A Percolation View of Novolak Dissolution. 4. Mechanism of Inhibitor Action

Hsiao-Yi Shih and Arnost Reiser*

Institute of Imaging Sciences, Polytechnic University, Brooklyn, New York 11201

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ABSTRACT: It is difficult to reconcile the large inhibition effects of some diazoquinones, and other additives, with a site-blocking model of dissolution inhibition; to produce an effect commensurate with the observed inhibition factors, one molecule of inhibition would have to disable up to 16 OH groups of the resin. The dual nature of OH groups which are simultaneously hydrogen donors and acceptors provides an amplification mechanism that can accommodate this requirement. Phenolic OH groups form aggregates in solutions of low polarity, and such aggregates, termed "phenolic clusters", are also formed in the coating solutions of novolak films. When inhibitors with strong hydrogen acceptor (carbonyl) groups are introduced into the system, they form hydrogen bonds with some of the OH groups, and in so doing polarize these groups. The polarized groups, in turn, become stronger hydrogen acceptors and cause a larger number of OH groups to collect in phenolic clusters. The coming together of hydrophilic sites in clusters disturbs the randomness of the percolation field and decreases the site connectivity. This lowers the diffusional flux of base through the matrix and with it the dissolution rate.

Introduction

We have recently presented a model of novolak dissolution based on the realization that novolak and other phenolic resins are amphiphilic materials.[1-4] A novolak film may thus be regarded as a hydrophobic solid in which hydrophilic sites, the OH groups of the phenols, are embedded. When a novolak film is immersed in aqueous base, the ions of the base are attracted to the hydrophilic sites and repelled by the hydrophobic regions, and they diffuse into the film by jumping from one hydrophilic site to the next. This mode of propagation is a typical percolation process, and a model of novolak dissolution may be constructed on the basis of percolation theory.[5,6] An excellent exposition of the subject is to be found in ref 7.

The dissolution of novolak in alkaline developers is controlled by the rate of diffusion of base into the resin film, and that depends on the density of hydrophilic percolation sites in the system.[2,3] When naphthodiazoquinones, or some other additives, are incorporated in the films, the rate of dissolution decreases dramatically. In the spirit of a percolation model, it seemed reasonable to assume that inhibitors function by blocking some of the OH groups of the resin. This idea was qualitatively in agreement with experience, but when it was tested quantitatively, it could not account for the large inhibition effects observed in commercial resist systems.

To quantify the inhibition effect in our investigations, we have characterized the inhibition efficiency of an additive by an inhibition factor, f_{ij}, defined as the derivative of the logarithm of the dissolution rate (log R) with respect to the concentration (c_i) of inhibitor i in resin j.[8]

$$-\frac{\mathrm{d}\log R}{\mathrm{d}c_i} = f_{ij} \qquad (1)$$

In an earlier paper[4] we have described the inhibition behavior of a group of aromatic ketones which display a wide range of inhibition efficiencies. We were able to determine the volume in the resin matrix made unavailable to percolation by the presence of the inhibitor.

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Table 1. Inhibition Factors and Hydrophobic Displacements of Inhibitors in Partly Methylated Novolak Resins[a]

inhibitor	$f_{ij}°$	M_i	α_i/α_B
benzophenone	0.55	182	1
xanthone	2.26	196	2.13
flavanone	6.80	223	3.78
diazoquinone inh	13.1	396	6.79
flavone	18.3	221	7.65
2,3-diphenylindenone	23.6	282	9.73
β-naphthoflavone	46.6	271	16.4
α-naphthoflavone	49.1	271	16.6

[a] $f_{ij}°$ is the inhibition factor of the additive in the base resin, α_i is the hydrophobic displacement volume of the inhibitor, α_B is that of benzophenone, and M_i is the molecular weight of the inhibitor.

This was termed the hydrophobic displacement volume (α_i) of the inhibitor. Table 1 gives the displacement volumes of a group of inhibitors relative to the displacement volume (α_B) of benzophenone, the least effective inhibitor of the group. If one assumes that benzophenone blocks just one OH group, the ratio α_i/α_B counts the number of OH groups that the inhibitor must disable in order to produce the observed inhibition effect. One may envision benzophenone eliminating one hydrophilic site from the percolation field, but it is hard to see how a single molecule of naphthoflavone could affect 16 OH groups of a novolak resin. For the inhibitors to create hydrophobic displacements commensurate with their inhibition factors, a mechanism is needed that would amplify the effect of the inhibitor on the resin. In other words, the resin must in some way cooperate with the inhibitor.

Resin–Inhibitor Interaction

A mechanism by which phenolic resins may cooperate with the inhibitor is hydrogen bonding between the OH groups of the resin and the keto groups of the inhibitor.

$$\mathrm{POH} + \mathrm{A} \rightleftharpoons \mathrm{POHA}$$

The OH groups act here as hydrogen donors, the keto groups as acceptors. The interaction is characterized by an equilibrium constant,[9]

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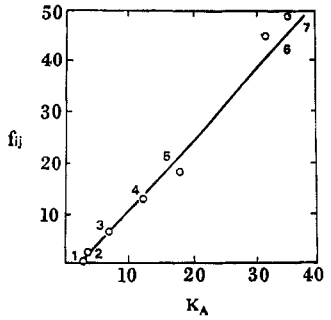


Figure 1. Inhibition factors of seven inhibitors plotted against the equilibrium constant K_A of their interaction with phenol in dilute benzene solution.

$$K_A = \frac{[POHA]}{[POH][A]}$$

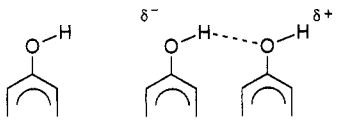
We have determined the interaction constants of phenol with various inhibitors in dilute benzene solution, using the absorbance of the OH stretching vibration in the IR spectrum to monitor the concentration of free phenol. The results are listed in Table 2, together with the inhibition factors of the additives in a standard novolak film.[4] The data, plotted in Figure 1, show that there is a linear relation between the interaction constants (measured in solution) and the inhibition factors (observed on solid resin films). Hydrogen bonding between the OH groups of the resin and the carbonyl groups of the additives appears to play a central role in the inhibition phenomenon.

How can hydrogen bonding affect the dissolution rate of the resin film? Simple blocking of sites cannot account for the large inhibition effect of some of the more successful inhibitors. We realized that in some way the effect of a single inhibitor molecule must be transmitted to a multiplicity of OH groups. It occurred to us that the dual nature of the phenol functionality with respect to hydrogen bonding could provide a solution to the problem.

Phenolic Clusters

It is well known that OH groups can act as both hydrogen donors and acceptors.[10-14] This ability of the OH groups, together with the amphiphilic nature of the resin, makes novolak compatible with many other resins.[15-17] It is, moreover, an excellent binder of pigments and fillers. In fact, the name "novolak" is derived from the resin's ability to substitute for that classical natural binder, shellac.

A closer look at an isolated phenol and at a hydrogen-bonded pair of OH groups shows that in the bonded pair, the partial positive charge on one of the hydrogens and the partial negative charge on the oxygen atom of the other phenol are increased by inductive bond polarization.



The inductive effect makes the phenolic dimer a stronger acid and causes the well-known hyperacidity of novolak.[18-21] Paniez et al. have described the same phenomenon in *p*-cresol oligomers.[22] By the same token, the oxygen atom of the phenol that carries the increased negative charge becomes a stronger hydrogen acceptor.

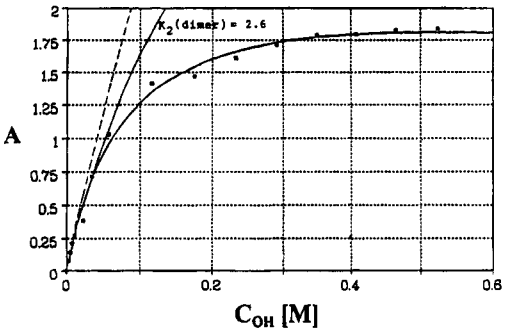


Figure 2. Absorbance of the free OH-stretching peak at 3614 cm^{-1} as a function of *p*-cresol concentration (mole/liter) in CCl_4.

Table 2. Inhibitor–Phenol Interaction and Inhibition Factors

inhibitor	f_{ij}	K_A
benzophenone	0.55	2.5
xanthone	2.26	4.0
flavanone	6.80	7.1
diazoquinone inh	3.1	12.4
flavone	18.3	18.0
β-naphthoflavone	46.6	30.1
α-naphthoflavone	49.1	35.3

There are many other manifestations of that phenomenon. We refer to the seminal paper by Cairns and Eglinton[23] on intramolecular hydrogen bonding in phenolic oligomers. Multiple hydrogen bonding in a variety of phenolic crystals has been described, e.g., in ref 24.

The inductive self-polarization of interacting OH groups is also the reason why hydrogen bonding of a phenol group with a phenolic dimer is preferable to interaction with a single phenol. The self-association of phenol in solvents of low polarity does not, therefore, stop at the dimer but leads almost immediately to larger aggregates that may be termed "phenolic clusters". Figure 2 shows the optical density of the free OH peak at 3614 cm^{-1} in the IR spectrum of a dilute CCl_4 solution of *p*-cresol, plotted as a function of the *p*-cresol concentration.[25] The broken line in the plot indicates the optical density that would be observed if the cresol molecules did not interact with each other. The curve marked $K_2 = 2.6$ represents the behavior in a dimerizing system. It can be seen from the position of the experimental points that free OH disappears very much faster than can be accounted for by a dimerization reaction. The formation of clusters of OH groups is a pervasive phenomenon in phenolic materials and solutions, and in that process phenolic dimers are only a transient stage.

The basic conditions for cluster formation exist also in solutions of novolak. Cluster sizes in pure novolak are probably quite small, but that clusters are actually formed can be demonstrated by solution viscosities. Figure 3 compares the viscosities of novolak solutions with those of partially methylated samples of the same novolak. The viscosity increase is clearly the result of interaction between the OH groups of adjacent chains, and that depends strongly on the resin content of the solutions. In the casting of a polymer film, the solution runs through the whole gamut of resin concentrations, and at the point of solidification a high proportion of OH groups will be contained in phenolic clusters. In fact, no free phenol groups can be detected in novolak films.

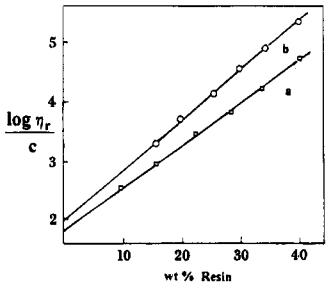


Figure 3. Logarithmic plot of the inherent viscosity of solutions of (b) novolak and (a) methylated novolak (30%).

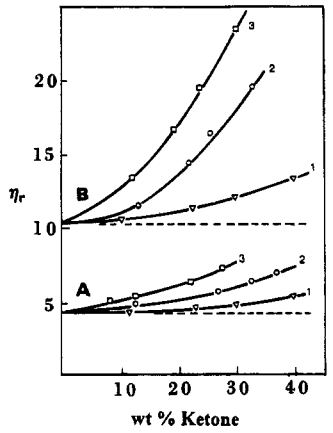
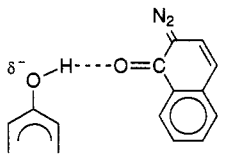


Figure 4. Relative viscosities of (A) 18.4% solutions of novolak and (B) 24.2% solutions of novolak containing the indicated concentrations of the inhibitors: (1) flavanone, (2) flavone, and (3) α-naphthoflavone.

Inductive Cluster Polarization

What happens when an inhibitor is introduced into the system? Inhibitors are stronger hydrogen acceptors than OH groups. The equilibrium constant for OH–OH interaction is of the order of 2–3; the interaction constants of phenol with inhibitors are listed in Table 2. The acceptor (carbonyl) group of the inhibitor will bond with the nearest OH group of the resin, and in so doing will inductively polarize its partner.



As a result, the oxygen atom of that OH group will become a much stronger hydrogen acceptor. It will bind the next OH group and polarize it, etc. The polarization effect does not stop at the OH group to which the inhibitor has become attached but propagates some distance into the sequence of hydrogen-bonded hydroxyls which represent the cluster. Here then is a mechanism by which a single inhibitor molecule can affect a multiplicity of hydrophilic sites. In fact, the inhibitor molecules have now become "attractors" of hydroxyl groups in the fractal sense.

The reality of cluster polarization and its effect on the strength of hydrogen bonding can be demonstrated by measuring the viscosity of novolak solutions to which inhibitors have been added. Figure 4 shows the viscosity increase brought about by the three acceptor-inhibitors flavanone, flavone, and α-naphthoflavone. The effect becomes stronger as the concentration of novolak in the solution increases, and it also depends

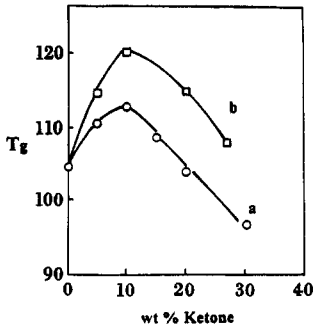


Figure 5. Glass transition temperature, T_g, of novolak films containing (a) flavone and (b) α-naphthoflavone.

on the acceptor strength of the inhibitor, as measured by the interaction constant K_A in Table 2. Enhanced interchain interaction in the presence of inhibitors can also be detected in solid resin films. Figure 5 shows the glass transition temperature of novolak films containing increasing concentrations of flavone and of α-naphthoflavone. It can be seen that up to a content of 10% of the inhibitors the glass transition temperature of the films increases. Beyond that point the glass transition temperature of the resin is lowered by the dilution effect of the (hydrophobic moiety of the) inhibitor.

We recall at this point that the inhibitors of this study have only one carbonyl functionality and can directly interact only with a single OH group. They could not cause the interchain bonding that is manifested in the viscosity increase or the increase in T_g shown in Figures 4 and 5, except for their inductive effect on adjacent OH groups. These experiments are convincing proof of the existence, and the importance, of the inductive polarization brought about by the inhibitor in a phenolic environment. We conclude that the primary effect of inhibitors is the inductive polarization of hydroxyl groups in their vicinity, leading to the formation of larger and more closely bound phenolic clusters than exist in pure novolak films.

Clusters in a Percolation Field. How can cluster formation affect base diffusion and with it the dissolution rate? Classical percolation theory contains the assumption of a random distribution of percolation sites.[5,6] We now find that in novolak and in other phenolic resins the distribution of hydrophilic sites is not strictly random and that the hydrophilic sites are grouped together in clusters. The consequences of this can be visualized more clearly if it is remembered that the hydrophilic OH groups are attached to the hydrophobic moieties of their phenols and that, therefore, hydrophilic clusters are of necessity adjacent to extended hydrophobic regions. As more of the hydrophilic sites become concentrated in a smaller number of clusters and smaller clusters are absorbed in larger ones, the hydrophobic areas surrounding the clusters grow and become more connected. This reduces the connectivity between site clusters and thereby lowers the diffusional flux through the system, and with it the rate of dissolution.

The effect of cluster formation on site connectivity is qualitatively illustrated in Figure 6. We have taken a two-dimensional percolation field of 100 cells and put one hydrophilic site (OH group) in each cell. The OH groups are attached to the polymer backone and have only a limited range of motion. We have simulated this situation by restraining the sites to positions within their cells. Within the cells we have randomized the

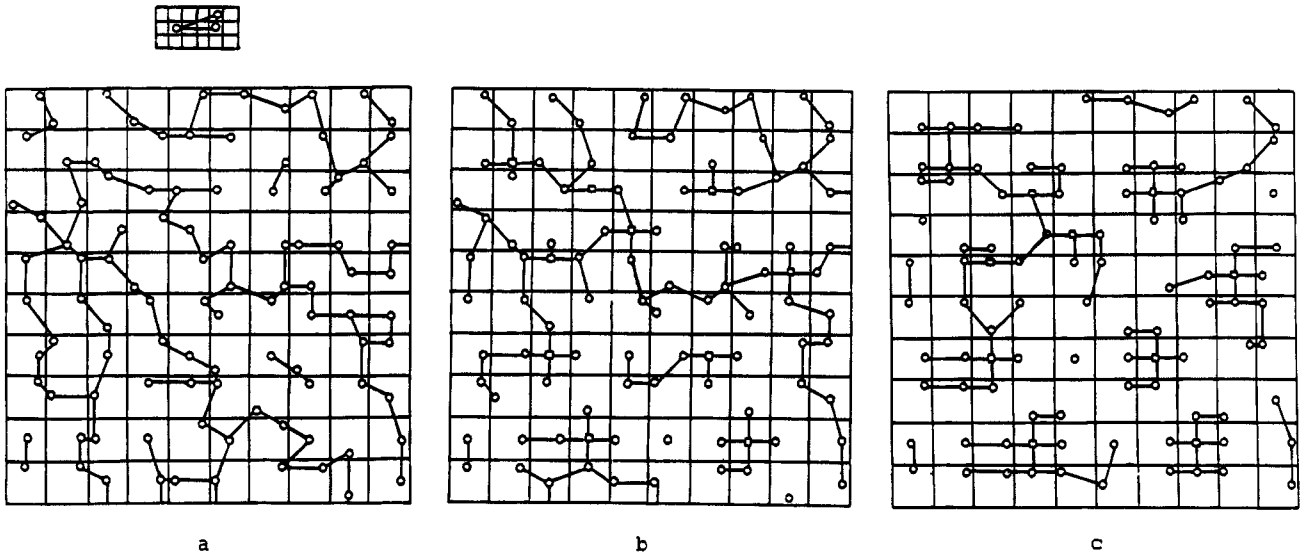


Figure 6. (a) Two-dimensional bond percolation field of 100 sites, representing a pure novolak film. (b) The same field containing 10 attractors binding 4 sites each. (c) Still the same field containing 10 attractors, but each binding up to 7 neighboring sites.

positions of the sites over nine positions available in each cell. The randomized field, representing a pure novolak film, is shown in Figure 6a. Since base diffusion occurs in a bond percolation field, we have drawn the open bonds, choosing as the critical site separation of an open bond[5] a value of 1.054 times the cell length (see the sketch at the head of Figure 6a). This produced the network of connected sites shown in Figure 6a, where 71 of the 100 sites are connected to a single network. That number represents the so-called "strength" of the field[5] and is a measure of its connectivity. A network which connects opposite edges of a field is termed an "infinite" network. In our representation, the field of pure novolak contains an infinite network of strength 0.71.

To simulate the introduction of inhibitors, we now choose at random 10 sites and let these represent the inhibitor molecules and act as attractors of hydrophilic sites. We allow the selected sites (marked by squares in Figure 6b) to attract 4 hydrophilic sites of their surroundings. As a consequence, the sites in the cells adjacent to the attractor migrate within their cells into the position nearest to the attractor. We connect again sites that are within 1.054 × cell length of each other. There is now an infinite network with a strength value of 56 in this field. The connectivity of the system has clearly decreased. Let us now simulate the effect of a stronger inhibitor (attractor). We let the attractor sites stay where they were but allow them to attract, as far as possible, up to 7 neighboring sites. The result is shown in Figure 6c. There is no infinite network left, and the largest finite network has 35 sites. It must be emphasized at this point that the figures are not more than a simple-minded illustration, but they make their point clearly: By collecting percolation sites into clusters, even to the mild degree shown in Figure 6b, the connectivity of the percolation field is lowered considerably.

Can the loss in connectivity brought about by cluster formation account quantitatively for the observed changes in the diffusive flux through the system? This question could be answered by a systematic computer simulation of the percolation process, using cluster numbers and cluster sizes as parameters. We have initiated such an effort. In the meantime, we would like to offer a treatment which approaches the problem from the point of view of a microheterogeneous percolation field. This is not a substitute for a proper statistical treatment of the system, but it establishes a framework in which simple assumptions produce answers that conform to experimental experience.

A Microheterogeneous Percolation Field: Inhibition vs Methylation. If the hydrophilic sites are grouped together in clusters, these hydrophilic clusters are surrounded mainly by the hydrocarbon moieties of the resin, and the two constitute qualitatively different regions of the system. That means that on the level of molecular dimensions the material is composed of a hydrophilic and diffusionally conductive microphase (a) and a hydrophobic, nonconductive microphase (b). The diffusivity of a two-phase system where one of the phases (b) is diffusionally nonconductive may be expressed as the product of the diffusivity of the a-phase, D_a, times the volume fraction, ϕ_a, of that phase.[26]

$$D = D_a\phi_a \tag{2}$$

The response of the overall diffusivity to the introduction of an infinitesimal quantity (dc_i) of inhibitor into the system is represented by the derivative dD/dc_i, which can be written in the form

$$\frac{dD}{dc_i} = \frac{dD_a}{dc_i}\phi_a + \frac{d\phi_a}{dc_i}D_a \tag{3}$$

The introduction of an inhibitor disturbs the balance between the two phases by transferring hydrophilic sites from small clusters, prevalent in pure novolak films, into larger clusters. This action enlarges the hydrophobic regions surrounding the hydrophilic clusters and in that way adds to the nonconducting b-phase. By the same token, it reduces the volume fraction of the conducting a-phase.

We shall assume that the addition of an infinitesimal quantity of inhibitor, dc_i, will not affect the diffusivity of the conducting phase but will only change the volume fraction of that phase. Hence, the first term on the right-hand side of eq 3 is zero, $dD_a/dc_i = 0$. In determining the nonzero derivative $d\phi_a/dc_i$ we make the assumption that the change in the volume fraction of the conductive phase, brought about by the polarization effect of the inhibitor, is proportional to the existing

volume fraction of that phase. This means that an additional quantity of inhibitor will polarize only that part of the phase that has not yet undergone polarization by previously introduced inhibitor molecules.

$$\frac{d\phi_a}{dc_i} = \text{constant} \times \phi_a \quad (4)$$

The constant in eq 4 is characteristic of the inhibitor; it is in fact ($\log e\, f_{ij}$). Combining (3) with (4), one obtains the dependence of the overall diffusivity on the inhibitor concentration c_i.

$$-\frac{dD}{dc_i} = \text{constant} \times D_a\phi_a = \text{constant} \times D \quad (5)$$

Substitution of the dissolution rate, R, for the diffusivity produces the Meyerhofer equation,[8] which is known to describe the effect of inhibitor concentration on dissolution rate.

$$-\frac{d \log R}{dc_i} = \text{constant} \quad (=f_{ij}) \quad (6)$$

If, instead of adding an inhibitor, we remove some of the hydrophilic OH groups by methylation, the dissolution rate decreases also,[2,3] but the mechanism of this change is quite different. The clusters in novolak are small and we are almost dealing with a homogeneous field. In that case we may assume that, in a first approximation, the volume fractions of the two phases will remain unchanged, $d\phi_a/dc_i = 0$. The change in the diffusivity of the conductive phase is now brought about by a change in the density of hydrophilic sites, and that is described by the quadratic scaling law of percolative diffusion[5,6]

$$D = \text{constant} \times (p - p_c)^2 \quad (7)$$

If we treat the methyl group as a quasi-inhibitor, the scaling law leads to a quite different dependence of the derivative dR/dc_i on the dissolution rate. By differentiating the scaling law and substituting for $(p - p_c)$ the identity "(constant $\times R)^{1/2}$" from the scaling law itself, one obtains the expression

$$\frac{dD_a}{dc_i} = \text{constant} \times \frac{dR}{dp} = \text{constant} \times \frac{1}{R^{1/2}} \quad (8)$$

which is quite different from Meyerhofer's equation (6).

We conclude that any lowering of the dissolution rate of novolak is the result of a perturbation of the percolation field. We now understand that there are two distinct ways in which the microheterogeneous percolation field of a phenolic resin can be disturbed: a change in composition changes the diffusivity of the hydrophilic phase (and that is the case in methylation and in other operations which directly affect the percolation parameter), or there can be a change in the volume fractions of the hydrophilic and hydrophobic phases. That is what happens when inhibitors are added to the systems. The recognition of these distinct mechanisms has clarified our understanding of the dissolution behavior of amphiphilic solids and has removed a troubling inconsistency from our earlier work.

Summary

Removal of hydrophilic sites from the percolation field cannot account for the large inhibition factors routinely observed in commercial resists. To produce effects of that magnitude, the resin must in some way cooperate with the inhibitor. A possible mechanism for such cooperation is hydrogen bonding between the carbonyl groups of the inhibitors and the hydroxyls of the phenolic resin. Hydrogen bonding is an interaction between just one hydrogen donor and a single hydrogen acceptor, but the large size of the inhibition effect demands that the effect of an inhibitor be relayed to a whole neighborhood of phenols. The dual nature of the OH group furnishes a possible relay mechanism. OH groups can function as both hydrogen bond donors and hydrogen acceptors and form hydrogen-bond clusters in solutions of low polarity. Such phenolic clusters are also formed in the coating solutions of novolak films. Cluster sizes in these systems depend on the interaction strength between the components. They are modest in solutions of pure novolak, but when inhibitors are introduced, their stronger acceptor groups (carbonyls) cause the formation of much larger clusters in resist films. The formation of clusters disturbs the randomness of the percolation field, it decreases the site connectivity, and that leads to a lowering of the dissolution rate.

Experimental Section

Materials. All experiments were carried out with a *m*-cresol-based novolak supplied by Hoechst Celanese Corp. The inhibitors (aromatic ketones) were purchased from Aldrich Chemical Co., Inc., and were used as received, except for α-naphthoflavone, which had to be purified by chromatography. The solvents, benzene, carbon tetrachloride, isoamyl acetate, and acetone, were supplied by Aldrich.

Dissolution Rates. Dissolution rates were measured by laser interferometry as described earlier.[1,2]

Infrared Spectra. The spectra of phenolic solutions were taken with an Analect FTIR instrument. The equilibrium constants of hydrogen bonding between the ketonic inhibitors and phenol in dilute benzene solution were derived from changes in the absorbance of the free OH-stretching peak at 3614 cm^{-1} caused by the introduction of increasing quantities of inhibitors. From the data the equilibrium constant was calculated using the expression

$$K_A = \frac{(1-x)}{x} \frac{1/[P]_c}{[A]_0/[P]_0 + x - 1}$$

Here $[P]_0$ is the initial concentration of phenol (0.01 M), $[A]_0$ is the original concentration of the acceptor ketone (0.01−0.2), and x is the ratio $x = [P]/[P]_0$. To obtain IR spectra of resist films, these were cast from isoamyl acetate solutions onto NaCl pellets.

Viscosities. These were measured at 25 °C with a set of Cannon-Manning semimicro viscometers supplied by the Cannon Instrument Co., State College, PA.

Glass transition temperatures were determined from SCM traces in the usual way.

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