Ashdin Publishing
Journal of Evolutionary Medicine
Vol:10 (2022) Article ID 236096, 02 page
DOI:10.4303/JEM/236096

Commentary

Progressions of Molecular Dynamics in Evolutionary Medicine

Elijah Tejada*

Department of Biochemistry, Weill Medical College, United States

**Address Correspondence to Elijah Tejada, E-mail: elijah_tej@yahoo.com*

Received: 30-November-2022; Manuscript No: JEM-23-87365; **Editor assigned:** 02-December-2022; PreQC No: JEM-23-87365 (PQ); **Reviewed:** 16-December-2022; QC No: JEM-23-87365; **Revised:** 21-December-2022; Manuscript No: JEM-23-87365 (R); **Published:** 28-December-2022; **DOI:** 10.4303/JEM/236096

Description

Many molecular dynamics simulations attempt to forecast and quantify uncommon occurrences, such the folding of a protein or a phase transition. Rare event simulation is frequently impractical, especially when the equations of motion are high-dimensional, as in molecular dynamics. For quickly calculating mean initial passage durations, transition rates, or reaction routes, many techniques have been presented. This article covers and examines current research in the area of rare event simulation and presents a novel methodology that integrates concepts from statistical mechanics and optimal control. The detailed optimum control method is similar to using Jarzynski's equation to calculate free energy, but it uses a faster sampling technique and (theoretically) provides variance-free estimators of the statistics for uncommon occurrences. We provide two numerical examples to demonstrate the new methodology and talk about how it compares to earlier approaches. The literature on rare event simulations in molecular dynamics is extensive. The fundamental theoretical framework for describing transition events has been provided by Transition State Theory (TST) and its expansions based on the reactive flux formalism since the 1930s.

TST, however, has a limited ability to convey data and does not permit the characterization of transition channels. The theory simply describes how this surface is traversed throughout the reaction. It is based on splitting the state space into two sets and placing a dividing surface between them, leaving set A on one side and the target set B on the other. Choosing an appropriate dividing surface may be challenging, and making the wrong decision will result in a very inaccurate rate estimate. If the unusual event is of the diffusive kind, where several distinct reaction pathways coexist, the TST estimate is thus very challenging to correct. As a result, several strategies that attempt to go beyond TST have been presented.

A crucial characteristic of many systems emerging in physics, chemistry, biology, etc. are rare but significant transition events between long-lived states. Simulations of Molecular Dynamics (MD) make it possible to examine and comprehend the dynamical behaviour of molecular systems. Even with the most powerful general purpose computers, accurate simulations for interesting (big) chemical systems in solution on timeframes beyond microseconds remain impossible. Due to the fact that many biological equilibrium processes are frequently linked to uncommon occurrences, this severely restricts the investigation of these processes using MD. Due to the average waiting time between the occurrences being orders of magnitude larger than the duration of the transition defining the event itself, these infrequent events necessitate unreasonably extended simulations. Because of this, it is impractically excessive to solve this problem directly using numerical simulation of the system before a sufficient number of events have been recorded for the majority of relevant systems. As a result, estimate and simulation of uncommon events are among the most difficult subjects in molecular dynamics.

The traditional anticancer platinum drugs, including cisplatin [cis-diamminedichloroplatinum (II)], transplatin [trans-diamminedichloroplatinum (II)], and oxaliplatin [1,2-diaminocyclohexane-oxalateplatinum (II), were widely used in clinical therapy with success against several common types of tumours, including ovarian, testicular The platinum drugs' method of action against tumours is believed to include the Pt (II) centre attacking DNA by covalent interactions with the N7 guanine atoms of the DNA molecule in order to hinder DNA replication and transcription, ultimately

causing cell death. Platinum drugs that cross nuclear membranes and interact with DNA are essential for increasing the effectiveness of cancer therapies. However, the diminished cytoplasmic absorption and higher outflow of these platinum agents limit their efficiency, hence reducing the likelihood that the DNA target will be reached. In reality, the platinum agents can quickly react with several cytoplasmic macromolecules, particularly thiol compounds, and lose their effectiveness before reaching the cell nucleus. As a result, research into the platinum agents' mechanisms of resistance and their interactions with bio-macromolecules has gained popularity in recent years.

Acknowledgement

Authors do not have acknowledgments currently.

Conflict of Interest

There are no conflicts of interest.