In our culture, the taboo against incest and inbreeding is highly pervasive. Most people have a sense that you shouldn’t ‘marry your cousin’ (and many states prohibit it!) or may know that the children of closely related individuals can suffer health and developmental consequences ranging from mild to severe. For centuries, agriculturists have known that inbred plants and animals tend to be less fit than their outbred counterparts. Even Charles Darwin, who married his first cousin Emma Wedgewood, often worried that his relatedness to his wife had negative effects on the health of his children, three of whom died at a young age [2].

But why is inbreeding so bad? What does parental relatedness have to do with the health and survival of an individual? As dedicated pet breeders you have a substantial interest in the quality, well-being, and reproductive capacity of your animals. Inbreeding represents a real potential threat to the health of your sugar gliders which means you have a responsibility to understand what can go wrong and how to protect your animals from harm.

Inbreeding and Inbreeding Depression

Inbreeding describes reproduction between individuals who are genetically similar. This usually means that parents are close relatives, although it is possible for individuals to have genes in common without sharing a recent common ancestor. Across a wide array of species inbreeding is associated with various deficits traits that relate to organisms’ fitness. In biology, the term fitness is used to describe an individual’s capacity to survive (viability) and reproduce (fecundity). On average, organisms with higher fitness are expected to produce more offspring over their lifespan and make a greater genetic contribution to the next generation.

The decreased health and survival that is observed in inbred offspring is known as inbreeding depression. Though not all species or populations are equally susceptible to inbreeding depression, in the wild, many species have innate tendencies, physiological mechanisms, and behavioral strategies that help them avoid mating with close genetic relatives. Inbreeding depression has been studied predominately in captive populations, like livestock, animals kept in zoos, or organisms studied in laboratories. However, inbreeding depression is also important to the study of conservation biology, the discipline that studies how to preserve diversity in nature and protect species from extinction. Inbreeding depression represents a threat to the survival of small endangered populations and therefore must be managed carefully if researchers want to save species from going extinct.
What Can Go Wrong?

Every species will respond differently to inbreeding: while some species have evolved to cope with it, others may experience severe effects. However, when inbreeding does have measurable consequences they are usually detrimental. Negative effects of inbreeding have been documented across a vast array of different types of wildlife, including plants, insects, snails, fish, birds, rodents, primates, and even humans. Inbreeding depression acts on traits that affect fitness characteristics including things like birth weight, growth rate, fertility, and ultimately survival. These effects can range from subtle deficiencies to dramatic developmental disorders or even lethal genetic conditions.

Some documented consequences of inbreeding depression include:
- reduced egg hatching rates in birds and insects [13, 17]
- low birth weight [1]
- low juvenile weight [9]
- reduced juvenile survival [6, 9, 12]
- increased susceptibility to disease [1, 3, 11]
- delayed onset of breeding [7]
- depressed sperm count in males [11]
- increased rate of miscarriages and stillbirths [2]
- shortened lifespan [9, 13]

Clearly, these are outcomes you want to avoid among your own animals. Inbreeding depression has consequences not only for the immediate health of individuals but for their well-being over their entire lifespan and the health and fertility of a population across generations.

Why Does it Happen? The Genetics Behind Inbreeding Depression

Most mammals have two copies of every gene in their body. Each copy is called an allele. The interaction of the two alleles of a gene produces an organism’s phenotype, the visible traits that an organism expresses. Every individual only has two alleles for each gene, but across an entire population there could be many more. Different combinations of alleles produce much of the physical variation we see across individuals.

One example of this in humans in blood type. Each person carries just two alleles that determine their blood type, but across the entire human population there are three blood type alleles: A, B, and O. An individual’s blood type is determined by the interaction of the two alleles they possess. A and B alleles are both dominant while the O allele is recessive:

<table>
<thead>
<tr>
<th>Allelic Combination (Genotype)</th>
<th>AA</th>
<th>AO</th>
<th>BB</th>
<th>BO</th>
<th>AB</th>
<th>OO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Type (Phenotype)</td>
<td>A</td>
<td>A</td>
<td>B</td>
<td>B</td>
<td>AB</td>
<td>O</td>
</tr>
</tbody>
</table>

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For almost every gene, one allele is inherited from a child’s mother and one allele is inherited from a child’s father. Mom and dad might both give you the same allele for a particular gene or they might each give you a different allele. When an individual has two copies of the same allele for a particular gene they are considered **homozygous** for that gene; if instead they have two different alleles they are called **heterozygous** for that gene. It is possible for a single individual to be homozygous for some traits in their genome but heterozygous at others.

When mom and dad are related they already share a high proportion of their genes. This means they probably have a lot of their alleles in common. Therefore, when they reproduce, they are limited in the array of different alleles they can pass on to their offspring. Inbred offspring are more likely to inherit matching alleles at any particular trait, leading to an increase in homozygosity across the genome.

Why does that matter? Remember that organisms express a gene based on the interaction between their own two alleles, and different combinations of alleles result in different outcomes. The interactions between alleles in the most simple patterns of inheritance can be characterized as **dominant** or **recessive**. A dominant allele is always expressed when an individual carries it in their genome, even if they only possess one copy. If an organism is homozygous for the dominant allele or heterozygous and carries one copy of the dominant allele they will still express the dominant trait. In contrast, a recessive allele only appears in an individual’s phenotype if they carry two copies, that is, if they are homozygous for the recessive allele.

In many systems heterozygosity correlates positively with overall fitness such that individuals who are heterozygous for more genes throughout their genome have a higher lifetime fitness [4]. Homozygosity itself is not inherently good or bad. The problem is that many mutations that disrupt an organism’s normal functioning are recessive alleles that can be successfully masked in heterozygous individuals but become damaging when they are expressed homozygously. They persist in a population because they are carried by heterozygous individuals who have the mutation in their genotype but don’t express it in their phenotype. This is true of many serious diseases in humans, including cystic fibrosis, sickle cell anemia, and Tay Sachs disease.

Those three disorders are extreme cases in which homozygosity at a single gene has devastating or fatal effects on the body. More often, complex functions like disease susceptibility, growth rate, or reproduction are influenced by multiple genes. As inbred individuals become increasingly homozygous across their genome over generations of inbreeding, they are more likely to accumulate recessive alleles homozygously and express deleterious mutations in their phenotype. Each individual mutation when considered independently may have only a small effect on an organism’s fitness, but when mutations accumulate at multiple sites across the genome the total effects can be severe.
When Heterozygosity Really Matters

For some traits, heterozygosity itself is very important for individual fitness. One example is the major histocompatibility complex (MHC), which is one of the most important components of the immune system in mammals. The MHC is made up of multiple genes which code for proteins that help the body recognize pathogens and coordinate an immune response. Individuals who are heterozygous at MHC genes can produce a greater variety of proteins that can detect a greater variety of pathogens, meaning that their bodies are inherently more prepared to fight off sickness and disease. MHC represents a clear case where you can imagine a direct relationship between homozygosity and decreased fitness resulting from an increased susceptibility to disease.

A Real World Case Study: Scandinavian Grey Wolves

Grey wolves had not been seen in Sweden for decades until a new breeding pack was discovered unexpectedly in 1983 [Vila]. DNA analysis suggests that this new pack was descended from a single pair of wolves who had migrated from a neighboring population in a nearby country, most likely Russia. Throughout the 1980s, the new Swedish population remained very small and very isolated. Inbreeding likely became common after the original founder wolves died and siblings began mating with each other [4]. Since its reappearance the Swedish population has closely monitored for conservation purposes, so researchers know detailed information about the relatedness of individuals, as well as demographic, genotypic, and fitness characteristics.

Studies on the captive zoo population of grey wolves showed that inbreeding depression could have devastating effects in these animals. The population maintained in Scandinavian zoos is descended from two pairs of wolves who were brought in from wild in the early 1950s and 60s. Both pairs were full siblings (brother and sister) and the siblings were mated to initiate a captive breeding program.

An analysis by researchers from the University of Stockholm observed some striking patterns in the fitness of inbred individuals. Researchers documented a dramatic increase in homozygosity and a dramatic loss of genetic variation in the population over time with real consequences for individual fitness. As expected, inbred wolves showed decreased heterozygosity at many points in their genomes [5]. Wolves who were more inbred were smaller, lived shorter lives on average, and had lower rates of reproduction [9]. Inbred wolves also developed a form of hereditary blindness. Wolves with the condition were born being able to see but lost their vision entirely within their first six months. The condition shows a complicated pattern of inheritance, but only appears within the most inbred families. These blind wolves were more likely to die before they reached reproductive age than sighted wolves [9].
Studies of the wild Swedish population throughout the 1980s, 90s, and early 2000s suggest that these wolves were experiencing high levels of inbreeding and suffering some of these same consequences. Analyses of genetic similarity and homozygosity showed that wolves in the wild had similar levels of genetic diversity as the captive zoo population [5]. Compared to the neighboring population in Russia and the historical Swedish population that was sampled through taxidermied museum specimens, the current population in Sweden has very low levels of genetic diversity [16]. Importantly, younger wolves were more homozygous than older wolves, indicating that homozygosity has increased since the population was founded [5]. In 2005 a group of researchers determined the degree of inbreeding for all the wolves in the population and found that many individuals shared as many alleles as if they were full siblings, even when they were descended from different sets of parents [10]. As seen in studies of captive populations, wolves who were more inbred had lower reproductive success than outbred individuals; pups born to inbred females have lower survival rates than those born to more outbred wolves [10].

Then, in 1991, a male wolf migrated from another population to join the Swedish group. The impact of his contribution of new genetic material were rapid and profound. The population suddenly expanded exponentially from a single breeding pack to over 100 individuals as fertility rates and pup survival increased [8]. Genes from the new migrant spread rapidly throughout the population and account for a significant increase in heterozygosity and restoration of genetic diversity since his arrival [16]. This single male wolf has a disproportionately large influence on the genetic make-up of the present population and the majority of births that have occurred since his arrival can trace their ancestry back to his genetics [8].

The Scandinavian grey wolf population is just one group that seems to have rapidly rebounded following the contribution of new genetic diversity to the reproductive pool. The increased success of populations following the arrival of diverse individuals is known as genetic rescue, and this is not the only compelling example of this phenomenon. It is important to remember that such dramatic improvements do not erase the damaging effects caused by generations of inbreeding. But the rapid rebound of this population following the contribution of a single individual speaks to how devastating inbreeding depression can be for the health of a group of animals.

How You Can Protect Your Animals

In the wild, juvenile sugar gliders are kicked out of their natal territories before they become capable of reproducing and disperse away from their birth sites to found or join new communities of gliders [14, 15]. This helps prevent gliders from reproducing with other individuals in their own family. In captivity, animals don’t have this option, which means breeders need to be responsible about managing where they house young animals once they reach reproductive age, around 8-12 months [15]. Current information provided by breeders’ experience within the United States indicates that reproductive ages are much closer to 3-4 months.
Maintain careful records of the individuals you use in your breeding programs to make sure you are always aware of the degree of relatedness and recent common ancestors between any two reproductive adults. Kinship charts such as this one http://glidernursery.webs.com/kinshipchart.htm are a helpful resource when thinking about arranging pairings between gliders. Keeping detailed pedigrees has the added benefit of allowing you to monitor the inheritance patterns of desirable traits like coat coloration, which can help you think about breeding for specific traits in the future. There is no way to determine how much inbreeding is “safe” versus “too much” so it is important to avoid inbreeding as much as possible. If your breeding population is small consider sharing animals with other breeders in your area to maintain genetic diversity in your own population.
References


Glossary

**Allele** - one of two or more versions of a single gene

**Conservation biology** - the biological discipline that studies works to preserve biodiversity in nature and protect endangered species and populations

**Dominant** - when one allele can mask the expression of another. A dominant allele is expressed even if there is only one copy.

**Fecundity** - fertility, the ability to reproduce; the reproductive rate of an individual or population

**Fitness** - the capacity of an individual or population to survive and reproduce. An organism’s fitness depends on both viability and fecundity. Individuals with higher fitness are expected to make a greater contribution to the next generation. Fitness is inscribed in an organism’s genotype and manifested through its phenotype.

**Gene** - a stretch of DNA that contains the information for a specific trait

**Genetic rescue** - a phenomenon in which inbreeding depression in a struggling population can be partially reversed by the introduction of genetically diverse reproductive adults from other populations

**Genotype** - an organism’s genetic sequence describing which alleles the individual possesses for a given trait

**Heterozygous** - when an organism possesses 2 different alleles for a given gene

**Homozygous** - when an organism possesses 2 copies of the same allele for a given gene

**Inbreeding** - the process of reproduction between individuals who are genetically similar. This often means that parents are close relatives; it is possible for individuals to have genes in common without sharing a recent common ancestor.

**Inbreeding depression** - the biological term used to describe reduced survival and fertility of the offspring of related individuals

**Major histocompatibility complex (MHC)** - an important component of the vertebrate immune system that allows the body to recognize and respond to invading pathogens. Heterozygosity at the multiple genes that encode MHC correlates closely with individual fitness and allelic diversity for MHC genes across a population correlates with population health and persistence.

**Mutation** - an event that changes the sequence of DNA inside an organism’s cells. Mutations might or might not affect an individual’s phenotype. If there is an effect, a mutation could improve an organism’s fitness but is more likely to disrupt normal functioning and cause some degree of damage.

**Outbreeding** - the process of reproduction between individuals who are genetically different and not closely related

**Phenotype** - the physical traits an organism expresses based on the interactions of its inherited genes with the environment

**Recessive** - describes an allele that must be present in two copies in order to be expressed in an organism’s phenotype

**Trait** - a genetically determined characteristic

**Viability** - the ability to live and survive; capable of normal growth and development