Unit 7.1. MCQs Set 1

Results

#1. Q1. Which concept laid the foundation for the “Principles of Inheritance and Variation” historically?

☐
(A). The cell theory
☐
(B). Mendel’s Laws of segregation and independent assortment
☐
(C). Hippocratic medicine
☐
(D). None

Gregor Mendel’s pea plant experiments revealed how traits are inherited, forming the basis for classical genetics.

#2. Q2. Mendel’s “Law of Independent Assortment” states that

☐
(A). Two alleles of a gene separate from each other during gamete formation
☐
(B). Genes for different traits assort independently if they are on different chromosomes
☐
(C). No crossing over occurs
☐
(D). None

Different gene pairs segregate independently when unlinked, giving novel phenotypic combinations in the offspring.

#3. Q3. Historical perspectives: The modern synthesis integrated genetics with

☐
(A). None
☐
(B). Darwin’s theory of evolution, explaining how gene frequencies change in populations
☐
(C). Hippocratic medicine
☐
(D). Infectious illusions

The “modern synthesis” merged Mendelian genetics with natural selection, forming the basis of population genetics.

#4. Q4. The human genome’s approximate size is

☐
(A). 3 million base pairs
☐
(B). 3 billion base pairs
☐
(C). 30,000 base pairs
☐
(D). 300 trillion base pairs

The haploid human genome has around 3×10^9 nucleotides.

#5. Q5. The “Human Genome Project” concluded around 2003, revealing that

☐
(A). Humans have ~100,000 genes
☐
(B). Humans have ~20,000–25,000 protein-coding genes
☐
(C). None
☐
(D). Genes are mostly large introns

Surprisingly, the human gene count was found to be around 20–25,000 protein-coding genes.

#6. Q6. Human genome evolution suggests

☐
(A). No trace of homology to other organisms
☐
(B). Shared genes with other primates, numerous evolutionary conserved elements
☐
(C). RBC doping
☐
(D). None

Humans share a high degree of genetic similarity with other primates, indicating common ancestry.

#7. Q7. Genotype-phenotype correlation tries to

☐
(A). Increase metabolism
☐
(B). Link specific genetic variations to observable traits, bridging molecular basis and outward expression
☐
(C). RBC doping
☐
(D). Infectious illusions

Understanding how certain genes or polymorphisms cause or influence phenotypic outcomes is key to modern genomics.

#8. Q8. Multi-OMIC approach includes

☐
(A). None
☐
(B). Integrating genomics, transcriptomics, proteomics, and metabolomics data for holistic correlation
☐

(C). RBC doping
☐
(D). Infectious illusions

By examining multiple molecular layers, we obtain a more comprehensive view of how genotype affects phenotype.

#9. Q9. Ayurveda’s concept of doṣa-prakṛti might be correlated with

☐
(A). None
☐
(B). Genetic and epigenetic profiles that predispose an individual’s metabolic and physiologic tendencies
☐
(C). RBC doping
☐
(D). Infectious illusions

Research suggests that traditional prakṛti classifications may reflect underlying genetic or metabolic predispositions.

#10. Q10. Using medicinal plants in correlation with doṣa-prakṛti might imply

☐
(A). None
☐
(B). Personalized herbal selection based on genetic/phenotypic constitution
☐
(C). RBC doping
☐
(D). Infectious illusions

Personalizing herbal therapy based on an individual’s prakṛti and genetic profile is part of the emerging field of “Ayurgenomics.”

#11. Q11. Basics of human genomics includes

☐
(A). Only RBC doping
☐
(B). Structure of chromosomes, gene organization (exons/introns), regulatory sequences, and polymorphisms
☐
(C). None
☐
(D). Infectious illusions

Human genomics encompasses the study of chromosomal structures, gene organization, regulation, and variation.

#12. Q12. Regulatory mechanisms of genetic variation might involve

☐
(A). Epigenetic modifications, promoter/enhancer usage, splicing variants
☐
(B). None
☐
(C). RBC doping
☐
(D). Infectious illusions

Gene regulation is influenced by epigenetic modifications, alternative promoter usage, and splicing variants.

#13. Q13. Genetic variation’s role in drug response ties into

☐
(A). None
☐
(B). Pharmacogenomics—some polymorphisms (e.g., CYP450) affect drug metabolism
☐
(C). Infectious illusions
☐
(D). RBC doping

Pharmacogenomics studies how individual genetic variations affect drug metabolism and response.

#14. Q14. Population genomics studies

☐
(A). None
☐
(B). The distribution of gene variants across populations and their evolution under selection, drift, and migration
☐
(C). RBC doping
☐
(D). Infectious illusions

This field examines how different gene frequencies arise and evolve in various populations.

#15. Q15. Disease genomics focuses on

☐
(A). None
☐
(B). Identifying genetic variants that predispose to or cause specific diseases, enabling targeted prevention or therapy
☐
(C). RBC doping
☐
(D). Infectious illusions

Disease genomics aims to pinpoint the genetic factors that influence disease risk and therapeutic responses.

#16. Q16. Pharmaco-genomics (pharmacogenetics) attempts to

☐
(A). None
☐
(B). Tailor drug choice and dose based on an individual’s genetic makeup for optimal safety and efficacy
☐
(C). RBC doping
☐
(D). Infectious illusions

Personalized therapy in pharmacogenomics seeks to optimize drug treatments based on genetic profiles.

#17. Q17. Nutrigenomics means

☐
(A). None
☐
(B). Studying how diet interacts with the genome, influencing metabolism and disease risk
☐
(C). RBC doping
☐
(D). Infectious illusions

Nutrigenomics explores the interplay between dietary factors and genetic predispositions.

#18. Q18. Scientific approaches for biomarker discovery might include

☐
(A). None
☐
(B). Using multi-omics data analysis and machine learning on big datasets to identify gene/protein/metabolite markers
☐
(C). RBC doping
☐
(D). Infectious illusions

Advanced analytical and computational techniques are essential for identifying robust biomarkers.

#19. Q19. “P4 medicine” stands for

☐
(A). None
☐
(B). Predictive, Preventive, Personalized, Participatory
☐
(C). Panchakarma-based protocols
☐
(D). RBC doping

P4 medicine is defined by the four pillars: Predictive, Preventive, Personalized, and Participatory approaches.

#20. Q20. “P5 medicine” extends P4 by adding

☐
(A). None
☐
(B). Promotive—actively enhancing overall health, not just preventing disease
☐
(C). RBC doping
☐
(D). Infectious illusions

P5 medicine adds a focus on health promotion to the P4 framework.

#21. Q21. Approach to integrative “Ayurgenomics” might be

☐
(A). None
☐
(B). Mapping doṣa-prakṛti with genetic expression patterns to refine personalized prevention or therapy
☐
(C). RBC doping
☐
(D). Infectious illusions

Integrating traditional prakṛti classification with genomic data is key to Ayurgenomics.

#22. Q22. Limitation in bridging Ayurveda & genomics is

☐
(A). None
☐
(B). The complexity of polygenic traits and the limited standardization of prakṛti classification, requiring large-scale studies
☐

(C). RBC doping
☐
(D). Infectious illusions

A major challenge is standardizing prakṛti assessment and managing the complexity of polygenic traits.

#23. Q23. Challenges in “delivery” of P4/P5 medicine in Ayurveda might be

☐
(A). None
☐
(B). Limited infrastructure for genetic testing, big data analytics, and standardized doṣa assessments
☐
(C). RBC doping
☐
(D). Infectious illusions

Improved infrastructure and standardization are necessary to deliver personalized and predictive medicine effectively.

#24. Q24. Development of a biomarker for “pittaja prakṛti” might

☐
(A). None
☐
(B). Involve multi-omics to identify consistent transcript or metabolite signatures associated with pitta-dominant physiology
☐
(C). RBC doping
☐
(D). Infectious illusions

A multi-omics approach may reveal distinct molecular signatures linked to the pitta constitution.

#25. Q25. Historically, genetic inheritance was widely recognized with Mendel, but in Ayurveda

☐
(A). None
☐
(B). The bīja (seed) concept indicates an early awareness of hereditary factors
☐
(C). RBC doping
☐
(D). Infectious illusions

Ayurveda’s bīja concept reflects early insights into hereditary transmission.

#26. Q26. “Variation” in genetics is explained by

☐
(A). None
☐
(B). Mutation, recombination, independent assortment, and crossing over
☐
(C). RBC doping
☐
(D). Infectious illusions

These mechanisms generate genetic diversity, which is fundamental to evolution.

#27. Q27. Population genomics in an Ayurvedic sense might explore

☐
(A). None
☐
(B). The distribution of prakṛti-based gene variants across different ethnic/regional groups
☐
(C). RBC doping
☐
(D). Infectious illusions

This approach investigates how genetic variants related to prakṛti vary across populations.

#28. Q28. Disease genomics, e.g., in diabetes, attempts

☐
(A). None
☐
(B). To identify risk alleles (such as TCF7L2) that predispose individuals to type 2 diabetes
☐
(C). RBC doping
☐
(D). Infectious illusions

Genome-wide association studies have identified risk alleles for complex diseases like type 2 diabetes.

#29. Q29. Pharmacogenomics can yield

☐
(A). None
☐
(B). Personalized drug dosing recommendations based on genetic variants (e.g., CYP450 polymorphisms)
☐
(C). RBC doping
☐
(D). Infectious illusions

Pharmacogenomic data can guide individualized drug dosing to minimize adverse reactions.

#30. Q30. Nutrigenomics might find that

☐
(A). None
☐
(B). Certain genotypes respond better to specific diets (e.g., low-carb versus low-fat), influencing weight loss and disease outcomes
☐
(C). RBC doping
☐
(D). Infectious illusions

Nutrigenomics investigates how genetic variation affects dietary response and health outcomes.

#31. Q31. Discovery of biomarkers requires

☐
(A). None
☐
(B). Advanced analytical platforms (e.g., microarrays, next-gen sequencing, proteomics) and rigorous validation in clinical populations
☐
(C). RBC doping

☐
(D). Infectious illusions

Robust discovery and validation methods are essential for identifying clinically useful biomarkers.

#32. Q32. Approaches to P4/P5 medicine in Ayurveda are limited by

☐
(A). None
☐
(B). Inadequate large-scale genomic data on prakṛti, challenges in standardizing assessment, and the high cost of genetic profiling
☐
(C). RBC doping
☐
(D). Infectious illusions

Significant financial and methodological challenges exist in integrating genomics with Ayurvedic principles.

#33. Q33. Challenges in “development & delivery” of P5 medicinal aspects might include

☐
(A). None
☐
(B). Regulatory issues regarding genomic data privacy and the need for extensive clinical trials correlating doṣa assessment with molecular biomarkers
☐
(C). RBC doping
☐
(D). Infectious illusions

Bridging traditional Ayurveda with modern genomics requires resolving regulatory and methodological challenges.

#34. Q34. The principle of “Predictive medicine” is to

☐
(A). None
☐
(B). Identify genetic or other risk markers early, preventing disease onset or mitigating severity
☐
(C). RBC doping
☐
(D). Infectious illusions

Early identification of risk markers allows for timely intervention and disease prevention.

#35. Q35. “Preventive medicine” complements predictive by

☐
(A). None
☐
(B). Implementing lifestyle and dietary interventions based on identified risk factors
☐
(C). RBC doping
☐
(D). Infectious illusions

Once high-risk individuals are identified, preventive measures such as diet and lifestyle modifications can be employed.

#36. Q36. “Personalized medicine” indicates

☐
(A). None
☐
(B). Tailoring therapy based on an individual’s genetic, phenotypic, and clinical data for optimal outcomes
☐
(C). RBC doping
☐
(D). Infectious illusions

Personalized medicine optimizes treatment by considering each individual’s unique profile.

#37. Q37. “Participatory medicine” suggests

☐
(A). None
☐
(B). Patients actively engaged in their health decisions through digital tools and shared decision-making
☐
(C). RBC doping
☐
(D). Infectious illusions

Patients’ active involvement in their treatment is a key component of participatory medicine.

#38. Q38. “Promotive medicine” in a P5 context means

☐
(A). None
☐
(B). Actively enhancing overall well-being, going beyond prevention
☐
(C). RBC doping
☐
(D). Infectious illusions

Promotive medicine seeks not only to prevent disease but also to enhance overall health and vitality.

#39. Q39. The approach of “Ayurgenomics” might unify

☐
(A). None
☐
(B). Ancient doṣa-based classification with modern multi-omic and big data analytics to refine personalized therapies
☐
(C). RBC doping
☐
(D). Infectious illusions

Ayurgenomics merges classical Ayurvedic principles with advanced genomic methodologies for personalized care.

#40. Q40. Historical perspective in genetics includes

☐
(A). None
☐
(B). Contributions by Darwin, Mendel, Watson & Crick, culminating in the Human Genome Project
☐
(C). RBC doping
☐
(D). Infectious illusions

The evolution of genetics from Darwin and Mendel to modern molecular biology and the Human Genome Project is well documented.

#41. Q41. Regulatory mechanisms of genetic variation might involve

☐
(A). None
☐
(B). Epistasis, enhancer/repressor activity, methylation, histone modification, splicing variants, and microRNAs
☐
(C). RBC doping
☐
(D). Infectious illusions

Multiple layers of regulation, including epigenetic and post-transcriptional mechanisms, control genetic variation and expression.

#42. Q42. Role of these variations in health includes

☐
(A). None
☐
(B). Certain polymorphisms can confer susceptibility or resistance to diseases and affect drug metabolism
☐
(C). RBC doping
☐
(D). Infectious illusions

Genetic variations can determine individual disease risk and influence treatment outcomes.

#43. Q43. Population genomics in disease might show

☐
(A). None
☐
(B). Variation in allele frequency for risk genes among ethnic groups, which can guide public health strategies
☐
(C). RBC doping
☐
(D). Infectious illusions

Population genomics studies can reveal how gene frequencies differ among populations, impacting disease risk.

#44. Q44. Exploratory genotype-phenotype correlation helps

☐
(A). None
☐
(B). Link doṣa-based phenotypes or disease states with specific SNP haplotypes
☐
(C). RBC doping
☐
(D). Infectious illusions

Correlating traditional Ayurvedic phenotypes with modern SNP data can help validate and refine prakṛti classification.

#45. Q45. The biggest challenge in practical “Ayurgenomics” is

☐
(A). None

☐
(B). Standardizing doṣa assessment, collecting large multi-omic datasets, and achieving robust statistical power
☐
(C). RBC doping
☐
(D). Infectious illusions

Challenges include variability in clinical assessment and the high costs associated with omics technologies.

#46. Q46. P4 & P5 medicine aim to reduce chronic disease burden by

☐
(A). None
☐
(B). Predicting high-risk individuals, preventing disease onset, personalizing therapy, involving patients, and promoting health
☐
(C). RBC doping
☐
(D). Infectious illusions

These integrated approaches encompass the entire spectrum from prediction to health promotion.

#47. Q47. Genetic approach to identify “biomarkers” for Ayurvedic rasāyana response might

☐
(A). None
☐
(B). Evaluate changes in transcript or metabolite levels pre- and post-rasāyana intervention
☐
(C). RBC doping
☐
(D). Infectious illusions

Measuring molecular changes before and after treatment can help in biomarker discovery.

#48. Q48. Limitations of “Ayurgenomics” research can include

☐
(A). None
☐
(B). Heterogeneity in classical textual interpretations, subjective prakṛti classification, and high omics costs
☐
(C). RBC doping
☐
(D). Infectious illusions

Variability in traditional assessments and high research costs are significant hurdles.

#49. Q49. Implementation-wise, “development & delivery” of P4 medicine might require

☐
(A). None
☐
(B). Digital health platforms, machine learning integration, standardized doṣa questionnaires, and genomic screening programs
☐
(C). RBC doping
☐
(D). Infectious illusions

Integrating modern digital tools and genomic screening with classical assessments is essential for practical

implementation.

#50. Q50. Overall, bridging genetics with Ayurvedic practice aims to

☐
(A). None
☐
(B). Provide a more holistic, personalized medicine by merging traditional doṣa-based wisdom with modern molecular insights
☐
(C). RBC doping
☐
(D). Infectious illusions

The goal is to enhance personalized healthcare by integrating Ayurvedic concepts with contemporary genetic research.

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