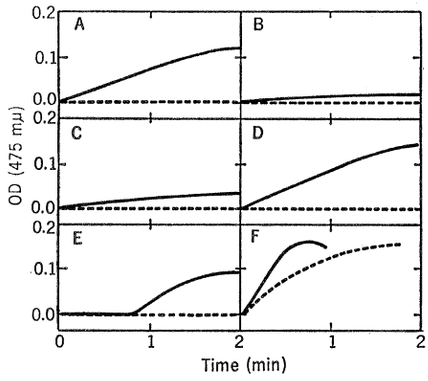

Fig. 3. Phenol oxidase activity in *Drosophila*. Oregon R (*R*/♂) wild-type males, solid lines; lozenge-glossy (*g*/♂) mutant males, dashed lines. The substrate for A–D was $2.4 \times 10^{-3}M$ L-tyrosine; for E, $2.4 \times 10^{-3}M$ L-tyrosine with $2.4 \times 10^{-4}M$ 2-amino-4-hydroxy-6,7-dimethyltetrahydropteridine cofactor; for F, $0.02M$ L-3,4-dihydroxyphenylalanine (L-dopa). Activation of homogenates was for from 90 to 110 minutes at 0°C for the monophenol oxidase assays and for 60 minutes at 0°C for the diphenol oxidase assays. (A) White pupal stage; (B) 6 hours after the white pupal stage (PWP); (C) 12 hours after PWP; (D) 24 hour PWP; (E) 24 hour PWP with cofactor; (F) 24 hour PWP–diphenol oxidase. Reactions were carried out in microcuvettes in a Cary recording spectrophotometer at 30°C; measurements were made at a wavelength of 475 mμ. PWP, Post-white-pupa.

to the *g*/♂ reaction mixture effected no increase in monophenol oxidase activity, although the added pteridine seemed to increase the rate of reaction after an initial lag in the *R*/♂ male extract (see Fig. 3D for comparison). Heat-treated extracts of *R*/♂ male pupae, when added to *g*/♂ male-pupal extracts, did not increase activity of the enzymes from the *g*/♂ males (not shown). Mixtures of *g*/♂ male and *R*/♂ male extracts had approximately half the activity of the *R*/♂ male samples; thus no inhibition of *R*/♂ male oxidase activity by *g*/♂ male extracts was indicated.

Cytological observations are that the initial failure of normal development of the accessory sex organs (*7*) and eyes (*8*) of the *g* mutant occurs within a few hours after the white pupal period in development; this period would coincide with the period of decreased oxidase activity in the wild-type fly (Fig. 3, A–D). The absence of monophenol oxidase activity, as well as the reduced diphenol oxidase activity in the *g* mutant, may result in reduction in the production of quinones necessary for either the initial development or secondary hardening of such structures as claws, female accessory sex organs, and basement membrane of the eyes. In turn, the malformed ommatidia of the eyes may alter production of pigment in the granules associated with them (*9*). An interesting difference at this point is between the light tan color of the smaller claws of the *g* mutants and the dark brown color of the claws of the *R* wild-type fly; such color is presumed to be a direct result of quinone interaction with proteins in hardening of the claws (*1*).

The mutants lozenge-spectacle (*s*), lozenge (*lz*), and lozenge-krivshenko (*k*), as representatives of mutants located at the other three pseudoallelic loci of the lozenge series, also show variations from wild-type oxidase activity, as will be reported later. Further study of these lozenge mutants and their phenol oxidases may lead to significant improvement in our understanding of developmental abnormalities and gene action in *Drosophila*.

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4. Preparation of activator [method adapted from Mitchell and Weber (*2*)]: Approximately 3 g of pupae in 60 ml of $0.1M$ potassium phosphate buffer, *p*H 6.3, was homogenized in a Virtis blender for 1 minute. The homogenate was immediately centrifuged at 0°C for 5 minutes at 18,000*g*, and the supernatant was adjusted quickly to 41 percent saturation with $(NH_4)_2SO_4$. The precipitate was dissolved in phosphate buffer (10 ml per gram of pupae) and either used immediately or stored at −8°C.
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Modulation of Elicited Behavior

Morse, Mead, and Kelleher (*1*) note the following basic relationship: "When responses have followed recurrent shock they tend to occur before the shock." The authors then question "the need for a response to precede an event in order to be reinforced by that event" (*1*, p. 217).

Using inescapable and unavoidable shock these investigators found that "the initially elicited pattern of maximal responding just after each shock was altered by the recurrent shock and by the added fixed-interval schedule to a pattern of maximal responding just before each shock." Subjects (monkeys) in this experiment had the option of being passive and receiving shock every 60 seconds or receiving shock after a 30-second period if they responded. Morse *et al.* found that most shocks were produced by responses; that is, subjects responded to receive shock after the 30-second period instead of not responding and receiving shock every 60 seconds. If the basic relationship as stated by them does obtain, it is puzzling why the frequency of responding before shock on a 30- or 60-second schedule alone is lower than when the 30- and 60-second schedules are both operative. This difference would not be expected and seems incompatible with their basic relationship. Further, an interpretation (relationship) not considered by the authors, but strongly suggested by their data, is that subjects prefer short, as opposed to long, delays before shock. Since the frequency of subject-initiated shock increased, the suggestion is that short delays are less aversive (preferred) than longer delays of shock. Data supporting this have been reported for both humans (*2*) and infra-humans (*3*).

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