Action on amphibians

The first World Congress of Herpetology (University of Kent, Canterbury, UK, 1989) brought together field biologists who observed the first apparent reappearance of various species of frogs, sufficient concern being expressed to cause the National Science Foundation (NSF) to convene a special workshop (February, 1990) to explore the matter. The organization of a special task force on declining amphibian populations (DAPTF), allied with the Species Survival Commission (IUCN, Gland, Switzerland) and related to the outcome of this workshop. Throughout this decade, substantial research has been conducted on amphibian populations and, although many questions remain and many species and several areas of the world appear unaffected, there have been extensive species declines and extinctions (10–14 recent species extinctions can be documented, and evidence of additional extinctions exists). At the third World Congress of Herpetology (Prague, Czech Republic, 1997), a symposium highlighted new evidence of declines and hypotheses for their causes. The need for an in-depth analysis of the problem and development of a general research strategy was expressed by many, and in the months since the congress new developments have lent a sense of urgency. For example, Bruce Babbitt (Secretary of the Interior, USA) has shown a personal interest in the matter, requesting several briefings. At the most recent (May, 1998), two other USA presidential cabinet officers, Donna Shalala (Health and Human Services, NSF), Neal Lane, and Bruce Babbitt joined the group later, and Bruce Babbitt pledged his support for developing a coordinated approach to the problems. Agreement was obtained on the fundamental points:

- The phenomenon is real – there has been documentation of species loss and/or decline in Cordilleran America from the Rocky Mountains and Cascades through the Sierra Nevada to Costa Rica and Panama, and the high paramos of Venezuela and Ecuador. Results from Australia are also convincing.
- Not all species in a given region are similarly affected. Some regions, especially high amphibian diversity, such as Colombia and Indonesia. Issues that field biologists do not normally face mean that investigators representing other approaches will have to be recruited. A sense of urgency exists concerning new fungal and viral diseases, and the increased impact of algal blooms. These issues were discussed by virologists and pathologists attending the workshop, but researchers in these disciplines are rarely involved in field work. Several participants emphasized the role of generalist stress. Environmental stress factors remain poorly understood. We need to understand natural versus anthropogenic stressors, and the impact of stressors on amphibian immune systems and physiology. In general, more field workers are required, especially in tropical areas.

Proposals for action

The participants developed the following specific proposals.

(1) Development of a new distributed institute: this came to be termed the ‘Cordilleran Initiative’. The institute would link agency and independent research units in western North America with researchers focusing attention on Costa Rica and Panama, although Mexican, Guatemalan, Honduran and other appropriate groups would also be included. The institute would also sponsor collaborative interaction, such as internships for training professionals, graduate students and post-doctoral fellows, as well as providing opportunities for regular communication via workshops and the Internet. Data management systems and communication systems are also a necessary component of such an institute. The approach would be multidisciplinary and would involve the development of rapid response teams.

(2) A series of specialized workshops:
- Amphibian diseases and immunity – the Centers for Disease Control in Atlanta, which should be invited to participate...
(this workshop was immediately funded by NSF and will take place in San Diego on 26–28 July 1998).

- Population and community ecology and monitoring – focused critical thinking is needed to deal with statistical issues in assessing population dynamics.
- Ecotoxicology – airborne contaminants are implicated in amphibian declines, and existing test protocols might be inappropriate.
- Developmental biology – malformations in amphibians demand investigation because normal developmental processes are being disrupted.
- Immediate action is being taken to set up an interagency Task Force on Amphibian Declines and Deformities (chaired by William F. Brown, science advisor to Bruce Babbitt) in the USA, which will be important in the establishment of an interdisciplinary and collaborative research program. Tasks include quantification of the direct and indirect factors affecting amphibian population dynamics, starting with the study of patterns of change through the use of historical records, field-based correlative data, and controlled, multilaboratory experiments. Organization of interdisciplinary incident response teams was also recommended. These teams could be assembled in ‘hot spots’ of amphibian decline to identify factors to mitigate these declines. Finally, the workshop participants called upon both public and private agencies and institutions for the funding and policy support required for future activities.

David B. Wake

Museum of Vertebrate Zoology, University of California, Berkeley, CA 94720-3400, USA

(wake@lbcb.berkeley.edu)

References


Mother’s boy or daddy’s girl?

Sex determination in Hymenoptera

Sex determination in hymenopteran insects is superficially simple: fertilized eggs develop as diploid females; unfertilized eggs develop as haploid males. But this apparent simplicity hides a more difficult question. How can two copies of a genome cause an embryo to develop as a male, when no gene that is present in one sex is absent in the other, and when all ratios among sex-determining genes are unchanged? Whiting proposed an answer to this conundrum based on his discovery of diploid males among mated progeny of the parasitoid Bracon hebetor1. In his model of complementary sex determination (CSD), heterozygotes at a multi-allelic sex-determining locus develop as females, whereas hemizygotes and homozygotes develop as males.

Diploid males have now been reported in more than 30 hymenopteran species – including sawflies, bees, ants, branroids and ichneumonid wasps – and CSD is believed to be the mechanism of sex determination in these species2,3. However, CSD cannot explain the difference between the sexes in species that do not produce diploid males, even after prolonged inbreeding. One such species is the chalcidoid wasp Nasonia vitripennis. A recent paper now provides evidence that femaleness in N. vitripennis is determined by the presence of a paternal genome, and maleness by its absence4. Dobson and Tanouye5 tested alternative theories of sex determination in N. vitripennis by mating triploid females to haploid males, some of which carried the paternal sex-ratio chromosome (PSR). Triploid females came from a polyplody strain of N. vitripennis that had been maintained by re-pooled matings of diploid males to triploid females (diploid males produce diploid sperm; triploid females lay abundant eggs, most of which are aneuploid, but a few have haploid or diploid chromosome complements and produce viable offspring).

In crosses using this strain, unfertilized eggs (whether haploid or diploid) produce diploid, triploid or tetraploid females5. The PSR chromosome is a B chromosome, transmitted via sperm, that causes the elimination of all paternal chromosmes (except itself) from early embryos. The results of Dobson and Tanouye’s crosses (Box 1) were incompatible with most published hypotheses about the mechanism of haplodiploid sex determination. Single-locus and multilocus CSD models could be rejected because unfertilized, heterozygous diploid eggs developed as males. The act of fertilization itself could not be female-determining (as proposed by Whiting) because eggs fertilized by PSR-bearing sperm developed as males. Finally, ‘maternal-effect’ and ‘genic-balance’ models could be rejected because these models predict a one-to-one correspondence between sex and ploidy. The only model that was not contradicted by the data was Binkley’s hypothesis that one or more loci are differentially modified (imprinted) in male and female germ lines, such that a paternal genome is necessary for female development. Imprints must be reversible in this model because a paternal allele in one generation is a maternal allele in the next.

Mother’s boy or daddy’s girl?

Sex determination in Hymenoptera

Sex determination in hymenopteran insects is superficially simple: fertilized eggs develop as diploid females; unfertilized eggs develop as haploid males. But this apparent simplicity hides a more difficult question. How can two copies of a genome cause an embryo to develop as a male, when no gene that is present in one sex is absent in the other, and when all ratios among sex-determining genes are unchanged? Whiting proposed an answer to this conundrum based on his discovery of diploid males among mated progeny of the parasitoid Bracon hebetor. In his model of complementary sex determination (CSD), heterozygotes at a multi-allelic sex-determining locus develop as females, whereas hemizygotes and homozygotes develop as males.

Diploid males have now been reported in more than 30 hymenopteran species – including sawflies, bees, ants, branroids and ichneumonid wasps – and CSD is believed to be the mechanism of sex determination in these species. However, CSD cannot explain the difference between the sexes in species that do not produce diploid males, even after prolonged inbreeding. One such species is the chalcidoid wasp Nasonia vitripennis. A recent paper now provides evidence that femaleness in N. vitripennis is determined by the presence of a paternal genome, and maleness by its absence. Dobson and Tanouye tested alternative theories of sex determination in N. vitripennis by mating triploid females to haploid males, some of which carried the paternal sex-ratio chromosome (PSR). Triploid females came from a polyplody strain of N. vitripennis that had been maintained by re-pooled matings of diploid males to triploid females (diploid males produce diploid sperm; triploid females lay abundant eggs, most of which are aneuploid, but a few have haploid or diploid chromosome complements and produce viable offspring).

In crosses using this strain, unfertilized eggs (whether haploid or diploid) produce diploid, triploid or tetraploid females. The PSR chromosome is a B chromosome, transmitted via sperm, that causes the elimination of all paternal chromosmes (except itself) from early embryos. The results of Dobson and Tanouye’s crosses (Box 1) were incompatible with most published hypotheses about the mechanism of haplodiploid sex determination. Single-locus and multilocus CSD models could be rejected because unfertilized, heterozygous diploid eggs developed as males. The act of fertilization itself could not be female-determining (as proposed by Whiting) because eggs fertilized by PSR-bearing sperm developed as males. Finally, ‘maternal-effect’ and ‘genic-balance’ models could be rejected because these models predict a one-to-one correspondence between sex and ploidy. The only model that was not contradicted by the data was Binkley’s hypothesis that one or more loci are differentially modified (imprinted) in male and female germ lines, such that a paternal genome is necessary for female development. Imprints must be reversible in this model because a paternal allele in one generation is a maternal allele in the next.

---

**Mother’s boy or daddy’s girl?**

**Sex determination in Hymenoptera**

Sex determination in hymenopteran insects is superficially simple: fertilized eggs develop as diploid females; unfertilized eggs develop as haploid males. But this apparent simplicity hides a more difficult question. How can two copies of a genome cause an embryo to develop as a male, when no gene that is present in one sex is absent in the other, and when all ratios among sex-determining genes are unchanged? Whiting proposed an answer to this conundrum based on his discovery of diploid males among mated progeny of the parasitoid Bracon hebetor. In his model of complementary sex determination (CSD), heterozygotes at a multi-allelic sex-determining locus develop as females, whereas hemizygotes and homozygotes develop as males.

Diploid males have now been reported in more than 30 hymenopteran species – including sawflies, bees, ants, branroids and ichneumonid wasps – and CSD is believed to be the mechanism of sex determination in these species. However, CSD cannot explain the difference between the sexes in species that do not produce diploid males, even after prolonged inbreeding. One such species is the chalcidoid wasp Nasonia vitripennis. A recent paper now provides evidence that femaleness in N. vitripennis is determined by the presence of a paternal genome, and maleness by its absence. Dobson and Tanouye tested alternative theories of sex determination in N. vitripennis by mating triploid females to haploid males, some of which carried the paternal sex-ratio chromosome (PSR). Triploid females came from a polyplody strain of N. vitripennis that had been maintained by re-pooled matings of diploid males to triploid females (diploid males produce diploid sperm; triploid females lay abundant eggs, most of which are aneuploid, but a few have haploid or diploid chromosome complements and produce viable offspring).

In crosses using this strain, unfertilized eggs (whether haploid or diploid) produce diploid, triploid or tetraploid females. The PSR chromosome is a B chromosome, transmitted via sperm, that causes the elimination of all paternal chromosmes (except itself) from early embryos. The results of Dobson and Tanouye’s crosses (Box 1) were incompatible with most published hypotheses about the mechanism of haplodiploid sex determination. Single-locus and multilocus CSD models could be rejected because unfertilized, heterozygous diploid eggs developed as males. The act of fertilization itself could not be female-determining (as proposed by Whiting) because eggs fertilized by PSR-bearing sperm developed as males. Finally, ‘maternal-effect’ and ‘genic-balance’ models could be rejected because these models predict a one-to-one correspondence between sex and ploidy. The only model that was not contradicted by the data was Binkley’s hypothesis that one or more loci are differentially modified (imprinted) in male and female germ lines, such that a paternal genome is necessary for female development. Imprints must be reversible in this model because a paternal allele in one generation is a maternal allele in the next.