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Autosomal recessive mutations affecting body colour in *Pyrrhocoris apterus* (Hemiptera: Pyrrhocoridae)

KAREL SLÁMA

Institute of Entomology, Czech Academy of Sciences, Drnovská 507, 161 00 Praha 6, Czech Republic; e-mail: slama@entu.cas.cz

Recessive mutations, *white* (*w*), *yellow* (*y*), *melanotic* (*m*), trihybrid crosses, pteridine pigments, melanins, recessive epistasis, *Pyrrhocoris apterus*

Abstract. Genetic characteristics of the first three mutants found in *P. apterus* L.; *white* (*w/w*) 1965, *yellow* (*y/y*) 1966 and *melanotic* (*m/m*) 1973 have been described in detail. Exact Mendelian proportions of 1 : 1 and 3 : 1 in all standard test crosses and absence of sexual linkage revealed that each of these mutations was inherited as a single autosomal recessive gene. The dihybrid and trihybrid crosses showed that the *w* gene is epistatic over *y*. The absence of linkage shows that each of the described mutant genes is situated on a different chromosome. During 30 years of sustained rearings of *P. apterus*, the *white* (*w/w*) and *yellow* (*y/y*) mutants never originated de novo, whereas the *melanotic* (*m/m*) mutants originated independently from the macropterous strain three times. Triple recessive (*w y m*) *white melanotic* strain has been maintained and used for some genetic investigations for over 20 years.

INTRODUCTION

The first mutation found in the European fire bug, *Pyrrhocoris apterus*, was a white body colour (*white*, *w*). It appeared sporadically among normal purple red larvae when brought from Europe to Harvard University, Cambridge, Massachusetts, in 1964. It was inherited as a single recessive autosomal gene (Rizki & Sláma, 1968). In 1966, in Prague, several larvae with a bright yellow pteridine pigment in the epidermal cells were found. This yellow body colour (*yellow*, *y*) was also inherited as an autosomal recessive trait. Finally, in 1973, a third recessive mutation (*melanotic*, *m*) was found, which was distinguished by enlarged black melanin pigmentation on the larval pronotum, while in the adult stage it could be recognized by extended black pattern on the wings.

The *white*, *yellow* and *melanotic* mutants of *P. apterus* have been used in a number of physiological and genetic investigations (Sláma & Socha, 1979; Socha, 1984; see Socha, 1993 for review). However, with the exception of *white* (Rizki & Sláma, 1968), the actual data related to inheritance of the other two mutants have never been published. The red, yellow and white pteridine pigments of the mutants are well suited for the study of the genetic control of pteridine biosynthesis (Socha & Němec, 1992, 1996), particularly as they are autonomous to each epidermal cell (Rizki & Sláma, 1968). They may also be used as markers in studies of induced somatic crossing over or induced somatic mutations, as was developed in *Drosophila melanogaster* (Graf et al., 1984; Vogel, 1992).

During the past decade, several new mutants of *P. apterus* have been selected and described in more detail by Socha (Socha 1984, 1993; Socha & Němec, 1992) and it will be possible to map genes on the chromosomes of this species. The present paper provides

essential information on the genetics and linkage of three mutations of *P. apterus*, i.e. *white* (*w*; *wh* in Socha, 1993), *yellow* (*y*; *yw* in Socha, 1984, 1993) and *melanotic* (*m*).

MATERIAL AND METHODS

Rearing of normal, wild-type *P. apterus* was established in 1959 (Sláma, 1960), starting with material collected in the vicinity of Slaný and Veltrusy (northwest from Prague, Czech Republic). The colony was replenished several times with the material collected near Veltrusy.

The larvae and adults of *P. apterus* L. were reared in half-litre glass jars supplied with linden seeds (*Tilia cordata* L.; Malvales: Tiliaceae) and with cotton-plugged vials with water. The rearing temperature was 27°C and photoperiodic conditions were 18L : 6D phase. Dissections of the melanotic females were performed in insect Ringer solution.

Standard genetic crosses were made between true-breeding mutants and wild-type individuals in order to examine the mode of inheritance of the *y* and *m* traits. Two- and three-point test crosses were employed for the study of gene interactions, the data related to inheritance of the *w* mutation were partly taken from Rizki & Sláma (1968). The results were analysed statistically by Chi-square test for fitness, the $P < 0.05$ probability was accepted for rejection of congruence between the obtained and expected values. In certain cases, statistical analysis was completed by the common analysis of variance (ANOVA).

RESULTS

The *yellow* (*y*) mutation

This mutation originated in October 1966 when rearings of *P. apterus* were contaminated accidentally by a synthetic analogue of insect juvenile hormone, methyl-7,11-dichlorofarnesoate (Romaňuk et al., 1967). A few *yellow* larvae appeared all in a single rearing jar, suggesting that they might be progeny from one pair of bugs heterozygous for *y*. A single pair bred true, giving all *yellow* progeny. The *yellow* mutants of *P. apterus* that exist today in various laboratories are all descendants of that single initial pair.

DESCRIPTION: The *y/y* larvae are distinguished by the bright yellow colour of the integument, which is due to the absence of the red and presence of some yellow pteridine pigment in cytoplasm of the epidermal cells. The bright yellow colour of the epidermis is particularly visible, but not limited, to integumental areas which are covered with the transparent cuticle. The parts of integument that are covered by black melanized cuticle in the wild type appear also black in the *y/y* mutants, although the yellow colour may occasionally permeate through the margins of the black areas. Fig. 1 shows that the last instar larvae of the *y/y* mutant look exactly the same as the *w/w* mutant, from which they actually differ only by their yellow body colour. The black cuticular melanin pigmentation of larvae or adults of *y/y* and *w/w* is the same as in the wild type (+/+). The white or yellow colour on the upper part of adult wings is not clear; it has always some light orange tinge. This shows that the synthesis of red pteridine pigment has not been completely abolished in the wings of *y/y* and *w/w* mutants. Both the *w/w* and *y/y* mutants lack the red pigment of the eyes which are brown instead of brown-red. The eye pigmentation becomes visible already at the end of embryonic life, so that the red, yellow or white phenotypic integumental coloration may be recognized as early as in the first larval instar.

Table 1 shows the results of crosses between the wild-type (purple red, +/+) individuals and the *yellow* (*y/y*) strain. A lack of difference between reciprocal crosses, a simple Mendelian ratio of 3 : 1 in the F_2 progeny and a 1 : 1 ratio in the backcross progeny indicate that *yellow*, as *white*, is a recessive autosomal gene.

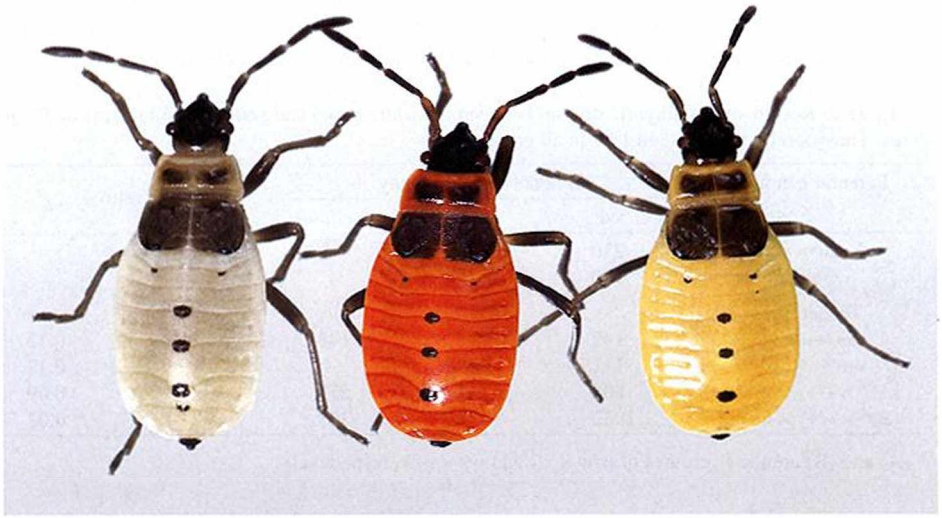

Fig. 1. The fourth larval instars of *P. apterus*. From left to the right: *white* (*w/w*) mutant, red (wild-type), and *yellow* (*y/y*) mutant.

TABLE 1. Results of crosses between the red (+/+) and *yellow* (*y/y*) mutants of *P. apterus*. The sex ratio for all progenies was normal, i.e. 1 : 1.

Parental genotype (♀ × ♂)	Phenotype of progeny (♂ + ♀)	Ratio	χ^2
y/y × *y/y*	*yellow* (2450)	–	–
+/+ × *y/y*	red (540)	–	–
y/y × +/+	red (420)	–	–
Test crosses			
+/*y* × +/*y*	192 red : 63 *yellow*	3 : 1	0.17
+/*y* × *y/y*	350 red : 339 *yellow*	1 : 1	0.01

During the 30 years of continuous rearing, the *y/y* mutant appeared to be the best adapted and fitted laboratory strain of *P. apterus*. It was much better in this respect even when compared with the wild-type red population. The *y/y* females showed increased fecundity and the larvae exhibited the fastest rate of growth. The *y/y* strain was most resistant against microsporidial or bacterial diseases. It was best suited for gregarious life, showing relatively good rates of survival under severe overcrowding conditions. The *y/y* mutants never reappeared spontaneously in our extensive rearings of *P. apterus*.

Interaction between the *w* and *y* genes

It has been mentioned that the *w* and *y* genes are part of a common biochemical pathway for the biosynthesis of the red pteridine pigments. Table 2 indicates clearly that the mutations are nonallelic, i.e., that they are controlled by two different genes. Moreover, the test cross revealed a 9 : 4 : 3 ratio (red, white and yellow respectively), suggesting that the homozygous *w* is epistatic over *y*. This result suggested clearly that the *w* epistatic

allele suppresses not only the synthesis of the red pteridine pigment (erythropterin), but also abolishes biosynthesis of a yellow erythropterin precursor. The *w* and *y* genes show independent assortment (Table 2) and are thus located on different chromosomes.

TABLE 2. Results of the dihybrid crosses between the *white* (*w/w*) and *yellow* (*y/y*) mutants of *P. apterus*. The sex ratio was close to 1 : 1 in all groups.

Parental genotype (♀ × ♂)	Phenotype of progeny			Expected ratio	χ^2
	red	*white*	*yellow*		
w/w × *y/y*	220	–	–	–	–
y/y × *w/w*	340	–	–	–	–
Test crosses					
w+/+y × *y/y*	142	–	148	1 : 0 : 1	0.12
w+/+y × *w/w*	111	105	–	1 : 1 : 0	0.17
(A)* *w+/+y* × *w+/+y*	808	344	275	9 : 4 : 3	0.69
(B)* *w+/+y* × *w+/+y*	1032	454	343	9 : 4 : 3	0.03

* (A) and (B) are the F_1 crosses of *w/w* × *y/y* and *y/y* × *w/w*, respectively.

Selection of the double recessive (*ww yy*) white strain

All *yellow* F_2 larvae (from Table 2; A, B) were either ++ *yy* (1/3) or were +*w yy* (2/3). Random mating between these *yellow* F_2 adults should yield a 1/9 proportion of the double recessive (*ww yy*), which are phenotypically white but homozygous for both the *w* and *y* genes. Table 3, cross (C), shows that such a random crossing among the indicated yellow progeny indeed resulted in 7.94 *yellow* : 1.07 *white* larvae, i.e. almost exactly the calculated 1/9th of the (*ww yy*) double recessive strain. Further observations indicated that larvae of this strain (*ww yy*) did not differ remarkably from the normal *white* (*w/w*) mutants in the quality and distribution of the white integumental areas, eye colour or other physiological criteria. The double recessive *ww yy* strain has been successfully maintained for almost 30 years. It has been used in a number of other genetical studies of this species (see Socha, 1984; Socha & Němec, 1992).

TABLE 3. Results of crosses between the normal red (+/+), *white* (*w/w*) and *yellow* (*y/y*) mutants and selection of the double recessive (*ww yy*) strain.

Parents (♀ × ♂)	Progeny			Actual proportions	Expected ratio	χ^2
	red	*white*	*yellow*			
(C) *yellow* F_2* × *yellow* F_2*	–	65	482	0 : 1.07 : 7.94	0 : 1 : 8	0.32
(D) +/+ × *ww yy*	254	–	–	–	–	–
ww yy × +/+	646	–	–	–	–	–
ww yy × *ww yy*	–	5430	–	–	–	–
(E) *ww yy* × ++ *yy*	–	–	620	–	–	–
(F) +*w* +*y*** × *ww yy*	134	283	140	0.96 : 2.03 : 1	1 : 2 : 1	0.29
(G) +*w* +*y*** × +*w* +*y***	892	401	319	8.92 : 4.0 : 3.2	9 : 4 : 3	1.17

* Progeny from crosses A and B in Table 2; ** progeny from cross D.

The genotypic difference between the double recessive *ww yy* and *ww* ++ can best be demonstrated by crossing with ++ *yy* with yellow progeny being produced in cross E

(Table 3). The 1 : 2 : 1 modification of the common 1 : 1 : 1 : 1 Mendelian ratio in the back cross (F) and the 9 : 4 : 3 ratio in the other test cross (G), confirm the epistatic function of *w* over *y*. Moreover, this also confirms that the genes are located on different autosomes.

The *melanotic* (*m*) mutation

The first larvae with the *melanotic* phenotype appeared in population of *P. apterus* selected for macropterism (see Honěk, 1995). The population originated from specimens collected in Slaný (30 km northwest from Prague), in October 1973. Later, in 1975, the *m* phenotype appeared independently again in a new macropterous strain from different locality, near Veltrusy. Finally, the *m/m* mutants were re-encountered in a new macropterous strain from Slaný in 1976. It is quite important to stress that serious problems were encountered with contamination of the rearings by synthetic analogues of insect juvenile hormone between 1973 and 1977 (cf. Sláma et al., 1974). Since 1976, however, several new macropterous strains from the same localities were established but the *m* phenotype was not found.

DESCRIPTION: The *m* phenotype may be easily recognized in the last instar larvae by black melanin pigmentation of the posterior part of the pronotum and side rims of the wing lobes. Occasionally, *m/m* larvae of the 5th instar show a slightly different shape of the wing lobes (Fig. 2). The best morphological identification of the *m* phenotype can be made in the adult stage; the *m/m* mutants show a black posterior pronotum and enlarged black dots on the wings, with a characteristic "melanotic" pattern on the wings (Fig. 3). The pteridine pigments of the epidermal cells, as well as the dark-red pigment of the eyes, are not affected.

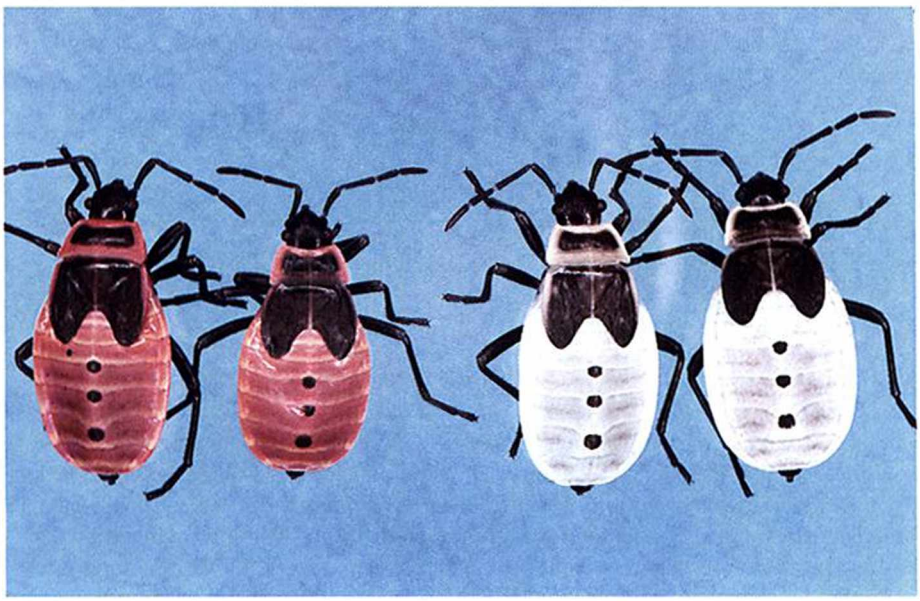

Fig. 2. The fifth (last) larval instars of *P. apterus*. From left to the right: red (wild-type), *melanotic* (*m/m*), *white* (*w/w*), and *white melanotic* (*ww mm*) mutants.

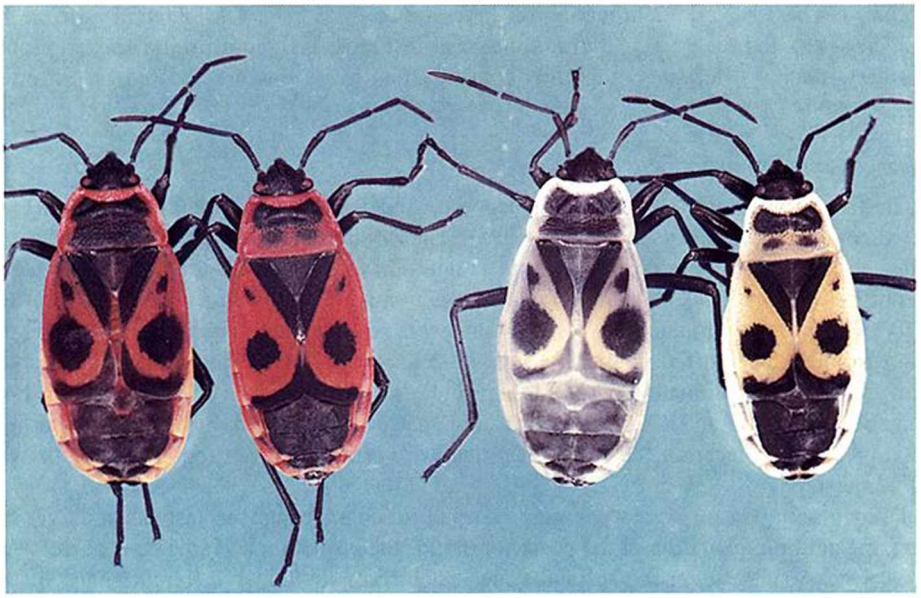

Fig. 3. Female adults of *P. apterus*. From left to the right: *melanotic* (*m/m*), red (wild-type), *white melanotic* (*ww mm*), and *white* (*w/w*).

The crosses between the *m/m* and the wild type (+/+) *P. apterus* revealed 3 : 1 and 1 : 1 Mendelian ratios in the test crosses (Table 4). When combined with the absence of any sex-linked differences, these results indicate that the *m* trait segregates as a single recessive autosomal gene.

TABLE 4. Results of initial crosses between the wild type (+/+) and the *melanotic* (*m/m*) mutants of *P. apterus*. The sex ratio in all progenies was normal, 1 : 1.

Parental genotypes (♀ × ♂)	Progeny phenotypes (♂ + ♀)	Expected	χ^2
m/m × *m/m*	*melanotic* (850)	–	–
+/+ × *m/m*	wild-type (220)	–	–
m/m × +/+	wild-type (320)	–	–
Test crosses			
+/*m* × +/*m*	124 w-t. : 36 *melanotic*	3 : 1	0.53
+/*m* × *m/m*	82 w-t. : 78 *melanotic*	1 : 1	0.1

In the normal brachypterous population of *P. apterus*, the strain homozygous for *m* gene shows a syndrome of reduced fecundity. The effect is stronger in older *m/m* females, usually, they are unable to lay more than two or three batches of eggs (5 and more batches in normal females). In the macropterous strain (stabilized to 80% long winged adults), the *m/m* females show further reduction of their fecundity down to 35% in comparison with the normal long-winged controls ($P < 0.05$ by ANOVA). Dissections of older *m/m* females with arrested oviposition revealed retention of ripe eggs within the ovaries. This was due

to melanisation and necrotisation of the common uterus, which was filled with retained mature eggs. This shows that the *m* gene controls metabolism of tyrosine and melanin not only in the integument (pronotum, wings), but also in certain internal organs.

Interactions between the *w*, *y* and *m* genes

The dihybrid crosses between the *m* and *w* revealed a 1:1:1:1 ratio in the backcrosses and 9:3:3:1 ratio in the offspring of F_1 intercrosses. Similar results were obtained in the dihybrid crosses between the *y* and *m* mutants (Table 5). The absence of any linkage indicates that the *w*, *y* and *m* genes are located on different autosomes. In order to prove this conclusion experimentally, we made trihybrid inter-crosses between the parents heterozygous for all three genes (*+w +y +m*). The distribution of the 6 phenotypic classes of the offspring was close to the expected ratio 27:12:9:9:4:3. The numbers of *white melanotic* and *yellow melanotic* phenotypic classes were smaller than expected (see the bottom of Table 5). This error was due to some difficulties in recognition of the *m* character in small larvae.

White melanotic and *yellow melanotic* progeny of the trihybrid crosses were crossed in individual pairs to establish a triple recessive homozygous strain (*ww yy mm*). This triple recessive strain was maintained in our laboratory for more than 20 years with slightly reduced female fecundity due to presence of the *m* gene.

TABLE 5. Results of crosses between the *white* (*w/w*), *yellow* (*y/y*) and *melanotic* (*m/m*) mutants of *P. apterus*. The sex ratio was 1 : 1 in all progenies.

Parental genotype (♀ × ♂)	Progeny phenotypes						expected	χ^2
	red	red/ *melan.*	*white*	*white/ melan.*	*yellow*	*yellow/ melan.*		
Dihybrid crosses for *w* and *m*								
+/+ × ww mm	164	–	–	–	–	–	–	–
+w +m × ww mm	119	129	129	92	–	–	1:1:1:1	7.81
+w +m × +w +m	549	154	179	36	–	–	9:3:3:1	12.2
Dihybrid crosses for *y* and *m*								
+/+ × yy mm	68	–	–	–	–	–	–	–
+y +m × yy mm	153	128	–	–	152	137	1:1:1:1	3.07
+y +m × +y +m	557	200	–	–	219	59	9:3:3:1	4.97
Trihybrid crosses								
+w +y +m × +w +y +m	509	183	242	70	191	38	27:12:9:9:4:3*	10.49

* Actual proportions found: 26.6 : 12.5 : 9.9 : 9.5 : 3.6 : 1.2.

DISCUSSION

The Mendelian inheritance of three single, recessive autosomal genes affecting body colour in *P. apterus* can be used as a good example of general hereditary rules (cf. Mendel, 1963; King, 1965). The integumental colours of the pteridines may be discerned in the late embryonic period or in the first larval instar, some 10 days after fertilisation of the eggs. In addition, the white or yellow coloration of epidermal cells are so different from the purple-red colour of wild-type larvae that they have been used as genetical markers in

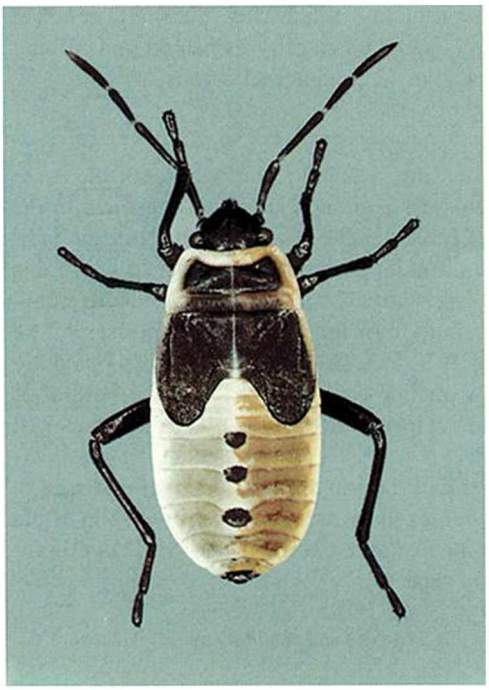

Fig. 4. Bilateral mosaicism in the 5th instar larva of *P. apterus* which was found among the heterozygote (+*w* +*y*) population.

the study of insect juvenile hormone action (Sláma & Socha, 1979).

In a review, Socha (1993) reports the existence of 7 mutants inherited as autosomal recessive traits, with 2 more mutants now being described (Socha, 1997 and pers. comm.). Further progress has been achieved in *P. apterus* by the selection of 2 new autosomal dominant and 3 sex-linked recessive traits (Socha, 1993). Unfortunately, possible linkage between these genes and the *w*, *y* and *m* genes has yet to be established. Socha (1993) suggested that the dominant gene *Apricot*, which also influences the body colour, is not linked with *w*, *y* or *m*. Moreover, Socha & Němec (1996) have described a new orange-yellow mutant of *P. apterus*, the *yolk body* (*yb*) which is also inherited as an autosomal recessive. The *yb* mutation differs, phenotypically, from *y* by orange-red colour of larvae and red coloration of the eyes. In addition, the complementation test between *y* and *yb* revealed that the two similar mutations are nonallelic; only red progeny is produced when crossed (Socha, 1997). It has been also concluded that *Pa* and *yb* are the only mutations which affect coloration of epidermal cells without any effect on coloration of the eyes (Socha & Němec, 1996).

The bright pigments in the epidermal cells of *P. apterus* (Merlini & Mondelli, 1962) and of other insects (Ziegler & Harmsen, 1969) are determined by different colours of pteridines. The purple-red pigment of *P. apterus* was always thought to be erythropterin (Merlini & Nasini, 1966). When *white* was isolated (Rizki & Sláma, 1968), it was assumed that the biosynthetic pathway of erythropterin was arrested at an intermediary stage which was characterised by a white (not colourless) pteridine. These assumptions have been later corroborated by Socha & Němec (1992), who provided experimental evidence for the replacement of erythropterin by leucopterin in the *white* mutant and, consequently, for the replacement of erythropterin by a yellow pigment xanthopterin in the *yellow* mutant. A more recent analysis of pteridine content in several mutants of *P. apterus* (Socha & Němec, 1996; Porcar et al., 1996) reveals quite specific and more detailed relationships between the genes and the ability of epidermal cells to synthesise the particular pteridine molecules; i.e., a situation similar to that found in *Drosophila* and in some other insects (cf. Socha & Němec, 1996).

With the description of the first *w/w* mutant of *P. apterus* (Rizki & Sláma, 1968) a curious bilateral mosaic of half-red, half-white body colour was reported. The existence of the mosaic provided evidence that the genetically controlled biosynthesis of pteridine pigments was not diffusible but it was autonomous to each epidermal cell. The origin of the bilateral mosaic was ascribed to somatic crossing over during the early cleavage divisions. In 1980, another specimen with bilateral white-red mosaicism (Fig. 4) was found. When mated with *w/w* males, the mosaic female produced 1 : 1 red and white progeny, which indicated that one side of the paired ovary was homozygous for the *w* allele whereas the other was homozygous for the +, red allele.

The appearance of *w*, *y* and *m* mutant genes was always associated with some unwanted contamination of the rearings by analogues of insect juvenile hormone. In case of *w* it was juvabione (methyl todomatuate) from American paper products (Bowers et al., 1965; Sláma & Williams, 1965); in case of *y* and *m* it was a synthetic compound, methyl ester of 7,11-dichloro-3,7,11-trimethyl-2-dodecenoic acid (Sláma et al., 1974). These compounds may inhibit embryonic development, cause serious developmental aberrations or produce ovicidal effects in *P. apterus* (Sláma & Williams, 1966). In addition, some synthetic juvenoids produced profound adverse developmental effects in embryos and metamorphosis stages of a number of insect species (Sláma et al., 1974 for review). The synthetic juvenoid compound (methoprene) exhibited direct mutagenic activity in assays on somatic cells of *Drosophila melanogaster* (Marec et al., 1987; Socha et al., 1988). These facts substantiate the above suggested association of juvenile hormone analogues with mutagenic effects in *Pyrrhocoris*.

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REFERENCES

BOWERS W.S., FALES H.M., THOMPSON M.J. & UEBEL E. 1965: Juvenile hormone: identification of an active compound from Balsam fir. *Science (Washington)* **154**: 1012–1021.

GRAF U., WÜRGLER A.J., KATZ H., FREI H., JUON H., HALL C.B. & KALE P.G. 1984: Somatic mutation and recombination in Drosophila melanogaster. *Environ. Mutagen.* **6**: 153–188.

HONĚK A. 1995: Factors and consequences of a non-functional alary polymorphism in Pyrrhocoris apterus (Heteroptera: Pyrrhocoridae). *Res. Popul. Ecol.* **37**: 111–118.

KING R.C. 1965: *Genetics. 2nd ed.* Oxford University Press, New York, 450 pp.

MAREC F., SOCHA R. & GELBIČ I. 1987: Mutagenicity testing of the juvenoid methoprene (ZR-515) by means of the Drosophila wing spot test. *Mutat. Res.* **188**: 209–214.

MENDEL G. 1963: *Experiments in Plant-Hybridisation. 16th ed.* Harvard University Press, Cambridge, 41 pp.

MERLINI L. & MONDELLI R. 1962: Sui pigmenti di Pyrrhocoris apterus L. *Gazz. Chim. Ital.* **92**: 1251–1261.

MERLINI L. & NASINI G. 1966: Insect pigments. IV. Pteridines and colour in some Hemiptera. *J. Insect Physiol.* **12**: 123–127.

PORCAR M., BEL Y., SOCHA R., NĚMEC V. & FERRÉ J. 1996: Identification of pteridines in the firebug, Pyrrhocoris apterus (L.) (Heteroptera: Pyrrhocoridae) by high-performance liquid chromatography. *J. Chromatography (A)* **724**: 193–197.

RIZKI T.A. & SLÁMA K. 1968: An autosomal recessive gene in Pyrrhocoris. *J. Hered.* **59**: 327–328.

ROMAŇUK M., SLÁMA K. & ŠORM F. 1967: Constitution of a compound with a pronounced juvenile hormone activity. *Proc. Natn. Acad. Sci. USA* **57**: 349–352.

SLÁMA K. 1960: Oxygen consumption during the postembryonic development of Pyrrhocoris apterus L. (Heterometabola: Heteroptera) and its comparison with that of Holometabola. *Ann. Entomol. Soc. Am.* **53**: 606–610.

SLÁMA K. & SOCHA R. 1979: Delay of juvenoid action from embryogenesis until metamorphosis unfounded. *Acta Entomol. Bohemoslov.* **76**: 289–299.

SLÁMA K. & WILLIAMS C.M. 1965: Juvenile hormone activity for the bug Pyrrhocoris apterus. *Proc. Natn. Acad. Sci. USA* **54**: 411–414.

SLÁMA K. & WILLIAMS C.M. 1966: "Paper factor" as an inhibitor of the embryonic development of the European bug, Pyrrhocoris apterus. *Nature (London)* **210**: 329–330.

SLÁMA K., ROMAŇUK M. & ŠORM F. 1974: *Insect Hormones and Bioanalogues*. Springer, Wien, New York, 477 pp.

SOCHA R. 1984: A genetic strain of Pyrrhocoris apterus (Heteroptera) last instar larvae with imaginal-like pigmentation of the wing lobes. *Acta Entomol. Bohemoslov.* **81**: 401–410.

SOCHA R. 1993: Pyrrhocoris apterus (Heteroptera) – an experimental model species: A review. *Eur. J. Entomol.* **90**: 241–286.

SOCHA R. 1997: Genetics of a yolk-body color mutation in Pyrrhocoris apterus (Heteroptera: Pyrrhocoridae). *Ann. Entomol. Soc. Am.* **90**: 358–362.

SOCHA R. & NĚMEC V. 1992: Pteridine analysis in five body-colour mutations of Pyrrhocoris apterus (Heteroptera, Pyrrhocoridae). *Acta Entomol. Bohemoslov.* **89**: 195–203.

SOCHA R. & NĚMEC V. 1996: Coloration and pteridine pattern in a new, yolk body mutant of Pyrrhocoris apterus (Heteroptera: Pyrrhocoridae). *Eur. J. Entomol.* **93**: 525–534.

SOCHA R., MAREC F. & GELBIČ I. 1988: Analysis of the genotoxic activities of 3 juvenile hormone analogues by the Drosophila wing spot test. *Acta Entomol. Bohemoslov.* **85**: 249–256.

VOGEL E.W. 1992: Tests for recombinagens in somatic cells of Drosophila. *Mutat. Res.* **284**: 159–175.

ZIEGLER I. & HARMSEN H. 1969: The biology of pteridines in insects. *Adv. Insect Physiol.* **6**: 139–203.

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