extracellular electrode. MEPCs were stored as FM tape recordings, and analyzed by averaging and/or semilogarithmic regression on a PDP-8 computer.

The fish MEPCs decayed as a single exponential function, and at all temperatures were significantly shorter than tetrapod MEPCs recorded under the same conditions (fig.) The fish MEPCs were also consistently shorter than published values for other twitch-type fibers: at 15 °C, the exponential time constant of decay (τ_D) for *T. novaezelandiae* MEPCs was approximately 1 msec, in contrast to 2 msec for mouse omohyoideus[14], 3 msec for toad sartorius[9], 5 msec for snake costocutaneous[15], and about 20 msec for chicken posterior latissimus dorsi[16], measured at the same temperature. In *Rana temporaria*, MEPCs from fast *m. pyriformis* fibers have decay time constants of 5 msec at 6 °C[17], which again is longer than τ_D for fish extraocular muscle (3–4 msec at 6 °C). Time constants reported for slow muscle fibers are generally several times longer than those of fast fibers at any given temperature[15,17], although in the chicken there is no significant difference between decay rates of fast and slow fibers[16]. Rise times for the fish MEPCs (T_G: the time elapsed between 20 and 80% of maximum deflection) were also quite fast, being about 120 µsec at 15 °C.

Apart from their short duration, *Trachurus* MEPCs were otherwise normal, with τ_D decreasing exponentially with temperature ($-\ln\tau_D = 41.66 - 83/RT$; R = gas constant, 8.32 J mole^{-1}deg^{-1}; T = absolute temperature, °K) (fig.). The Arrhenius temperature coefficient for rate of MEPC decay was 83 kJ mole^{-1}, which is within the range of other vertebrate preparations: mouse 66.2 kJ[14], toad 75.9 kJ[9], chicken 73–86 kJ[16]. There is a hint of a break around 17 °C in the Arrhenius plot of fish MEPCs, which may represent a phase change in membrane lipid, as reported for locust muscle[18].

The effect of membrane potential on τ_D was also comparable to that of tetrapod preparations[9,19,20], showing an e-fold increase in τ_D per 100 mV hyperpolarization in the voltage range between +25 and −110 mV. Voltage-clamped τ_D and T_G were somewhat longer than in extracellular MEPCs, which was probably due to non-focal clamping. In healthy cells, where MEPCs produced an extracellularly recorded voltage deflection of 350–500 µV, resting membrane potential was about −60 mV.

Drug effects on *Trachurus* MEPCs were also normal: at 15 °C, neostigmine (6 µM) increased τ_D about 2-fold, and 0.5 M ethanol increased τ_D approximately threefold.

The correlation reported here, between the fast decay of fish MEPCs and the increased unsaturation and fluidity of fish CNS lipids, is in general agreement with lipid hypotheses of anesthetic action. Provided that the increased fluidity of neural membranes is also reflected in the postsynaptic muscle membrane, it seems clear that changes in lipid fluidity do influence the time course of synaptic events; however this does not preclude additional sites or mechanisms for anesthesia.

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2 Trudell, J.R., Anesthesiology *46* (1977) 5.
3 Richards, C.D., Keightley, C.A., Hesketh, T.R., and Metcalfe, J.C., in: Molecular mechanisms of anesthesia, vol.2, p.337. Ed. B.R. Fink. Raven Press, New York 1980.
4 Gage, P.W., Physiol. Rev. *56* (1976) 177.
5 Gage, P.W., and Hamill, O.P., Br. J. Pharmac. *57* (1976) 263.
6 Torda, T.A., and Gage, P.W., Br. J. Anaesth. *49* (1977) 771.
7 Gage, P.W., McBurney, R.N., and Schneider, G.T., J. Physiol. Lond. *244* (1975) 409.
8 Gage, P.W., McBurney, R.N., and van Helden, D., J. Physiol. Lond. *274* (1978) 279.
9 Gage, P.W., and McBurney, R.N., J. Physiol., Lond. *244* (1975) 385.
10 Kreps, E.M., Avrova, N.F., Chebotarëva, M.A., Chirkovskaya, E.V., Levitina, M.V., Pomazanskaya, L.F., and Pravdina, N.I., Comp. Biochem. Physiol. *52B* (1975) 293.
11 Cossins, A.R., and Prosser, C.L., Proc. natl Acad. Sci. USA *75* (1978) 2040.
12 Known in Australia as *Trachurus mccullochi,* but recently determined to be *T. novaezelandiae;* Stephenson, A.B., and Robertson, D.A., J. R. Soc. N.Z. *7* (1977) 243.
13 Hudson, R.C.L., J. exp. Biol. *50* (1969) 47.
14 Dreyer, F., Müller, K.-D., Peper, K., and Sterz, S., Pflügers Arch. *367* (1976) 115.
15 Dionne, V.E., and Parsons, R.L., Nature *274* (1978) 902.
16 Harvey, A.L., and van Helden, D., J. Physiol., Lond. *317* (1981) 397.
17 Miledi, R., and Uchitel, O.D., Nature *291* (1981) 162.
18 Anderson, C.R., Cull-Candy, S.G., and Miledi, R., Nature *268* (1977) 663.
19 Kordas, M., J. Physiol., Lond. *204* (1969) 493.
20 Magleby, K.L., and Stevens, C.F., J. Physiol., Lond. *223* (1972) 151.

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Reduction of fitness in *Drosophila* adults surviving parasitization by a cynipid wasp

Y. Carton and J.R. David[1]

Laboratoire de Biologie et Génétique Evolutives, CNRS, F-91190 Gif-sur-Yvette (France), June 21, 1982

Summary. Drosophila adults emerging from larvae which successfully eliminated a parasite egg through the formation of a cellular melanotic capsule, were characterized by a decrease of their size and other biometrical traits and by an increase of the variability among individuals. Adults females also exhibited a significant reduction of offspring number due to lower oviposition rate and lower egg hatchability.

With respect to their impact upon natural populations, parasitic wasps are comparable to predators[1] since the development of the wasp implies the death of the host. The persistence of the host population is possible because parasite females cannot discover and attack all the possible hosts individuals and also because, in some cases, defense reactions may kill the parasite.

Natural populations are the theater of a classical coevolu-

Table 1. Influence of parasitization on biometrical traits of *Drosophila* adults

Character		Control (group 1)	Parasitized No capsule (group 2)	With capsule (group 3)	Comparison (t) 1-2	1-3	2-3	Coefficient of variation 1	2	3
Female	Fresh weight	107.56±0.51	97.21±2.47	82.60±1.24	4.12**	18.6**	5.2*	2.56	9.14	8.06
	Wing length	220.90±0.86	214.45±1.61	210.10±1.13	3.5**	7.6**	2.2*	2.10	2.38	2.90
	Thorax length	105.30±0.40	103.36±0.98	100.43±0.64	1.8	6.4**	2.5*	2.04	3.06	3.46
	Sternopleural chaetae	19.23±0.30	16.91±0.64	18.13±0.36	3.2	2.3*	1.6	8.38	11.96	10.62
	Abdominal chaetae	45.27±0.42	43.91±0.90	42.30±0.51	3.4	7.6**	1.5	4.95	6.48	6.52
	Ovariole number	40.10±0.86	39.67±1.40	35.50±0.72	0.3	4.1**	2.6*	11.61	10.00	10.99
	Number of individuals	30	11	30						
Male	Fresh weight	84.53±0.56	71.94±1.25	66.90±0.48	8.4**	58.3**	26.4**	3.54	6.70	3.89
	Wing length	191.93±0.65	188.23±1.56	181.03±0.75	2.2*	10.8**	4.1**	1.82	2.88	2.24
	Thorax length	93.03±0.47	92.46±0.71	86.47±0.51	0.6	9.4**	6.8**	2.71	2.67	3.15
	Sternopleural chaetae	18.07±0.33	17.77±0.59	17.13±0.40	1.3	1.9	0.9	9.74	11.52	12.71
	Abdominal chaetae	38.00±0.48	36.85±0.72	35.13±0.54	1.4	4.1**	1.8	6.77	6.73	8.28
	Number of individuals	30	16	30						

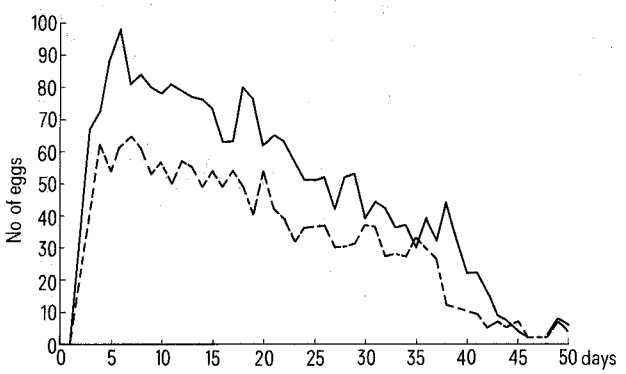
Flies which emerge from parasitized culture either contain (group 3) or do not (group 2) a melanotic capsule. Average values are expressed in mg$\times 10^{-2}$ (weight) or mm$\times 10^{-2}$ (length). n, number of flies; comparisons between the 3 groups with t-test (*$p<0.05$; **$p<0.01$).

Table 2. Comparison of fitness characters between control and parasitized (capsule containing) females

Traits	No.	Control	No.	Parasitized	Comparison
Maximum daily egg production	8	87.0± 6.8	8	62.3± 6.8	2.02*
Egg production at 10 days	8	664.4± 88.4	8	478.2± 60.4	1.62
Egg production at 20 days	8	1422.4±154.2	8	988.6±120.5	2.07*
Total egg production	8	2146.1±262.2	8	1375.0±226.8	2.07*
Egg hatchability (%)	8	96.6± 4.5	8	79.0± 1.7	3.89**
Female longevity (days)	8	36.9± 2.85	8	34.4± 4.7	0.45

Comparisons done with t-test (*$p<0.05$; **$p<0.01$).

tionary scenario[2-6]: the host is continuously selected for increased resistance since a part of the next generation is produced by adults which survived parasitization; the parasite, on the other hand, is selected for an increased virulence since only successful individuals give rise to the next generation. *Drosophila* parasitic wasps should be a convenient model for studying such interactions[7]. Previous studies[8] have shown that under laboratory conditions *Drosophila* adults emerging from an infected culture may be classified into 3 groups: 1. individuals which were not parasitized by the female wasp; 2. individuals which succeeded in killing the parasite egg by forming a cellular melanotic capsule; 3. individuals in which the parasitic egg 'disappeared' without any capsule formation. Observing a melanotic capsule within an adult fly demonstrates that it has overcome parasitization. However, the frequency of capsules in laboratory populations of *D. melanogaster* is generally low[8]. The discovery that a strain of the cynipid wasp *Leptopilina boulardi* induced a high frequency of capsules made it possible to study the physiological and genetical properties of surviving *Drosophila*. In this paper, we show that surviving a parasitic infection is accompanied by physiological modifications which persist in the adult flies and result in a reduction of the individual fitness. The implications of this observation in the coevolutionary process are discussed.



Egg production curves for control (—) and parasitized (capsule containing) females (- - -). In all cases, females were mated to control males.

Methods. Experiments were done with a strain of *D. melanogaster* collected in Malaucène near Avignon, France. The parasite *L. boulardi* originated from Brazzaville (R.P. du Congo). This strain may represent a geographic race or subspecies which differs from the type strain by various morphological characters[9] and also by a partial sexual isolation[10]. *Drosophila* larvae were deposited in culture vials in groups of 30 to avoid larval competition. A female wasp was then introduced in each vial and allowed to oviposit in young larvae. After 10 days, emerging *Drosophila* adults were observed and classified into 2 categories: those containing a capsule and those, less numerous, which did not. These adults were compared to control flies grown in similar conditions but without contact with a parasite. Various biometrical traits were measured according to previously described techniques[11] and egg production was also measured. Experiments were done at 25 °C, using a killed-yeast medium[12].

Results. Results of the biometrical analysis are given in table 1. In all cases, the average values observed in capsule-containing flies are much lower than in controls, the most affected traits being the weight and the wing length. A few individuals which emerged from parasitized cultures but

which did not contain a capsule, were also measured and, with only 1 exception (the number of sternopleural chaetae in females) gave intermediate values between the 2 other groups.

The variability among individuals is also an effective measure of the physiological state of a population since a lower variability may be considered as an indication of better physiological conditions[13]. Since different traits were measured, a relative estimate, i.e. the coefficient of variation, was used[14]. In almost all instances, we observed an increase of variability in the 2 groups which were exposed as larvae to the parasitic wasp (table 1).

Duration of development, from egg to adult emergence, was measured in parasitized and control flies but failed to show any significant difference. Egg production was measured on the 2 categories of females and the curves are shown in the figure. Some statistical comparisons are also given in table 2 for these flies.

The fecundity of the parasitized females was much lower than that of control ones for all periods of their life but their longevities were not significantly different (table 2). It is known that under optimum laboratory conditions egg production is associated with ovariole number[15]. Dividing the maximum daily egg production (table 2) by the average ovariole number (table 1) gives the rate of egg production. In the control flies, the value (2.1 eggs per ovariole per day) is similar to previous values found for *D. melanogaster*[15]. In the capsule-containing females, the rate (1.8 eggs) is lower, suggesting a persistent alteration of the female physiology, independent of the reduction in the ovariole number. This is also demonstrated by the analysis of egg hatching (table 2). In spite of the fact that, in all cases, the females were mated to control males, the capsule-containing group produced much fewer viable offspring than the control group. Although a more precise analysis remains desirable, this difference may be attributed to an impaired fertilization in parasitized females.

Discussion. Insect larvae containing a living parasite and which are expected to die exhibit numerous behavioral and physiological disturbances (see Vinson and Iwantsch[16] for a review). Finding that parasitized larvae, which overcame infestation, showed a disturbed development and produced physiologically abnormal adults, could appear at first to be a trivial observation. This matter has, however, hardly been documented in the case of parasitic wasps and its possible effects upon natural populations have not been considered. Our results demonstrate that the *Drosophila* larvae which encapsulated the parasite egg produced much smaller and more variable adults than the controls. *Drosophila* adults which emerged from parasitized cultures but did not contain a capsule, showed quite similar modifications. It is not known if these individuals survived after the death and resorption of the parasite egg without capsule formation or if the larvae were just stung by the female wasp without egg deposition. This observation however suggests that the venom, which is inoculated during the sting[17], could play a major role in the infection of the *Drosophila* larva.

Most interesting is the observation that some physiological alterations persist in the adult stage of *Drosophila* females. The reduction of egg production, and hence of their fitness, seems to be mainly a consequence of the reduction of the ovariole number. However, the lower rate of oogenesis and also the lower hatchability of the eggs suggest that some physiological alterations still persist in the adult stage.

Such phenomena are likely to occur in natural populations: the host species will be propagated, at each generation, by normal individuals which escaped parasitization and by individuals resistant to parasites but which have a reduced fitness. Presumably, this fitness difference slows down the dissemination of resistance genes in the host population and thus favors the virulence of the parasite. The proportion of resistant *Drosophila* in natural populations is not well known but, at least in 2 cases, a significant proportion of flies containing melanotic capsules has been found[18].

1 We thank Mrs F. Frey and E. Pla for help in the experiments.

2 Price, P.W., Evolutionary biology of parasites. Princeton Univ. Press, Princeton 1980.
3 Pimentel, D., and Stone, F.A., Can. Ent. *100* (1968) 655.
4 Chabora, P.C., and Pimentel, D., Ann. ent. Soc. Am. *63* (1970) 474.
5 Janzen, D.H., in: Evolutionary strategies of parasitic insects and mites, p.154. Ed. P.W. Price. Plenum Press, London/New York 1975.
6 Clarke, B., in: Genetic aspects of host parasite relationships, p.87. Eds A.E.R. Taylor and R. Muller. Blackwell Scient. Publ., Oxford 1976.
7 Carton, Y., Proc. 3rd Eur. Multicol. Parasit., p.65. Cambridge 1980.
8 Carton, Y., and Kitano, H., Biol. J. Linn. Soc. *16* (1981) 227.
9 Nordlander, G., Ent. scand. *11* (1980) 428.
10 Carton, Y., and Claret, J., Ecol. Ent. *7* (1982) 239.
11 David, J.R., Aquilo, Ser. Zool. *20* (1979) 49.
12 David, J.R., and Claret, M.F., Bull. biol. Fr. Belg. *99* (1965) 369.
13 David, J.R., Cohet, Y., and Fouillet, P., Exp. Geront. *10* (1975) 17.
14 David, J.R., Bocquet, C., de Scheemaeker-Louis, M., and Pla, E., Archs Zool. exp. gén. *118* (1978) 481.
15 Cohet, Y., and David, J.R., Oecologia *36* (1978) 295.
16 Vinson, S.B., and Iwantsch, G.F., Q. Rev. Biol. *55* (1980) 143.
17 Beard, R.L., Ann. ent. Soc. Am. *65* (1972) 90.
18 Carton, Y., Bouletreau, M., Van Lenteren, J., and Van Alphen, J., in: The genetics and biology of *Drosophila*. Ed. M. Ashburner. Academic Press, London/New York/San Francisco 1982, in press.

0014-4754/83/020231-03$1.50 + 0.20/0