Mechanisms of protein adhesion to bare and carbonate-coated silver nanoparticles

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Silver nanoparticles (AgNPs) are some of the most commonly used nanomaterials because of their powerful antimicrobial properties. Concerns regarding a release of AgNPs into the environment are rooted in the fact that we still lack a complete understanding of what controls this (eco)toxicological behavior. One relatively unexplored factor that may be important for controlling their toxicity and/or bioavailability is the adhesion of AgNPs to microbial biomolecules such as proteins. Using a environmental proteomics approach that combines matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) mass spectrometry with chemical derivatization techniques, we explored the binding mechanisms of proteins to two types of commercially available AgNPs. We studied a number of proteins and peptides based on their silver-binding affinity either as shown in previous studies or as we demonstrated by UV-Vis spectrophometry and polyacrylamide gel electrophoresis. MALDI mass spectra collected on conjugates of AgNPs and peptides (synthetic peptides and protein digestion fragments) often exhibited intense Ag adduct signals indicating strong association of, in some cases, up to four Ag atoms. Clear differences in the MALDI mass spectra of various protein fragments bound to bare and carbonate-coated AgNPs suggest differential binding based on nanoparticle surface chemistry. When the derivatizing agent DEPC was added to conjugates of protein fragments and carbonate-coated AgNPs, some residues were inaccessible to derivatization. This suggests that basic amino acids (e.g. Lys, His, Arg) are important in binding to the carbonate shell. In addition to presenting a probable mechanism for protein adhesion to AgNP, these results suggest that nanoparticle coatings may significantly influence their reactivity with microorganisms in the environment.

Carbon isotope excursions at times of mass extinction

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Without exception mass extinction events are associated with perturbations of the carbon isotope system. These take the form of rapid negative shifts (e.g. the end-Permian and Guadalupian events), positive shifts (Late Devonian and Cenomanian/Turonian events) and complex series of oscillations (end-Triassic and early Jurassic events). Understanding the cause(s) of these shifts lies at the heart of many mass extinction models. Negative excursions have been variously attributed to (a) methane sourced by gas hydrate dissociation, (b) thermogenic sources from major igneous intrusions beneath large igneous provinces (the Svensen hypothesis), (c) volcanogenic carbon dioxide directly exhaled during flood basalt volcanism, (d) the local signature of C recycling in a stratified water column (the Kuspert Model) and (e) collapse of primary productivity (Strangelove Ocean Model). In contrast, positive excursions are typically explained by a single mechanism: elevated organic C burial during phases of eustatic rise and ocean anoxia. The latter mechanism leaves a manifest geological record and so is easily tested but the mechanisms for negative excursions are either only indirectly inferred from the geological record or are difficult to quantify. Not surprisingly debates rage on the origin of negative excursions. An overlooked factor in many of these debates is the relative timing of excursions and contemporaneous extinction losses. For many events (e.g. Guadalupian, early Jurassic events) the perturbations postdate the extinctions losses indicating that they record destabilisation of the C cycle in the aftermath of a biotic crisis but that they are not directly implicated in the extinction mechanism.