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Kinetic model of impurity poisoning during growth of calcite

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The central role of the organic component in biologically controlled mineralization is widely recognized. These proteins are characterized by a high proportion of acidic amino acid residues, especially aspartate, Asp. At the same time, biomineralization takes place in the presence of a number of naturally-occurring, inorganic impurities, particularly Mg and Sr. In an attempt to decipher the controls on calcite growth imposed by both classes of modifiers, we have used *in situ* AFM to investigate the dependence of growth morphology and step kinetics on calcite in the presence of Sr^{2+}, as well as a wide suite of Aspartic acid-bearing polypeptides. In each case, we observe a distinct and step-specific modification. Most importantly, we find that the step speed exhibits a characteristic dependence on impurity concentration not predicted by existing crystal growth models. While all of the impurities clearly induce appearance of a "dead zone," neither the width of that dead zone nor the dependence of step speed on activity or impurity content can be explained by invoking the Gibbs-Thomson effect, which is the basis for the Cabrera-Vermilyea model of impurity poisoning. Common kink-blocking models also fail to explain the observed dependencies. Here we propose a kinetic model of inhibition based on a "cooperative" effect of impurity adsorption at adjacent kink sites. The model is in qualitative agreement with the experimental results in that it predicts a non-linear dependence of dead zone width on impurity concentration, as well as a sharp drop in step speed above a certain impurity content. However, a detailed model of impurity adsorption kinetics that give quantitative agreement with the data has yet to be developed.

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