An interfacial energy mechanism for the complete inhibition of crystal growth by inhibitor adsorption

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We present a unified model for complete crystal-growth inhibition based on the thermodynamics of interfaces. The premise for our model is that the adsorption of inhibitor leads to a reduction in interfacial tension or edge energy for the crystal surface or step, respectively. In our formulation, the work to add a layer or grow a step increases due to the difference in interfacial tensions or edge energies for surfaces with and without an adsorbed inhibitor. For a large enough difference in interfacial tensions or edge energies, complete inhibition of growth is realized when the total work does not decrease as more crystals are formed. We demonstrate that our model can provide a theoretical description of critical subcooling data for hydrates and ionic crystals, and critical supersaturation data for various crystal systems. © 2005 American Institute of Physics. [DOI: 10.1063/1.2060689]

I. INTRODUCTION

It has been demonstrated for a number of crystal-forming substances that the adsorption of various polymers, ions, and other species onto the growing surface can lead to either complete inhibition or to a reduction in growth rate. The inhibition of crystal growth due to adsorption is important for both natural and industrial processes. Antifreeze proteins (AFPs) and antifreeze glycoproteins (AFGPs) found in arctic fish and other organisms can adsorb onto growing ice crystals and induce complete inhibition of growth for small subcoolings. For the prevention of hydrate formation during the production of natural gas, the premium fuel of this century, various synthetic polymers (kinetic inhibitors) have been shown to delay the onset of natural gas hydrate formation. As with AFPs, complete hydrate crystal-growth inhibition can be induced for relatively small subcoolings or large concentrations of polymer. Polyelectrolytes can inhibit the unwanted precipitation of mineral salts such as BaSO4 and SrSO4 scale in oil and gas production and CaCO3 scale in heat exchangers and cooling towers. Synthetic polymers, natural proteins, and some smaller molecules can also influence biomineralization (e.g., kidney stone formation). In addition to macromolecular inhibitors, there is a large body of experimental studies on multivalent ions and other inhibitors including the so-called tailor-made additives.

Vastly different models have been proposed to describe observed inhibition due to inhibitor adsorption at the crystal surface. Most studies are based on a molecular view of inhibitors on the crystal surface. No unified model has yet been presented to explain the laboratory observations, and little attention has been paid to the thermodynamics of impurity adsorption on growing crystals. Some works on thermodynamic aspects have been carried out by Wu and Nancollas and van der Leeden et al. We will take a different approach from the works in Ref. 24 and 25 to offer an interfacial thermodynamic framework for the description of crystal-growth inhibition.

In this work we will examine the inhibition of crystal growth from an interfacial energy approach and focus on those systems where addition of adsorbing molecules leads to complete inhibition of growth. This paper is organized as follows. We will first present an overview of major experimental observations regarding crystal-growth inhibition (not intended to be an exhaustive review) in Sec. II. Then we will examine some of the mechanisms that have been proposed by various research groups to describe complete growth inhibition in Sec. III. In Sec. IV we will briefly identify some inconsistencies in the proposed mechanisms. Next, in Sec. V we will propose a new mechanism to explain how adsorbed molecules and ions can completely inhibit crystal growth, and in Sec. VI we will compare the model based on our proposed new mechanism with experimental data from various crystal-forming systems. At the end, in Sec. VII, we draw some conclusions. In this work we will not investigate the mechanism of adsorption itself (e.g., why molecules adsorb preferentially in particular planes or why some molecules adsorb more strongly than others) or how adsorption could influence nucleation. In Ref. 26 we employ the basic ideas from this work to propose a model to explain crystal-growth rate reduction (as opposed to complete inhibition) for various growth mechanisms.
FIG. 1. Critical subcooling as a function of additive concentration for two AFGP samples (data from Ref. 29) and for AFP 1 (founder) (data from Ref. 30) on ice. The solid curves are based on Eq. (23) with $K=2.5$ mg/mL for AFGP 1–5, $K=3.2$ mg/mL for AFGP 7, and $K=1.8$ mg/mL for AFP 1.

II. PAST EXPERIMENTS AND OBSERVATIONS

A. AFP and AFGP

There are various structures for AFPs and AFGPs, and the effectiveness of AFPs and AFGPs is determined by the magnitude of thermal hysteresis (lowering of the freezing temperature) induced by macromolecular adsorption on ice crystals (see Ref. 1, 2, 27, and 28 for reviews). The molecular weights of these proteins can vary from a few thousands for most AFPs to tens of thousands for some AFGPs. The lowering of the freezing point due to AFPs and AFGPs is not a colligative effect, because there is a difference between melting and freezing temperatures. To measure this difference, ice crystals in the presence of inhibitors are observed at various temperatures to find the temperature where crystal growth begins and the temperature where the crystals begin to melt. This temperature difference or thermal hysteresis (the so-called critical subcooling) is a function of the concentration of the inhibitor. Typical data of critical subcooling versus concentration are plotted in Fig. 1 for AFGP molecules (data from Knight et al.29) and AFP 1 (data from Scott et al.30).

B. Hydrate inhibitors

Most of the work on kinetic inhibitors has focused on the effect of the inhibitors on the delay time until hydrates are formed. Poly(vinylpyrrolidone) (PVP) was originally found to affect the onset of crystal formation and growth—Lederhos et al. refer to it as a first-generation inhibitor.31 Since then, poly(vinylcaprolactam) (PVCap) and its copolymers have been found to be more effective.3,31 Larsen et al.3,32 found that PVCap at high enough concentrations could even stop the growth of crystals up to a critical subcooling (in the same way as AFP and AFGP do—see Fig. 2), whereas PVP only slowed the hydrate growth. PVCap and PVP both have vinyl backbones with rings and amide groups in the pendant group. Other water-soluble polymers with similar properties (some with vinyl backbone and high hydrogen-bonding capability) have been examined and found ineffective to inhibit hydrate formation and growth—poly(ethylene oxide) (PEO), poly(vinyl alcohol), and poly(acrylamide).3,33 King and co-workers33,34 used neutron scattering to show that inhibitors such as PVP and PVCap indeed adsorb on the hydrate surface, while a noninhibiting polymer (i.e., PEO) does not.

The effect of kinetic inhibitors on hydrates is in some ways similar to the effects of AFP and AFGP on ice growth. In a hydrate-induction time study AFP was used to slow the onset of tetrahydrofuran (THF) hydrate formation and was observed to have an effectiveness similar to that of PVP.35 Likewise, PVP was used in inhibition of ice formation experiments and was found to hinder the onset of ice formation from solution with a similar effectiveness as an AFP molecule.36

C. Other observations of complete growth inhibition

For many inhibitor/crystal systems, complete inhibition of either growth normal to a face or step growth (depending on what is being measured) can be observed when the driving force (usually referred to in terms of supersaturation) is reduced to a critical value for a fixed concentration of the inhibitor. An example is shown in Fig. 3 for the inhibition of ammonium oxalate monohydrate (AO) crystals with Mn$^{2+}$ on the {100} face.37 Supersaturation $s$ and supersaturation ratio $S$ are defined by38

FIG. 2. Critical subcooling as a function of wt. % PVCap on sII-type hydrate with THF (data from Ref. 3). The solid curve through the data is based on Eq. (23) with $K=0.03$ (wt. fraction) and the dashed curve through the data is based on Eq. (25) with $p=0.54$. The dotted curves show the estimated colligative effects of methanol and the PVCap polymer on thermodynamic freezing-point depression.

FIG. 3. Growth rate in the {100} direction for AO crystals both in the absence (diamonds) and presence (squares) of Mn$^{2+}$ inhibitor. Data taken from Ref. 37.
Mechanism of inhibition of crystal growth


\[ s = \frac{a_s}{a_s'} - 1 = S - 1, \]

where \( a_s \) is the activity of the crystal-forming molecule in solution or the mean ionic activity for lattice ions in solution and \( a_s' \) is the equilibrium activity of the molecule or mean equilibrium ionic activity of the ions. Often, the supersaturation and supersaturation ratio are approximated as

\[ s = \frac{c_s}{c_s'} - 1 = S - 1, \]

where \( c_s \) represents the concentration of crystal-forming species and \( c_s' \) is the equilibrium concentration. Supersaturation ratio and driving force are related by

\[ n \ln S = \frac{\Delta \mu}{kT}, \]

where \( n \) is the number of species in the crystal-forming compound (e.g., \( n = 1 \) for nonionic crystals and \( n = 2 \) for ionic crystals such as NaCl, BaSO₄, etc.), \( \Delta \mu \) is the driving force for crystallization, \( T \) is the temperature, and \( k \) is the Boltzmann constant.

For some crystals, there appear to be two supersaturation barriers. The first barrier below which growth does not occur is the subject of this paper, while the appearance of a second barrier above which growth increases dramatically appears to be due to kinetic-adsorption effects. Many of the systems that have been studied involve ionic impurities, where inhibitor adsorption on the crystal surface arises due to charge interactions. However, there are examples of other low-molecular-weight inhibitors where the chemistry of the inhibitor is similar to that of the crystal-forming molecules, giving rise to adsorption of the inhibitors on the crystal surface. One problem that has been encountered for some of these systems is that the measurements are done at such low-impurity concentrations and supersaturations that inhibition from sample impurities can be observed even in the absence of an added inhibitor. Another problem is the inherent inaccuracy in determining the critical supersaturation given the scatter in the growth data as can be seen in Fig. 3. Here, the value of the critical supersaturation depends on the fitted model through the data. However, much of the data for complete inhibition using adsorbed molecules leads to a reduction in growth rate or complete growth inhibition if the adsorbed molecules are close enough together.

The Gibbs-Thomson equation (sometimes called the Kelvin effect) in one form relates the equilibrium subcooling to the curvature of the crystal (or new phase). For growth of a \( F \) face (that is, a flat face or surface) through step formation and growth, the Kelvin effect is expressed as a two-dimensional (2D) step growth inhibition, originally suggested by Cabrera and Vermilyea for adsorption on terraces [crystal surface as shown in Fig. 4(a)]. Bliznakov and Kirkova also presented early models for step growth inhibition, with related expressions developed later by Davey and Mullin and Davey, but these authors did not describe the models in terms of the Kelvin effect.) The idea is that adsorbed-immobile inhibitors pin the advancing step in various places, and the step becomes curved between the adsorbed inhibitors and the step velocity is reduced. The critical radius of a 2D nucleus is given by

\[ r^* = \frac{\kappa a}{\Delta \mu}, \]

where \( a \) is the surface area occupied per building block (molecular or ionic compound), \( \kappa \) is the specific edge energy, and \( \Delta \mu \) is the total driving force for crystallization [derivation for Eq. (4) is given later]. Ideally, when the spacing between adsorbed inhibitor molecules on the step, \( l \), is less than twice the critical \( 2r^* \), the curvature of the step would need to be less than the critical radius and the step cannot proceed. A schematic of this mechanism is given in Fig. 4.

If supersaturation is realized by lowering the temperature, the critical subcooling can be related to various system parameters as presented in the following. The driving force is zero at the equilibrium (or melting) temperature \( T_e \). Using the relationship between chemical potential and enthalpy, we can write (for relatively small subcooling)
\[ \Delta \mu_j = \frac{\Delta h_j \Delta T}{T_e}, \]  

where \( \Delta h_j \) is the change in enthalpy of species \( j \) between the two phases and \( \Delta T \) is the change in temperature below the equilibrium temperature \( T_e \) (subcooling). The one-component version of Eq. (5) can be written as

\[ \Delta \mu = \frac{L \Delta T}{T_e}, \]

where \( L \) is the latent heat of fusion. Combining Eqs. (4) and (6) gives the expression for critical subcooling,

\[ \Delta T = \frac{\kappa a T_e}{r L}. \]

Kubota et al.\(^{49}\) derived an expression for critical subcooling based on the model of Kubota and Mullin\(^{41}\) and Langmuir adsorption. Their expression, which has some similarities with Eq. (7), is based on a relative step velocity of zero.

For crystal faces that do not have steps, the traditional three-dimensional (3D) version of the Gibbs-Thomson equation\(^{46}\) has been used primarily in the AFP literature\(^{45}\) to explain growth inhibition due to adsorbed inhibitors. The critical radius of a 3D nucleus is given by\(^{80}\)

\[ R^* = \frac{2 \sigma V_{bi}}{\Delta \mu}. \]

where \( \sigma \) is the interfacial tension between the new phase \( \beta \) and the old phase, \( V_{bi} \) is the molecular volume of species \( j \) in the new phase, and \( \Delta \mu_j \) is the driving force of species \( j \). It should be noted that \( r' \) and \( R^* \) are generally different, depending on the relationship between \( \kappa \) and \( \sigma \). The combination of Eqs. (5) and (8) leads to the expression for the equilibrium subcooling as a function of the curvature (one form of the Gibbs-Thomson equation),

\[ \Delta T = \frac{2 \sigma T_e V_{bi}}{R^* \Delta h_j}. \]

Equation (9) has been used by various authors to explain complete growth inhibition of a \( K \) face (that is, a kinked face or surface) on a crystal when the adsorbed inhibitor on the crystal surface has a spacing of less than two times the critical radius of curvature for a particular subcooling. Then the crystal cannot grow, because growth would require a curvature less than the critical value. If the spacing is greater than twice the critical radius then retarded growth occurs.\(^{26}\)

### B. Interfacial tension and edge energy changes

A number of authors have explained experimental results for inhibitors in terms of increases in interfacial tension or specific edge energy. van der Leeden et al.\(^6\) found that the increase in induction time and decrease in growth rate in the presence of an inhibitor could be explained by an increase in specific edge energy associated with 2D nucleation. At the same time they also noted that an increase in edge energy due to inhibitor adsorption is not consistent with thermodynamics. In a related paper, van der Leeden et al.\(^{25}\) proposed a model whereby inhibitors do not alter interfacial tension and edge energy by adsorbing on the nuclei, but act as additional 3D and 2D nucleation sites.

He et al.\(^7\) analyzed induction time data for SrSO\(_4\) crystals by assuming that induction time depends solely on the rate of 3D nucleation in solution and is independent of growth. They found that the increase in induction time caused by inhibitors could be explained by an increase in interfacial tension for the crystal nuclei with adsorbed inhibitor. Fernandez-Diaz et al.\(^{53}\) came to the same conclusion with the same analysis for phosphero-modified polyacrylic acid inhibitor with BaSO\(_4\) crystals.

In a related line of argument, Hall and Lips\(^{52}\) suggested a mechanism for AFP/AFGP activity whereby a lack of protein adsorption at a step would lead to an increase in edge energy (described as line tension in their paper) for 2D nucleation. As a result of increased edge energy, the barrier to 2D nucleation would be increased.

### IV. CRITIQUE OF THE PROPOSED MECHANISMS

In this work we present an interfacial thermodynamic framework for the study of crystal-growth inhibition based on surface energies. We have concerns about both the molecular-based Kelvin effect and the mechanisms advocating surface/edge energy increases.

Hall and Lips\(^{52}\) provide a critique of both the 2D and 3D Kelvin effect as a possible mechanism for AFP/AFGP behavior. One deficiency they cite is that this mechanism requires very strong adsorption. For macromolecules that adsorb strongly, a relatively constant level of adsorbed material is typically reached at a low bulk, equilibrium concentration. Above this concentration, the adsorbed amount (and thus the spacing) would change little. With the Kelvin effect, the critical subcooling should remain practically constant as protein concentration increases in the bulk phase above this level. However, Fig. 1 shows that critical subcooling increases significantly well up to 40 mg/mL for AFGP 1–5.\(^{29}\) For the Kelvin effect to describe these data, the changes in spacing would need to be quite large and the adsorbed amount would have a large increase.

Other concerns with the Kelvin model for AFP inhibition relate to the mobility of the inhibitor on the crystal surface. The Kelvin effect requires essentially immobile inhibitors on the surface (irreversible adsorption). Hall and Lips suggest that any immobile material should be trapped in the crystal if the critical subcooling is surpassed. However, Raymond and DeVries found relatively little AFP engulfed in the crystal.\(^{44}\) Another issue of the Kelvin effect applied to macromolecular inhibitors is that there is no reason to believe adsorbed macromolecules would necessarily be immobile. Although there is evidence for partial engulfment of adsorbed polymer in some crystals,\(^{44,45,53}\) irreversibility of adsorption is not commonly accepted.\(^{54,55}\) There are many examples of polymer displacement in the literature (larger molecules displacing smaller ones in polydisperse samples, small displacer molecules forcing polymers to desorb, etc.). Thus the reasoning that crystal-forming molecules could not adsorb and displace...
the macromolecule is not necessarily sound. The issue of immobility of adsorbed-molecular inhibitor becomes a concern especially for lower molecular weight inhibitors. For these, a dynamic equilibrium is expected and an interfacial energy approach is perhaps better suited than the molecular view of immobile stoppers.

Knight and Wierzbicki56 discuss some of the issues concerning inhibitor mobility and engulfment for AFGP molecules. Visualization of the large molecules as particles with variable contact angles or wettability with the crystal surface helps to explain some of the observed results. However, small-inhibitor molecules, this approach may not be applicable.

Certainly, atomic force microscope (AFM) images showing step distortion in the presence of the inhibitors40,57 could be explained by a step-pinning mechanism. However, they may also be explained by local variations in interfacial tension caused by kinetic effects.

Mechanisms based on an increase in interfacial tension or edge energy also appear to have deficiencies. Based on Eq. (4), an increase in edge energy will only increase the critical radius. Thus, changes in the number or activity of 2D nucleation sites and increases in edge energy can explain a reduction in growth rate, but they cannot explain the complete inhibition of growth at some additive concentration except perhaps at very large concentrations. In addition, mechanisms based on increases in edge energy alone do not address growth of K faces.

An increase in interfacial tension due to adsorption of molecules would violate laws of thermodynamics (as mentioned by van der Leeden et al.6 and Liu58). Nevertheless, the increase in interfacial tension is still often suggested as a mechanism for inhibition. The Gibbs adsorption isotherm is given by

\[ -d\sigma_s = \sum_i \Gamma_i d\mu_i, \]

where \( \sigma_s \) is the interfacial tension of the surface, \( \Gamma_i \) is the surface excess of species \( i \) (approximate number of molecules per area) and depends on the definition of dividing surface, and \( \mu_i \) is the chemical potential of species \( i \). Let us assume that \( \Gamma_i \) is approximately the same as the number of adsorbed molecules of species \( i \) per unit interfacial area at the solid surface. For a two-component (inhibitor and water) system when we apply the Gibbs-Duhem equation to the bulk external phase (and note that each species has the same chemical potentials in the bulk and at the interface at equilibrium), then we can rewrite Eq. (10) as

\[ -d\sigma_s = \left[ -\Gamma_w \frac{n_p}{n_w} + \Gamma_p \right] d\mu_p, \]

where \( n_p \) and \( n_w \) are the number of molecules of inhibitor and water, respectively, in the bulk solution phase. At constant \( T \) and constant external pressure \( P \), \( d\mu_p \) increases when the amount of inhibitor in the bulk solution increases because \( (d\mu_p/d\sigma)_{T,P} > 0 \), where \( x \) is the mole fraction of the inhibitor in solution. If \( \Gamma_p > 0 \) (and thus \( \Gamma_w < 0 \)), then an increase in inhibitor concentration in the solution results in a decrease of interfacial tension.

V. OUR PROPOSED MECHANISM

The basic premise of our proposed model is that the adsorption of inhibitor molecules leads to a lowering of interfacial tension on the crystal surface. We do not treat the surface as having heterogeneous properties (adsorbed inhibitor sites with inhibitor-free surface in between), but assume a uniform interfacial tension reduction due to inhibitor adsorption. The growth of the crystal through addition of molecules leads to a temporary difference between interfacial tensions of surfaces before and after growth until inhibitor molecules can readсорb on the newly formed surface. If the difference in surface energy caused by crystal growth is equal in magnitude to the decrease in energy caused by molecules in solution or melt becoming crystalline, then the crystal will not grow. Essentially, the energy barrier to growth will be too large for growth to occur. The resulting form of the equation for complete inhibition is the same, irrespective of the mechanism of growth. Here we will show how our proposed model is developed for the growth of K faces and F faces. For F faces we will specifically focus on 2D nucleation and step propagation.

A. Growth of a K face (Continuous growth)

We first consider the work to form a new layer on the K face of a crystal. A 2D schematic for this process is shown in Fig. 5. Here \( \sigma \) is the interfacial tension between the fresh layer and solution, \( \sigma_{ns} \) is the interfacial tension between the fresh layer and the crystal, and \( \sigma_c \) is the interfacial tension between old crystal and solution. The interfacial tension of the original crystal face is lowered by the presence of the adsorbed inhibitor, and \( \sigma_{ns}=0 \) due to the identical material on both sides of the interface between fresh layer and crystal (previously adsorbed inhibitor is displaced when the fresh layer is formed). When the fresh layer is formed, the new interface between crystal and solution will not initially have an adsorbed inhibitor—a solid surface must be formed prior.
to adsorption—and thus will have a larger interfacial tension as compared with the old crystal surface. The work to form this layer must account for this difference in interfacial tensions. The work to add a fresh layer of crystal \(n_m\) molecules with a temporarily higher interfacial tension compared to the previous layer is

\[
W = -n_m \Delta \mu + n_m a \sigma + n_m a \sigma_{ns} - n_m a \sigma_s
\]

where \(\Delta \sigma = \sigma + \sigma_{ns} - \sigma_s\), and \(a\) is the average effective area per molecule or ionic compound on the crystal surface exposed to solution. Because \(\sigma > \sigma_s\), \(\Delta \sigma\) will be positive and act to increase the work of layer formation. In order for the crystal to grow \(\partial W / \partial n_m < 0\) (that is, Gibbs free-energy must decrease), and the threshold of growth will occur when \(\partial W / \partial n_m = 0\). The critical driving force from Eq. (12) is, therefore,

\[
(\Delta \mu)_c = a \Delta \sigma,
\]

which is the condition for complete growth inhibition. Equation (13) is one of the two key equations in this paper. Despite its simplicity, its strength lies in its unified application to a broad range of problems.

**B. 2D nucleation for a \(F\) face**

Essentially, the idea here is the same as what was seen for the \(K\) face. A new layer of crystal is forming on an old layer. We could again write Eqs. (12) and (13) to apply to the formation of a new layer on a \(F\) face rather than on the \(K\) face. Here, we will specifically examine the formation of 2D nuclei on the crystal surface to show an alternative derivation of Eq. (13). The proposed mechanism has some similarities to the work of van der Leeden et al.\(^{25}\) and Wu and Nancollas\(^{24}\) in that the work of 2D nucleus formation is affected by the adsorbed inhibitor. However, the model is distinctly different. In our proposed model when a 2D nucleus forms on the substrate the nucleus will initially be uncovered (as above, the surface needs to form before adsorption can occur). Thus, the interfacial tension between nucleus and solution will be larger than the tension between crystal with adsorbed inhibitor and solution. We postulate that the adsorbing material would not adsorb onto a 2D cluster (of subcritical size) and thus lower the energy of the nucleus for kinetic reasons.

Let us consider the formation of a 2D nucleus on a substrate as shown in Fig. 6. The work to form the nucleus is given by

\[
W = \kappa p - n_m \Delta \mu + A(\sigma + \sigma_{ns} - \sigma_s),
\]

where \(p\) is the perimeter of the nucleus, \(A\) is the area of the nucleus, \(\sigma\) is the interfacial tension between the nucleus and solution, \(\sigma_{ns}\) is the interfacial tension between the nucleus and the substrate, and \(\sigma_s\) is the interfacial tension between substrate and solution. For a circular-2D nucleus, the work can be written as

\[
W = 2\pi r \kappa - \pi r^2 \left( \frac{\Delta \mu}{a} - \Delta \sigma \right),
\]

where \(a\) is the area per crystal-forming unit \((A/n_m)\), \(r\) is the radius, and \(\Delta \sigma = \sigma + \sigma_{ns} - \sigma_s\). Taking the derivative of \(W\) with respect to \(r\) and setting it equal to zero allows the critical radius to be determined,

\[
r^* = \frac{\kappa}{\left( \frac{\Delta \mu}{a} - \Delta \sigma \right)}.
\]

The work to form a critical-2D nucleus could be written as\(^{59,60}\)

\[
W^* = \frac{\pi \kappa a^2}{(\Delta \mu - a \Delta \sigma)}.
\]

For a 2D nucleus on its own substrate (same crystal) in the absence of an inhibitor, then \(\Delta \sigma = 0\) and Eq. (16) reduces to Eq. (4). In the presence of an inhibitor, \(\sigma\) (fresh interface without adsorbed inhibitor) will be greater than \(\sigma_s\) (old interface with adsorbed inhibitor). If we assume that no adsorbed inhibitor resides at the interface between nucleus and substrate, then \(\sigma_{ns} = 0\) as we postulated above. Thus \(\Delta \sigma\) will be positive and the critical radius increases as a result of adsorption. This will lead to a decrease in the 2D nucleation rate and a decrease in crystal-growth rate. When \(\Delta \sigma\) is large enough, the denominators in Eqs. (16) and (17) go to zero and a critical nucleus cannot be formed. This would lead to complete inhibition of growth (critical subcooling) via a 2D nucleation-mediated growth mechanism for a smooth \(F\) face, and the condition for complete inhibition is again given by Eq. (13). However, the definition of \(a\) varies depending on the growth mechanism. For growth on a \(F\) face, \(a\) represents the area per crystal-forming molecule on the step. For growth on a \(K\) face, \(a\) is the average exposed area of a molecule on the face. For the same molecule, the latter value for \(a\) would most likely be larger than the former.

For the case of 2D nucleation it could be argued that if \(\Delta \sigma > 0\), then growth on the crystal may more readily occur by the formation of spherical caps rather than 2D disks. However, estimates of nuclei size for small \(\Delta \sigma / \sigma\) show that the cap height may not rise above the molecular diameter in most cases. Thus, the cap model would be inadequate to describe growth in line with Ref. 60. Also, systems with 2D nucleation-mediated growth (both with or without inhibitor) have been observed. When the formation of spherical caps is more likely than 2D nucleation, Eq. (13) may not describe the condition for nucleation. For such a case, growth inhibition can still be explained through the inhibition of step growth as described below.
C. Spiral growth for a $F$ face

For the spiral growth mechanism, the growth rate $G$ is related to the critical radius and step velocity by $G \sim v/r^*$. The spiral arm can only begin spreading when its radius of curvature is greater than $r^*$. The growth rate will tend to zero when the step velocity goes to zero; this condition is discussed in Sec. V D. We will discuss spiral growth in more detail in Ref. 26 with a focus on growth rate reduction.

D. Growth of steps

For both spiral growth and 2D nucleation-mediated growth of a $F$ face, growth is also inhibited by reduction in the step velocity. Step velocity can be thought of as a 2D version of normal growth for a $K$ face. The condition for complete inhibition of step growth may differ from Eq. (13) if inhibitor adsorbs preferentially on the steps rather than on the flat terraces. If we consider the work of formation of a one-dimensional (1D) cluster of crystal-forming molecules or species at a flat step similarly to what was done by Kashchiev (see Fig. 7), then we can write

$$W = -n_m \Delta \mu - n_m d \kappa + n_m d \kappa_1 + n_m d \kappa_1 + \eta_m d \sigma + 2e = -n_m (\Delta \mu - d \Delta \kappa - a \Delta \sigma) + 2e,$$

where $d$ is the effective length of the molecule, $\kappa$ is the edge energy between the substrate (bulk crystal) and old phase, $\kappa_1$ is the edge energy between the newly formed cluster and old phase, $\kappa_1$ is the edge energy between the cluster and substrate, and $e$ is the energy of the end of the cluster. For a kinked step (not shown) the expression for work would be the same as given in Eq. (18) except without the $2e$ term, and the following analysis would still hold. Similar to what we did for the 2D nucleus, we assume that $\kappa_1$ is zero, because the material in the cluster and the substrate are identical. In addition, the newly formed cluster will not initially have adsorbed material on the surface, so $\Delta \kappa = \kappa_1 - \kappa_1 > 0$. Thus, only for $\Delta \mu > d \Delta \kappa + a \Delta \sigma$ will the work decrease with an increase in $n_m$. By setting the derivative of Eq. (18) equal to zero, we find the condition of complete inhibition to be

$$\Delta \mu = d \Delta \kappa + a \Delta \sigma.$$  \hspace{1cm} (19a)

For the case where inhibitor adsorbs preferentially at the step, then $d \Delta \kappa \gg a \Delta \sigma$ and

$$\Delta \mu = d \Delta \kappa.$$  \hspace{1cm} (19b)

Equation (19b) is analogous to Eq. (13). Under the condition where inhibitor adsorption occurs only on the step terrace and not on the kinks or step sites, specifically, we would expect Eq. (19a) to reduce to Eq. (13) [we could also arrive at Eq. (13) using the same arguments for formation of the fresh layer as was done in Sec. V A for the growth of a $K$ face]. Equations (19a) and (19b) are the other key, basic equations of this work in addition to Eq. (13).

VI. COMPARISONS WITH EXPERIMENTS

To confirm the validity of our model given by the key new expressions from Eqs. (13) and (19a), we will analyze various sets of literature data. We will first show how the model can explain the critical subcooling (thermal hysteresis) data for AFPs and AFGPs on ice. Then we will demonstrate that the model can explain critical subcooling data for kinetic inhibitors of hydrates. Finally, we will compare our model with the data for complete inhibition (critical supersaturation) of various other crystals in the presence of lower-molecular-weight inhibitors (ions, etc.).

A. AFP and AFGP critical subcooling

To compare the model with the data for critical subcooling, we combine our expression for complete growth inhibition, Eq. (13), with Eq. (6) to obtain an expression for critical subcooling,

$$\Delta T = \frac{T_c \Delta \sigma}{L}.$$  \hspace{1cm} (20)

For AFP and AFGP (or any other inhibitor), we need to write expressions for the change of interfacial tension difference, $\Delta \sigma$, with protein concentration. Using the concept of relative adsorption and an assumption that the protein-free interfacial tension $\sigma$ does not change with concentration, we write the Gibbs adsorption equation [Eq. (10)] in the following form for a two-component mixture assuming a monodisperse macromolecule, an ideal solution, and $[\Gamma_{\sigma} d \mu_{\sigma}] < [\Gamma_{\sigma} d \mu_{\sigma}]$:

$$-d \sigma = -d (\sigma - \sigma) = d \Delta \sigma = \Gamma_p kT \ln x,$$  \hspace{1cm} (21)

where $x$ is the mole fraction of the inhibitor (in this case, protein). The fluid nonideality does not change the results that are of interest in this work. An adsorption expression relating $\Gamma_p$ and $x$ needs to be assumed. Over a moderate range of concentrations, a Langmuir isotherm in the following form can be used to describe the adsorption of macromolecules such that

$$\Gamma_p = \frac{\Gamma_{\sigma} x}{K + x},$$  \hspace{1cm} (22)

where $K$ is the Langmuir constant and $\Gamma_{\sigma}$ is the upper limit of $\Gamma_p$. This assumption will be discussed further in Sec. VI B. Combining Eqs. (20)–(22), approximating $T$ as constant (since the subcooling data covers only about 1 K) and equal to $T_c$, approximating both $L$ and $K$ as constant, and integrating we obtain the expression for critical subcooling.
FIG. 8. Critical subcooling vs ln(1+c/K) or ln(K+x) to demonstrate the linearity based on Eq. (23) for AFP/AFGP on ice and kinetic inhibitor on hydrates. The data are the same as given in Figs. 1 and 2. K values are chosen to maximize fit ($R^2$) for the line. (a) AFGP 1–5 with $K =$2.5 mg/mL, AFGP 7 with $K=3.2$ mg/mL, and AFP 1 (flounder) with $K =1.8$ mg/mL all on ice. (b) PVCap on sII hydrate with K=3 wt.%.

\[
\Delta T = \frac{kT^2a\Gamma_m}{L} \ln(K+x) + M_1,
\]

where $M_1$ is a constant. In Eq. (23) $x$ is the model fraction, but at low concentrations it can be replaced by mass fraction or concentration ($c$) with only a change to the definition of the constant, $M_1$. With an appropriate value for $K$, a plot of $\Delta T$ vs ln($K+x$) or ln($K+c$) should yield a straight line (the values of $K$ and $M_1$ depend on the system and on units of $x$ or $c$). Ideally, if the adsorption expression adequately describes adsorption over the entire concentration range, the value of $M_1$ should be such that $\Delta T=0$ when $x=0$ ($M_1 = -kT^2a\Gamma_m \ln K/L$). In this case a plot of $\Delta T$ vs ln($1+x/K$) or ln($1+c/K$) should yield a straight line through the origin.

Figure 8(a) shows $\Delta T$ plotted against ln($1+c/K$) for AFGP 1–5, AFGP 7, and AFP 1, with $K$ values chosen to optimize the fit of the line as given in the figure caption. It should be noted that there is significant uncertainty in the value of $K$, as it can be varied over a fairly large range with very little change in the $R^2$ for the fitted line. From the slopes of the lines we can estimate $\Gamma_m$, and using $K$ we can estimate the adsorbed amount for any concentration. However, it must be realized that the protein used in one case, AFGP 1–5, is a blend of various size molecules. In this case Eq. (21) is technically not appropriate and needs to be replaced by equations where adsorbed amounts and chemical potentials of every molecule size are included. If for the moment we neglect the polydispersity, we can estimate the apparent adsorbed amount from the slope by using $a=9.6$ Å$^2$, $L=6030$ J/mol, and $T_s=273$ K. From the slope we estimate (on a molar basis) $\Gamma_m=6.8 \times 10^{-8}$ moles/m$^2$ for AFGP 1–5, $4.5 \times 10^{-8}$ moles/m$^2$ for AFGP 7, and $5.9 \times 10^{-8}$ moles/m$^2$ for AFP 1. Over the range of concentrations studied, the adsorbed amounts range from about $(3–6) \times 10^{-8}$ moles/m$^2$ for AFGP 1–5, $(2–4) \times 10^{-8}$ moles/m$^2$ for AFGP 7, and $(3–6) \times 10^{-8}$ moles/m$^2$ for AFP 1. The trends are as expected, although it is difficult to compare AFGP molecules to AFP molecules due to the difference in structure and chemistry. For AFGP 1–5, the average molecular weight is approximately 20 000; this would give an adsorbed amount in mass of about 1 mg/m$^2$, which is in the range of expected adsorbed amount for strongly adsorbing macromolecules of that size. Within the same class of compounds, the lower-molecular-weight AFGP 7 does not adsorb as strongly. Figure 1 shows the subcooling data with the results from Eq. (23). It is clear that the proposed model is consistent with the data for AFP and AFGP thermal hystereses. Also, the changes in the amount of adsorbed protein needed to explain the results for our proposed model are far less than what would be necessary for the Kelvin effect. Finally, it should be noted that the values of $\Delta \sigma$ necessary for complete growth inhibition of ice from Eq. (20) are relatively small with $\Delta \sigma<1$ mJ/m$^2$ for $\Delta T$ of up to 2.5 K.

B. Kinetic inhibitors for hydrates

Here we employ the proposed model to examine the data for critical subcooling of hydrates with kinetic inhibitors. However, since the crystals are multicomponent, Eq. (5) should be used instead of Eq. (6). Anklam and Firoozabadi\textsuperscript{50} have shown that for the nucleation of hydrates, the driving force will be zero for all components except water. For monodisperse inhibitors, then, Eq. (23) can be used provided that Langmuir adsorption [Eq. (22)] holds over the concentration range of interest and that $L$ (enthalpy difference between water and ice) is replaced by $\Delta h_{wb}$ (enthalpy difference between water in solution and water in the hydrate). Note that we are still assuming that $T$ is constant and approximately equal to $T_s$ and that $\Delta h_{wb}$ is constant.

Figure 8(b) shows that Eq. (23) fits the critical subcooling data for hydrate-kinetic inhibitors (data from Larsen et al.\textsuperscript{2} for PVCap inhibitor with molecular weight of about 1700) with $K=3$ wt. %. The model result is replotted in Fig. 2 (solid curve) to reveal its good description of the data. It appears that the adsorbed amount of polymer varies significantly over the concentration range. As above, the uncertainty for the value of $K$ is large due to the fact that it can be varied over a large range with only a small change in $R^2$.

It may appear somewhat surprising that Eq. (23) describes the data so well considering the polydispersity of the inhibitor and the significant range of temperatures (i.e., adsorption may not necessarily be described by an isotherm and $K$ may vary with temperature). However, the curve from Eq. (23) does not go through the origin in Fig. 8(b); that is, a plot of $\Delta T$ vs ln($1+c/K$) does not give a straight line through the origin (not shown). This suggests that Eq. (22) only describes adsorption over a limited range of concentrations. It could be argued that even if the effect of temperature change...
is not significant, the Langmuir adsorption isotherm [Eq. (22)] does not adequately describe homopolymer adsorption like it may for the adsorption of globular proteins. There has been much work to model polymer adsorption using a variety of methods (e.g., self-consistent-field theory). Although these models are much more complicated than a simple adsorption isotherm expression such as Eq. (22), we can see if any expression does an adequate job of describing adsorption. An examination of model adsorption isotherms for homopolymers based on a self-consistent-field theory shows that a Freundlich isotherm follows the model isotherm much better than a Langmuir isotherm (not shown). The Freundlich adsorption expression is given by

$$\Gamma_p = b c^p,$$  
(24)

where $b$ and $p$ are empirical constants ($0 \leq p \leq 1$). Combining Eqs. (20), (21), and (24), with $L$ (or $\Delta h_{\text{m}}$), $b$, and $p$ assumed independent of temperature, and integrating we find

$$\Delta T = \frac{akT^2bc^p}{L_p}.$$  
(25)

If Eq. (24) adequately describes adsorption, then a plot of $\Delta T$ vs $c^p$ should give a line through the origin. In fact, we find that the data are reasonably linear and the resulting model based on Eq. (25) with $p=0.54$ describes the data well in Fig. 2 (dashed curve). Obviously, we find an even better fit if the model is not forced to go through $\Delta T=0$ at $c=0$.

Another difficulty in using the model to describe the behavior of polymeric inhibitors is the polydispersity. As mentioned with AFGPs 1–5 above, from Eq. (10) the change in interfacial tension is from the summed contributions of every chain length. Thus even a small change in the overall adsorbed amount may arise due to large changes in the adsorbed amounts of individually sized chains. Particularly we would expect that as the surface is covered, the amount of adsorbed material of large molecular weight will increase preferentially to that of the lower-molecular-weight material. One possible artifact of the use of equations for a monodisperse sample to describe a polydisperse sample is that Eq. (23) predicts a significantly nonzero $\Delta T$ in Fig. 8(b) for a polymer concentration of zero (see Fig. 2) and the fit from Eq. (25) deviates at low concentrations in Fig. 2.

As an additional note for both kinetic inhibitors and AFPs/AFGP, it must be mentioned that crystals are certainly not isotropic, so the above analysis could be applied to each crystallographic plane—a, $\sigma$, and $\sigma_r$ will depend on the crystal plane. Thus it is not that surprising that just beyond the critical subcooling (or at low levels of added inhibitor) the crystal morphology is quite different with and without an inhibitor as has been often observed for ice with AFP/AFGP and hydrates with kinetic inhibitors. It is probable that the critical subcooling for one plane is different than the others and the degree of growth inhibition depends on the plane and direction. It is further expected that the first plane to grow when critical subcooling is surpassed will be that with less adsorbed protein and smaller values for $a$.

### C. Other $F$ face growth measurements

Aside from the data on AFPs/AFGPs and hydrate kinetic inhibitors, various groups have determined and analyzed the observable critical supersaturation $s_c$ in the presence of an inhibitor below which step growth (and face growth) does not occur. The relationship between this critical supersaturation and inhibitor concentration $c$ varies from system to system. Much of the data has been shown to be fitted with a power-law relationship when supersaturation is small

$$s_c = Bc^m,$$  
(26)

or a reciprocal relationship

$$\frac{1}{s_c} = \frac{C_2}{c} + C_3,$$  
(27)

where $B$, $m$, $C_2$, and $C_3$ are constants. As we will see, critical supersaturation data can also be described using our model from Eq. (13) or (19b). Unfortunately, unlike the data for AFPs and AFGPs, most of the data consist of relatively few points with much scatter. Therefore we are limited in the comparisons we can make.

For the growth of a $F$ face where the inhibitor adsorbs on the terraces and thus Eq. (13) holds, we can combine Eqs. (3), (21), and (22) with Eq. (13) to find

$$\ln S_c = \left(\frac{\Delta \mu}{nkT}\right)_c = (a/n)\Gamma_m \ln(1 + x/K) + M_2.$$  
(28)

Thus, a plot of $\ln S_c$ vs $\ln (1 + x/K)$ should be linear and, in most cases, a plot of $\ln S_c$ vs $\ln (1 + x/K)$ should be linear and go through the origin if $\ln S_c=0$ when $x=0$. The exception to this is the data from Rashkovich and Kronsky, which gives nonzero $\ln S_c$ in the absence of an inhibitor as mentioned earlier (presumably due to impurities). For the case where $x$ is small compared to $K$, adsorption will behave linearly with $\Gamma_m/K$ as the proportionality constant and

$$\ln S_c = \left(\frac{a}{n}\right)\left(\frac{\Gamma_m}{K}\right)x + M_3.$$  
(29)

In most cases (except for the data from Ref. 15), $M_3=0$ so that $\ln S_c=0$ when $x=0$. This would result in a linear relationship between critical supersaturation and inhibitor concentration for small supersaturations where $\ln S_c=\ln(s+1) = s$. It is interesting to note that the model of Kubota and Mullin would also predict a linear relationship between $x$ and $x$ for linear adsorption (large $K$); our model in some cases predicts similar behavior as models based on the step-pinning mechanism.

It turns out that a linear relationship between critical supersaturation and inhibitor concentration has been observed for some systems. Figure 9 shows a plot of $\ln S_c$ versus inhibitor concentration for a potassium dihydrogen phosphate (KDP) crystal inhibited by Fe$^{3+}$ impurity (from step velocity measurements in the (001) direction on the prism face). Linear behavior is certainly within the realm of experimental error. Rashkovich and Kronsky also comment on linear behavior when Cr$^{3+}$ is used. Sangwal takes the data from Ref. 15 for inhibition of KDP and finds
should note that two supersaturation barriers, denoted \( s_c \) and \( s_d \), respectively, exist. Sangwal also finds a linear relationship for \( s_c \) when plotted as \( \ln s_c \) versus inhibitor concentration. However, Sangwal is still able to fit the data with a line when \( s_d \) is plotted as \( \ln s_d \) versus inhibitor concentration. Even if this is the case, it is still possible to determine the critical supersaturation for a single step and thus the one relevant for our analyses. At \( s_d \), growth completely stops, so that is the critical value used for the above analyses. At \( s_c \), step velocity undergoes a sharp transition from gradual to rapid. Land et al. suggest that \( s_c \) is the critical supersaturation for a single step and thus the one most relevant for our analyses. Even if this is the case, Sangwal shows a linear relationship between \( s_c \) and \( c \) as well as between \( s_d \) and \( c \).

Unfortunately, without adsorption isotherms for these systems, it is difficult to determine if adsorption truly lies in the linear region. Kubota et al. did calculate adsorption isotherms for Pb\(^{2+} \) adsorbing on NaCl and compared this to crystal-growth data. Complete inhibition is certainly observed in the linear region of the isotherm, but only three critical supersaturations are determined, and each of these has significant scatter so a linear relationship between critical supersaturation and concentration cannot be uniquely confirmed. These measurements will be revisited in Ref. 26 to show agreement between the adsorption isotherms and predicted growth based on our model.

Not all critical supersaturation data follow a linear relationship with impurity concentration. Sangwal and Mielniczek-Brzoska observe that for many inhibitors on particular faces of AO crystals, Eq. (26) with \( m=0.5 \) or some other value less than 1 often fits the data. However, these data may also be explained by our model and be fitted with a line when plotted as critical supersaturation versus \( \ln(1 + x/K) \) using Eq. (28). Examples of this include Mn\(^{2+} \) and Co\(^{2+} \) as shown in Fig. 10. For Fig. 10(a) a value of \( K=0.9 \times 10^{-5} \) (mole fraction) was used, while for Fig. 10(b) a value of \( K=2 \times 10^{-5} \) (mole fraction) was used. In Fig. 10(a), multiple values for \( \ln s_c \) are given. The values determined from the original data, it seems, depend on the number of data points used in the fitting procedure. This again highlights the inherent scatter in the literature data—very different conclusions could be reached depending on the values used.

Kubota et al. present the data for sucrose inhibition using raffinose determined from the data of Albon and Dunning. To compare the data with their model based on step pinning [form of Eq. (27)], they find a linear relationship when \( 1/s_c \) vs \( 1/c \) is plotted. However, as we show in Fig. 11, the data can be plotted using our Eq. (28) \([\ln S_c \text{ vs } \ln(1+x/K)]\) with \( K=1.5 \) mol %.

Sangwal found that critical supersaturation can be reasonably described by a linear relationship with the impurity concentration for Al\(^{3+} \) on KDP. However, the data are slightly better fits when plotted versus \( \ln(K+c) \) [based on our Eq. (28)] with \( K=7 \) ppm as shown in Fig. 12. In a related

![FIG. 9. Critical supersaturation vs inhibitor concentration for KDP inhibition with Fe\(^{3+} \). The data show a linear relationship as would be expected based on Eq. (29) for linear adsorption. The data are for step velocity measurements in the (001) direction on the prism face and are taken from Ref. 15.](image)

![FIG. 10. Critical supersaturation plots showing that Eq. (28) adequately describes the data with (a) \( K=0.9 \times 10^{-5} \) (mole fraction) for Mn\(^{2+} \) on AO and (b) \( K=2 \times 10^{-5} \) (mole fraction) for Co\(^{2+} \) on AO, both for normal growth in the (100) direction. The data are taken from (a) Refs. 37 and (b) 61.](image)

![FIG. 11. Critical supersaturation plot for sucrose inhibition with raffinose showing that Eq. (28) describes the data with \( K=1.5 \) mol %. The data are for step velocity measurements in the (010) direction on the (100) face (Ref. 62) and are taken from Ref. 39.](image)
work, Kubota et al.49 report critical subcooling data for Al\textsuperscript{3+} on KDP crystals. The driving forces and inhibitor concentrations were larger than what were used for the data in Fig. 12. The data can be explained by our Eq. (23) with \(K=4\) mg/L, as shown in Fig. 13 [note that the curve based on Eq. (23) is very similar to the one shown by Kubota et al.\textsuperscript{49} from their work based on the model of Kubota and Mullin\textsuperscript{41} for step pinning (Kelvin effect) with a larger Langmuir constant of about 15 mg/L]. The fact that the \(K\) values that we have determined for the two sets of data for KDP with Al\textsuperscript{3+} are quite comparable shows that our model proposed here can consistently describe the critical subcooling and critical supersaturation data.

We based much of the above analyses of data on Eqs. (28) and (29), which were derived assuming that Eq. (13) (adsorption on step terraces) is the condition of complete growth inhibition. The analyses would remain unchanged if Eq. (19b) (adsorption at the steps) were used as the condition for complete growth inhibition. When the inhibitor adsorbs preferentially at the step, Eq. (19b) must be used with analogous expressions of Eqs. (21), (22), (28), and (29). The 2D analog to the Gibbs adsorption equation for ideal solutions as given in Eq. (21) (at constant temperature) is

\[ -d\kappa = d\Delta \kappa = \Gamma_e kT d \ln x, \]  

where \(\Gamma_e\) is the edge excess (approximately the number of adsorbed inhibitor molecules per length). Assuming Langmuir adsorption at the step we have

\[ \Gamma_e = \frac{\Gamma_{em} x}{K_e + x}. \]  

where \(\Gamma_{em}\) is the maximum edge excess and \(K_e\) is the Langmuir constant for step adsorption. Combining Eqs. (30) and (31) with Eq. (19b) we find for a critical supersaturation

\[ \ln S_c = \left( \frac{\Delta \mu}{n k T} \right)_c = (d\ln) \Gamma_{em} \ln (K_e + x) + M_4. \]  

As was shown above, if the value of \(K_e\) is much larger than \(x\), adsorption is linear and we find a linear relationship between critical supersaturation and inhibitor concentration such that

\[ \ln S_c = (d\ln) \left( \frac{\Gamma_{em}}{K_e} \right)_p x + M_5. \]  

The forms of Eqs. (32) and (33) are identical to those of Eqs. (28) and (29), and thus the comparisons of our model to experimental data and the values of \(K\) determined above would be unchanged if Eqs. (32) and (33) had been used. However, the differences between Eqs. (13) and (19b) may become important when analyzing growth rate data where both \(\Delta \sigma\) and \(\Delta \kappa\) may need to be considered (e.g., growth of a F face with preferential adsorption at the steps). Unfortunately this means that it is difficult to distinguish what type of adsorption is present (i.e., adsorption at terraces or at steps) based on comparisons of these equations with critical supersaturation data.

VII. CONCLUSIONS

A model for crystal-growth inhibition based on an interfacial energy approach is proposed. Two new and simple equations, \((\Delta \mu) = a \Delta \sigma\) and \((\Delta \mu) = d \Delta \kappa\), give the critical driving force due to inhibitor adsorption for different mechanisms of growth and adsorption [see Eqs. (13) and (19a)]. The proposed mechanism of inhibition and the model based on the mechanism appear to describe the observed experimental data for the critical subcooling of ice, hydrates, and ionic crystals in the presence of inhibitors and for the critical supersaturation in the presence of inhibitors for various other systems.

The step-pinning model has been widely used to describe the behavior of crystal-growth inhibitors. Here, we present a different approach to model the effect of interface inhibition—one based on the thermodynamics of the surfaces rather than the behavior of individual molecules. There are some similarities between the two models such as the prediction of a linear relationship between critical supersaturation and impurity concentration in the range of linear adsorption. However, the two models are fundamentally different in mechanism.

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