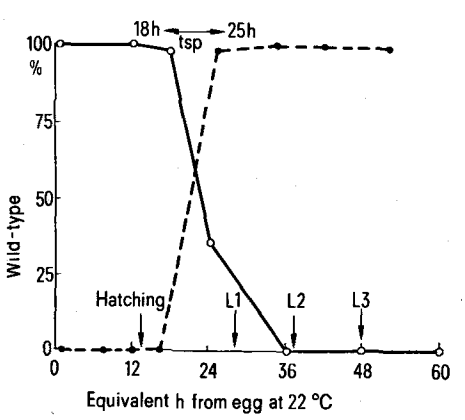

Fig. 1. Determination of the temperature-sensitive period for the roller phenotype of f48ts. During their early embryogenesis fertilized eggs were distributed to drops of fresh medium at the permissive (17 °C) temperature or restrictive (22 °C) temperature. The drops were shifted up from 17 to 22 °C (●---●) 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-

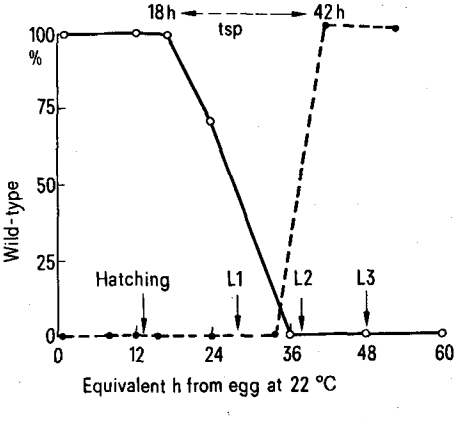

Fig. 2. Determination of the temperature-sensitive period for the f48ts dumpy phenotype. Same method as in figure 1. Shift up (from 17 to 22 °C): ●---●; shift down (from 22 to 17 °C): ○—○.

ments (figures 1 and 2). A TSP is defined as the interval between the earliest time of shift down (from 22 °C to 17 °C) at which the mutant phenotype is observed, and the latest time of shift up (from 17 °C to 22 °C) at which the mutant phenotype occurs. Figure 1 shows that the roller phenotype TSP is entirely situated in the first larval stage (L_1). It begins a few hours after hatching and is extending from 18 to 25 h of development at 22 °C after fertilized egg deposition. Figure 2 shows that the period for the dumpy phenotype is more extended. It includes: later L_1 stage, all the L_2 stage, and the beginning of the L_3 stage (from 18 to 42 h of development at 22 °C, after fertilized egg deposition).

Mutant expression at 22 °C. f48ts length growth curves at 17 °C and 22 °C (figure 3) show that dumpiness expression starts at the beginning of the L_3 stage, near the end of the dumpy phenotype TSP. Roller phenotype is never seen in the larvae (from L_1 through L_4) and is only expressed in the adult stage.

Mode of inheritance. Genetic studies prove that f48ts carries a single autosomal recessive mutation. Homozygous 48/48 hermaphrodites reared at 22 °C are mated, at this same temperature, with wild-type males (+/+).

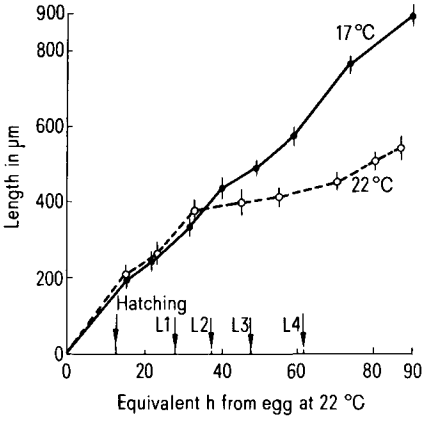

Fig. 3. Length growth curves of f48ts. Evolution of the f48ts length during development is determined at 17 °C (●—●) and 22 °C (○---○). Eggs during early embryogenesis are deposited on fresh medium. At various times, worms are killed with a moderate heat and their lengths measured.

Heterozygotic hermaphrodites (+/48), easily recognized by their wild-type phenotype, are picked, and roller or dumpy recombinants are selected from their self-fertilizing progeny. We did not find any recombinant among a total of 8.093 progeny. This means that if 2 closely linked mutations were involved, they should be distant by less than 0.02 C.M. Thus it is justified to assume that 1 gene is involved. As yet no data on localization and allelism are available. The fact that a single pleiotropic mutation can lead to both roller and dumpy phenotypes was already observed in *C. elegans* Bristol, by Brenner[7] with non-temperature-sensitive mutants (alleles of dpy-2 gene, on linkage group II).

The A and B tests defined by Hirsh et al.[8] prove non-maternal inheritance of the 2 temperature-sensitive phenotypes. These 2 tests show that the mutant allele of the gene studied must be expressed in the zygote in order that the mutant phenotype be realized. This is not suprising since the TSPs are both late, and indicates that the roller and dumpy phenotypes of f48ts are not due to an oocyte component.

Conclusions. The fact that the roller phenotype is only seen in the adult was already noted with non-temperature-sensitive roller mutants[7] which include the only temperature-sensitive roller previously isolated[9]. These observations show that if the adult cuticle is modified, and recent studies of Higgins and Hirsh[9] agree with this conception, it would only alter the adult cuticle. The fact that we find for the f48ts roller phenotype, a temperature-sensitive period earlier than the formation of the adult cuticle is not incompatible with this hypothesis, since the time at which the restrictive temperature affects the gene product can precede the moment of the phenotypic expression of the mutation. Thus it would be interesting to compare the biochemical composition of the larval cuticles and the adult cuticle in this roller mutant.

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Circles in spermatocyte chromatin loops. Electron microscopy and AgAs-NORs studies[1]

Maria Luiza Beçak, Sylvia Mendes Carneiro and K. Fukuda[2]
Serviço de Genética – Instituto Butantan, Caixa Postal 65, São Paulo (Brazil), 31 May 1977

Summary. We describe the production of circles in chromomeric loops during the pachytene stage of the spermatocytes. These circles are found attached to chromatin or already free in the nucleoplasm. Each circle measures an average of 3700 Å in circunference. We suggest that such circles might indicate the presence of tandem repetitions.

Thomas et al.[3] demonstrated that repeated sequences of the DNA in the eukaryotes can produce rolling circles by denaturation and annealling. According to the rolling helix model[4], a single copy of a repeated sequence may form a circle. This circle migrates along the DNA helix, maintaining the base-pairing. During the migration, repairs could be made enzymatically and excision of these circles could be the 1st step of replication. Again, the findings of Hourcade et al.[5] demonstrated that the rolling circles could account for the amplification in the nucleoplasm. In this article we describe the origin of

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