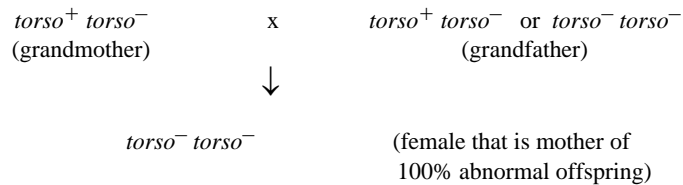


S1. Our understanding of maternal effect genes has been greatly aided by their identification in experimental organisms such as *Drosophila melanogaster* and *Caenorhabditis elegans*. In experimental organisms with a short generation time, geneticists have successfully searched for mutant alleles that prevent the normal process of embryonic development. In many cases, the offspring will die at early embryonic or larval stages. These are called **maternal effect lethal alleles**. How would a researcher identify a mutation that produced a recessive, maternal effect lethal allele?

Answer: A maternal effect lethal allele can be identified when a phenotypically normal mother produces only offspring with gross developmental abnormalities. For example, let's call the normal allele *N* and the maternal effect lethal allele *n*. A parental cross between two flies that are heterozygous for a maternal effect lethal allele would produce 1/4 of the offspring with a homozygous genotype, *nn*. These flies are viable because of the maternal effect. Their mother would be *Nn* and provide the *n* oocyte with a sufficient amount of *N* gene product so that the *nn* flies would develop properly. However, homozygous *nn* females cannot provide their oocytes with any normal gene product. Therefore, all their offspring are abnormal and die during early stages.

S2. A maternal effect gene in *Drosophila*, called *torso*, is found as a recessive allele that prevents the correct development of anterior- and posterior-most structures. A wild-type male is crossed to a female of unknown genotype. This mating produces 100% larva that are missing their anterior- and posterior-most structures and therefore die during early development. What is the genotype and phenotype of the female fly in this cross? What are the genotypes and phenotypes of the female fly's parents?

Answer: Since this cross produces 100% abnormal offspring, the female fly must be homozygous for the abnormal *torso* allele. Even so, the female fly must be phenotypically normal in order to reproduce. This female fly would have had a mother that was heterozygous for a normal and abnormal *torso* allele and a father that was either heterozygous or homozygous for the abnormal *torso* allele.



This female fly is phenotypically normal because its mother was heterozygous and provided the gene products of the *torso*⁺ allele from the nurse cells. However, this homozygous female will produce only abnormal offspring, because it cannot provide them the normal *torso*⁺ gene products.

S3. An individual with Angelman syndrome produced an offspring with Prader-Willi syndrome. Why does this occur? What are the sexes of the parent with Angelman syndrome and the offspring with Prader-Willi syndrome?

Answer: These two different syndromes are most commonly caused by a small deletion in chromosome 15. In addition, genomic imprinting plays a role, since genes in this deleted region are differentially imprinted depending on sex. If this deletion is inherited from the paternal parent, the offspring develops Prader-Willi syndrome. Therefore, in this problem, the individual with Angelman syndrome must have been a male, since he produced a child with Prader-Willi syndrome. The child could be either a male or female.

S4. In yeast, a haploid petite mutant also carries a mutant gene that requires the amino acid histidine for growth. The *petite his*⁻ strain is crossed to a wild-type strain to yield the following tetrad:

2 cells: *petite his*⁻
2 cells: *petite his*⁺

Explain the inheritance of the *petite* and *his*⁻ mutations.

Answer: The *his*⁻ and *his*⁺ alleles are segregating in a 2:2 ratio. This indicates a nuclear pattern of inheritance. By comparison, all four cells in this tetrad have a *petite* phenotype. This is a suppressive petite that arises from a mitochondrial mutation.

S5. Let's suppose that you are a horticulturist who has recently identified an interesting plant with variegated leaves. How would you determine if this trait is nuclear or cytoplasmically inherited?

Answer: Make reciprocal crosses involving normal and variegated strains. In many species, plastid genomes are inherited maternally, although this is not always the case. In addition, a significant percentage of paternal leakage may occur. Nevertheless, when reciprocal crosses yield different outcomes, an organellar mode of inheritance is possibly at work.

S6. A phenotype that is similar to a yeast suppressive petite was also identified in the mold *Neurospora crassa*. Mary and Hershel Mitchell identified a slow-growing mutant that they called *poky*. Unlike yeast, which are isogamous (i.e., produce one type of gamete), *Neurospora* is sexually dimorphic and produces male and female reproductive structures. The “female” reproductive structure is the ascogonium and the “male” reproductive structure is the antherium. When a *poky* strain of *Neurospora* was crossed to a wild-type strain, the results were different between reciprocal crosses. If a *poky* mutant was the female parent, all the spores exhibited the *poky* phenotype. By comparison, if the wild-type strain was the female parent, all the spores were wild type. Explain these results.

Answer: These genetic studies indicate that the *poky* mutation is maternally inherited. This is because the cytoplasm of the female reproductive cells provides the offspring with their mitochondria. Besides these genetic studies, the Mitchells and their collaborators showed that *poky* mutants are defective in certain cytochromes, which are iron-containing proteins that are known to be located in the mitochondria.