Model Questions & Answers

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Q.1. Mention the functions of Topoisomerase I and II?

Ans: DNA topoisomerase is a nuclease that breaks a phosphodiester bond in a DNA strand. This reaction is reversible, and the phosphodiester bond reforms as the enzyme leaves.

Type-I topoisomerase, produces a transient single strand break; this break in the phosphodiester backbone allows the two sections of DNA helix on either side of the nick to rotate freely relative to each other, using the phosphodiester bond in the strand opposite the nick as a swivel point. Type-I topoisomerases are subdivided into two sub classes: Type-IA and Type-IB topoisomerases. Type-IA topoisomerases form a covalent intermediate with the 5' end of DNA, while the IB topoisomerases form a covalent intermediate with the 3' end of DNA.

A second type of DNA topoisomerase is type II topoisomerase, makes a transient double-strand break in the helix and forms a covalent linkage to both strands of the DNA helix at the same time. It is also split into two sub classes: Type-IIA and Type IIB topoisomerases. The first type-II topoisomerase (Topo-II) to be described was isolated from *E. coli* and named DNA gyrase.

Bothe type I and Type I topoisomerases change the linking number of DNA. Type-IA topoisomerases change the linking number by one and type-IB topoisomerase change the linking number by any integer, while type IA and type IB topoisomerases change the linking number by two. In prokaryotes Topo-II changes the positive suoercoil into a negative supercoil or of increasing the number of negative supercoils by 2. These enzymes are activated by sites on chromosomes where two double helices cross over each other. Once a topoisomerase II molecule binds to such a crossing site, the protein uses ATP hydrolysis to perform the following set of reactions efficiently:

i) It breaks one double helix reversibly to create a DNA gate.
ii) It causes the second, nearby double helix to pass through this break; and
iii) It then reseals the break and dissociates from the DNA.

All type II topoisomerases also catalyze *catenation* and *decatenation*, that is the linking and unlinking, of two different duplexes. Nalidixic acid, ciprofloxacin and novobiocin binds to *E. coli* DNA gyrase and inhibit its action.

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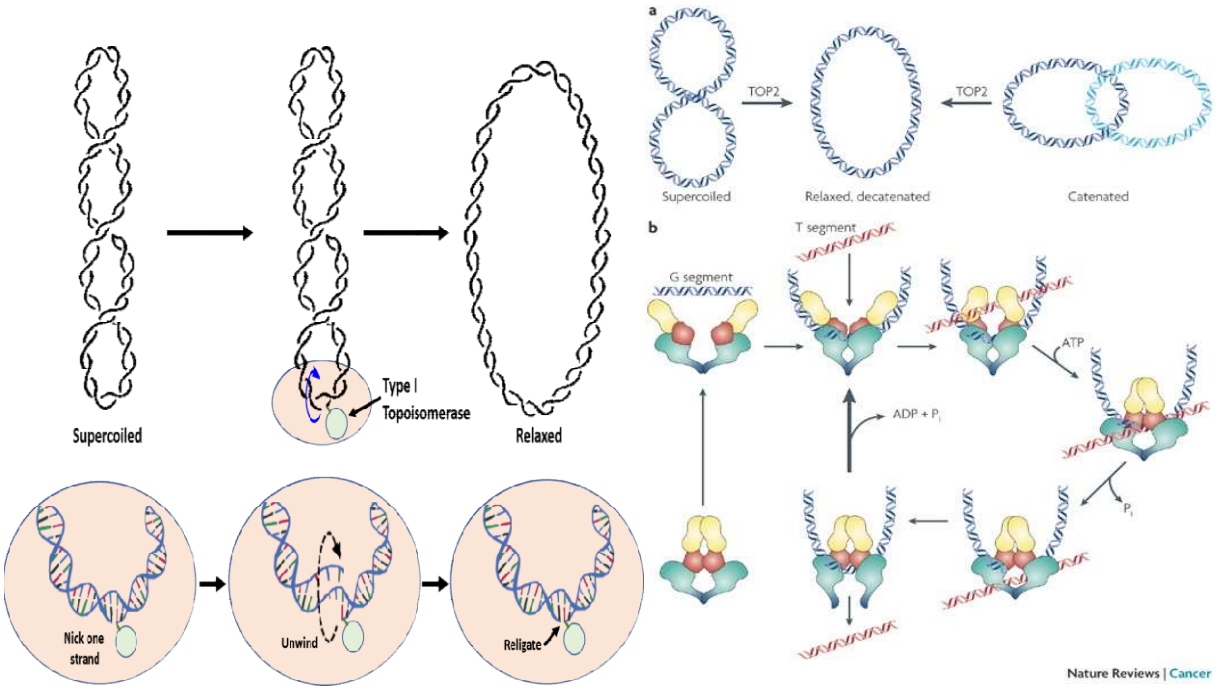

Fig: Functions of topoisomerase I and II

Q.2. Describe the molecular structure of troponin?

Ans: Troponin complex is a component of skeletal and cardiac muscle thin filament and plays a central role in the regulation of skeletal and cardiac muscle contraction, connecting changes in intracellular Ca^{+2} concentration with generation of contraction. It is a globular protein, consists of three subunits- troponin T (Tn T), troponin C (Tn C) and troponin I (Tn I) and is located on polymerized actin together with tropomyosin, forming muscle thin filament.

Tn T is of 259 amino acid residues, and has binding sites for tropomyosin. Tn C has 159 amino acids and bear four Ca-binding sites, control position of tropomyosin on F-actin.

Tn I is a 23KD peptide with 179 amino acids, possessing binding sites for F-actin and stop binding of actin with myosin.

The structure of the troponin complex suggests that the Ca^{+2}-binding of the regulatory Tn C site displace the carboxyl-terminal portion of Tn I from actin/ tropomyosin. Thereby altering mobility and/or flexibility of the troponin/tropomyosin strand on the actin filament.

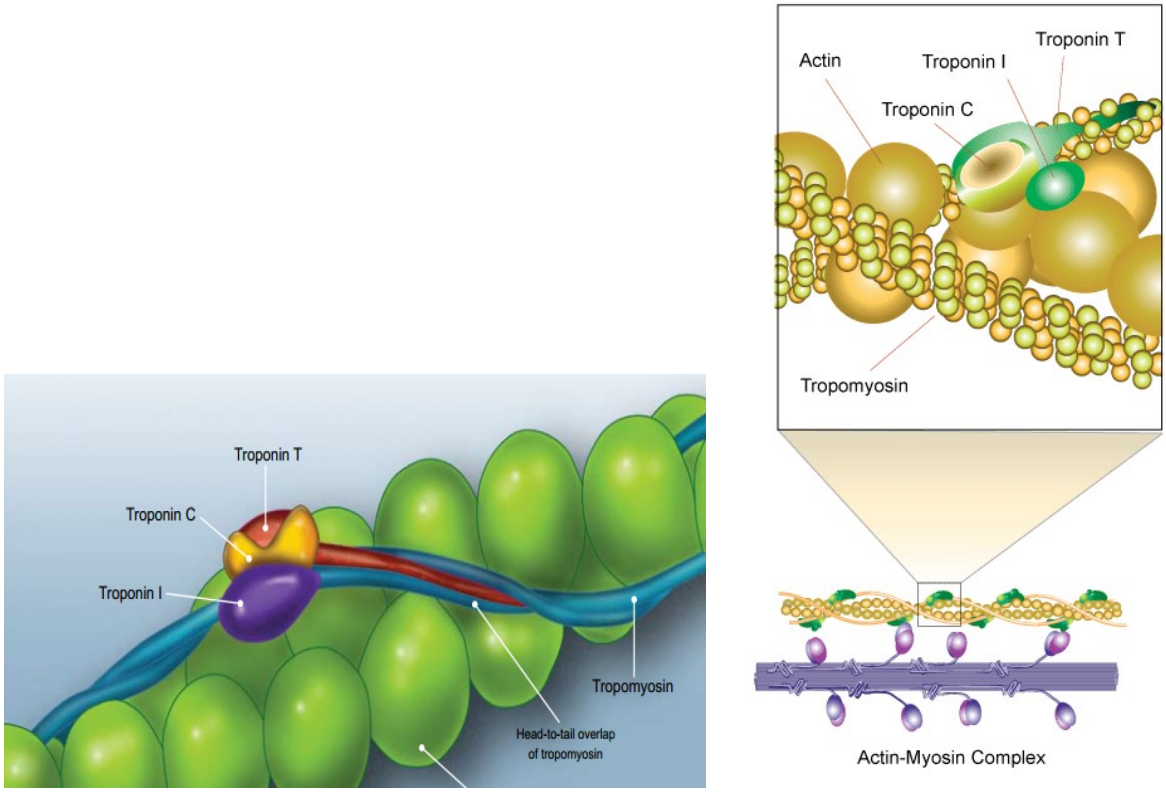

Fig: Structure of Troponin

Q.3. What is Hardy-Weinberg law? Mention the conditions affecting the Hardy-Weinberg Equilibrium ?

Ans: In 1908, G. H. Hardy, a British mathematician, and W. Weinberg a German physician, independently discovered a rule that relates allelic and genotypic frequencies in a population of diploid, sexually reproducing individuals.

The rule has three aspects:

i) The allelic frequencies at an autosomal locus in a population will not change from one generation to the next (allelic-frequency equilibrium).

ii) The genotypic frequencies of the population are determined in a predictable way by the allelic frequencies (genotypic- frequency equilibrium).

iii) The equilibrium is neutral. That is, if it is perturbed, it will be reestablished within one generation of random mating at the new allelic frequencies (if all the other requirements are maintained).

Definition: The principle of genetic equilibrium states that relative frequencies of various kinds of genes in the gene pool of a large and randomly mating sexual, panmictic population tend to remain constant from generation to generation in the absence of evolutionary forces, such as mutation, selection and gene flow.

This is called **Hardy-Weinberg principle or Hardy-Weinberg law of equilibrium.**

The principle based on certain assumptions. The following major assumptions are necessary for the Hardy-Weinberg equilibrium to hold.

a) Extremely large population size: The equilibrium in gene and genotype frequencies occurs only in large sized populations. In small populations, there will be significant sampling errors and random fluctuations in the gene frequency by chance, the so called **genetic drift or random drift.**

b) **Random mating** i.e. each individual of the population has equal opportunity of mating with any other individual of that population. A population in which the probability that any given male and female mate is equal for all individuals is termed **panmictic population**. If members of a population choose individuals of a particular phenotype as mates more or less often than at random, the population is engaged in **assortative mating**. The most important form of assortative mating is inbreeding. It is mating between closely related individuals. Outbreeding is the mating of genetically unrelated individuals. The chance of inbreeding is high in small populations. The extent of inbreeding occurring in a population is measured by inbreeding coefficient. The inbreeding coefficient (expressed as F) is the probability that two alleles of a given gene in an individual are identical by descent. In most species, including all mammals, inbreeding depression, which can result from increased homozygocity for harmful alleles. The number of harmful alleles present in the gene pool of a population is referred to as the **genetic load**.

c) Allele frequencies are the same in males and females.

d) **Biparental mode of reproduction**: Hardy-Weinberg principle is applicable only for biparent sexually reproducing species. Unisexual or asexually reproducing populations do not follow Hardy-Weinberg law.

e) **No natural selection** i.e. all genotypes are equal in viability and fertility. No individual will have a reproductive advantage over another individual because of its genotype.

f) **No mutation**; if a particular locus shows a high mutation rate then there will be a steady increase in the proportion of mutant alleles in a population.

g) **No migration**; allelic and genotypic frequencies may change through the loss or addition of alleles through migration (immigration or emigration) of individuals from or into a population.

In summary, the Hardy-Weinberg equilibrium holds for an infinitely large, randomly mating population in which mutation, migration, and natural selection do not occur.