BRIEF REPORTS

O. HALKKA, L. HALKKA, M. RAATIKAINEN and A. VASARAINEN: Transmission of genes for colour polymorphism in *Philaenus*.

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Colour polymorphism in natural populations of *Pilaenus spumarius* (L.) (Homoptera, Cercopidae) has been the subject of at least 27 publications. In most parts of the world, a number of colour forms occur in each population, in such ratios that the most frequent form, in the females, seldom exceeds 10 % of the total of this sex. In southern Finland, the frequency of most of the colour forms ranges between 3 and 5 per cent. Thus, in crossing experiments, if any of the colour forms is observed in three successive generations with a frequency significantly higher than 5 %, this implies that transmission is due to hereditary determinants either in the cytoplasm or in the chromosomes.

In crossing meadow spittlebugs for genetic analysis, we started with 60 pairs in the P generation. Of these, 24 yielded F1 progeny (HALKKA *et al.*, 1966). In the F2 generation, only 9 progenies with very small sibships could be scored. These 9 progenies were enough, however, to demonstrate beyond doubt the existence of chromosomal determinants specific for each of the colour phenotypes. Six of the nine progenies resulted from sib matings in the F1 and their outcome is presented in the pedigrees below.

(9)	typ ♀ × typ ♂	(P)	(57)	typ ♀ × typ ♂	(P)
	typ ♀ × typ ♂	(F1)		typ ♀ × typ ♂	(F1)
	typ ♀ typ ♀ typ ♂ typ ♂	(F2)		typ ♀	(F2)
(30)	mar ♀ × typ ♂	(P)	(19)	fla ♀ × tri ♂	(P)
	lop ♀ × typ ♂	(F1)		tri ♀ × tri ♂	(F1)
	lop ♀	(F2)		tri ♀	(F2)
(59)	tri ♀ × typ ♂	(P)	(41)	lop ♀ × typ ♂	(P)
	tri ♀ × typ ♂	(F1)		lop ♀ × typ ♂	(F1)
	tri ♀	(F2)		lop ♀ lop ♀ typ ♂ typ ♂	(F2)

The code numbers used for the crosses are the same as were used in our analysis of the F1 generation (HALKKA *et al.*, 1966). The F1 and F2 progenies of crosses 9 and 57 produced only *typica* phenotypes. Obviously, in the males of the P generation, no unexpressed colour genes were present.

Cross 30, on the other hand, is intelligible if it is assumed that in the P generation male an unexpressed *lop* gene was present. This gene was inherited by the F1 female parent and further by the single female which alone constitutes the F2.

In cross 19, a *tri* gene, originally present in the P male, is transmitted through the F1 generation to an F2 female.

Cross 59 shows transmission of *tri* and cross 41 transmission of *lop* through three generations in the female line.

Three of the nine F2 progenies are the result of non-sib matings in the F1 generation, and are given in the family trees below.

(A) lce ♀ × typ ♂ mar ♀ × tri ♂ (P)
lce ♀ tri ♂ (F1)
tri ♀ typ ♀ typ ♀ typ ♂ (F2)
(B) lop ♀ × typ ♂ tri ♀ × typ ♂ (P)
lop ♀ tri ♂ (F1)
tri ♂ (F2)
(C) mar ♀ × typ ♂ mar ♀ × tri ♂ (P)
mar ♀ typ ♂ (F1)
mar ♀ (F2)

On case A, the *tri* gene is transmitted from P male to F1 male to F2 female, and the *lce* gene from P female to F1 female. Occurrence of *typ* individuals in the F2 shows that both the F1 parents are heterogyzotes.

In case B, the *tri* gene is transmitted from P female to F1 male to F2 male and the *lop* gene from P female to F1 female.

In case C, the *mar* gene expressed in the F2 female was inherited either in the female line or through the F1 male, in which, as in general in males, this gene was not expressed.

Summarizing, the pooled evidence of these crossings shows that:

(1) The colour genes (or perhaps "supergenes", as defined by E. B. FORD) are transmissible through 3 generations, from one sex to another as well as in the same sex.

(2) The genes are chromosomal and not cytoplasmic.

(3) Since *Philaenus* males are XO as regards the sex chromosomes and since *tri* is inherited from male to male, this gene must be autosomal. The other genes may be allelomorphs of *tri*.

The genes determining colour polymorphism in *Philaenus* also regulate the response of the meadow splittlebug to climatic and other factors in the natural environment. Recent papers on this subject include those of HUTCHINSON (1964), BEREGOVOY (1966), HALKKA, RAATIKAINEN and VILBASTE (1967), HALKKA, RAATIKAINEN, VASARAINEN and HEINONEN (1967) and FARISH and SCUDDER (1967).

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K.-H. GUSTAVSON and J. VERNEHOLT: The XYY syndrome in a prepubertal boy.

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The results of an XYY sex-chromosome constitution in man appears to be variable. Some cases are normally developed fertile males with normal intelligence (HAUSCHKA *et al.*, 1962). In other cases there are abnormalities of the external genitalia (DUNN *et al.*, 1961; FRACCARO *et al.*, 1962; VIGNETTI *et al.*, 1964) and/or mental retardation (FRACCARO *et al.* 1962; WILTON and LEVER, 1966; RICCI and MALACARNE, 1964; COURT BROWN *et al.*, 1964).

The XYY sex-chromosome constitution has gained increasing interest since JACOBS *et al.* in 1965 reported an increased incidence of this karyotype among mentally subnormal patients with aggressive behaviour and tall stature. No abnormal physical features distinguished these patients from normal men (PRICE *et al.*, 1966) but their personalities showed extreme emotional instability combined with an incapacity to tolerate even mild frustrations. They began their criminal activities at a very young age but there was no significant family history of crime or mental illness (PRICE and WHATMORE, 1967). Recent investigations indicate that there is an increased frequency of electroencephalographic abnormalities and epilepsy in XYY patients (FORSSMAN, 1967; FORSSMAN and HAMBERT, 1968).

There are only a few reports with detailed clinical descriptions of prepubertal patients with an XYY sex-chromosome constitution (DUNN *et al.*, 1961; SANDBERG *et al.*, 1963; KOSENOW and PFEIFFER, 1967). We have recently studied an 8 1/2 year-old boy, who was admitted to the Paediatric clinic, University Hospital, Uppsala, because of difficulties at school and clumsy movements. At the time of the patient's birth his mother was 31 and his father 37 years of age. Both parents were normal and in good health. A paternal first cousin of the patient was mentally subnormal and of tall stature. The family history was otherwise negative. The mother gave no history of abortions. She had had two pregnancies; the first resulted in a normal girl, whose subsequent development was normal. The second pregnancy, resulting in the birth of the present patient, was uneventful. He was born 2 weeks after term. The delivery was normal. The birth weight was 4250 g and height 56 cm. The child's psychomotor development was somewhat retarded and he had obvious difficulties in keeping abreast at school. He was abnormally sensitive, lacked initiative, became very easily tired and had great difficulty in establishing contact with