

energy required for movement, reaction, etc., thus an increase in the likelihood of reactivity. Enzymes would also probably be more flexible, with fewer bonds overall again to encourage movement and reactivity.

- Many colonies can have similar morphologies and not be related. While some bacteria produce very distinctive colonies that are readily identifiable, most don't, and so morphology alone cannot be used for the majority of microbes. As we learned in Chapter 4, several tests are generally necessary to truly establish the identity of an organism. In this case, colony morphology, coupled with microscopy and metabolic tests, would be far more useful than colony morphology alone.

CHAPTER 7 Microbial Genetics

Multiple Choice

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|------|-------|-------|-------|-------|
| 1. B | 6. C | 11. C | 16. D | 21. D |
| 2. D | 7. D | 12. A | 17. D | 22. C |
| 3. B | 8. D | 13. B | 18. D | 23. D |
| 4. A | 9. B | 14. C | 19. B | 24. C |
| 5. B | 10. C | 15. B | 20. B | 25. A |

Fill in the Blanks

- uracil, thymine
- linear, more
- triphosphate deoxyribonucleotides
- leading, lagging
- transcription, translation
- self-terminating, enzyme-dependent
- codons
- mRNA, tRNA, rRNA, translation
- specific, more than one
- A, P, E
- promoter, operator, one or more genes
- inducible, repressible
- inducible
- base-excision repair
- auxotrophs
- homologues
- transformation
- F plasmid, pilus (conjugation pilus)

Short-Answer Questions for Thought and Review

- Plasmids carry “extra” genes that are not essential to survival, but which can enhance growth potential and in some cases confer pathogenicity upon the microbes that possess them. Resistance plasmids, for example, carry genes coding for antibiotic resistance (enzymes to degrade antibiotics). Virulence plasmids code for a variety of proteins, enzymes, structures, or toxins that influence pathogenicity. F plasmids, though present to stimulate conjugation, can lead to the inadvertent transfer of antibiotic resistance or other harmful genetic elements.

2. The genotype is not entirely translated into phenotype. Cells in general have many more genes than they have expressed proteins. Additionally, not all genes that are used are used at the same time, so while one set of genes may be producing a given phenotype, expression of a different set of genes may give you something different (with the two sets never being expressed at the same time).
3. Eukaryotic mRNA is first made as pre-mRNA which has to be spliced to remove introns and leave exons, it codes for only one polypeptide at a time, and it is not translated until fully transcribed and transported out of the nucleus. Prokaryotic mRNA does not have introns, can code for more than one protein in some cases, and can be translated as it is being transcribed.
4. Wobble allows for redundancy in the genetic code which cuts down on mistakes in translation. If several codons call for the same amino acid and differ only in the last slot, it is highly probably that the right amino acid will be used in the majority of cases. This protects against mutational changes to the proteins produced. Wobble cuts down on energy because the cell does not have to make a separate, specific tRNA for every single amino acid codon combination possible (per the text, rather than making 64 or so codons, *E. coli* can make 40 and get the same level of translation).
5. In prokaryotes, replication, transcription, and translation all occur in the cytoplasm of the cell (though replication and transcription will be more localized to the nucleoid region). In eukaryotes, replication occurs in the nucleus, transcription occurs in the nucleus, and translation occurs in the cytoplasm or bound to the endoplasmic reticulum. Some of these functions also occur in the mitochondria and chloroplasts.
6. Rates of mutation: $X = [(6 - 5) / 5] \times 100 = 20\%$, $Y = 460\%$, $Z = 200\%$.
Order of increasing mutagenic potential = X, Z, Y.
7. The genetic assumption of the Ames test is that all DNA is essentially the same structurally and functionally within all cells. Thus, if something damages DNA in one cell type, there is every reason to believe it will damage DNA in another cell type.

Critical Thinking

1. RNA viruses have a higher mutational rate because they have nothing to compare their new genomic strands to in order to see if there are any mistakes. With dsDNA there is always a second strand paired to the first; if pairing can't occur it is a signal that a mistake has been made and it can be repaired. With single strands, there is no check template, thus mutations are not caught based on structural anomalies and can slip through.
2. The mRNA sequence is 5' — AUG GCC GAU GCU GCC UCC CUA CUC UUU GUA — 3'; the protein sequence is NH₂ — f-Met — Ala — Asp — Ala — Ala — Ser — Leu — Leu — Phe — Val — COOH.
3. The RNA transcript is 5' — CGU GAU AGA CGC GUA — 3'; if you substitute the first G with an A you get a start codon: 5' — C AUG AUA GAC GCG UA — 3' (protein would start at AUG and skip the first C and last UA to give f-Met — Ile — Asp — Ala); if you insert an A after the first G you also get a start codon: 5' — CG AUG AUA GAC GCG UA — 3' (protein would start at the AUG, skip the first CG and last UA and give the same protein as above); if you delete the second A in the sequence you will get a start codon: 5' — CGUG AUG ACG CGU A — 3' (protein would start at the AUG, skip the first CGUG and the last A and give f-Met — Thr — Arg).

Concept Building Questions

1. The actual genes themselves and the DNA composing them never directly interact with the environment in any functional way. DNA may be present outside of the cell, but it is inert unless picked up by another cell during some sort of horizontal gene transfer process. Structure, however, interacts everyday with the environment and will function accordingly. Metabolism is dependent on pulling in nutrients and excreting wastes. Thus it is the physical part of the cell and the functional parts of the cell (the *products* of the genes) that interface with “the world,” determine associations and successful reproduction, and ultimately form the basis of evolutionary selection.
2. In the presence of galactose, the cell produces a symporter and the catabolic genes needed to import galactose and break it down. Galactose is probably serving as an inducer for an inducible operon. Galactose keeps the operon on and prevents repression. If galactose runs out, there is no more galactose to bring into the cell and eventually all galactose inside the cell is used up. The repressor is now free to bind to the operator of the operon to shut off synthesis of transporters and enzymes. No new transporters are placed on the cell surface and the ones that are there can be retrieved into the cell by endocytosis and recycled along with the enzymes. A very few symporters are probably left on the cell surface to “sense” for more galactose. If galactose shows up again, it can be brought into the cell, can interact with the repressor, and re-initiate promotion of the operon.
3. Hydrophobic amino acids will not form hydrogen bonds with water in an aqueous environment and thus will not be stable in the presence of water. One of two things could happen to the mutant protein: either the amino acid could distort the protein by folding inward to “hide” from the water, or two proteins could associate at the hydrophobic residues to “hide” each other, thus forming aggregates. In both cases, the protein will no longer interact normally with other proteins or molecules in the environment. If it is distorted, normal binding sites will most likely have been lost, and if it is aggregated, there is a good chance important interaction sites will be blocked.

CHAPTER 8 Recombinant DNA Technology

Multiple Choice

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|------|------|------|------|
| 1. D | 3. B | 5. B | 7. C |
| 2. C | 4. C | 6. B | |

Fill in the Blanks

1. introns
2. sticky, blunt, sticky
3. PCR (polymerase chain reaction)
4. size, shape, charge
5. DNA, RNA
6. DNA fingerprinting
7. transgenic