

INBREEDING and FITNESS

By Diana Walstad (Oct 2026)

Sadly, many popular aquarium fish species have been degraded by continuous inbreeding. A hobbyist obtains a bag of full siblings and breeds them. The sibling progeny are distributed to other hobbyists that also mate siblings. Eventually, the fishes' fitness will decline from ID (*inbreeding depression*). Hobbyists often associate ID with gross deformities. They may ignore gradual reductions in fitness (reproduction, survival, disease resistance, and longevity)—or blame it on poor tank conditions, novel pathogens, etc. Fitness traits, which involve numerous alleles with small effects, are reportedly 5 times more vulnerable to ID than morphological traits [1].

For this article, I chose scientific studies that I believe best show inbreeding's effect on fitness. I ask readers not to negate studies with invertebrates. After all, DNA—the target of inbreeding—is the universal biomolecule shared by all sexually reproducing plants, animals, and invertebrates. The DNA codes differ, but the underlying genetic mechanisms (meiosis, recombination, etc.) are the same.

Anecdotal information and inbreeding studies with just a few fish, a single trait, and only one generation of inbreeding can be misleading.

In contrast, experimental studies like Parrett's 2025 study [2] with bulb mites provide meaningful information. Results were based on 36 replicate strains created by mating a male and female sibling for 5 sequential generations. The "fitness metric" was the number of adult offspring produced by a single mating pair after 3 days of egg laying. This metric is comprehensive in that it includes mating success, number of eggs laid, egg hatch rate, and survival of larva to adulthood.

Fig 1 from Parrett's study [2] shows—in a nice dose-dependent manner—decreasing fitness with increased inbreeding over 5 generations of sibling matings. Graph shows considerable individual variation, mainly due to inbreeding's "scatter-shot" effect on DNA. For example, at Generation 1, sibling pairs produced a median of ~80 adult offspring, yet one individual pair produced ~120 and other pairs produced ~40.

The underlying causes of ID are: (1) increased DNA homozygosity; and (2) the loss of allelic richness.

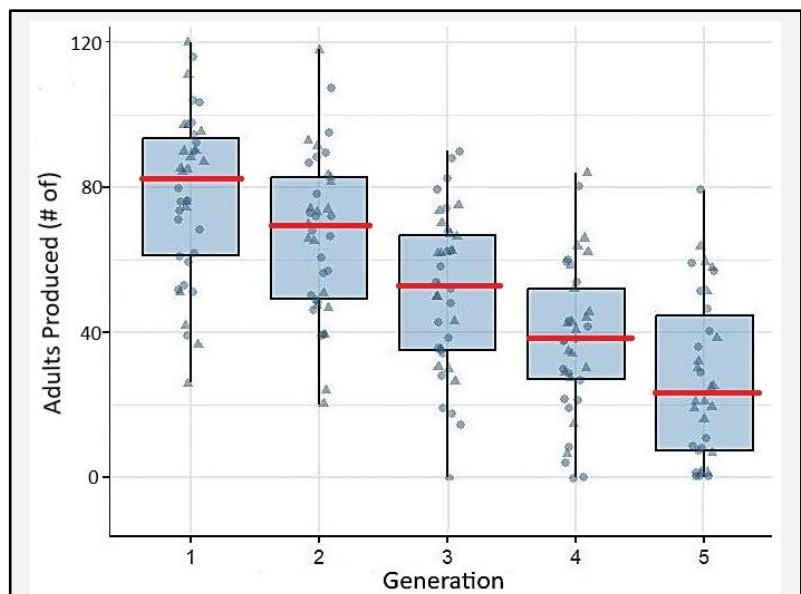


Fig 1 Inbreeding Decreases Fitness of Bulb Mites [2]

Graph shows 'Adults Produced' by each sibling pair. Inbreeding consisted of 5 sequential generations of sibling matings. Each generation's *box plot* shows the median number (50th percentile) as a thick red line for 36 replicate strains. (Due to reproductive failure, 7 of the starting 36 replicate strains went extinct during the inbreeding.) Top and bottom of the box represent the 75th and 25th percentiles, respectively. {Slightly redrawn from Parrett's Fig S11 [2]}

DNA Homozygosity

Inbreeding leads to increased DNA homozygosity where identical bad alleles can pair up at a genetic locus and reduce fitness. Bad alleles can easily become *fixed* in small populations (all individuals carry them). In contrast with a large, non-inbred populations, a bad allele is typically paired with a normal dominant allele, such that the bad allele is not expressed and the animal suffers no reduced fitness.

Homozygosity involves segments of chromosomes that are *identical by descent*, because they were inherited from the same ancestor(s). The more shared ancestry, the greater the homozygosity.

The inbreeding coefficient (F) quantifies inbreeding and DNA homozygosity (See **Box**). There are many tools for determining F , but I feature here *runs of homozygosity* (ROHs). Scientists applaud this “new-generation” method [4]. It can be used—without a pedigree—to determine the *actual* homozygosity of *individuals* rather than just *predict* population *averages*. Investigators sample SNPs within the animal’s genome (entire DNA) to find ROHs, which are chromosome segments that are homozygous due to inbreeding. Length of these segments can range up to ~50 million base pairs (Mb). Longer segments represent more recent inbreeding; shorter segments, more distant inbreeding. (Over time, genetic recombination breaks up long segments into shorter segments.)

Fig 2 depicts ROHs in the chromosomes of 14 wild sheep [5]. The sheep were members of an island population isolated for thousands of years, so it was quite inbred. The population of ~6,000 sheep averaged $F=0.24$ (range 0.18-0.50), virtually identical to that obtained by mating full siblings. The 7 individuals with the most ROHs (Fig 2) would be the most inbred; the 7 with the least ROHs would be the least inbred.

Fig 2 nicely shows inbreeding’s random, scatter-shot effect on each animal’s DNA and their chromosomes. This implies that *all* traits (fertility, longevity, growth, behavior, morphology, etc.) are vulnerable to ID.

The investigators [5] reported that—for every 10% increase in F —a lamb’s chance of survival was reduced by 60%.

Inbreeding Coefficient (F)

Inbreeding is quantitated via the inbreeding coefficient (F). The greater the inbreeding, the higher the F . An $F=0$ means that all allelic pairs within the animal’s genome contain two different alleles. It is the heterozygous condition, exemplified by large wild populations or the progeny of an outcross.

Mating full-siblings or doing a backcross produces progeny with an $F=0.25$, meaning that the DNA of the progeny is 25% homozygous. (Multiply F by 100 to get % DNA homozygosity.)

F can be calculated from pedigree [3] or by new genomic methods such as measuring ROHs.

{See Glossary at end of article for other definitions.}

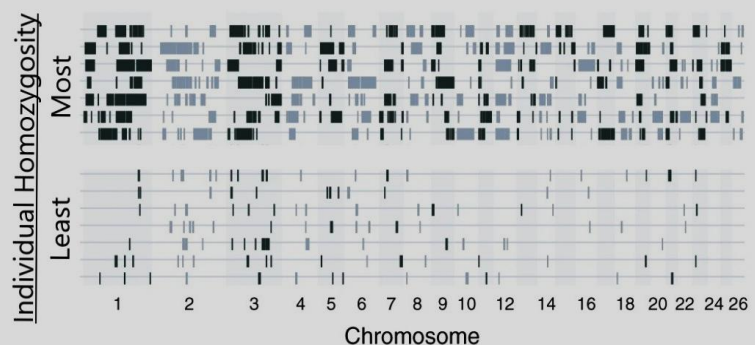


Fig 2 Inbreeding’s Scatter-shot Effect on DNA [5]

Dark rectangles represent ROHs (runs of homozygosity) longer than 5 million base pairs in the 7 most inbred and the 7 least inbred individual sheep of an inbred, wild population.

{I adapted Fig 1a from Stoffel’s 2021 paper [5].}

Allelic Richness

Large wild populations typically carry a wide variety of different alleles at each genetic locus. Many of the alleles are rare (i.e., exist at low frequencies). These wild populations are *allelic rich*. Inbreeding causes a much faster loss of rare alleles than more common alleles [6]. Since homozygosity measurements focus mainly on the more common alleles, F does not accurately measure allelic richness.

Also, F -levels and homozygosity are sometimes not relevant to fitness. For example, one could cross 2 different strains of inbred guppies to get entirely heterozygous offspring with an $F = 0$. Nevertheless, if both parents are *allelic poor*, the offspring will also be allelic poor. For once an allele is lost, it cannot be easily regained. Only long-term evolution (hundreds of generations) or outcrossing to strains carrying those—or similar—rare alleles would bring back the lost fitness.

Allelic richness increases a population's ability to adapt to stress, environmental changes, or to new, more virulent forms of *potential* pathogens. [Fish are always in an evolutionary “arm's race” with common fish pathogens (e.g., Ich, flukes, mycobacteria, *Aeromonas*, etc.).]

Decreased allelic richness is particularly relevant to immunity. That is because MHC genes for disease resistance contain dozens of distinct alleles required to recognize a wide variety of pathogens. Because rare alleles are quickly lost during inbreeding, highly polymorphic genes like MHC are particularly degraded—and there goes disease resistance!

In a study [7] where guppies were experimentally infected with the skin fluke *Gyrodactylus turnbulli*, a common guppy pest, ornamental guppies (10 different strains) had a 10-fold higher peak infestation compared to wild guppies. The wild guppies had enough immunity to control their infections. The “allelic poverty” of ornamental guppies compared to wild guppies has been shown repeatedly [8, 9]. For example, wild guppies were found to have 15-16 alleles at a key MHC gene site for immunity, whereas 3 ornamental guppy strains had only 1-3 [9].

The poor survival of captive-bred fish released into the wild is often attributed solely to long-term captive breeding with its benign environment. However, captive breeding often involves some inbreeding due to small population sizes and artificial selection for desired traits. The result is a loss of allelic richness, not just heterozygosity.

Inbreeding and Stress

Some inbred animals never show problems until they are stressed.

Bijlsma's 2000 study [10] with fruitflies shows how much inbreeding can threaten populations when stressed. (The stressors in this study were heat and ethanol.)

I admire this study for several reasons. Unlike studies monitoring a single trait (e.g., juvenile survival), fitness was defined as colony extinction, which includes multiple traits (i.e., mating success, egg laying, egg hatching, and juvenile survival). This comprehensive metric—similar to the one used in the bulb mite study [2]—captures inbreeding's total and “real world” effect. Second, inbreeding's impact was tested with several inbreeding levels and **lots** of replicates.

The investigators first prepared 6 inbred lines from a heterozygous stock population of *Drosophila* fruitflies. Fruitflies were inbred to different F levels via their maintenance—for 7 generations—at small population sizes. [F increases as N decreases by standard formula ($F = 1 \div 2N$).] **Table 1** shows the 6 designated population sizes (N) and the resulting F . N32 lines were created by maintaining a population size of 16 breeding pairs for each of the 7 generations; N2 lines were inbred via 7 consecutive sibling matings. The table shows that some of the more inbred lines went extinct during the inbreeding. For example, 60% of the N2 lines went extinct. To ensure enough surviving replicates for later experiments, the investigators wisely started many more replicates (i.e., 30) for the more inbred lines.

After line/strain preparation, the investigators randomly selected 10 replicates from each F -level and expanded their populations, allowing the flies to grow normally for 3 generations in large bottles with a carrying capacity of 350-450 larvae.

For testing, the fruitflies were transferred to small vials. Each of the 6 F -levels was started with 50 vial colonies (i.e., 10 replicate lines per F times 5 replicate vials per line). All colonies were maintained at 20N (10 females plus 10 males) for 8 generations during testing.

Fig 3 shows the results as the % of the 50 starting vial colonies that went extinct over those 8 generations. The upper 'Control' graph, representing flies maintained at

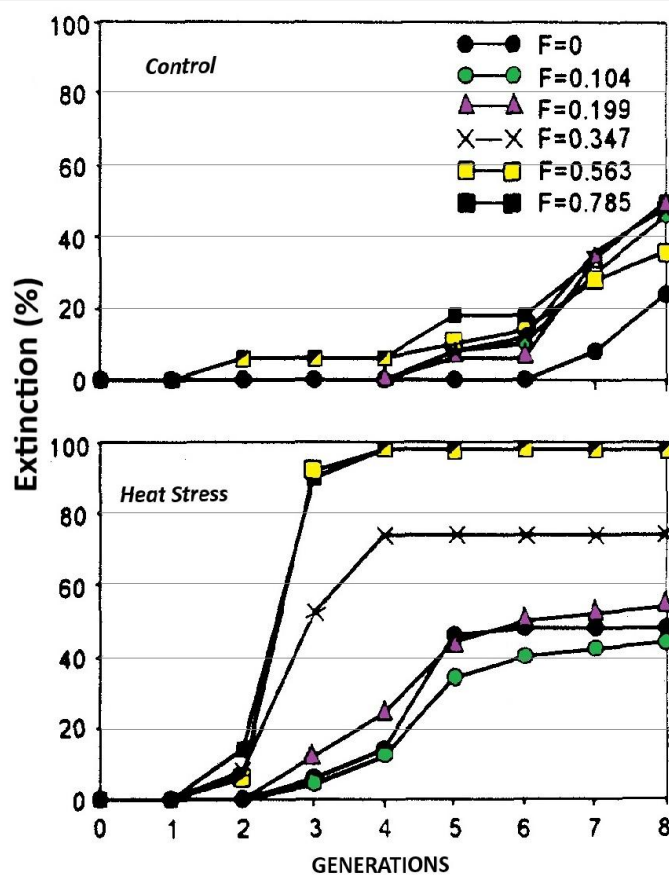


Fig 3 Stress Dramatically Increases ID

'Extinction %' represents the percent of 50 vial colonies per inbreeding level (F) that went extinct over 8 consecutive generations. Top graph shows results for control colonies maintained at a normal 25°C; bottom graph, results for colonies maintained at 28.5°C.

{Redrawn from material in Bijlsma's Fig 2 [10]}

Table 1 Preparation of *Drosophila* Lines

Flies were inbred to each of the 6 'Inbreeding Levels (F)' by maintaining them for 7 generations at the designated population sizes (N). 'Extinction (%)' is the % of the 10-30 replicate lines that went extinct during their preparation.

Inbreeding Level (F)	Population Size (N)	Extinction (%)
0	Base (∞)	0
0.104	N32	0
0.199	N16	0
0.347	N8	20
0.563	N4	25
0.785	N2	60

{Adapted from Bijlsma's Table 1 [10]}

normal temperature, shows that overall, the inbred lines did fairly well. [The fact that all flies were maintained at N20 during testing might explain the ~20% extinction of the non-inbred, $F=0$ lines. (Maintaining an N20 for multiple generations would generate moderate inbreeding.)]

In stark contrast, inbreeding devastated matching lines subjected to 'Heat Stress' (flies maintained at 28.5°C). The bottom graph of Fig 3 shows that 2 of the most inbred lines ($F = 0.563$ and 0.785) suffered ~90% extinction as early as Generation 3. By Generation 4, all their colonies had gone extinct.

In addition, Bijlsma [10] showed that stress's impact was accelerated at higher inbreeding levels, meaning that inbreeding and stress do not act independently but synergistically. Severely inbred fish will be more vulnerable to stress than expected.

Inbreeding's Long-term Effect

Bijlsma [10] also showed negative effects ~50 generations after the original inbreeding.

For this second experiment, the investigators had set aside flies from the same lines prepared earlier (Table 1). These flies were not tested until much later. Rather, they were allowed to propagate normally in large bottle cultures for 2 years (~45 generations) at population sizes that would hinder inbreeding. The bottle colonies were meant to represent flies that had gone through a *population bottleneck* (period of inbreeding) and then allowed to expand and supposedly recover.

The investigators tested the 'recovered' lines for extinction, just as they had tested matching lines in experiments two years earlier. Would the previous inbreeding impact fitness?

The answer was a resounding "Yes!" Results (Fig 4) show that under control conditions the inbred lines appeared to have recovered somewhat over the 2 years of normal growth. None of them—even the most inbred—had extinction rates above 40%. Under heat stress, though, inbreeding's long-term effect was readily apparent. Inbred lines—compared to the non-inbred lines ($F=0$)—were going extinct at dramatically high rates. About 80% of the most inbred lines ($F = 0.563$ and 0.785) had gone extinct by Generation 5.

Bijlsma's study [10] also documented extinction rates due to the continuous mating of siblings, plus the impact of ethanol stress. (Results were similar to those shown here.)

Does Purging Help?

By increasing DNA homozygosity, inbreeding can purge (remove) bad alleles from populations due to the death and infertility of individuals carrying those bad alleles in the homozygous condition. To reduce the purging factor in Bijlsma's study, the investigators purposely tested the flies at least 3 generations after the inbreeding. Thus, the flies (Figs 3 and 4) should have been purged—cleaned up, genetically speaking—before being tested. However, I fail to see any fitness improvement due to inbreeding.

Purging is no magic bullet [6]. It removes major, lethal alleles efficiently but not the mildly harmful ones that typically determine fitness. Moreover, purging is an exceedingly slow process. Perez (2021) [11] showed that it required about 100 generations of slow inbreeding to purge most deleterious alleles from *Drosophila* populations.

Both Perez [8] and Bijlsma [10] argue that purging only increases fitness in the environment in which the purging took place. When the environment changes, new alleles for fitness may be required.

Conservation biologists advise against purging. It not only may not work, it is risky. The inbreeding required for purging can drive small, captive populations to extinction.

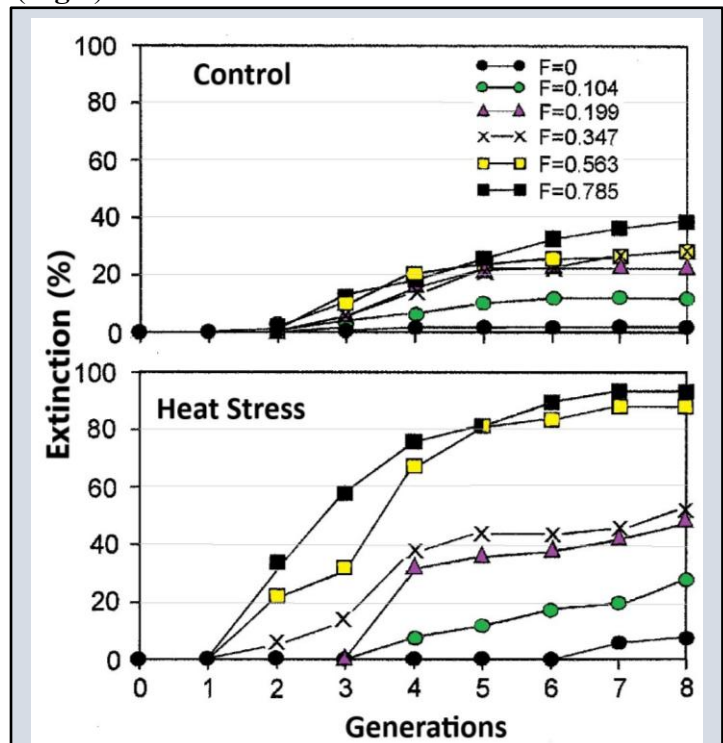


Fig 4 Long-term Effects of Inbreeding

Figure 4 shows that inbreeding has long-term effects, particularly under stressful conditions. 'Extinction (%)' results are for fruitfly lines/strains that were replicates of those inbred earlier (See Table 1), except that these strains were not tested until 2 years (~45 generations) later. During those 2 years, the flies were maintained normally (i.e., no inbreeding, no stress, etc.). See Fig 3 for details of their testing.

{Redrawn from material in Bijlsma's Fig 3 [10]}

Discussion

Most hobbyists are aware that inbreeding reduces fitness, but not how much, or that negative effects are long-term, or that inbreeding greatly increases a fish's vulnerability to stress.

The sensitivity of inbred fish to stress has important implications for aquarium hobbyists. An inbred fish may perform well in its original home, but do very poorly when transferred to a new tank. An inbred fish may lack rare MHC alleles for resisting new variants of potential pathogens.

There is considerable variation in inbreeding's effect (Fig 1). Thus, a few fish breeders will succeed with inbred fish—assuming the fish are never stressed. Rare examples of successful inbreeding are the exception—the lottery winners, so to speak—, not the rule. The Channel Island foxes of California have managed—via natural purging over thousands of years—to thrive despite being severely inbred. That said, how many similarly small, isolated populations have quietly gone extinct?

Fish breeding should be more than just putting fish into a tank and collecting offspring. Captive breeding may become the last resort for rescuing the many fish species whose native habitats are fast disappearing. The appalling fragility of ornamental guppy strains, severely inbred for decades, may predict the eventual fate of newer species in the hobby. There may come a time when there will be precious few wild and/or non-inbred fish for outcrossing to increase heterozygosity and allelic richness. Hobbyist efforts to maintain the genetic integrity of captive fish need to be encouraged. Hopefully, a greater awareness of inbreeding's devastating impact on fish fitness will someday get the same attention as environmental factors (water quality, fish diseases, etc.) are currently getting in the aquarium hobby.

{Note: This article was first published in the ALA Newsletter [Sept 2026 issue (#267)] of the American Livebearers Association (<https://livebearers.org/>)}

References

1. DeRose MA and DA Roff. 1999. A comparison of inbreeding depression in life-history and morphological traits in animals. *Evolution* 53: 1288-92.
2. Parrett, JM *et al.* 2025. Fighting to persist: Genetic rescue increases long-term fitness despite elevated genetic load. *bioRxiv*, 2025-09. Figure made available under a CC-BY 4.0 International license.
3. From: Nikolay's Genetics Lessons. 2015. How to calculate inbreeding from a pedigree chart. From <https://www.youtube.com/watch?v=1H8mhSHCWxw> (accessed Feb. 2026)
4. Kardos M, Luikart G and FW Allendorf. 2015. Measuring individual inbreeding in the age of genomics: marker-based measures are better than pedigrees. *Heredity* 115: 63-72.
5. Stoffel MA *et al.* 2021. Genetic architecture and lifetime dynamics of inbreeding depression in a wild mammal. *Nature Communications* (2021) 12:2972 (Fig 1a is from an "open access" paper made available for distribution via <https://creativecommons.org/licenses/by/4.0/>)
6. Allendorf FW *et al.* 2013. *Conservation and the Genetics of Populations* (2nd Edition). Wiley-Blackwell Publishers (West Sussex, UK), 602 pp.
7. Smallbone W, Ellison A, Poulton S, van Oosterhout C, and J Cable. 2021. Depletion of MHC supertype during domestication can compromise immunocompetence. *Molecular Ecology* 30: 736-746.
8. Bleakley BH *et al.* 2008. Are designer guppies inbred? Microsatellite variation in five strains of ornamental guppies, *Poecilia reticulata*, used for behavioral research. *Zebrafish* 5: 39-48.
9. van Oosterhout C *et al.* 2006. Evolution of MHC class IIB in the genome of wild and ornamental guppies, *Poecilia reticulata*. *Heredity* 97: 111-118.
10. Bijlsma R *et al.* 2000. Does inbreeding affect the extinction risk of small populations? predictions from *Drosophila*. *J Evol Biol* 13: 502-514.
11. Perez-Pereira N *et al.* 2021. Long-term exhaustion of the inbreeding load in *Drosophila melanogaster*. *Heredity* 127: 373-383

GLOSSARY

alleles are components of genes. A gene is made up of pairs of alleles, one allele from the mother, one from the father. The two alleles in a pair interact with each other to determine a trait.

allelic richness means that a population has a large number of different alleles distributed among its individuals. The greater the allelic richness, the greater the genetic diversity, and the greater the population's capacity to adapt to change.

“bad alleles”- recessive alleles that—due to a mutation—no longer code for a normal protein; scientists often refer to bad alleles as a population's “genetic load”

base pair (bp) is two complementary nucleotides (i.e., adenine, thymine, cytosine, or guanine) joined together to form the “rungs” on DNA's double-helix “ladder.” [Human genome (haploid) has ~3 billion bp; guppy, ~740 million bp.]

box plot summarizes statistical data in a visually friendly way with boxes showing 25th, 50th (median) and 75th percentiles. Vertical bars outside each box are for outliers (more extreme values). See Fig 1.

***F* (Inbreeding Coefficient)** quantifies the inbreeding level. It predicts the average homozygosity. The mating of full siblings produces progeny with an $F=0.25$, meaning that 25% of their DNA is homozygous.

gene- segment of DNA that determines a trait. A gene is made up of pair(s) of alleles.

heterozygosity means that the alleles within an allelic pair differ; in the homozygous condition, they are identical.

homozygosity means that the alleles within an allelic pair are identical; in the heterozygous condition, they differ. Inbreeding simultaneously increases homozygosity and decreases heterozygosity.

ID (inbreeding depression)- loss of fitness due to inbreeding

IBD (identical by descent) refers to segments of DNA that are identical (homozygous) because they were inherited from the same ancestor.

locus- the physical position of a gene on a chromosome

Mb- megabase; unit of DNA length equal to 1 million bases; 1 Mbp = 1 million base pairs

median is the 50th percentile (middle number in a group sorted from smallest to largest value). Statisticians use medians—instead of averages—when a data set is severely skewed by outliers (extreme values).

MHC (major histocompatibility complex) is a large group of vital proteins that help the immune system differentiate between healthy body cells and foreign invaders. In humans, MHC molecules are known as Human Leukocyte Antigens (HLA).

N (population size) often means all individuals in a population, but here I am referring to populations composed entirely of breeding pairs. For example, 10 reproducing males plus 10 reproducing females = 20N.

population bottleneck is a sharp drop in population size that causes inbreeding and a big loss in genetic variety.

purging removes bad alleles from a population by the death or infertility of individuals that carry those alleles. (Inbreeding generates homozygosity, increases the pairing of bad alleles, and thus, can accelerate purging.)

recombination is a natural process during meiosis whereby two cuts occur in a chromosome to generate a segment that swaps places with another chromosomal segment. Recombination shuffles existing maternal and paternal genes into new combinations, but it does not create new genes and alleles.

ROHs (runs of homozygosity) are segments of homologous chromosomes that are identical due to inbreeding. (Homologous chromosomes are paired chromosomes.)

SNPs (single nucleotide polymorphisms) represents a single switching of nucleotide bases at a specific place in an individual's DNA. Humans have millions of SNPs, whereby an individual's unique SNP pattern can be used for determining ancestry, personalized medicine, etc. Although SNPs are mutations, most have no impact on function or fitness.

Diana Walstad is the author of *Ecology of the Planted Aquarium*. For more information and other articles, check out: <http://dianawalstad.com/aquariums>