Copyright © 1993 by the Genetics Society of America

Conflict Between Feminizing Sex Ratio Distorters and an Autosomal Masculinizing Gene in the Terrestrial Isopod *Armadillidium vulgare* Latr.

Thierry Rigaud and Pierre Juchault

Université de Poitiers, Laboratoire de Biologie Animale, URA CNRS 1452, F-86022 Poitiers Cedex, France

Manuscript received May 6, 1992
Accepted for publication October 12, 1992

ABSTRACT

Female sex determination in the pill bug *Armadillidium vulgare* is frequently under the control of feminizing parasitic sex factors (PSF). One of these PSF is an intracytoplasmic Wolbachia-like bacterium (F), while the other (f) is suspected of being an F-bacterial DNA sequence unstably integrated into the host genome. In most wild populations harboring PSF, all individuals are genetic males (*ZZ*), and female phenotypes occur only due to the presence of PSF which overrides the male determinant carried by the *Z* chromosome (females are thus *ZZ* +F or *ZZ* +f neo-females). Here we report the effects of the conflict between these PSF and a dominant autosomal masculinizing gene (*M*) on phenotypes. The *M* gene is able to override the feminizing effect of the f sex factor and, consequently, male sex may be restored. However, *M* is unable to restore male sex when competing with the F bacteria. It seems that the main effect of *M* is to delay the expression of F bacteria slightly, inducing intersex phenotypes. Most of these intersexes are functional females, able to transmit the masculinizing gene. The frequency of *M* and its effects on the sex ratio in wild populations are discussed.

MATERNALLY transmitted elements which distort population sex ratios are known to exist in a number of arthropods. In insects, cytoplasmic microorganisms generally kill one of the sexes, usually the males (CHANG *et al.* 1991; UYENOYAMA and FELDMAN 1978; WILLIAMSON and POULSON 1979; WERREN, SKINNER and HUGER 1986). In Amphipod and isopod crustaceans, sex ratio distorters (often cytoplasmic microbes) reverse genetic males into functional neo-females (BULNHEIM 1978; GINSBURGER-VOGEL, CARRE-LECUYER and FRIED-MONTAUFIER 1980; JUCHAULT, LEGRAND and MOCQUARD 1980; LEGRAND, LEGRAND-HAMELIN and JUCHAULT 1987). Owing to maternal transmission, these feminizing factors induce the production of highly female-biased broods.

The consequences of such sex ratio distorters spreading through populations have been investigated extensively (BULL 1983; RIGAUD, MOCQUARD and JUCHAULT 1992; TAYLOR 1990; UYENOYAMA and FELDMAN 1978; WERREN 1987). In Crustacea, feminizing factors may invade populations, leading to an increased female-biased sex ratio. In gonochoric species, this increased female ratio could drive a population to extinction if the feminizing power of the sex ratio distorter were too high. In accordance with FISHER (1930), theoretical models have assumed that certain nuclear genes restoring the male function (*i.e.*, conferring a resistance to feminizing factors) could be selected and restore the 1:1 sex ratio in female-biased populations (TAYLOR 1990; WERREN 1987). However, whereas they are known in numerous plants with cytoplasmic male sterility, such genes have been rarely investigated in animals (COUVET *et al.* 1991).

One of the most documented examples of sex ratio distorters can be found in the Isopod *Armadillidium vulgare* (JUCHAULT and LEGRAND 1981a,b, 1989; JUCHAULT, LEGRAND and MOCQUARD 1980). In this species, maternally transmitted parasitic sex factors (PSF) tend to replace homo-heterogametic sex determination (♂*ZZ*/♀*WZ*). One of these PSF is a Wolbachia-like bacterium (F) found in the cytoplasm of host cells (RIGAUD *et al.* 1991). The nature of the second sex factor (f) remains unknown, but experimental data suggest that f could be a segment of the F bacterial genome unstably integrated into the host genome (LEGRAND and JUCHAULT 1984). The two PSF of *A. vulgare* are present in the most wild populations which, consequently, are female-biased. In several populations, the nuclear female sex-determining allele (*W*) has disappeared, and all individuals are genetic males, *i.e.*, *ZZ* males without PSF, and *ZZ* +F or *ZZ* +f neo-females reversed by the PSF. In the wild, *ZZ* +F neo-females produce highly female-biased broods (85% on average), whereas *ZZ* +f neo-females produce broods of more variable sex ratios (65% female on average), due to a more unstable transmission rate of f to offspring (JUCHAULT, LEGRAND and MOCQUARD 1980).

These high mean proportions of females are due to two kinds of brood: a majority of neo-females produce all-female broods, whereas a few produce male-biased broods or broods with high ratios of intersexes in *ZZ* +F strains. The intersexes are of three types: (i) female intersexes (iF) which are functional females with small

male endopodites, (ii) male intersexes (iM) which are sterile individuals with incomplete male gonopods and (iii) functional males with female genital apertures (♂og). The two first types (iF and iM) harbor F. They begin their sexual development by a male stage, which then stops, allowing female development to occur. On the other hand, ♂og are individuals with initial female development followed by male development (JUCHAULT 1966). The production of males, which allows wild populations to survive, suggests that control of the PSF transmission or expression is possible. Such a control has been shown in lineages harboring F bacteria: the bacterial transmission to offspring was limited by certain genotypes, inducing the production of highly male-biased broods by ZZ+F neo-females (RIGAUD 1991; RIGAUD and JUCHAULT 1992).

A masculinizing gene (*M*) has been discovered in wild populations of *A. vulgare* harboring PSF (JUCHAULT and LEGRAND 1976). The masculinizing properties of this dominant autosomal gene have been demonstrated by crossing males harboring *M* with genetic females (*WZ*). The result of these crosses can be described by the following equation:

$$(♂)ZZ\ Mm \times (♀)WZ\ mm \rightarrow$$
$$ZZ\ Mm\ (♂) + WZ\ Mm\ (♂\ \text{or}\ ♂\text{og}) + ZZ\ mm\ (♂) + WZ\ mm\ (♀) \quad (1)$$

In this cross, half the *WZ* individuals are reversed into functional males by the *M* gene inhibiting the female determinant carried on the *W* chromosome. The presence of ♂og in progenies denotes the presence of *M* in one of the progenitors: this phenotype results from a delayed effect of *M* on the female sex-determining gene. Although this gene is suspected to be responsible of high male ratios in strains carrying PSF, this has not been formally demonstrated. However, it has been shown that the presence of *M* is correlated with the production of high intersex ratios in broods of *ZZ* +F neo-females (LEGRAND, JUCHAULT and MOCQUARD 1974).

The aim of this paper is to test whether the *M* nuclear gene is able to restore male sex when parasitic feminizing sex factors (F or f) are present, as it does in the presence of the nuclear sex-determining gene carried on the *W* chromosome.

MATERIALS AND METHODS

To test the effect of the *M* gene, crosses between males carrying *M* and neo-females of f and F strains were performed. In order to ascertain whether the *M* gene was present in these male genotypes, ♂og from crosses described in Equation 1 were used as progenitors. They issued from a hybrid strain selected over 20 years. The *WZ Mm* genotypes of these ♂og were ensured by crossing them with *WZ* females beforehand (RIGAUD 1991).

These ♂og were mated with *ZZ* +f and *ZZ* +F neo-females from the Niort population (France) selected for thelygeny over 10 years. In these laboratory strains, *M* is known to be absent (RIGAUD and JUCHAULT 1992). For the control series *ZZ* +F and *ZZ* +f neo-females of the Niort strains were mated with males of the same origin. Neo-females of these control series were sisters of neo-females paired with ♂og, so their characteristics for PSF transmission are expected to be very close. At each generation, both males and females were bred about 12 months after their birth (female weight was between 70 and 100 mg). This lag time was necessary for obtaining puberal individuals and females large enough to breed sizeable broods. Mating took place in small circular boxes, and immediately after birth, the offspring were isolated from their parents in wider rectangular boxes, to avoid mortality due to high densities. Offspring were then sexed 16 weeks later and the males and the females were separated to avoid sib-mating. Under the experimental rearing conditions (20°, LD 18:6), each female produced one, two or three broods, and all broods from the same mother were added up to estimate the sex ratio. In this paper, the word "progeny" always refers to the sum of all offspring from all broods from a single mother.

TABLE 1

Genotypes and phenotypes expected from the crosses: ♂og (*WZ Mm*) × neo-female (*ZZ mm* + PSF)

PSF nontransmitted		PSF totally transmitted	
		M > PSF	*M* < PSF
WZ Mm = ♂ or ♂og	*WZ Mm* + PSF =	♂	♀
WZ mm = ♀	*WZ mm* + PSF =	♀	♀
ZZ Mm = ♂	*ZZ Mm* + PSF =	♂	♀
ZZ mm = ♂	*ZZ mm* + PSF =	♀	♀

These phenotypes are expected from the transmission rates of PSF (F or f) and from the dominance of the masculinizing gene over PSF.

The following criteria were used to analyze these crosses:

- In the f strain, the proportion of female (♀/offspring) and the proportion of ♂og (♂og/♂ + ♂og) were calculated.
- In the F strain, since iF are functional females, the proportion of females was calculated as follows: iF + ♀/offspring. The proportion of intersexes among female phenotypes (iF + iM/♀ + iF + iM) was also calculated.

These ratios allowed us to compare the ability of *M* to restore the male sex in various strains. In order to obtain average values of these ratios for a group of crosses (mean female ratio = MFR, and mean intersex ratio = MIR), a regression method was used, as described in RIGAUD, JUCHAULT and MOCQUARD (1991). Comparison between groups could then be made by using covariance analysis and the Snedecor *F* test.

RESULTS

Because of the homo-heterogametic composition of the progenitors, and according to the transmission rate of the PSF to offspring, the expected phenotypes of crosses between *WZ Mm* ♂og and *ZZ mm* neo-females are those described in Table 1. If *M* had the same masculinizing effect on PSF as on the *W* sex-determining gene, female ratios in progenies would be between ¼ and ½. If the feminizing effect of PSF overrode *M*,

TABLE 2

Phenotypes of offspring in progenies of the six types of crosses

A		B			C			D				E				F			
♂	♀	♂	♀	♂og	♂	♀	i	♂	♀	♂og	i	♂	♀	♂og	i	♂	♀	♂og	i
55	44	30	10	2	0	47	0	3	35	0	18	9	27	1	22	0	23	0	48
12	92	42	15	5	1	49	1	21	97	1	22	1	12	6	18	1	31	1	44
57	72	50	22	18	11	37	4	15	91	0	38	0	40	0	12	1	56	0	59
117	39	72	44	17	0	84	0	15	100	0	25	0	26	0	8	0	42	0	28
4	103	114	53	9	0	15	0	16	61	0	24	5	27	0	5	1	40	1	116
0	35	43	8	6	5	40	0	1	13	0	1	0	59	1	29	2	29	1	33
15	70	163	118	36	2	48	3	11	85	4	10	1	22	2	15	50	32	13	4
40	110	55	70	26	2	15	1	0	17	0	1	0	38	0	14	2	9	2	16
11	77	22	39	17	0	70	0	5	66	2	26	10	15	2	4	9	99	4	109
0	110	14	54	33	10	39	1	11	8	0	5	0	49	0	9	12	9	4	2
13	79	131	102	44	0	112	0	14	45	3	31	0	26	0	15	7	60	1	68
16	73	11	49	32	0	74	0	10	58	6	29	0	36	0	18	1	89	0	92
43	91	48	85	36	0	70	3	5	80	0	7	1	33	0	11	0	58	0	1
7	96	81	118	55	6	66	0	4	29	0	8					2	11	0	3
2	74	73	55	11	12	8	0	3	41	0	23					0	30	0	19
0	33	151	100	64	0	91	0	0	27	1	8					8	40	1	14
5	103							9	55	4	18					0	11	0	29
48	11							5	22	0	13					6	41	2	35
50	21							5	60	0	41					0	106	0	3
								7	39	0	8					0	75	0	33
MFR = 72.9% ± 6.0		MFR = 38.4% ± 2.4			MFR = 94.7% ± 2.7 MIR = 1.5% ± 0.7			MFR = 88.9% ± 1.5 MIR = 25.5% ± 2.3				MFR = 93.8% ± 2.9 MIR = 30.5% ± 3.2				MFR = 92.6% ± 3.6 MIR = 45.7% ± 4.7			

Each line of a column represents the progeny of one mother. A = Niort ♂ × Niort *ZZ* + f neo-♀; B = ♂og × Niort *ZZ* + f neo-♀; C = Niort ♂ × *ZZ* + F neo-♀; D = ♂og × *ZZ* + F neo-♀; E = Niort ♂ × iF; F = ♂og × iF. MFR = mean female ratio ± SEM; MIR = mean intersex ratio ± SEM; i = iF and iM intersexes.

female ratios in progenies would be between ¼ and 1.

The different types of crosses, the composition of each progeny and the mean values of female and intersex proportions are given in Table 2. When a single female produced more than one brood, significant differences between sex ratios of each brood were never observed.

Crosses in the f strain: In control crosses (♂ × *ZZ* +f neo-female), the female ratios were variable, but a majority of progenies was highly female-biased (Table 2, column A, and Figure 1). The progenies with lower female ratios reflected a variability in transmission of the feminizing factor. No ♂og were observed in these progenies.

In progenies from crosses of ♂og × *ZZ* +f neo-females (Table 2, column B, and Figure 1), the female ratio was always between ¼ and ½ (χ^2 NS for extreme values). This distribution highly differed from that of preceding crosses, and corresponded to the expected proportions described in Table 1 for dominance of *M* over f, with a variable transmission of the feminizing factor to offspring (as observed in the control series). In these progenies, there was a positive correlation between the ♂og/♂ + ♂og proportion and the female proportion (R = 0.81 after arcsine transformation of data).

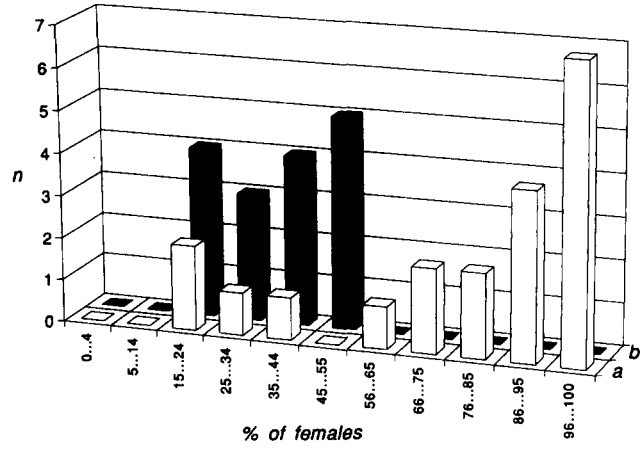

FIGURE 1.—Distribution of the percentage of females in the control crosses: Niort males × *ZZ* +f neo-females (a) and in crosses: ♂og × *ZZ* +f neo-females (b). n = number of progenies.

Crosses in the F strain: In control series, the majority of neo-females produced highly female-biased progenies (Table 2, column C). The single male-biased progeny could be related to a depressed bacterial transmission to offspring, seeing that males never harbor bacteria (this was verify by electron microscopy and by using physiological tests described in RIGAUD 1991). Such a low F transmission recall an effect of the resistance polygenic system, unrelated to the *M*

gene (RIGAUD 1991; RIGAUD and JUCHAULT 1992). In all progenies, intersex ratios were very low, and no ♂og were observed. Among these intersexes, the proportion of sterile individuals (iM) was very low (<3% in all progenies). This observation was also valid for all the ensuing crosses.

The mean female ratio in crosses between ♂og and *ZZ* +F neo-females (Table 2, column D) was approximately the same as in the control series (F = 4.02; d.f.: 1,35; NS). The high proportions of neo-females and iF intersexes showed that the *M* gene did not prevent the transmission of F to offspring. The mean intersex ratio among female phenotypes was significantly higher in this series than in the control (F = 79.83; d.f.: 1,35; $P < 0.001$). The intersex/females + intersex ratio varied between progenies, but never exceeded 50%. We put forward the hypothesis that individuals harboring both *M* and F (*WZ Mm* and *ZZ Mm* genotypes, see Table 1) could develop into intersexes, regardless of their homo-heterogametic composition. As this theoretical value of ½ of intersexes was a maximum only in experimental crosses, we may assume that these genotypes could develop into neo-females or intersexes equally easily. It thus seemed that F could override the masculinizing effect of *M* totally or partially.

A number of iF intersexes from these broods were mated with genetic males (Table 2, column E). Progenies from these crosses showed a mean female ratio very similar to that of preceding crosses (F = 2.64; d.f.: 1,32; NS). The intersex proportion also showed the same distribution between 0 and 50%, with a similar mean value (F = 1.45; d.f.: 1,32; NS). This result confirmed that iF intersexes transmitted the *M* gene. Thus, unlike in the f strain, the masculinizing gene could be carried by either a male or a female phenotype.

The effect of *M* when homozygous was investigated by crossing *WZ Mm* ♂og with iF (Table 2, column F). Here again, the mean female ratio did not differ from that of the control series (F = 0.18; d.f.: 1,35; NS). The mean intersex ratio was higher than in control series (F = 84.60; d.f.: 1,35; $P < 0.001$) and was also higher than in crosses involving iF or ♂og only (F = 17.4; d.f.: 1,52; $P < 0.005$). In this series, the variation of the intersex ratios between broods was greater than in the preceding. This greater dispersion could be a result of variable success in the conflict between F and M. Regardless of the homo-heterogametic possible combination, these crosses theoretically produced ¼ *mm* +F individuals (which must develop toward the female phenotype), ½ *Mm* +F individuals (which could develop toward both female or intersex phenotypes, see below) and ¼ *MM* +F individuals.

To confirm the possibility of obtaining *MM* genotypes, males from one progeny of previous crosses (♂og × iF) were mated with *WZ* females (Table 3). Two out of nine male progenitors produced all-male progenies. This result showed that approximately ¼ of the male progenitors were *MM*. In other progenies, the female ratios were either ¼ or ⅜ (χ^2 NS), corresponding to the results of crosses between *ZZ Mm* and *WZ Mm* males and *WZ mm* females, respectively.

TABLE 3

Phenotypes in progenies of the crosses: ♂ × *WZ mm* ♀

♂	♀	♂og	Percent of females
28	10	16	18.5
11	18	15	40.9
43	0	29	0.0
30	29	27	33.7
35	45	18	45.9
20	0	12	0.0
43	35	25	34.0
45	37	38	30.8
70	26	5	25.7

Each set of progeny was composed of two successive broods. The male progenitors came from the crosses: ♂og × iF (column F, Table 2), from the following progeny: 9 ♂ + 99 ♀ + 4 ♂og + 109 i.

DISCUSSION

Consequences of *M*/PSF conflict on phenotypes: This study showed that the masculinizing gene *M* has different effects according to the PSF with which it competes.

The *M* gene was able to restore male sex in f strains, as it was in strains consisting of genetic females. However, the inheritance and mode of action of the *M* gene appeared to be different from those of the paternal sex ratio (psr) factors observed in *Nasonia vitripennis* (WERREN, SKINNER and CHARNOV 1981) or in *Orchestia gammarellus* (GINSBURGER-VOGEL 1989), where these paternally inherited factors are parasitic and are able to induce all-male broods. In genetic *A. vulgare* females, the *W* female determinant inhibits the *Z* male determinant in the early stages of postembryonic development. This allows for female differentiation by preventing development of the androgenic gland (JUCHAULT 1966). Like the *W* chromosome gene, f should inhibit the male gene carried by the *Z* chromosome at an early stage and is always overridden by *M*. This great similarity of action between the female determinant carried on the *W* chromosome and the f factor supported the hypothesis that f is located on the *A. vulgare* genome, and acts as a female sex determining gene. In the ♂og which appear in these strains, female traits begin to differentiate (female genital apertures are traces of this), then, after the *M* effect, a functional male phase replaces female differentiation. This supported the hypothesis that *M* overrides the f effect, but cannot prevent this feminizing factor being transmitted. Fur-

thermore, in f lineage progenies, a positive correlation between the ♂og proportion and the female proportion has also been observed. This may indicate that the efficiency of feminization by f could increase in proportion with this factor's transmission rate. It would thus appear that the main difference between the *W* chromosome gene and the f factor concerns their transmission rate to progenies: whereas *W* is transmitted strictly to ½ of the offspring, f is often transmitted more extensively.

M does not restore male sex when female sex determination is under the control of F bacteria, even when homozygous. In crosses described in this paper, the few males and ♂og were due to a nontransmission of F to offspring, unrelated to an *M* effect on F. However, the high proportion of iF and iM intersexes in these crosses indicated that the *M*/F interaction was not without effect on the phenotype. We must reiterate that the appearance of iF or iM is not always correlated to the presence of *M*: a few intersexes are sometimes present in crosses free of *M*. Unlike ♂og, an initial male differentiation occurs in iF and iM (JUCHAULT 1966). This shows a more or less late expression of the F feminizing effect. We may thus state that *M* enhances the frequency of this late expression of F. Although our standard genetical method did not allow us to put forward a hypothesis as to how the *M* gene operates, we may nevertheless suggest that *M* slows down proliferation of the bacteria, which could prevent expression of the feminizing effect for a while. Differences in the effect of *M* according to female determinants might reflect a fundamental difference between the modes of action of F and f. The cytoplasmic localization of F suggests that the way in which it inhibits expression of the male determinant is more complex than that of the other two female determinants.

Consequences of *M*/PSF conflict on sex ratio in wild populations: The *M* gene is only observed in wild populations harboring PSF. This gene is logically nonselected in equilibrated sex ratio populations, according to FISCHER's (1930) predictions. In female-biased populations, where f is the only female determinant, such a male restorer gene is expected to be selected and spread (TAYLOR 1990). Theoretically, if *M* were to appear in a totally f-invaded population, where every females are *ZZ* +f neo-females and males *ZZ*, the sex ratio should be determined by the frequencies of f and *M* (TAYLOR 1990). At equilibrium, the *M* gene should be established and the population reach a 1:1 sex ratio, with ½ *Mm* +f males and ½ *mm* +f females (the situation would then be identical to the homo-heterogametic system: ♂*XY*/♀*XX*). This situation presupposes total transmission of the f factor to offspring.

Surprisingly, *M* is more frequent in F/f mixed populations (about 30%) (JUCHAULT, RIGAUD and MOCQUARD 1992) than in populations harboring only f (<10%) (JUCHAULT and LEGRAND, 1981b). These frequencies are lower than those expected according to the model of TAYLOR (1990). However, in *A. vulgare*, f transmission rate may vary between females (results in this paper), between generations of one lineage (RIGAUD, MOCQUARD and JUCHAULT 1992), and even between the broods of a given female (LEGRAND and JUCHAULT 1984). With variations such as these, an irregular but often substantial proportion of males could be produced at each generation in a f sex-determined population. Thus, individuals carrying *M* should not be the only males contributing to the gamete pool. Owing to the unpredictability of the f transmission, establishment of *M* should be difficult to reach in wild populations.

The situation is quite different in populations where F and f are the only two female determinants. Here, *M* could be carried by both male and female phenotypes (with the iF contribution), thus its spread through the population would be easier. The *MM* individuals arising from this double transmission could facilitate this spread (such individuals have been observed in the Niort population) (RIGAUD 1991). However, since *M* is only partially effective against F, the efficiency of male restoration is not 100%, and restoration of the 1:1 sex ratio in such populations is impossible. Moreover, it has recently been shown that *M* favors the spread of f in these populations: males or ♂og harboring both *M* and f are able to transmit the feminizing factor to their offspring, which increases the probability of f transmission (JUCHAULT, RIGAUD and MOCQUARD 1992).

Thus, while *M* could potentially restore the male sex, it is poorly selected with respect to the factor against which it is effective in wild populations. We could argue that this is due to the complexity of sex determination by parasitic sex factors in *A. vulgare*. Moreover, population dynamics parameters (*e.g.*, spatial organization, the founder effect, migration rates) could also favor the diversity of the possible evolutionary pathways, as suggested by GOUYON and COUVET (1987) for cytoplasmic-induced gynodioecy in plants.

We are grateful to J. P. MOCQUARD for helpful discussions and critical reading of this paper, and to two anonymous reviewers for their comments.

LITERATURE CITED

BULL, J. J., 1983 *Evolution of Sex Determining Mechanisms*. Benjamin/Cummings Publ. Co., Menlo Park, Calif.

BULNHEIM, H. P., 1978 Interaction between genetic, external and parasitic factors in sex determination of the crustacean amphipod *Gammarus duebeni*. Helgol. Cuissens. Meerresunters. **31:** 1–33.

CHANG, K. S., T. SHIRAISHI, F. NAKASUJI and N. MORIMOTO, 1991 Abnormal sex-ratio condition in the walnut leaf beetle,

Gastrolina depressa (Coleoptera, Chrysomelidae). Appl. Entomol. Zool. **26:** 299–306.

COUVET, D., A. ATLAN, E. BELHASSEN, C. GLIDDON, P. H. GOUYON and F. KJELLBERG, 1991 Coevolution between two symbionts: the case of cytoplasmic male-sterility in higher plants, pp. 225–249 in *Oxford Surveys in Evolutionary Biology*, edited by D. J. FUTUYAMA and J. ANTONOVICS. Oxford University Press, New York.

FISHER, R. A., 1930 *The Genetical Theory of Natural Selection*. Oxford University Press, England.

GINSBURGER-VOGEL, T., 1989 Déterminisme des anomalies de sex ratio à hérédité paternelle chez le crustacé amphipode *Orchestia gammarellus* Pallas. Invertebr. Reprod. Dev. **16:** 183–194.

GINSBURGER-VOGEL, T., M. CARRE-LECUYER and M. C. FRIED-MONTAUFIER, 1980 Transmission expérimentale de la thélygénie liée à l'intersexualité chez *Orchestia gammarellus* (Pallas). Analyse des phénotypes sexuels dans les descendances de femelles normales transformées en femelles thélygènes. Arch. Zool. Exp. Gen. **122:** 261–270.

GOUYON, P. H., and D. COUVET, 1987 A conflict between two sexes, females and hermaphrodites, pp. 245–261 in *The Evolution of Sex and Its Consequences*, edited by S. C. STEARNS. Biskhauser Verlag, Berlin.

JUCHAULT, P., 1966 Contribution à l'étude de la différenciation mâle chez les crustacés isopodes. Thèses de doctorat d'état. Université de Poitiers, France.

JUCHAULT, P., and J. J. LEGRAND, 1976 Etude génétique de l'intersexualité des mâles à ouvertures génitales femelles chez l'oniscoïde *Armadillidium vulgare* Latr.: interprétation et modalités de la transmission héréditaire. C. R. Soc. Biol. **2:** 429–433.

JUCHAULT, P., and J. J. LEGRAND, 1981a Contribution à l'étude qualitative et quantitative des facteurs contrôlant le sexe dans les populations du Crustacé Isopode terrestre *Armadillidium vulgare* Latr. II. Populations hébergeant le facteur féminisant F (bactérie intracytoplasmique). Arch. Zool. Exp. Genet. **122:** 65–74.

JUCHAULT, P., and J. J. LEGRAND, 1981b Contribution à l'étude qualitative et quantitative des facteurs contrôlant le sexe dans les populations du Crustacé Isopode terrestre *Armadillidium vulgare* Latr. III. Populations n'hébergeant le facteur féminisant F. Arch. Zool. Exp. Genet. **122:** 117–131.

JUCHAULT, P., and J. J. LEGRAND, 1989 Sex determination and monogeny in terrestrial isopods *Armadillidium vulgare* (Latreille, 1804) and *Armadillidium nasatum* (Budde-Lund, 1885). Monit. Zool. Ital. (N.S.) Monogr. **4:** 359–375.

JUCHAULT, P., J. J. LEGRAND and J. P. MOCQUARD, 1980 Contribution à l'étude qualitative et quantitative des facteurs contrôlant le sexe dans les populations du Crustacé Isopode terrestre *Armadillidium vulgare* Latr. I. La population de Niort (Deux-Sèvres). Arch. Zool. Exp. Genet. **121:** 3–27.

JUCHAULT, P., T. RIGAUD and J. P. MOCQUARD, 1992 Evolution of sex determining mechanisms in a wild population of *Armadillidium vulgare*: competition between two parasitic sex factors. Heredity **69:** 382–399.

LEGRAND, J. J., and P. JUCHAULT, 1984 Nouvelles données sur le déterminisme génétique et épigénétique de la monogénie chez le crustacé isopode terrestre *Armadillidium vulgare* Latr. Genet. Sel. Evol. **16:** 57–84.

LEGRAND, J. J., P. JUCHAULT and J. P. MOCQUARD, 1974 Analyse préliminaire de l'intersexualité féminisante chez le crustacé *Armadillidium vulgare* Latr. (isopode oniscoïde). C. R. Acad. Sci. Paris **278:** 2979–2982.

LEGRAND, J. J., E. LEGRAND-HAMELIN and P. JUCHAULT, 1987 Sex determination in crustacea. Biol. Rev. **62:** 439–470.

RIGAUD, T., 1991 Déterminisme extrachromosomique du sexe chez *Armadillidium vulgare* Latr. (Crustacé, Isopode): modifications du sex ratio par une bactérie endocytobiotique et conséquences sur l'évolution des génotypes sexuels dans les populations. Thèse d'Université, Université de Tours.

RIGAUD, T., and P. JUCHAULT, 1992 Genetic control of the vertical transmission of a cytoplasmic sex factor in *Armadillidium vulgare* Latr. (Crustacea, Oniscidea). Heredity **68:** 47–52.

RIGAUD, T., P. JUCHAULT and J. P. MOCQUARD, 1991 Experimental study of temperature effects on sex ratio of broods in terrestrial crustacea *Armadillidium vulgare* Latr. Possible implication in natural populations. J. Evol. Biol. **4:** 603–617.

RIGAUD, T., J. P. MOCQUARD and P. JUCHAULT, 1992 The spread of parasitic sex factors in populations of *Armadillidium vulgare* Latr. (Crustacea, Oniscidea): effects on sex ratio. Genet. Sel. Evol. **24:** 3–18.

RIGAUD, T., C. SOUTY-GROSSET, R. RAIMOND, J. P. MOCQUARD and P. JUCHAULT, 1991 Feminizing endocytobiosis in the terrestrial crustacean *Armadillidium vulgare* Latr. (Isopoda): recent acquisitions. Endocytobiosis Cell Res. **7:** 259–273.

TAYLOR, D. R., 1990 Evolutionary consequences of cytoplasmic sex ratio distorters. Evol. Ecol. **4:** 235–248.

UYENOYAMA, M. K., and L. W. FELDMAN, 1978 The genetics of sex ratio distortion by cytoplasmic infection under maternal and contagious transmission: an epidemiological study. Theor. Popul. Biol. **14:** 471–497.

WERREN, J. H., 1987 The coevolution of autosomal and cytoplasmic sex ratio factors. J. Theor. Biol. **124:** 317–333.

WERREN, J. H., S. W. SKINNER and E. L. CHARNOV, 1981 Paternal inheritance of a daughterless sex ratio factor. Nature **293:** 467–468.

WERREN, J. H., S. W. SKINNER and A. M. HUGER, 1986 Male-killing bacteria in a parasitic wasp. Science **231:** 990–992.

WILLIAMSON, D. L., and D. F. POULSON, 1979 Sex ratio organisms (Spiroplasmas) of *Drosophila*, pp. 175–207 in *The Mycoplasmas*, Vol. III, edited by R. F. WHITCOMB and J. G. TULLY. Academic Press, New York.

Communicating editor: R. E. DENELL