

## Quadrant II – Transcript and Related Materials

**Programme:** Bachelor of Science (Third Year)

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**Unit:** 03

**Module Name:** Kappa particles in Paramecium and Mitochondria in yeast

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### Notes:

#### Kappa Particles in Paramecium:

One of the most striking and spectacular example of cytoplasmic inheritance due to symbiont bacteria is noted in the most common ciliate protozoan *Paramecium aurelia*. In 1943, T. M. Sonneborn reported that some strains of *P. aurelia* contain kappa particles and are known as **killer strain**.

They are bounded by a membrane and contain a little bit of cytoplasm with DNA. The strains of *Paramecium* in which the kappa particles are absent are called **sensitive strain**. The sensitive strains are killed by the **killer strain**.

The destruction of sensitive strain occurs through secretion of a toxic substance called **paramecin**. This toxic substance is believed to breakdown the food vacuole membrane of the sensitive strain. Paramecin is diffusible in the liquid medium.

When killers are allowed to remain in a medium for a time, they are not killed. It means that paramecin has no effect on killers. Paramecin is associated with a particular kind of kappa that occurs in about 20 percent of a kappa population.

These kappa bacteria possess a **refractile protein** containing 'R' body and are called **brights** because they are infected with a virus that controls the synthesis of a viral protein as well as R protein body in kappa bacterium.

The virus may act as the toxin in the killing response and R body facilitates the penetration of the toxin. The non-bright kappa bacteria may also contain virus but the virus may be in provirus state in them.

The killer character of *Paramecium* has a nuclear as well as cytoplasmic basis. The existence of kappa particles is determined by presence of nuclear dominant gene K. Kappa particles, like other bacteria, multiply through fission.

But their multiplication in the cytoplasm of *Paramecium* depends on the presence of a dominant nuclear gene K which helps to make an environment necessary for the bacteria to reproduce.

When killer strain of *Paramecium* conjugates with sensitive strain under appropriate condition for brief period and no cytoplasm exchange occurs, two kinds of clones result- one from the original killer cell which contains allele K (Kk) and kappa particles and the other from the original sensitive cell which carries the allele k (kk) and lacks kappa particles.

It indicates that homozygous (either KK or kk) strains become heterozygous following an exchange of K and k genes without cytoplasmic exchange.

Following autogamy (a process of self- fertilisation within one undivided cell resulting in homozygosity), half the progeny (50%) are sensitive *Paramecia*. But all progenies of sensitives following autogamy will be sensitive's.

In this conjugation, following autogamy of killers, 50% progeny will receive Kk genotype with cytoplasmic kappa particles other 50% progeny will receive kk genotype with cytoplasmic kappa particles. But it will be sensitive, because kappa cannot reproduce in the cells unless a K allele is present in the nucleus and, as a consequence the kappa is eliminated.

On the other hand, in this conjugation the product of autogamy of sensitive strain obtained after conjugation are all sensitive. All through, 50% progeny of autogamy have KK genotype without cytoplasmic kappa particles because no

cytoplasm has been transferred in this conjugation. Remaining 50% progeny of autogamy of sensitive's have kk genotype and no cytoplasmic kappa particles.

Under some conditions of conjugation persists much longer; a long connection is established between conjugants (killer and sensitive). In this conjugation, cytoplasm as well as nuclear genes are exchanged. As a consequence both ex-conjugants will receive the genotype Kk and the cytoplasm with kappa particles.

Therefore, conjugation for longer period with cytoplasmic exchange will produce all killer strains. Autogamy of both ex-conjugants produces homozygotes KK (killer) and kk (sensitive) cell in the 1 : 1 ratios, respectively, as expected from Mendelian segregation.

Therefore, conjugation for shorter period without cytoplasmic exchange does not follow the Mendelian pattern of inheritance. Hence it confirms the cytoplasmic basis of inheritance of killer trait.

## **MITOCHONDRIA IN YEAST / PETITE MUTATIONS IN YEAST**

Under aerobic conditions, yeast grows with distinctive colony morphology. Under anaerobic conditions, the colonies are smaller, and the structures of the mitochondria are reduced. Occasionally, when growing aerobically, small, anaerobic like colonies appear; but in these colonies, the mitochondria appear perfectly normal. These colonies are caused by petite mutations. All petites represent failures of mitochondrial function, they usually lack one or another cytochrome.

**When petites are crossed with the wild-type, three modes of inheritance emerge.**

- a. The segregational petite, caused by mutation of a chromosomal gene, exhibits Mendelian inheritance.
- b. The neutral petite is lost immediately upon crossing to the wild-type.
- c. The suppressive petite shows variability in expression from one strain to the next but is able to convert the wild-type mitochondria to the petite form.

In the **first** type called **segregational petites** when an individual is crossed with a normal strain, a 1 : 1 ratio of normal: petite results after segregation. This

suggests Mendelian inheritance and the petite strain has originated due to a mutation in nuclear genes.

**Neutral petites** seem to have mitochondria that entirely lack DNA. When neutral petites are crossed with the wild-type to form diploid cells, the normal mitochondria dominate. During meiosis, virtually every spore receives large numbers of normal mitochondria; the progeny are, therefore, all normal.

**Suppressive petites** could exert their influence over normal mitochondria in one of two ways. Suppressive petite mutants are found to have mutant DNA in their mitochondria. The suppressive mitochondria might simply out-compete the normal mitochondria and take over; they might simply reproduce faster within a cell. The suppressive petites when crossed to normal cells of yeast show the petite trait in the progeny but in non Mendelian ratios. The mutant mitochondria replicate and transmit the mutant phenotype to the progeny cells.