

Embryo and seed abortion in plants

WIENS ET AL. REPLY—Our letter to *Nature*¹ reported extensive (97.5 per cent) genetically mediated developmental failure of ovules and consequent loss of reproductive capacity in *Dedeckera eurekaensis* (Polygonaceae), a rare, palaeoendemic, monotypic genus from the Mojave desert of California.

Bawa *et al.* question both the uniqueness and interpretation of our results. They indicate that mimosoid legumes have exceptionally low fruit/flower ratios (per cent fruit set), but relatively high seed/ovule ratios (per cent seed set) giving these plants overall seed set similar to *Dedeckera* (2–3 per cent). We have confirmed these results in African *Acacia* and North American *Prosopis*. Apocynaceae and Asclepiadaceae behave similarly, as do many woody groups, and plants reproducing vegetatively. These reproductive systems, however, are not comparable to that of *Dedeckera*.

In mimosoid legumes low fruit set is genetically programmed by the maternal genome. The great majority of ovules are probably unfertilized, or if fertilized, probably abort before the activation of the zygotic genome at differentiation, or their maturation is physiologically inhibited by earlier developing fruits. Such ovules never enter the potential seed pool.

Maternal fitness, however, is determined by the absolute number of fully viable seeds produced. In spite of the low fruit set per inflorescence, a mimosoid may produce hundreds or possibly thousands of inflorescences with one to several fruits and thousands of seeds.

In *Dedeckera* more than 95 per cent of the ovules are fertilized (flowers are uniovulate) and develop to various stages. The few filled seeds that mature occur randomly in the inflorescence: any ovule can potentially enter the seed pool. The 97.5 per cent abortion rate among *Dedeckera* ovules, however, represents only a portion of the reproductive loss. Large, vigorous plants of *Dedeckera* may produce about 64,500 flowers (and ovules). Of the 2.5 per cent of filled seeds only 3.5 per cent germinate spontaneously, and only 11 per cent are without post-developmental abnormalities. Most *Dedeckera* plants probably produce less than 50 fully viable seeds annually. Some plants are apparently totally sterile. Seed inviability of this magnitude seems unprecedented among any evolutionary successful species.

Severely reduced seed sets (inbreeding depression) are often encountered when typically outcrossing plants are selfed. Such results are of little interest. The similarly low seed sets obtained in *Dedeckera* from both outcrossing and inbreeding are precisely what make it so unusual. The genetic load in *Dedeckera* is perhaps so high that rare, recombinant gametes produced upon selfing may have as great a probability of producing a viable embryo/ endosperm as outcrossed progeny. A number of phylogenetically and geographically rare plants (monotypic families) seem to have reproductive capacities similar to *Dedeckera*. These endangered plants may also exhibit unusual genetic phenomena not heretofore encountered. They require urgent study.

The failure of ovular development in general is attributable to several causes. Nonrandom abortion (maternally controlled) is exceedingly common and likely under fixed genetic control of the maternal genome¹⁶. Random ovule abortion is primarily attributable to genetic load and developmental selection¹⁷.

Our previous letter on *Dedeckera* is essentially concerned with reduction of fitness, and presents a difficult evolutionary conundrum. If the loss of fecundity resulted in continued population decline, why would selection not reverse the trend? We proposed a heterosis (balanced-load) model that allowed for the survival of unique, highly heterozygous genotypes in an environment stressed by increasing aridity. Such genotypes should suffer drastically reduced fecundity from genetic load. Selection, however, must first ensure individual survivorship, then fecundity. This could ultimately lead to extinction.

How can such a situation be reconciled with sibling rivalry/parent-offspring conflict hypotheses? How can they explain the high loss of seed viability, germinability, and the presence of extensive postgermination developmental abnormalities, all of which follow the release of seeds from the maternal plant? Likewise, how can they account for embryo deaths similar to those known to be controlled by developmentally lethal genes¹⁸.

These highly anthropomorphic, sociobiological hypotheses are best not applied to plants. We suggest they cannot be tested critically, and have no mechanism to explain their operation. The causal-mechanistic genetic based hypotheses, although not without difficulties, are founded on established phenomena and Occam's razor dictates their acceptance.

D. WIENS

E. J. KING

Department of Biology,
University of Utah,
Salt Lake City, Utah 84111, USA

D. L. NICKRENT

Department of Plant Biology,
University of Illinois,
Urbana, Illinois 61801, USA

C. L. CALVIN

Department of Biology,
Portland State University,
Portland, Oregon 97207, USA

N. L. VIVRETTE

Ransom Seed Laboratory,
P.O. Box 300
Carpinteria, California 93013, USA

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