Abstract 1409

Quantification of Learning Advances in a Science CURE: Providing Learning Objectives to Corroborate and Validate the Advantages of Experiential Education

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Course-based Undergraduate Research Experiences (CURE) seek to supply college students with hands-on experiences to develop research skills and complex thinking. As students progress through their education and enter their professional careers, improvement of these critical skills is imperative. CUREs make it possible for students to experience the enchantment of science in its authentic form by engaging in research. Regardless of race, socioeconomic status or family obligations, CUREs have been gaining popularity as an essential model to expose every student to the process of science firsthand. This study aims to gauge student learning objective mastery quantitatively using Anticipated Learning Outcomes (ALOs) designed specifically for the Biochemistry Authentic Student Inquiry Laboratory (BASIL) CURE curriculum. Likert scale-based analysis is employed to evaluate the level of content mastery student responses demonstrate. Assignment questions were designed to correspond to the most critical learning objectives. Learning gains were evaluated across several semesters and represent fully in person, hybrid and online teaching modalities. Analysis shows greater mastery of bio-informatic ALOs during remote learning. The data show that quality education can be enhanced and improved by technology. The student's mastery of wet-lab ALOs coincided with our findings that lab courses need enhanced strategies to teach critical STEM lab-research skills in an online setting. This work will allow for wider CURE adoption through quantitative assessment and instruction strategies targeted to identifying learning mastery gaps. Ultimately, research based curricula can serve as a platform to expose every undergraduate student to vital STEM research experiences.

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Abstract 1451

Comparing effectiveness of two antibodies (Aducanumab and Gantenerumab) on reducing amyloid-beta plaques

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Alzheimer's disease (AD) is a degenerative neurological disorder that destroys memory and other important cognitive functions. As time progresses, brain cell connections, as well as the brain cells themselves, atrophy and die. AD is caused by a missense mutation in the amyloid-beta peptide within the amyloid precursor protein (APP). The mutation results in glutamine being replaced with glutamic acid. Previously conducted studies showed that mutated forms of the amyloid-beta peptide fragment have a greater tendency to stick together and form protein clumps or aggregates. The abnormal build-up of aggregates in and around the brain cells has been found to be strongly associated with the development of Alzheimer's disease, therefore, it appeared crucial to study the methods that reduce these build-ups. Attempts to treat this disease have produced antibodies that bind to the mutated amyloid-beta peptide and clear the aggregated amyloid precursor protein out of the brain. The overall goal of this project is to use 3D printed protein models to show interactions leading to a clearer explanation of the efficacy variations between antibodies. One antibody, Aducanumab, is currently in Phase 3 clinical trials and has been fast-tracked by the U.S. Food and Drug Administration. Aducanumab functions by specifically binding to the mutated amyloid-beta peptide and clearing aggregates out of the brain. This antibody binds to a smaller linear epitope formed by amino acids 3–7 of the amyloid-beta peptide. Using Jmol, protein visualization software, the Aducanumab (6CO3) PDB was manipulated to highlight multiple hydrophobic interactions, shown in a dark salmon color, and 2 hydrogen bonds, shown in white. The small binding location, flexibility provided by fewer strong interactions, and high affinity for aggregates at a high density make the antibody ideal for clearing out large aggregates. Another antibody, Gantenerumab, is still undergoing testing in order to ensure safety and efficacy. This antibody functions by binding to a longer linear epitope formed by amino acids 3–11 of the amyloid-beta peptide. Unlike Aducanumab, Gantenerumab interacts with peptides through 2 salt bridges in addition to 3 hydrogen bonds and multiple hydrophobic interactions. Along with hydrogen bonds in white and hydrophobic interactions in dark salmon, the Gantenerumab (5CSZ) PDB was manipulated to show negative side chains of the salt bridge, labeled in red, while the positive side chains were labeled in blue. The increased number and strength of interactions reduces the flexibility of this antibody, thus making it difficult to easily bind and clear aggregated peptides. While both antibodies bind to a similar region of the amyloid-beta peptide and function to