

well (Figure 1 c). Inside the cells, the globules are stable, but once the enzymes in the form of minute vesicles are discharged, the globules lose their identity. These vesicles are the carriers of protease digestive enzymes which were tested histochemically, using Burstone and Folk method³. The young digestive glands show more positive reaction to aminopeptidase activity than the mature ones (Figure 1 d). Thus, the glandular epidermis releases the enzymes after bursting of cells (Figure 1 e). The number of vesicles formed seems to depend on the intensity of the stimulus generated by the captured prey.

On the other hand, the absorptive glands are glomerulus or discoid, located in the pits covered by epidermal flap. These glands are mainly concerned with absorption of the digested food materials. These glands are comprised of absorptive cells,

two endodermal layers with distinct casparian strips in their radial wall (Figure 1 f), and communicatory cells which are in direct contact with vascular strands (Figure 1 g). The casparian strips in the endodermal layers are similar to those in the developing wall of root endodermis cells. These thickenings prevent the backflow of the absorbed substances through apoplast. A similar role was ascribed to these thickenings by Bruni and Modensei⁴ and Fahn⁵.

According to Heslop-Harrison¹ and Lutge^{6,7}, the secretion of digestive enzymes and reabsorption of digested food materials are by the same secretory gland in two different phases. This study reveals that in addition to the presence of digestive glands, the inner epidermal and sub-epidermal layer of cells also have an important role in the secretion of digestive enzymes in *N. khasiana* Hk.f.

1. Heslop-Harrison, Y., in *Perspective in Experimental Biology* (ed. Sunderland, N.), Pergamon Press, Oxford, vol. 2, pp. 463-476.
2. Lloyd, F. E., *The Carnivorous Plants*, Chronica Botanica Co., Massachusetts, USA, 1942.
3. Burstone, M. S. and Folk, J. E., *J. Histo. Cytochem.*, 1956, 4, 217-226.
4. Bruni, A and Modensei, P., *Nor. J. Bot.*, 1983, 3, 7-84.
5. Fahn, A., *New Phytol.*, 1988, 108, 229-257.
6. Lutge, U., *Planta*, 1965, 66, 331-344.
7. Lutge, U., *Flora (Jena)*, 1965, 155, 228-236.

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Rare-male mating advantage in *Drosophila*

Rare-male mating advantage or minority male mating advantage is one of the best studied examples of negative frequency-dependent selection. When the rare type has higher fitness than the common type, selection is balancing. Initially, the rare genotype will increase in frequency, if there are no other selective forces operating against it, but as soon as the rare type becomes more common, its advantage disappears which leads to equilibrium. Interestingly, genetic variability may be maintained without any genetic load at equilibrium under negative frequency dependence or rare-male mating advantage. Thus rare-male mating advantage is important from the viewpoint of population genetics as it plays an important role in the maintenance of genetic polymorphism in natural populations and also promotes outbreeding and exchange of genes among different populations.

In 1951, C. Petit¹ (University of Paris, France) discovered rare-male mating advantage in *Drosophila melanogaster* by employing wild-type and *Bar* eye mutant flies. It was demonstrated that *Bar* males were disadvantaged in competition for mates when they were more common than the wild-type in the population of

competing males. This is an example of one-sided rare-male mating advantage in favour of the wild-type males when present together with the mutant males. Similar results were found with other sex-linked mutations in *D. melanogaster*²⁻⁴. In 1966, Lee Ehrman⁵, while studying mating success of different inversion karyotypes of *D. pseudoobscura*, found that two types of males were equally successful in mating when present in equal ratios. However, when present in unequal proportions, the rare type of males were more successful irrespective of their karyotypes. In general, Ehrman reported two-sided rare-male mating advantage in *D. pseudoobscura*. Since then a number of reports have been documented which suggest that rare-male mating advantages is of common occurrence in *Drosophila* (for reviews, see refs 6-11). Rare-male mating advantage has so far been reported in *D. melanogaster*, *D. pseudoobscura*, *D. persimilis*, *D. immigrans*, *D. pavani*, *D. gaucha*, *D. tropicalis*, *D. willistoni*, *D. equinoxialis*, *D. funebris*, *D. ananassae*, and *D. bipectinata* (see Table 1). It has been shown to occur for mutants, inversion karyotypes, isozyme variants, geo-

graphic strains, strains reared at different temperatures and having behavioural differences. Rare-male mating advantage has been shown to occur for inversion karyotypes in natural population of *D. funebris*¹² and *D. pseudoobscura*¹³. This unique phenomenon has subsequently been shown in other animals also such as flour beetles *Tribolium castaneum*¹⁴, a parasitic wasp *Mormoniella vitripennis*¹⁵, ladybird *Adalia bipunctata*^{16,17} and *Musca domestica*¹⁸. In *Adalia bipunctata*, rare-male effect has been demonstrated for different morphs in a natural population. The generality of this phenomenon where rare-males are favoured in mating has become accepted, but has given rise to much discussion and controversy concerning its causes. Thus more extensive work on this phenomenon might be of interest. Bryant *et al.*¹⁸ suggested that rare-male effect might be an artifact caused by marking the houseflies for identification during experiment. Markow¹⁹ has shown that the fluctuation in mean sexual vigour of males caused by sampling errors might result in spurious rare-male advantage. Differential storage conditions can also bias the outcome of a rare-male effect^{20,21}.

Table 1. Experimental evidence for rare-male mating advantage in different species of *Drosophila*

Species	Aspect surveyed	Details	Ref.
<i>D. melanogaster</i>	Genetic background	Mutant used for identification of strains	Anxolabehere ²⁷
	Mutants	—	Bundgarad and Christiansen ²⁸
	Phenotypic included variations	Flies peppermint scented	Dal Molin ²⁹
	Mutant	—	Magalhaes and Rodrigues-Pereira ^{30*}
	Position in vial	Males not sampled randomly with respect to place in vial	Markow ¹⁹ shown to be an artifact due to nonrandom sampling
	Genetic background	Male ratio varied <i>Bar</i> forked vs wild-type	Markow <i>et al.</i> ³¹
	Mutants	White vs wild-type	Petit ^{1-3*} Petit and Nouaud ^{4*}
	Geographic origin	—	Petit and Nouaud ⁴
	Mutants	Multiple-choice test and single female test	Spiess and Schwer ³²
	Genetic background	—	Tardif and Murnik ³³
	Mutants	Multiple choice test	Spiess and Kruckeberg ³⁴
	<i>D. immigrans</i>	Geographic origin	—
<i>D. pavani/D. gaucha</i>	Species	<i>D. pavani</i> vs <i>D. gaucha</i>	Ehrman, Koref-Santibanez and Falk ³¹
<i>D. pseudoobscura</i>	Geographic origin	Female and male ratio varied	Ehrman ⁵
	Temperature of rearing mutant vs wild-type karyotypes	simultaneously Male ratio varied	
	Karyotypes	Density effect	Ehrman ³⁷
	Karyotypes	Homokaryotype vs heterokaryotype	Ehrman ³⁸
	Mutants	—	Ehrman ^{39*}
	Karyotypes and/or allozymes	Amy(ST) vs Amy(CH)	Ehrman, Anderson and Blatte ^{40*}
	Inversions and mutant	Three types tested simultaneously	Leonard and Ehrman ⁴¹
	Karyotypes (AR vs PP)	Male ratio varied Ratio of both sexes varied simultaneously	Spiess ⁴⁸
<i>D. persimilis</i>	Karyotypes	—	Spiess (see Ehrman ⁵)
	Geographic origin (temperature of rearing - 25 or 15)	Male ratio varied	Spiess and Spiess ⁴²
<i>D. paulistorum</i>	Geographic origin	—	Ehrman ^{43*}
<i>D. tropicalis</i>	Geographic origin	—	Ehrman and Petit ⁴⁴
<i>D. willistoni</i>	Geographic origin	—	Ehrman and Petit ⁴⁴
<i>D. equinoxialis</i>	Geographic origin	—	Ehrman and Petit ⁴⁴
	Karyotypes	—	Ehrman <i>et al.</i> ⁴⁵
	Geographic origin	—	Ehrman <i>et al.</i> ^{45*}
<i>D. ananassae</i>	Mutants and wild-type	Sepia vs wild type Cardinal vs wild-type	Singh and Chatterjee ⁴⁶
<i>D. bipunctata</i>	Mutant and wild-type	Cut vs wild type	Singh and Sisodia ⁴⁷
<i>D. funebris</i>	Karyotypes	Inversions in a natural population	Borisov ¹² (doubtful, only one-sided)

*One-sided.

Because the evolutionary implications of this phenomenon are potentially important, a number of explanations have been proposed to account for the minority male mating advantage (see the reviews by Spiess⁹ and Knoppien¹⁰). To explain the rare-male effect, Ehrman and Spiess²² suggested sampling and habituation hypothesis. According to this hypothesis, the nature of cue is different for different male types. The females become conditioned against mating with the males that first court them during their unreceptive period after eclosion. Since these males would usually be the more frequent type, the rare-male type would gain mating advantage when the females become sexually active as they are able to break through the habituation by its slightly different cues. Rare-male mating advantage can also occur when the two types of males differ greatly in mating behaviour, one male type being more vigorous than the other. It has been demonstrated experimentally that frequency-dependent sexual selection in *D. pseudoobscura* is influenced by age of females, exposure to other flies, and previous mating experience^{23,24}. The rare-male mating advantage is also influenced by the sex-ratio^{25,26}. The cause of rare-male effect is not yet resolved fully and indeed it is likely that there is no single mechanism which is responsible for rare-male mating advantage. It is likely that a combination of specific mechanisms is responsible, and a set of mechanisms operating in one species may be different from those in other species.

1. Petit, C., *Bull. Biol. France Belg.*, 1951, 85, 392-418.

2. Petit, C., *Bull. Biol. France Belg.*, 1954, 88, 435-443.
 3. Petit, C., *Bull. Biol. France Belg.*, 1958, 92, 248-329.
 4. Petit, C. and Nouaud, D., *Evolution*, 1975, 29, 763-776.
 5. Ehrman, L., *Anim. Behav.*, 1966, 14, 332-339.
 6. Petit, C. and Ehrman, L., *Evol. Biol.*, 1969, 3, 177-223.
 7. Ehrman, L. and Propper, J., *Am. Sci.*, 1978, 66, 216-222.
 8. Ehrman, L. and Parsons, P. A., *Behaviour, Genetics and Evolution*, McGraw Hill, New York, 1981.
 9. Spiess, E. B., *Am. Nat.*, 1982, 119, 675-693.
 10. Knoppien, P., *Biol. Rev.*, 1985, 60, 81-117.
 11. Haj-Ahmad, Y. and Hickey, D. A., *Nature*, 1982, 299, 350-352.
 12. Borisov, A. I., *Genetika*, 1970, 6, 61-67.
 13. Salceda, M. V. and Anderson, W. W., *Proc. Nat. Acad. Sci. USA*, 1988, 85, 9870-9874.
 14. Sinnock, P., *Am. Nat.*, 1970, 104, 469-476.
 15. Grant, B., Snijders, G. A. and Glessner, S. F., *Evolution*, 1974, 28, 259-264.
 16. Majerus, M., O'Donald, P. and Weir, J., *Heredity*, 1982, 49, 37-49.
 17. Muggleton, J., *Heredity*, 1979, 42, 57-65.
 18. Bryant, E. H., Kence, A. and Kimball, K. T., *Genetics*, 1980, 96, 975-993.
 19. Markow, T. A., *Behav. Genet.*, 1980, 10, 553-556.
 20. Knoppein, P., *Drosoph. Inf. Serv.*, 1985, 61, 101.
 21. Knoppein, P., *Behav. Genet.*, 1987, 17, 403-425.
 22. Ehrman, L. and Spiess, E. B., *Am. Nat.*, 1969, 103, 675-680.
 23. Pruzan, A. and Ehrman, L., *Behav. Genet.*, 1974, 4, 159-164.
 24. Pruzan, A., *Evolution*, 1976, 30, 130-145.
 25. Sharp, P. M., *Genet. Res.*, 1982, 40, 201-205.
 26. Lechien, J., Derroncourt-Sterpin, C. and Elens, A., *Genetica*, 1990, 80, 189-194.
 27. Anxolabehere, D., *Genetics*, 1980, 95, 743-755.
 28. Bundgaard, J. and Christiansen, F. B., *Genetics*, 1972, 71, 439-460.
 29. Dal Molin, C., *Am. Nat.*, 1979, 113, 951-954.
 30. Magalhaes, L. E. and Rodrigues-Pereira, M. A. Q., *Experientia*, 1976, 32, 309-310.
 31. Markow, T. A., Richmond, R. C., Mueller, L., Sheer, I., Roman, S., Laetz, C. and Lorenz, L., *Genet. Res.*, 1980, 35, 59-64.
 32. Spiess, E. B. and Schwer, W. A., *Behav. Genet.*, 1978, 8, 155-168.
 33. Tardif, G. N. and Murnik, M. R., *Behav. Genet.*, 1975, 5, 373-379.
 34. Spiess, E. B. and Kruckeberg, J. F., *Am. Nat.*, 1980, 115, 307-327.
 35. Ehrman, L., *Behav. Genet.*, 1972, 2, 79-84.
 36. Ehrman, L., Koref-Santibanez, S. and Falk, C. T., *Drosoph. Inf. Serv.*, 1972, 48, 36-37.
 37. Ehrman, L., *Am. Nat.*, 1967, 101, 415-424.
 38. Ehrman, L., *Genet. Res.*, 1968, 2, 135-140.
 39. Ehrman, L., *Proc. Natl. Acad. Sci. USA*, 1970, 65, 345-348.
 40. Ehrman, L., Anderson, W. W. and Blatte, L., *Behav. Genet.*, 1977, 7, 427-432.
 41. Leonard, J. E. and Ehrman, L., *Genetics*, 1983, 104, 713-716.
 42. Spiess, L. D. and Spiess, E. B., *Am. Nat.*, 1969, 103, 155-172.
 43. Ehrman, L., *Behav. Genet.*, 1970, 1, 111-118.
 44. Ehrman, L. and Petit, C., *Evolution*, 1968, 22, 649-658.
 45. Ehrman, L., Spassky, B., Pavlovsky, O. and Dobzhansky, T., *Evolution*, 1965, 19, 337-346.
 46. Singh, B. N. and Chatterjee, S., *Genet. Sel. Evol.*, 1989, 21, 447-455.
 47. Singh, B. N. and Sisodia, S., *Genetika*, 1997, 29, 41-48.
 48. Spiess, E. B., *Am. Nat.*, 1968, 102, 363-379.

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Confinement of electrons in a Penning trap

Ion traps have been in use for over three decades for the confinement of atomic ions and other charged particles. They have also been used for precision experiments in atomic physics, nuclear physics and quantum optics¹⁻⁴. We have initiated the setting up of ion traps with a view to carrying out precision spectroscopic studies of trapped ions. Our initial

endeavour to trap electrons in a Penning trap has been successful. In this short note, we describe our experimental set-up to trap and detect electrons.

Our ion trap (Figure 1 a and b) consists of a cylindrical, three-electrodes system with symmetrical structure: two endcap electrodes and a ring electrode. This arrangement gives rise to a quadrupole

electric potential with cylindrical symmetry of the form⁵

$$\phi(r, z) = \frac{U_0}{2r_0^2} (x^2 + y^2 - 2z^2), \quad (1)$$

where r_0 is the ring radius. The potential $\phi(r, z)$ has a saddle point at the trap centre, with a minimum in the axial