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CORE JOBS · INTERVIEW QUESTIONS

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Genetics, Cell and Molecular Biology Interview Questions

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Mendelian genetics, gene interaction and linkage mapping

1. What does the law of independent assortment say, and when does it fail?

Alleles of different genes separate independently into gametes, so a dihybrid AaBb makes four gamete types in equal numbers. It holds for genes on different chromosomes or far apart on one chromosome. For linked genes, parental gamete types outnumber recombinant types, so the law fails.

2. What is the difference between incomplete dominance and codominance?

In incomplete dominance the heterozygote shows an intermediate phenotype, such as pink flowers from red and white parents, because one functional allele gives too little product. In codominance both alleles are fully expressed side by side, as in the AB blood group. Both give a 1:2:1 phenotype ratio in the F2.

3. Why does recessive epistasis turn the dihybrid F2 ratio of 9:3:3:1 into 9:3:4?

The homozygous recessive genotype at one gene masks the effect of the other gene. Plants of genotype aaB_ and aabb then look the same, so the 3 and the 1 classes merge into one class of 4. The genes still assort independently and the 16 combinations are unchanged.

4. A testcross of a dihybrid gives 1000 progeny, of which 140 are recombinant. What is the map distance between the two genes?

The recombination frequency is $140/1000 = 0.14$, or 14%. One per cent recombination equals one map unit (1 cM), so the genes are 14 map units apart. Over a short distance like this, double crossovers are rare, so the estimate holds.

WORKING

1. Recombination frequency $= \dfrac{\text{recombinants}}{\text{total progeny}}$
2. $= \dfrac{140}{1000} = 0.14$
3. 1% recombination equals 1 map unit (1 cM), so the distance is $0.14 \times 100 = 14$ map units

Microbial genetics, gene transfer and transposable elements

5. How do transformation, conjugation and transduction differ as ways of moving DNA between bacteria?

Transformation is uptake of free DNA from the surroundings by a competent cell. Conjugation passes DNA, usually a plasmid, from donor to recipient through direct cell contact and a pilus. Transduction carries bacterial DNA inside a phage particle that infects the recipient. Only conjugation needs cell contact, and only transduction needs a phage.

6. Why does the recipient in an Hfr by F minus cross usually stay F minus?

In an Hfr cell the F factor is integrated into the chromosome, and transfer starts inside it. The genes needed to make the cell F positive lie at the far end, so they enter last. The transfer of the whole chromosome takes about 100 minutes, and the mating pair usually breaks apart before that.

7. In an interrupted mating, gene A enters the recipient at 8 min and gene B at 20 min. Assume the whole 4600 kb chromosome transfers in 100 min at a constant rate. How far apart are the two

genes?

The chromosome transfers at $4600/100 = 46$ kb per minute. Gene B enters $20 - 8 = 12$ minutes after gene A, so the genes are about $12 \times 46 = 552$ kb apart, if the transfer rate stays constant.

WORKING

1. Transfer rate $= 4600\ \text{kb}/100\ \text{min} = 46\ \text{kb/min}$
2. Time between entries $= 20 - 8 = 12$ min
3. Distance $= 12 \times 46 = 552$ kb

Population genetics and evolution

8. What does genetic equilibrium in a population state, and what conditions does it need?

Allele frequencies stay constant from generation to generation, and genotype frequencies settle at p^2, $2pq$ and q^2 after one round of random mating. This needs a large population, random mating, and no selection, mutation or migration. It is the baseline that shows when evolution is acting.

9. An autosomal recessive disorder affects 1 in 10000 people in a population at genetic equilibrium. What percentage of people are carriers?

At equilibrium the affected frequency is $q^2 = 1/10000$, so $q = 0.01$ and $p = 0.99$. Carriers are heterozygotes, with frequency $2pq = 2 \times 0.99 \times 0.01 = 0.0198$, so about 1.98% of people, roughly 1 in 50, are carriers.

WORKING

1. Affected people $q^2 = 1/10000 = 10^{-4}$, so $q = 0.01$
2. $p = 1 - q = 0.99$
3. Carriers $2pq = 2 \times 0.99 \times 0.01 = 0.0198$
4. As a percentage, $0.0198 \times 100 = 1.98\%$

10. What is the difference between aneuploidy and polyploidy?

Aneuploidy is the gain or loss of single chromosomes, such as 2n+1 or 2n-1, usually from nondisjunction. It upsets gene dosage and is often lethal in animals. Polyploidy is the gain of whole chromosome sets, such as 3n or 4n. Gene dosage stays balanced, so plants tolerate it far better.

11. Why are female placental mammals a mosaic for X-linked genes?

For dosage compensation, one X in each female cell is silenced early in development. A non-coding RNA coats that X, and methylation and histone changes make it heterochromatin. The choice is random per cell and passed to all daughter cells. Patches of cells therefore express different X chromosomes, as in the coat of a tortoiseshell cat.

Cell structure and the cell cycle

12. What are the main checkpoints of the eukaryotic cell cycle and what does each one check?

The G1/S checkpoint checks cell size, nutrients and DNA damage before replication starts. The G2/M checkpoint checks that DNA replication is complete and undamaged before mitosis. The spindle assembly checkpoint checks that every chromosome's kinetochores are attached to the spindle before anaphase. The

first two act by controlling cyclin-CDK activity; the spindle checkpoint blocks the APC/C, the enzyme complex that triggers anaphase.

13. How do mitosis and meiosis differ in their outcome?

Mitosis is one division that makes two daughter cells genetically identical to the parent, with the same chromosome number, for growth and repair. Meiosis is two divisions after one round of DNA replication and makes four cells with half the chromosome number, haploid cells from a diploid parent. Crossing over and independent assortment in meiosis I make those cells genetically different from each other.

14. A culture grows exponentially from 1×10^5 cells to 1.6×10^6 cells in 24 h. What is the doubling time?

The cell number rises 16-fold ($1.6 \times 10^6 / 10^5 = 16$), which is 4 doublings ($\log_2 16 = 4$). Four doublings in 24 h give a doubling time of $24/4 = 6$ h, assuming growth stays exponential at a constant rate.

WORKING

1. Fold increase $= 1.6 \times 10^6 / 10^5 = 16$
2. Number of doublings $n = \log_2 16 = 4$
3. Doubling time $= 24\ \text{h} / 4 = 6$ h

Cell communication and signal transduction

15. How does a G protein coupled receptor pass a signal into the cell?

Ligand binding changes the receptor so that it makes a heterotrimeric G protein swap GDP for GTP. The alpha subunit then separates from the beta-gamma pair and activates an effector, such as adenylyl cyclase, which makes cAMP, or phospholipase C, which makes IP3 and DAG. The alpha subunit's own GTPase activity switches it off.

16. How do intracellular nuclear receptors differ from cell-surface receptors?

Nuclear receptors bind small lipid-soluble ligands, such as steroid hormones, that cross the membrane. The ligand-receptor complex acts as a transcription factor, so the response takes hours. Cell-surface receptors bind water-soluble ligands that cannot enter the cell, and signal through second messengers and kinase cascades within seconds to minutes.

17. A receptor has a dissociation constant of 10 nM for its ligand. What percentage of receptors is occupied when the free ligand concentration is 40 nM?

At equilibrium, fractional occupancy is $[L]/(K_d + [L]) = 40/(10 + 40) = 0.8$, so 80% of receptors are occupied. This assumes one class of identical, independent binding sites and a free ligand concentration that stays at 40 nM.

WORKING

1. Fractional occupancy $= \dfrac{[L]}{K_d + [L]}$
2. $= \dfrac{40}{10 + 40}$
3. $= 0.8$
4. As a percentage, $0.8 \times 100 = 80\%$

Genes and chromosomes, mutation and DNA repair

18. What is the difference between base excision repair and nucleotide excision repair?

Base excision repair fixes small damage to a single base, such as uracil or an oxidised base. A glycosylase removes the base, and then an endonuclease, polymerase and ligase finish the job. Nucleotide excision repair fixes bulky lesions that distort the helix, such as UV thymine dimers, by cutting out a short stretch of nucleotides around them.

19. Why is a single base insertion usually more damaging than a single base substitution?

A substitution changes at most one codon, so it is silent, missense or nonsense. An insertion of one base shifts the reading frame from that point on. Every later codon is read differently, so the protein is usually nonfunctional and often ends early at a new stop codon.

20. How does the Ames test show that a chemical is mutagenic?

It uses Salmonella strains that cannot make histidine. They are plated with the test chemical on medium with only a trace of histidine, enough for a few divisions. A mutagen causes reversions that let colonies grow, so more colonies than in the control means it is mutagenic. A liver extract is added to convert chemicals that only become mutagenic after metabolism.

Gene expression

21. How do leading strand and lagging strand synthesis differ in DNA replication?

The leading strand is made continuously in the 5' to 3' direction towards the fork, from a single primer. The lagging strand is made in short fragments, each from its own RNA primer, away from the fork. Primers are then replaced with DNA and the fragments are joined by DNA ligase.

22. Why does the lac operon switch on fully only when lactose is present and glucose is low?

Lactose, as allolactose, binds the repressor and releases it from the operator, which allows transcription. But strong transcription also needs CAP bound to cAMP, which helps RNA polymerase bind the promoter. Low glucose raises cAMP. With glucose present, cAMP is low and transcription stays weak even when lactose is there.

23. What processing does a eukaryotic pre-mRNA go through before it leaves the nucleus?

It gets a 7-methylguanosine cap at the 5' end, introns are removed by the spliceosome so that exons are joined, and a poly(A) tail is added at the 3' end. Together these protect the mRNA from exonucleases, help its export from the nucleus and help its translation.

24. The coding sequence of a gene, from the start codon to the end of the stop codon, is 1203 nucleotides long. How many amino acids does the polypeptide translated from it have, before any processing?

The 1203 nucleotides make $1203/3 = 401$ codons. The last one is the stop codon, which codes for no amino acid, so the polypeptide has 400 amino acids before any processing, such as removal of the first methionine.

WORKING

1. Number of codons $= 1203/3 = 401$
2. The last codon is the stop codon and codes for no amino acid
3. Amino acids in the polypeptide $= 401 - 1 = 400$

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