Codominance

Codominance is a type of inheritance wherein both the alleles of a gene are fully expressed in heterozygote. Consequently, the phenotype of the offspring is a combination of the alleles. Thus, the trait is neither dominant nor recessive.

AB blood type: A person inheriting the alleles I^A and I^B will have a type AB blood because I^A and I^B are codominant and therefore express together.

MN blood types in human

The MN locus codes for one of the types of antigens on red blood cells. Foreign MN antigens do not elicit a strong immunological reaction, and therefore the MN blood types are not routinely considered in blood transfusions. At the MN locus, there are two alleles:

the L^M allele, which codes for the M antigen; and

the L^N allele, which codes for the N antigen.

Homozygotes with genotype L^ML^M express the M antigen on their red blood cells and have the M blood type.

Homozygotes with genotype L^NL^N express the N antigen and have the N blood type.

Heterozygotes with genotype L^ML^N express both the M and the N antigens and the MN blood type.

Cystic Fibrosis in Human

The gene responsible for cystic fibrosis resides on the long arm of chromosome 7. It encodes a protein termed *cystic fibrosis transmembrane conductance regulator*, abbreviated CFTR. CFTR acts as a gate in the cell membrane and regulates the movement of chloride ions into and out of the cell. Patients with cystic fibrosis have a mutated, dysfunctional form of CFTR that causes the

channel to stay closed, and so chloride ions build up in the cell. This buildup causes the formation of thick mucus and produces the symptoms of the disease.

Most people have two copies of the normal allele for CFTR. They produce only functional CFTR protein. Those with cystic fibrosis possess two copies of the mutated CFTR allele, and produce only the defective CFTR protein. Heterozygotes produce both functional and defective CFTR protein. Thus, at the molecular level, the alleles for normal and defective CFTR are codominant, because both alleles are expressed in the heterozygote. However, because one normal allele produces enough functional CFTR protein to allow normal chloride transport, the heterozygote exhibits no adverse effects, and the mutated CFTR allele appears to be recessive at the physiological level.