

Bioavailability of aducanumab in alzheimer's disease treatment.

Walid Khan*

Department of Pharmacology and Therapeutics, United Arab Emirates University, Al Ain, United Arab Emirates

Received date: May 15, 2023, *Manuscript No.* AJPTI-23-104775; **Editor assigned date:** May 17, 2023, *Pre QC No.* AJPTI-23-104775 (PQ); **Reviewed date:** June 01, 2023, *QC No.* AJPTI-23-104775; **Revised date:** June 08, 2023, *Manuscript No.* AJPTI-23-104775 (R); **Published date:** June 16, 2023.

Accepted on 25th June, 2023

Description

Aducanumab, a ground-breaking medication, has sparked hope in the treatment of Alzheimer's disease. Developed by Biogen, this monoclonal antibody targets amyloid-beta plaques, a hallmark of the disease. Bioavailability refers to the proportion of a drug that reaches the systemic circulation, and it plays a vital role in determining its efficacy. The bioavailability of Aducanumab and the factors that influence its absorption, distribution, metabolism, and excretion, shedding light on its pharmacokinetic profile.

Aducanumab targets and binds to amyloid-beta plaques, which are known to accumulate in the brains of individuals with Alzheimer's disease. These plaques are believed to contribute to the cognitive decline and neuronal damage observed in the disease. By binding to the plaques, Aducanumab aims to facilitate their removal, potentially slowing down disease progression and preserving cognitive function. The development of Aducanumab has been a journey marked by both challenges and breakthroughs. Initial Phase 1b clinical trials showed promising results, demonstrating a reduction in amyloid-beta plaques and suggesting a potential cognitive benefit. Building upon these findings, Phase 3 clinical trials were conducted to further evaluate the safety and efficacy of Aducanumab.

Aducanumab is administered intravenously, allowing for direct infusion into the bloodstream. The intravenous route ensures rapid and complete absorption of the medication, bypassing the challenges associated with oral administration, such as degradation in the gastrointestinal tract and first-pass metabolism. By directly targeting amyloid-beta plaques in the brain, Aducanumab's bioavailability is optimized, leading to increased therapeutic potential. Following administration, Aducanumab is distributed throughout the body. The distribution process is influenced by factors such as plasma protein binding, tissue permeability, and the Blood-Brain Barrier (BBB). Aducanumab's large molecular size and the potential for binding to amyloid-beta plaques may affect its distribution pattern. While Aducanumab can cross the BBB, further research is needed to understand its penetration into

different brain regions and its binding affinity to target sites. Factors like inflammation and disease progression may also impact the distribution of Aducanumab within the brain.

Aducanumab undergoes metabolic processes that can influence its bioavailability. The primary mechanism of metabolism for monoclonal antibodies is proteolysis via enzymes such as proteases and peptidases. The elimination of Aducanumab and its metabolites is primarily through renal excretion. Studies have shown that a substantial portion of Aducanumab is excreted unchanged in the urine. However, further research is needed to understand the complete excretion pathway and the impact of renal function on the elimination of Aducanumab.

Aducanumab's bioavailability is a crucial factor in determining its effectiveness in treating Alzheimer's disease. The intravenous route of administration ensures rapid and complete absorption, maximizing its therapeutic potential. While the distribution and metabolism of Aducanumab are still being explored, its ability to cross the BBB and target amyloid-beta plaques in the brain holds promise. Understanding the excretion pathways will aid in optimizing dosing regimens and monitoring the drug's clearance from the body. Continued research into the bioavailability of Aducanumab will provide valuable insights and contribute to the on-going development of this ground-breaking medication for Alzheimer's disease.

*Correspondence to:

Walid Khan,
Department of Pharmacology and Therapeutics,
United Arab Emirates University, Al Ain,
United Arab Emirates,
E-mail: walidkhan@gmail.com