國立中山大學115學年度學士後醫學系招生考試試題答案疑義釋疑公告

科目	題號	釋疑答覆	釋疑結果
普通生物及生化概論	34	The Golgi apparatus is involved in processing and packaging proteins and lipids for secretion or delivery to other organelles. It's like a post office for the cell. The first option is “formation of extracellular enzymes”: The Golgi modifies and packages enzymes that are secreted outside the cell. For example, digestive enzymes are processed in the Golgi before being released. So, number 1 is a function. The second option is “modification of proteins”: This includes adding carbohydrate groups (glycosylation), phosphorylation, and other changes. So, number 2 should be correct. The third option is “formation of peptide bonds through condensation reactions”: Peptide bonds are formed during protein synthesis, which happens on the ribosomes in the cytoplasm or rough ER. The Golgi doesn't synthesize proteins; it modifies existing ones. So, peptide bond formation isn't a function of the Golgi. That should be incorrect. The forth option is “synthesis of certain polysaccharides”: For plant cells, it's involved in making cell wall components like pectins and hemicellulose. In animal cells, it might be involved in glycosaminoglycans for the extracellular matrix. So, yes, synthesis of certain polysaccharides is a function. The correct answer is D: three of the four options are correct. For option 1: Formation of extracellular enzymes. The Golgi doesn't form the enzymes; it modifies and packages them for secretion. The formation (synthesis) happens in the ER. But the question says "formation," which might be ambiguous. In cell biology, "formation" could refer to the final processing steps. But typically, synthesis is in the ER, and modification is in Golgi. However, for **extracellular enzymes**, the Golgi is crucial for making them functional and ready for secretion. So, it's considered part of the formation process. The question says "formation," which might imply synthesis, but in context (you could see the option 3: formation of peptide bonds. This is not correct.), it's the preparation for extracellular release.	維持原公布答案(D)

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普通生物及生化概論	78	Glucose is a small molecule that can easily diffuse from the blood into the interstitial fluid. Throughout the body, including the muscle and adipose tissues, the glucose concentration is in near-simultaneous equilibrium with blood glucose levels. For pancreatic beta-cells, they sense the elevated glucose concentration in the surrounding interstitial fluid, which triggers the secretion of insulin. Concurrently, in muscle cells and adipocytes, this interstitial glucose is poised for cellular uptake, awaiting the insulin-dependent translocation of GLUT4 transporters.	維持原公布答案(D)

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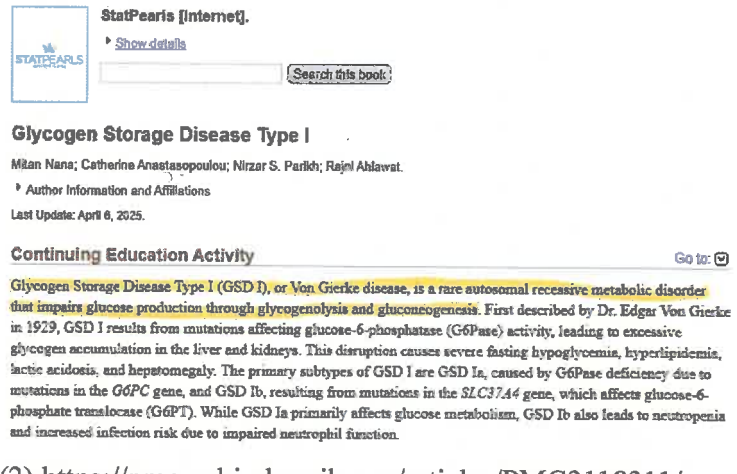
科目	題號	釋疑答覆	釋疑結果
普通生物及生化概論	88	本題重點在於「during fasting」情境下，G6Pase 缺乏「directly impaired」哪些生化途徑的生理功能。在肝臟中，G6Pase 催化 G6P → glucose + Pi，是 glycogenolysis 與 gluconeogenesis 的最後共同步驟（final common step）。缺乏此酶時，肝臟雖能將糖原分解至 G6P，但無法釋放游離葡萄糖進入血液，導致兩個途徑的血糖維持功能均被直接阻斷。以下為文獻或專業書籍的支持: (1) https://www.ncbi.nlm.nih.gov/books/NBK534196/  StatPearls [Internet]. Show details Search this book Glycogen Storage Disease Type I Milan Nana; Catherine Anastasopoulou; Nirzar S. Parikh; Rajni Ahlawat. Author Information and Affiliations Last Update: April 6, 2025. Continuing Education Activity Go to: Glycogen Storage Disease Type I (GSD I), or Von Gierke disease, is a rare autosomal recessive metabolic disorder that impairs glucose production through glycogenolysis and gluconeogenesis. First described by Dr. Edgar Von Gierke in 1929, GSD I results from mutations affecting glucose-6-phosphatase (G6Pase) activity, leading to excessive glycogen accumulation in the liver and kidneys. This disruption causes severe fasting hypoglycemia, hyperlipidemia, lactic acidosis, and hepatomegaly. The primary subtypes of GSD I are GSD Ia, caused by G6Pase deficiency due to mutations in the *G6PC* gene, and GSD Ib, resulting from mutations in the *SLC37A4* gene, which affects glucose-6-phosphate translocase (G6PT). While GSD Ia primarily affects glucose metabolism, GSD Ib also leads to neutropenia and increased infection risk due to impaired neutrophil function. (2) https://pmc.ncbi.nlm.nih.gov/articles/PMC3118311/ **Metabolic alterations** **In GSDI, the fasting hypoglycemia results from the blockage of the last step of glycogenolysis and gluconeogenesis. However, a few patients show an unexpected tolerance to fasting [61].** (3) https://www.sciencedirect.com/topics/pharmacology-toxicology-and-pharmaceutical-science/glycogen-storage-disease-type-1#chapters-articles Chapter Glucose Metabolism and Hormonal Regulation Glycogen Storage Diseases Genetic deficiency of enzymes of the liver related to glycogen breakdown can lead to the engorgement of the liver with glycogen. Thus, patients with Hers disease (type VI) lack liver phosphorylase and must therefore rely on gluconeogenesis to maintain blood glucose. Patients with Von Gierke disease (type I) lack glucose 6-phosphatase, cannot get glucose from either glycogenolysis or gluconeogenesis, and therefore can withstand only limited starvation. Presumably, they have increased glycogen because increased levels of glucose 6-phosphate promote glycogen synthesis and inhibit glycogen breakdown, as described previously. In McArdle disease (type V), muscle phosphorylase is absent (indicating that there are separate muscle and liver isoforms from separate genes) and exercise capacity is limited. 雖然生化教科書在描述糖原分解牽涉的酶轉換步驟時可能將 G6Pase 置於「肝臟葡萄糖輸出」部分，但在代謝整合與臨床脈絡（尤其是禁食時血糖穩態）下，將 G6Pase 缺乏視為同時直接損害兩個途徑。	維持原公布答案(D)