BUTTERWORTH HEINEMANN

Polymer Vol. 36 No. 18, pp. 3511–3521, 1995

0032-3861/95/$10.00 + 0.00

Effect of the inhibitor on the curing of an unsaturated polyester resin

X. Ramis and J. M. Salla*
Laboratori de Termodinàmica de la ETSEIB, Universitat Politècnica de Catalunya, Diagonal 647, Barcelona 08028, Spain
(Received 14 June 1994; revised 19 December 1994)

Differential scanning calorimetry has been used to study the influence of hydroquinone as an inhibitor on the curing of an unsaturated polyester resin using benzoyl peroxide as an initiator. The isothermal kinetic analysis was performed by isoconversional adjustment $\ln t = A + E/RT$. This adjustment makes it possible to predict the variations in the activation energy with the degree of conversion without the need to know the rate equations. The results obtained through this adjustment were compared with those obtained by applying an autocatalytic model. The kinetic parameters obtained by both methods permitted the simulation of the curing process inside and outside the experimental range of temperatures. From the experimental results and the kinetic analysis, it was possible to elucidate the dual mechanism of inhibition and retardation exerted by hydroquinone. With the knowledge of the inhibition mechanism, we proposed a method for using the induction times to determine the kinetic parameters associated with the decomposition of the initiator and/or the instantaneous inhibitor content in unsaturated polyester resins.

(Keywords: unsaturated polyester resin; inhibition; curing kinetics)

INTRODUCTION

It is well known that the addition of certain substances may prevent the polymerization of monomers and unsaturated resins. These substances react with the radicals of initiation and/or propagation, becoming non-radical species or radicals of very low reactivity. Substances that cause a great decrease in the polymerization rate when added in small quantities can be divided into two types, according to their effectiveness, as inhibitors or retarders. Inhibitors neutralize all the free radicals, whether they come from the initiator, the active centres of the prepolymer chains or the monomer. The polymerization is completely stopped until the inhibitor has been consumed. Retarders are less effective than inhibitors, and only neutralize a fraction of the radicals. In this case, polymerization may occur, but at a lower rate. Some authors[1,2] relate the mechanism responsible for inhibition to the deactivation of the centres of initiation or to the reduction in the rate at which they are generated, whereas they relate retardation to the interruption of the propagation of the chains. Thus, when the inhibitor has been exhausted, if the inhibition is ideal, the polymerization rate and the polymer formed will be the same as in the absence of the inhibitor. With a retarder the polymerization rate is slower and the polymer formed may have a different length and number of chains.

In general the inhibitor and the reactive species in the reaction medium will compete for the primary radicals from the initiator. If a powerful inhibitor is used, there is complete inhibition up to very low concentrations of inhibitor, and the initiation and propagation of the polymerization are completely arrested. In this case, the induction time before the start of polymerization is the time necessary for the inhibitor initially present in the reaction medium to have been completely consumed by the primary radicals from the initiator[3–5]. Thus, the greater the amount of inhibitor, the greater the induction time. As the reaction of the inhibitor with the radicals of the initiator is very rapid, the induction time will also be proportional to the rate of decomposition of the initiator.

Quinones, such as 1,4-benzoquinone and hydroquinone, are the class of inhibitors most used for unsaturated polyester resins. The mechanism governing the process of inhibition with quinones has been widely studied. It is complex owing to the existence of many reactive processes, the possible regeneration of the inhibitor and the formation of species that may also act as an inhibitor[6]. Novák *et al.*[7] have studied the behaviour of hydroquinone and 1,4-benzoquinone during the curing of unsaturated polyester resins with an organic peroxide as an initiator. They made a polarographic analysis of the changes in the concentration of the inhibitor, studied the curing kinetics and measured the viscosity of the resins during the curing. On the basis of these studies, Novák[8] proposed the following mechanism of inhibition for hydroquinone: Each molecule of hydroquinone reacts with two radicals of the peroxide, forming a molecule of 1,4-benzoquinone. While there is hydroquinone in the reaction medium, the polymerization is completely arrested. When the hydroquinone is exhausted, the curing starts. It occurs at

* To whom correspondence should be addressed

a lower rate than without inhibitor owing to the retarding effect of the 1,4-benzoquinone that is formed and reacts with the propagation radicals of the growing chains.

Though it is desirable for the inhibitor to have a minimum effect on the curing of the resins, in some cases the inhibitor has been found to have an important influence on the curing process and on the properties of the reticulated materials, since it acts as a capturer of radicals, preventing or hindering chain growth. Some studies[8] have shown how the type and amount of inhibitor can modify the reactivity of the resin, the temperature of the mould, the colour, the mechanical properties of the cured material and the degree of conversion. Thus, for example, results of the study of the hydrolysis of cured polyester resins and research into styrene–fumaric acid copolymerization have shown that the length of the crosslinking styrene chains between polyester chains depends on the styrene content and the formulation of the resin, and is a function of the inhibitor[7].

It therefore seems clear that the inhibitor plays an important role in the curing of unsaturated polyester resins, and any study of this process must state clearly the amount and type of inhibitor present in the resin. One of the problems lies in the fact that it is difficult to establish the amount of inhibitor in commercial resins, since it is incorporated during the manufacture of the prepolymer, and is consumed during the manufacture and storage of the resin. This means that the resin evolves, and the amount of inhibitor is variable over time. Owing to the inhibition and retardation, in many cases the polymerization rates observed and the kinetic parameters tabulated in the literature are unreproducible unless the inhibitor and/or retarder have been eliminated before the polymerization, or their exact content is known. For unsaturated polyester resins it is not common to eliminate the inhibitor from the commercial formulations, because of the great tendency of the resin to polymerize. It is therefore essential to know the inhibitor content of the resins at the moment of polymerization.

In the present work we have studied the influence of hydroquinone as an inhibitor on the curing of unsaturated polyester resins catalysed with benzoyl peroxide. From isothermal curings in d.s.c. we have established the curing kinetics by different procedures, which allowed us to elucidate the mechanism of inhibition and retardation of the hydroquinone and show the influence of hydroquinone in the different stages of curing and its kinetic parameters. We also propose a method for using the calorimetric induction times to determine the inhibitor content of the resin and the kinetic parameters associated with the decomposition of the initiator.

THEORETICAL KINETIC ANALYSIS

Isoconversional adjustment ln t = A + E/RT (α = ct)

Assuming that the whole curing process is a single reaction with a single activation energy, then if the Arrhenius law is fulfilled the reaction rate can be expressed as[9]:

$$\frac{d\alpha}{dt} = kf(\alpha) = k_0 \exp\left(-\frac{E}{RT}\right) f(\alpha) \qquad (1)$$

where α is the degree of conversion; k is the rate constant, assumed to depend on the temperature according to the Arrhenius law; $f(\alpha)$ is a function of the degree of conversion; k_0 is the Arrhenius frequency factor; E is the activation energy; R is the universal gas constant; and T is the curing temperature. Considering that for a given degree of conversion $f(\alpha)$ takes the same form regardless of the curing temperature, it is possible to determine the activation energy for a given degree of conversion without needing to know $f(\alpha)$. Reorganizing equation (1) and integrating it between a curing time $t = 0$ where $\alpha = 0$ and a time t with a degree of conversion α, and taking logarithms, for a given degree of conversion α, at constant T, we obtain that:

$$\ln t = A + \frac{E}{RT} \qquad (2)$$

For each degree of conversion, A is a constant that takes the following value:

$$A = \ln\left(\int_0^\alpha \frac{d\alpha}{f(\alpha)}\right) - \ln k_0 \qquad (3)$$

Equation (2) is a linear relation between the logarithm of time necessary to reach a given degree of conversion and the inverse of the curing temperature. By applying equation (2) at a series of temperatures it is possible to determine from the slope of this linear relation the activation energy at different degrees of conversion, and thus to see how the reaction process evolves. This procedure, which has been applied with very good results to study the effect of the promoter in the curing of polyester resins in our previous work[10], allows us to determine the activation energy at different degrees of conversion without knowing the equation $f(\alpha)$, which may vary during the curing process. This method will be used in the present work to show and quantify the effect of the inhibitor in the curing process. The times needed to reach the different degrees of conversion will be determined from isothermal curing in d.s.c. at different curing temperatures. The procedure will also be used to simulate the curing inside and outside the experimental interval of temperatures used to establish the adjustment. In the curing of epoxy resins, some authors[11,12] have used the adjustment of equation (2), applied between two temperatures, to calculate the activation energy and to obtain the master curve for curing time *versus* degree of conversion by displacement of the time.

Autocatalytic model

In the traditional study of curing kinetics it is assumed that the whole reaction process can be assimilated to a single reaction with a single activation energy that remains constant throughout the curing process. The study is based on the isothermal rate equation[13]:

$$\frac{d\alpha}{dt} = kf(\alpha) = k(1-\alpha)^n \alpha^m \qquad (4)$$

where the function $f(\alpha)$ takes a given value for the whole curing process. In the autocatalytic model $f(\alpha) = (1-\alpha)^n \alpha^m$, where n and m are the so-called reaction orders. This model can be used[9] when the reactions have an induction time and when the maximum reaction rate appears between 30 and 40% of the conversion, as happens in most curing reactions of unsaturated

polyester resins. In this procedure the isothermal curing is followed in d.s.c. at several temperatures. Then the experimental rate equation is determined, i.e. the degree of conversion α and the reaction rate $d\alpha/dt$ according to the time, for the whole reaction. The reaction mechanism is adjusted, and the goodness of the adjustment of the experimental results is checked with a kinetic equation. Thus, for each temperature we obtain the reaction orders and the rate constant. Finally, using the Arrhenius equation for the dependence of the rate constant with the temperature, it is possible to determine the activation energy and the frequency factor. This method will also be used to show the influence of the inhibitor in the curing process and to simulate the curing. For obtaining the reaction orders and the rate constant, the experimental degrees of conversion and reaction rates will be adjusted using a multilinear regression. The results obtained by this procedure will be compared with those obtained by the previous isoconversional adjustment. The most important difference between the two procedures is that in isoconversional adjustment it is not necessary to know the equation $f(\alpha)$. Furthermore, this may vary during the curing process, whereas in autocatalytic model a single rate equation that remains constant throughout the whole curing process is assumed.

EXPERIMENTAL

Materials

The unsaturated polyester resin (UPE) used contains phthalic anhydride, maleic anhydride and propylene glycol in a molar ratio of 3 : 2 : 5 determined by ^{1}H n.m.r. The number-average molecular weight of the UPE was 1696 g mol^{-1} and the equivalent molecular weight per mole C=C was 465 g mol^{-1}. The UPE was supplied by Reposa under the commercial name Estratil A-228, with 35% styrene as a crosslinking agent and between 70 and 125 ppm of hydroquinone as an inhibitor, which was not eliminated. As an initiator a dispersion of benzoyl peroxide (BP) was used, with a peroxide content of 50%, supplied by Akzo Chemie under the commercial name Lucidol BW-50T. Merck hydroquinone (HQ) was used. All the samples contained 2% initiator. The inhibitor content added to the commercial formulation varied between 0 and 0.13%, according to the experiment. All the weight percentages of initiator and inhibitor in the work are in parts per hundred with respect to the resin + styrene system.

Differential scanning calorimetry

The calorimetric measurements were made with a Mettler DSC 30 calorimeter. The isothermal curing was performed in the temperature range 50–80°C in a nitrogen atmosphere. The weight of the samples was 20 mg. In the chosen temperature range there was evidence that thermal polymerization did not take place and that the initiation was due exclusively to the thermal decomposition of the initiator. The curing times were 120 to 2500 min, depending on curing temperature and inhibitor content. The calorimetric curve returned to the baseline at the end of each experiment, so the material could only continue curing at a very low rate not detected by the calorimeter. After the isothermal curing, a dynamic scan of 0 to 250°C was made at a heating rate of 10 K min^{-1} to determine the residual heat. Several dynamic curings were also made at 0 to 250°C and at heating rates of 2.5–10 K min^{-1} to determine the total reaction heat.

RESULTS AND DISCUSSION

Thermograms

The signal was divided into unit weight for the purposes of comparison and to standardize the thermograms. As an example, *Figure 1* shows the isothermal d.s.c. thermograms at different temperatures for the samples cured with 0.025% of added HQ. The kinetic effect of the temperature can be observed. At higher temperatures the induction, peak and curing times are

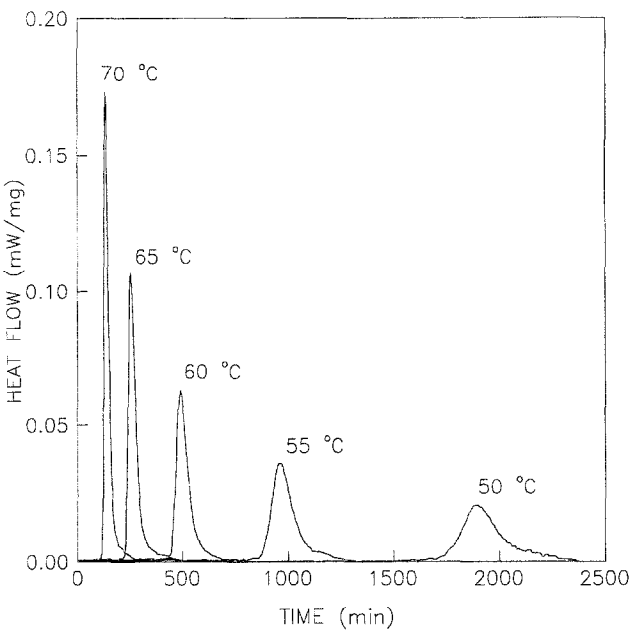

Figure 1 Isothermal d.s.c. thermograms for UPE samples cured with 0.025% of added HQ

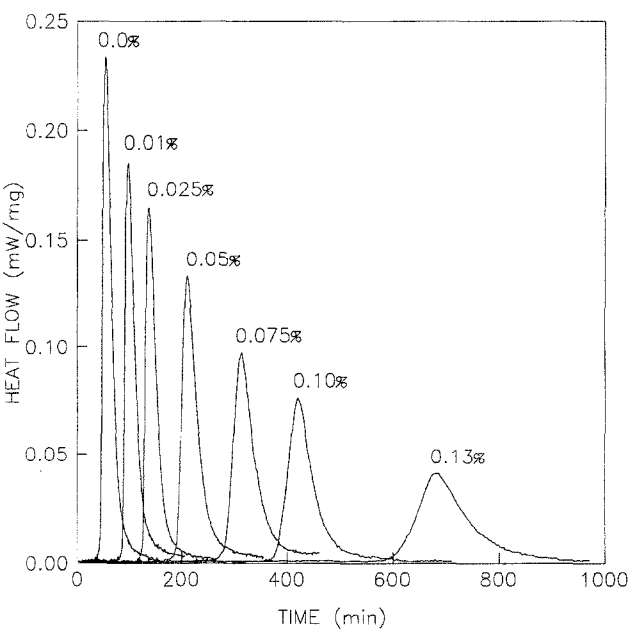

Figure 2 Isothermal d.s.c. thermograms for UPE samples cured at 70°C with different amounts of added HQ

lower and the reaction rates higher. To show the important effect of HQ on the curing process, *Figure 2* compares the isothermal thermograms at 70°C corresponding to the curing with different amounts of added HQ as indicated. We can clearly see how the HQ influences the induction time and the reaction rate. The greater the amount of inhibitor, the greater the induction time and the lower the curing rate, since the inhibition effect is more pronounced. The fact that, once the process has started, the HQ content not only affects the induction time but also the curing rate may indicate the existence of the following double mechanism of inhibition and retardation. During the induction time the HQ reacts with the primary radicals from the initiator, inhibiting the curing. While there is HQ in the reaction medium, the curing is completely stopped. When the HQ is exhausted, the curing starts, but it is slower than in the absence of HQ. This may be due to the retarding effect of some species formed during the induction time owing to a reaction between the HQ and BP, which reacts with the propagation radicals. The existence of this double mechanism of inhibition and retardation would be in accordance with the inhibition mechanism of the HQ proposed by Novák[8] and discussed above, where the species formed during the induction time would be 1,4-benzoquinone.

Determination of the decomposition constant of the BP

If we accept that the inhibitor is completely consumed during the induction period and that the polymerization reaction does not begin until the inhibitor is exhausted due to the reaction with the primary radicals of the initiator, it is easy to relate the consumption of initiator, the amount of inhibitor present, the decomposition constant of the initiator and the induction time. In support of this, some authors[14,15] have found that during the induction time the concentration of free radicals, measured by electron spin resonance (e.s.r.), is constant owing to the balance between the radicals generated by the initiator and those rapidly consumed due to the inhibition effect. When the inhibitor is exhausted, the curing starts and the concentration of free radicals in the reaction medium increases.

Free radicals are generated by thermal decomposition of the initiator according to:

$$I \xrightarrow{k_d} 2R\cdot \qquad (5)$$

where I represents the peroxide initiator, R· the free radicals formed and k_d is the decomposition constant of the initiator, which governs the previous process. The factor 2 in equation (5) refers to the formation of two free radicals for each decomposed molecule of initiator.

The rate of disappearance of the initiator can be expressed as:

$$\frac{d[I]}{dt} = -k_d[I] \qquad (6)$$

Reordering the above equation and integrating between the time at which the initiator is added $t = 0$ and the induction time t_i, we obtain:

$$[I] = [I]_0 \exp(-k_d t_i) \qquad (7)$$

where [I] represents the concentration of initiator at a given moment, in our case at the induction time, and $[I]_0$ represents the initial concentration of initiator at zero time when nothing has yet decomposed.

If each molecule of inhibitor reacts with two free radicals of the initiator (which seems logical in the case of HQ owing to the existence of two hydroxyl groups in the molecule), for each molecule of initiator consumed one molecule of inhibitor is used. Then, the amount of initiator consumed until the inhibitor is completely exhausted is equal to the initial inhibitor content and may be calculated by the difference between the initial initiator and the initiator existing at the induction time, expressed according to equation (7):

$$[Z]_0 = [I]_0 - [I] = [I]_0\{1 - \exp(-k_d t_i)\} \qquad (8)$$

where $[Z]_0$ is the initial inhibitor concentration. It is interesting to note how equation (8) relates in a simple way to the initial initiator and inhibitor concentrations with the decomposition constant of the initiator and with the induction time. If we know three of these parameters, it is always possible to determine the fourth.

As we have said, in many cases it is difficult to know the instantaneous amount of HQ in the commercial formulations. With the decomposition constant of the peroxide initiator, the initial peroxide concentration and the induction time determined by calorimetry, by applying equation (8) it is possible to estimate the amount of HQ in the formulation. Though the decomposition constants of the peroxides are generally tabulated, they have been obtained in different conditions to those in which the peroxide decomposes during the curing. Several authors[16–18] agree that the values of the decomposition constant of benzoyl peroxide can vary slightly with the solvent used, with the concentration of initiator and with the presence of inhibitor. Therefore, we first used equation (8) to calculate the decomposition constant k_d of the peroxide from samples cured at 70°C, adding high amounts of inhibitor. Thus, the effect of the inhibitor in the commercial resin is not very important compared with the effect of the added inhibitor. We assumed that the HQ content of the samples is the perfectly known amount added, plus 70 ppm, which is the minimum content of the commercial resin. The induction times were calculated from the experimental thermograms, corresponding to curing at 70°C (*Figure 2*). *Table 1* gives a summary of the data used and the results obtained from this calculation. We can see how the k_d of the BP hardly varies with the amount of hydroquinone used, and its value is very close to those tabulated for most conventional solvents[18]. This allows us to confirm that the inhibition mechanism assumed for the HQ and used to deduce equation (8) is correct.

Determination of the HQ content in the commercial resin

Accepting, as we have seen, that the k_d of the BP does not vary with the HQ content, it is now possible to use the constants calculated to estimate the HQ content in the

Table 1 Decomposition constants at 70°C of benzoyl peroxide calculated by equation (8)

HQ (%)	BP (%)	t_i (min)	k_d (s^{-1})
0.138	1.0	540	1.14×10^{-5}
0.108	1.0	375	1.20×10^{-5}
0.083	1.0	282	1.19×10^{-5}
0.057	1.0	184	1.21×10^{-5}

commercial resin, when no additional HQ has been added.

Taking an average value for the k_d at 70°C of $1.18 \times 10^{-5}\,s^{-1}$, by applying equation (8) we calculated the initial HQ content in the commercial resin. The induction time was obtained from the d.s.c. thermogram for the curing of the commercial resin at 70°C without adding HQ. We calculated an initial HQ content of 0.01% (equivalent to 100 ppm) in the commercial resin. Though in the present work we will refer always to HQ contents as those added to the commercial formulation, it must be borne in mind that in all cases this content must be increased by 0.01% for the HQ in the resin.

From the above analysis it is deduced that, from the calorimetric induction time and the initial initiator and inhibitor content, it is possible to calculate the decomposition constant of the BP, which apparently shows no important variation with the HQ content. Furthermore, if we know the k_d of the peroxide, from the initial peroxide content and the induction time it is possible to calculate the initiator consumed during the induction time, which for certain systems coincides with the initial inhibitor content. This last method may be employed generally to calculate the HQ content of commercial resins.

Degree of conversion α and reaction rate dα/dt

Assuming that the curing process may be studied through the thermal effect that it produces, the reaction advance will be proportional to the heat released. The maximum degree of conversion will be reached when all the links that may do so have reacted, and the reaction rate will be proportional to the calorimetric signal.

It has been found experimentally that the material does not undergo complete isothermal curing, since it is possible to detect a residual heat in a dynamic postcuring. It has also been found that the sum of the isothermal heat (ΔH_{iso}) and the residual heat (ΔH_{res}) takes a value near to $300\,J\,g^{-1}$ for any HQ content, and that it is always lower than the heat obtained in dynamic experiments. The fact that the sum of the isothermal heat and the residual heat is lower than the dynamic heat may be attributed to the fact that isothermally part of the heat cannot be registered by the calorimeter at the start and end of the reaction because it falls below the sensitivity of the device when working at low temperatures, and to the fact that part of the heat is released during the stabilization time of the d.s.c., when it is working at high temperatures[19,20]. Bearing in mind these considerations, as explained in our previous article[10], we calculated the degrees of conversion and the reaction rates using the following expressions:

$$\alpha_t = \frac{\Delta H_t(\Delta H_{tot} - \Delta H_{res})}{\Delta H_{iso}\Delta H_{tot}} \quad (9)$$

$$\left(\frac{d\alpha}{dt}\right)_t = \frac{(dH/dt)_t(\Delta H_{tot} - \Delta H_{res})}{\Delta H_{iso}\Delta H_{tot}} \quad (10)$$

where ΔH_t and $(dH/dt)_t$ are respectively the isothermal heat released up to a time t and the calorific power generated in a time t. In this analysis we took as the total reaction heat (ΔH_{tot}) a value of $350\,J\,g^{-1}$, calculated as an average value of that obtained dynamically between 0 and 250, at heating rates of between 2.5 and 10 K min^{-1}, since this heat is the maximum that can be determined experimentally. As the isothermal heat cannot be determined completely, ΔH_t and $(dH/dt)_t$ were corrected in equations (9) and (10) bearing in mind the part of the heat that has not been registered isothermally.

Figures 3 and *4* show, for the purpose of comparison, the degrees of conversion against time and the reaction rates against degrees of conversion, calculated by equations (9) and (10), for samples cured at 70°C with the indicated amounts of added HQ. As stated above, the effect of the inhibitor is shown in the induction times and in the reaction rates. The greater the amount of inhibitor, the longer the induction time, and when curing has started it occurs at a lower rate. This again shows the

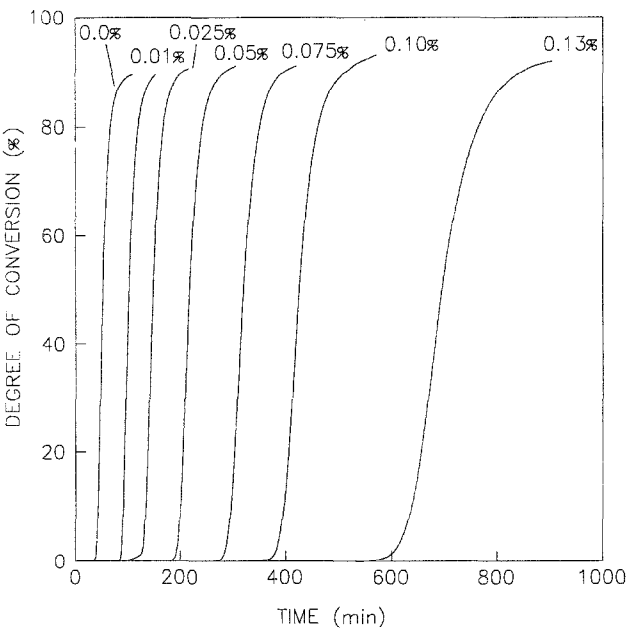

Figure 3 Degrees of conversion against curing times for UPE samples cured at 70°C with different amounts of added HQ

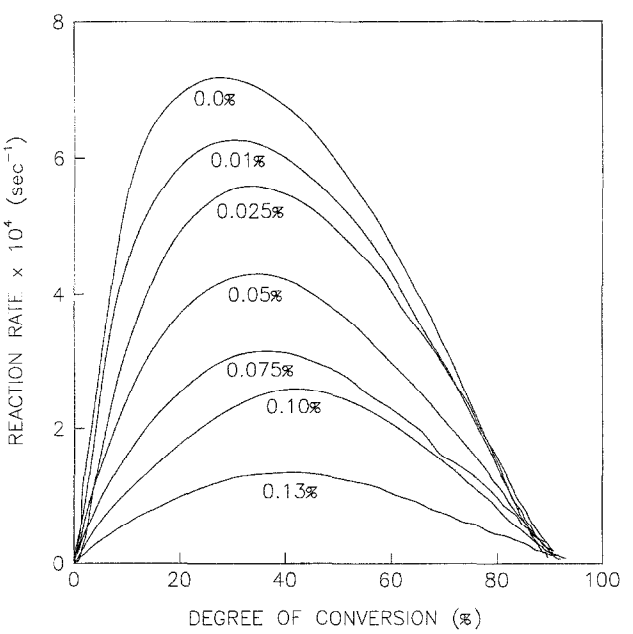

Figure 4 Reaction rates against degrees of conversion for UPE samples cured at 70°C with different amounts of added HQ

existence of a double mechanism of inhibition and retardation. We can also see how the last degree of conversion reached isothermally is practically constant, regardless of the inhibitor content used.

Isothermal kinetic analysis by adjustment $ln\,t = A + E/RT\ (\alpha = ct)$

For the isothermal kinetic analysis of the curing process with different amounts of HQ as the inhibitor, the adjustment given by equation (2) will be applied at different degrees of conversion. Though A is a complex constant related to the kinetic equation according to equation (3), we will now describe how in the induction time, where the degree of conversion is zero, A has a very simple expression related to the initial initiator and inhibitor content.

By taking equation (8), which gives us the amount of inhibitor in the resin or the consumption of initiator during the induction time (which is the same), and clearing the induction time, we obtain:

$$k_d t_i = -\ln\left(\frac{[I]_0 - [Z]_0}{[I]_0}\right) \qquad (11)$$

If the decomposition constant of the initiator follows the Arrhenius equation $k_d = k_{0d}\exp(-E_d/RT)$, by replacing this in equation (11), reordering and taking logarithms, we obtain:

$$\ln t_i = \ln\left(\frac{-\ln\{([I]_0 - [Z]_0)/[I]_0\}}{k_{0d}}\right) + \frac{E_d}{RT} \qquad (12)$$

where k_{0d} and E_d are respectively the frequency factor and the activation energy associated with the decomposition of the peroxide initiator, t_i is the induction time ($\alpha = 0$), and $[I]_0$ and $[Z]_0$ are the initial initiator and inhibitor concentrations respectively.

If we compare equation (12) with equation (2) applied to the induction time, when the degree of conversion is zero, we can clearly see how the constant A is related simply to the frequency factor of the decomposition process of the peroxide and to the initial initiator and inhibitor contents by the following equation:

$$A = \ln\left(\frac{-\ln\{([I]_0 - [Z]_0)/[I]_0\}}{k_{0d}}\right) \qquad (13)$$

For the kinetic analysis of the curing process using three different amounts of HQ, we used equation (2) applied to different degrees of conversion. The curing times were obtained from the curves of degree of conversion vs. curing time. These curves were calculated from the isothermal curing in d.s.c. by application of equation (9). *Figure 5* gives an example of the experimental curves for degree of conversion vs. time at different curing temperatures for the system where 0.025% HQ has been added to the commercial formulation. For each degree of conversion, by equation (2) we correlated the logarithm of curing time to the inverse of curing temperature. *Figure 6* shows the curves of $\ln t$ vs. $1/T$ for the same system. From these curves the constant A can be determined as the ordinate at the origin, and from the slope the activation energy can be determined. Results similar to those shown in *Figures 5* and *6* were obtained for all the formulations studied.

Table 2 shows the activation energies and the constants A calculated from the adjustments $\ln t = A + E/RT$ according to the degree of conversion for three systems using different amounts of HQ as inhibitor. In all cases, the adjustment had a regression coefficient of $0.99 < r < 1.00$. *Figure 7* shows the activation energy according to the degree of conversion for the three systems.

As is confirmed by the good regression coefficients obtained, *Figure 6* shows the existence of a linear dependence between $\ln t$ and $1/T$ in the range of temperatures tested for all the degrees of conversion and for any amount of HQ used. As we had supposed,

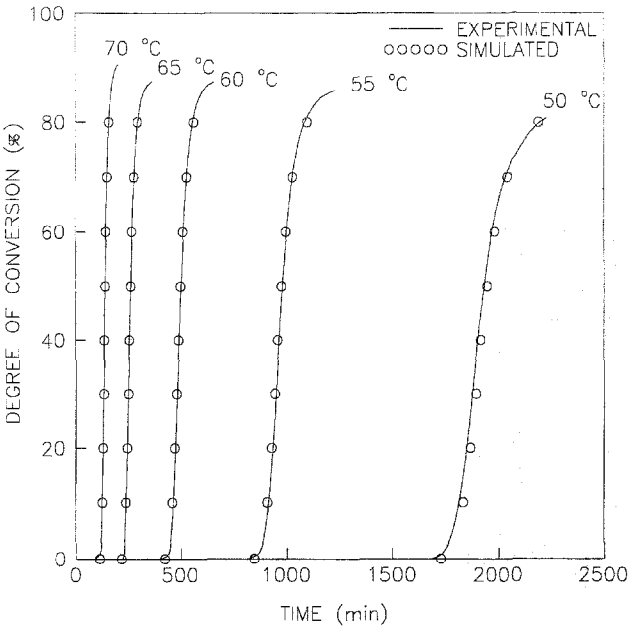

Figure 5 Experimental and simulated curves by adjustment $\ln t = A + E/RT$ for degree of conversion against curing time for UPE samples cured with 0.025% of added HQ

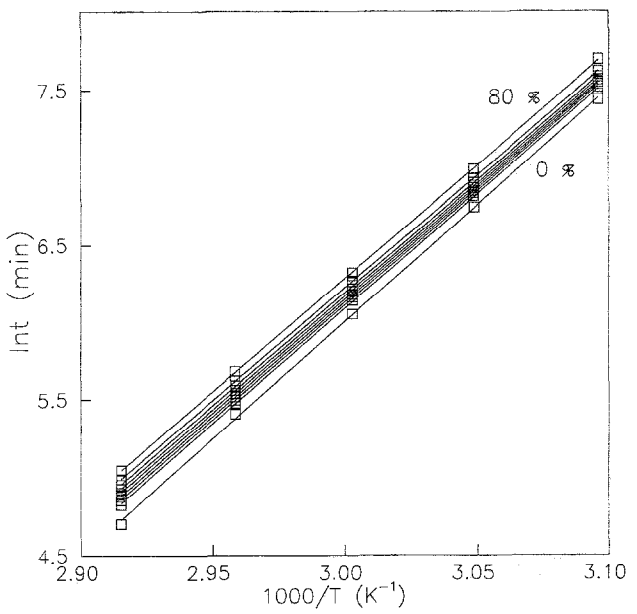

Figure 6 Correlation of the logarithm of time against inverse of temperature for different degrees of conversion according to the equation $\ln t = A + E/RT$ for UPE samples cured with 0.025% of added HQ. Conversion levels are 0, 10, 20, 30, 40, 50, 60, 70 and 80% (upwards)

Table 2 Kinetic parameters at different degrees of conversion obtained by adjustment according to the equation $\ln t = A + E/RT$ from isothermal curing with different amounts of added HQ

	0.0% HQ		0.01% HQ		0.025% HQ	
α (%)	E (kJ mol^{-1})	A	E (kJ mol^{-1})	A	E (kJ mol^{-1})	A
0	126.4	−40.75	126.6	−39.95	126.0	−39.45
10	121.4	−38.80	121.8	−38.10	123.4	−38.44
20	120.7	−38.52	121.3	−37.92	123.1	−38.30
30	119.0	−37.86	120.7	−37.66	122.7	−38.14
40	117.6	−37.30	120.0	−37.97	122.2	−37.94
50	116.5	−36.90	119.5	−37.18	121.8	−37.77
60	115.5	−36.46	118.6	−36.85	121.4	−37.61
70	114.9	−36.21	117.7	−36.47	121.2	−37.49
80	116.2	−36.58	117.9	−36.48	121.8	−37.67

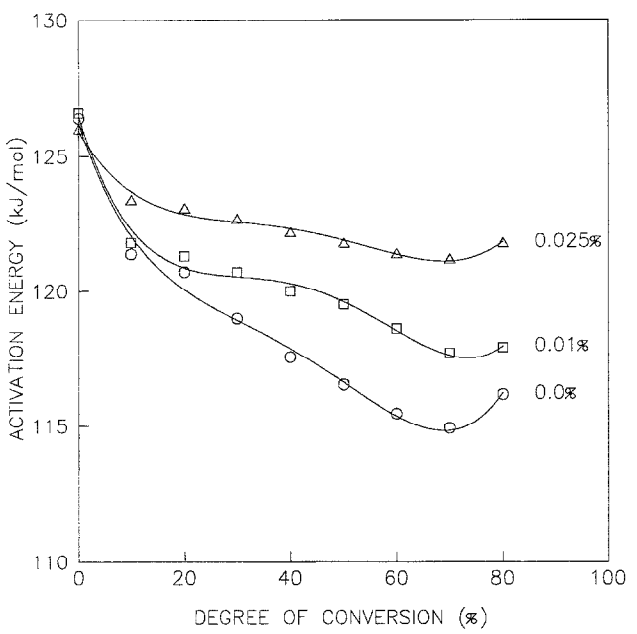

Figure 7 Activation energies against degree of conversion for three curing systems with different amounts of added HQ

this indicates that for a given degree of conversion and hydroquinone content the reaction takes place according to a uniform mechanism regardless of the curing temperature.

Table 2 and *Figure 7* show how the activation energy varies not only with the degree of conversion but also with the HQ content, though this variation is slightly more pronounced when the HQ content is lower. For any amount of HQ the activation energy takes a maximum value at the start of the reaction, then decreases and finally, after reaching a minimum, shows a certain tendency to increase. This behaviour, already observed in previous works[10], has been explained by the autocatalytic effect of the curing process when the reaction has started, and by the phenomena of gelling and vitrification and the increase in viscosity when the degree of conversion increases. The decrease in the activation energy has been mainly attributed to the autocatalytic effect, which increases the reaction rate owing to an increase in the free radicals in the reaction medium. The increase in the activation energy in the final parts of the curing can be attributed to vitrification, the increase in viscosity and the exhaustion of the reactive species, which will tend to decrease the reaction rate. It is also found that when the degree of conversion increases, the decrease in the activation energy is lower when the HQ content is greater. This can be explained by the fact that the more HQ there is, the lower is the effect of autoacceleration (*Figure 4*), since many propagation radicals are consumed by the inhibitor, and there are therefore fewer free radicals in the reaction medium. In general, for each degree of conversion (except $\alpha = 0$) we observe that the activation energy increases slightly when the inhibitor content increases. This may explain the decrease observed in the curing rate when the inhibitor content increases (*Figure 4*). As we have said, the inhibitor reacts with the propagation radicals and leads to there being fewer free radicals in the reaction medium and the reaction being retarded at a lower rate with a greater activation energy.

If the mechanism assumed for the inhibition of the HQ is correct, a comparison between equations (2) and (12) shows that the activation energy at the induction time ($\alpha = 0$) must be equal to the activation energy associated with the decomposition of the peroxide, since at this moment the curing has not yet started and the only process that has taken place is the decomposition of the peroxide and its reaction with the HQ. *Table 2* shows that the activation energy obtained at the induction time ($\alpha = 0$) is practically constant with the inhibitor content, and its value is consistent with the values tabulated for the decomposition of the BP[18]. The results obtained for the activation energy, its independence on the HQ content at zero degree of conversion and its increase during curing when the HQ content increases, all confirm the correctness of the mechanism of inhibition and retardation proposed for the hydroquinone.

By application of equation (13), from factor A calculated at the induction time ($\alpha = 0$) it is possible to determine the frequency factor associated with the decomposition of the initiator k_{0d} if we know the initial initiator and inhibitor content. *Table 3* shows the k_{0d} of the BP calculated from the constants A for $\alpha = 0$ and the activation energies at the same conversion for the three systems studied. The inhibitor content in the samples was calculated as that added plus that estimated to be contained in the commercial resin, which was 0.01%. The activation energy and the frequency factors do not show an important variation with the inhibitor content. As stated above, this indicates that they are characteristic

Table 3 Activation energies and frequency factors for the decomposition of the benzoyl peroxide calculated by equations (12) and/or (13) of samples cured with different amounts of added HQ

HQ (%)	E_d (kJ mol^{-1})	k_{0d} (s^{-1})
0.0	126.4	1.85×10^{14}
0.01	126.6	1.69×10^{14}
0.025	126.0	1.81×10^{14}

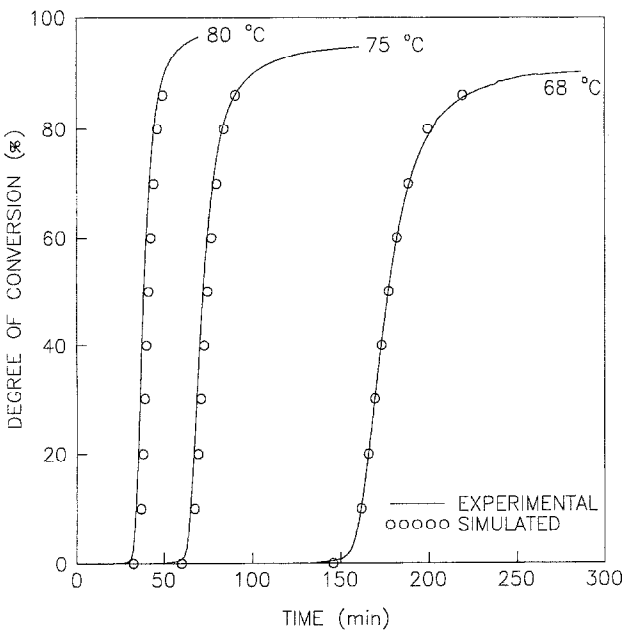

Figure 8 Experimental curves and curves simulated by extrapolation of the adjustment $\ln t = A + E/RT$ for degree of conversion against curing time for UPE samples cured with 0.025% of added HQ

parameters of the initiator system that do not depend on the amount of inhibitor used. Muzumdar and Lee[21] obtained similar results by application of the adjustment $\ln t = A + E/RT$ to the gel time ($\alpha < 0.1\%$). For two different inhibitors and initiators they found that the activation energy did not vary with the inhibitor content, and was characteristic of the initiator system.

If we know the activation energy and the frequency factor associated with the decomposition of peroxide, and these parameters do not vary significantly with the initiator and inhibitor content, equation (12) can be used to calculate the induction time of any resin at a given temperature for any amount of inhibitor and initiator, without the need for experimental measurements.

Simulation by adjustment ln t = A + E/RT (α = ct)

Owing to the good linearity obtained for all the formulations used in the curves of $\ln t$ vs. $1/T$ summarized in *Table 2*, it is possible to use them to calculate for a given temperature the time necessary to reach a given degree of curing, and thus simulate the curves for degree of conversion vs. curing time. For all the formulations used we have obtained an excellent agreement between these experimental curves and those simulated. *Figure 5* shows an example of the experimental curves used to obtain the adjustment, and the curves simulated by this adjustment for the system where 0.025% HQ has been added to the commercial formulation. *Figure 8* shows the same curves as in *Figure 5* for the same system, but for curing temperatures different from those used to deduce the adjustment. It is interesting to observe how, outside the experimental interval of temperatures where it was necessary to extrapolate the adjustments $\ln t = A + E/RT$, the agreement is still good, which indicates that for a given degree of conversion the mechanism still remains invariant up to higher temperatures. Similar simulation results were obtained for the other two formulations studied.

Isothermal kinetic analysis by autocatalytic model

Though from the isoconversional adjustment it has been shown that the activation energy, and therefore the reaction mechanism, can vary not only with the HQ content but also with the degree of conversion, it may be interesting to calculate the kinetic parameters by the autocatalytic model, where we assume a single reaction mechanism that remains constant during the whole curing process, to see whether they are sensitive to the inhibitor content and to compare them with those obtained by isoconversional adjustment $\ln t = A + E/RT$.

Table 4 summarizes the kinetic parameters obtained by the autocatalytic model. It shows the rate constants and the reaction orders corresponding to curing at different temperatures with different amounts of HQ added as an inhibitor. *Figure 9* shows a sample comparison of the curves of reaction rate vs. degree of conversion obtained experimentally and those predicted according to autocatalytic adjustment for the system in which 0.025% HQ has been added as an inhibitor. We can see a good agreement between the experimental and predicted curves. Similar results were obtained for the other two systems tested. In all cases the autocatalytic adjustment had a good regression coefficient ($0.99 < r < 1.00$).

Table 4 shows how the rate constant k varies with the

Table 4 Kinetic parameters at different curing temperatures obtained by adjustment according to an autocatalytic model for three systems with different amounts of added HQ

	0.0% HQ				0.01% HQ				0.025% HQ			
T (°C)	$k \times 10^4$ (s^{-1})	n	m	$n+m$	$k \times 10^4$ (s^{-1})	n	m	$n+m$	$k \times 10^4$ (s^{-1})	n	m	$n+m$
70	54.7	2.01	1.03	3.04	43.1	2.03	0.98	3.01	37.6	1.78	0.97	2.75
65	33.3	2.14	0.93	3.07	25.2	2.02	0.96	2.98	22.8	1.93	0.98	2.91
60	19.8	2.19	0.80	2.99	15.1	2.04	0.93	2.97	13.9	1.82	1.06	2.88
55	11.6	2.19	0.82	3.01	8.9	1.83	0.98	2.81	7.8	1.84	1.09	2.93
50	6.8	2.26	0.81	3.07	5.3	1.82	1.07	2.89	4.7	1.98	1.11	3.09

temperature and with the amount of inhibitor. For the three formulations, as the curing temperature increases, so does k. This reveals the kinetic effect of the temperature, which increases the reaction rate when it increases. At a given temperature the rate constant decreases when the HQ content increases, which is in accordance with the decrease in the reaction rate observed when the hydroquinone content increases (*Figure 4*). Though the reaction orders do not remain strictly constant, their variation with the temperature and with the inhibitor content is too small to be attributed to a change in mechanism, and must be attributed to fluctuations due to the adjustment.

The activation energy and the frequency factor were determined respectively from the slope and from the ordinate at the origin of the linear representation of $\ln k$ against $1/T$, assuming that the rate constant depends on the temperature according to the Arrhenius law:

$$\ln k = \ln k_0 - \frac{E}{R}\left(\frac{1}{T}\right) \qquad (14)$$

A good linearity ($0.99 < r < 1.00$) was obtained in all cases. *Table 5* presents the activation energies and frequency factors calculated from the Arrhenius equation (14) for the three formulations. We can see how the activation energy, calculated by the autocatalytic model, is practically constant with the inhibitor content, whereas the frequency factor decreases slightly when the hydroquinone content increases. This allows us to justify the decrease in the reaction rate and in the rate constant observed (*Figure 4* and *Table 4*) when the HQ content increases.

Table 5 Activation energies and frequency factors calculated by autocatalytic model for samples cured with different amounts of added HQ

HQ (%)	E (kJ mol^{-1})	k_0 (s^{-1})
0.0	96.2	2.41×10^{12}
0.01	96.4	2.05×10^{12}
0.025	95.8	1.44×10^{12}

Simulation by autocatalytic model

It was found that the reaction orders calculated by autocatalytic adjustment did not show an important variation with the curing time. If we suppose that the constancy of the reaction orders and the linearity of the Arrhenius equation are maintained inside and outside the experimental temperature interval, by autocatalytic adjustment it is possible to simulate the curing for other temperatures different from those used to obtain the constants and the reaction orders. Bearing in mind these considerations, we simulated the curing by the autocatalytic model. The rate constants were calculated by the Arrhenius equation, and for the reaction orders we took constant values calculated as the average value of those obtained at different temperatures (*Table 4*).

Figure 10 compares the curves of reaction rate vs. degree of conversion simulated at different temperatures by autocatalytic adjustment with those obtained *a posteriori* experimentally by d.s.c. curing for the system in which we added 0.025% HQ inhibitor.

In general, there is a correct adjustment between the calculated curves and the experimental ones. As was to be expected, within the temperature interval used to deduce the kinetic parameters ($T = 68°C$) the agreement is good. It is interesting to see how outside this interval ($T = 75$–$80°C$), where it was necessary to extrapolate the Arrhenius equation and assume constants of the reaction orders, the agreement is still acceptable, which indicates that the mechanism remains invariant up to higher temperatures. Equivalent results have been obtained for the other two systems studied.

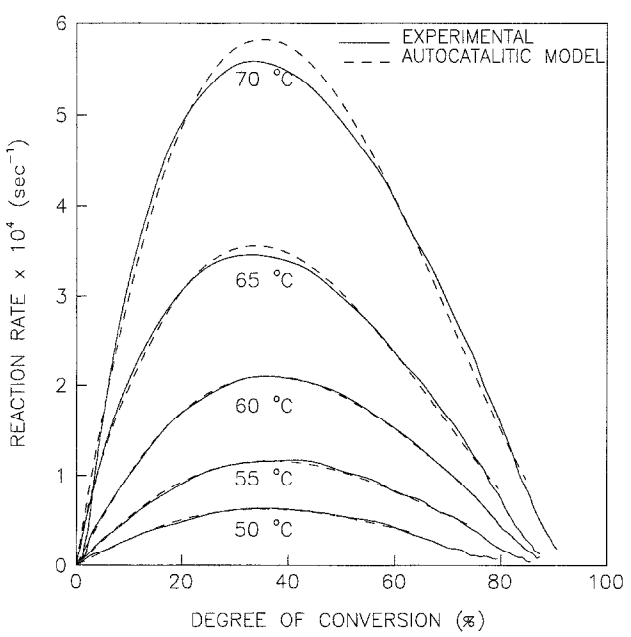

Figure 9 Experimental curves and curves predicted by autocatalytic adjustment for reaction rate against degree of conversion for UPE samples cured with 0.025% of added HQ

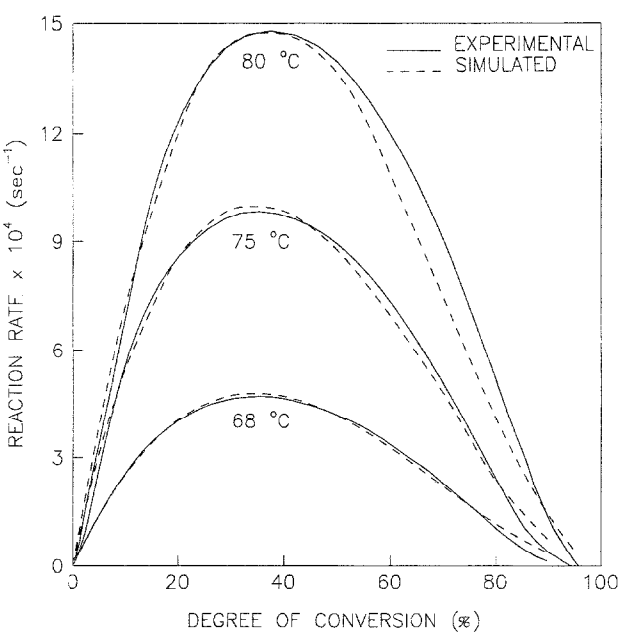

Figure 10 Comparison between the experimental curves for reaction rate against degree of conversion and curves simulated by autocatalytic adjustment for UPE samples cured with 0.025% of added HQ

The autocatalytic adjustment and the isoconversional adjustment $\ln t = A + E/RT$ give different activation energy values: in the first case a single activation energy describes the process, whereas in the second the activation energy varies with the degree of conversion. However, in both cases it is possible to describe the curing correctly and simulate the curing inside and outside the experimental interval of temperatures with good results (*Figures 5*, *8*, *9* and *10*). As the methods describe the same curing process, according to the method used the differences between the activation energies must be compensated by the other kinetic parameters of adjustment. This means that mathematically the two adjustments may be correct even if they have different activation energy values.

The two methods used describe the curing process, but do so in a different way. Besides serving to describe the whole process, the adjustment $\ln t = A + E/RT$ can be applied to the induction time, which allows us to characterize the processes that have taken place during this time before the curing starts. Depending on the information available, it also allows us to calculate the inhibitor content of the commercial resin, or the decomposition constant of the initiator, or the initiator consumed during the induction, or to predict the induction time. Furthermore, the method may be applied to each degree of conversion, reflecting the different stages through which the curing evolves. On the other hand, the autocatalytic model only allows us to describe the curing once it has started, but what happens during the induction time cannot be described. Moreover, in the autocatalytic model we assume that the reaction mechanism remains constant throughout the whole curing process, when it has been shown that this is not the case, and the mechanism varies as the conversion increases. In the autocatalytic model we have assumed a given equation $f(\alpha)$, which in reality does not necessarily describe the curing exactly. In the autocatalytic model we use many parameters of adjustment, and it may happen that different groups of parameters lead to the same result. Therefore, it is very difficult to give a physicochemical interpretation to the parameters obtained by the autocatalytic model, and these must simply be taken as parameters of mathematical adjustment. In the case of the adjustment $\ln t = A + E/RT$ this does not happen, since no assumptions are made about the mechanism governing the process.

Though in certain cases the autocatalytic model may be useful in the curing of unsaturated polyester resins, in general the adjustment $\ln t = A + E/RT$ shows better and wider results, since it reflects the whole reaction process (from the instant at which the initiator is added) without needing to make assumptions about the equation for $f(\alpha)$. Furthermore, as we have seen, the activation energies calculated by this adjustment show perfectly the inhibitor effect of the whole curing process.

CONCLUSIONS

By d.s.c. analysis we have found that hydroquinone inhibits and retards the curing process of a polyester resin with initiator, but does not have an important effect on the final degree of conversion reached. The greater the content of hydroquinone, the greater is the induction time and the lower the curing rate.

The studies performed by d.s.c. and the kinetic parameters calculated by different procedures have allowed us to elucidate the existence of a double mechanism of inhibition and retardation in the curing of polyester resins with initiator and with hydroquinone. In this mechanism, during the induction time the hydroquinone reacts with the primary radicals of the initiator and the polymerization is completely halted. When the hydroquinone is exhausted, the curing starts, but it occurs at a lower rate than in the absence of inhibitor owing to the retarding effect of some species formed during the induction period by reaction between the hydroquinone and the initiator, which reacts with the propagation radicals

If we know the mechanism of inhibition of the hydroquinone it has been seen that the induction time, the decomposition constant of the initiator and the initial concentrations of initiator and inhibitor are related simply by equation (8). If we know three of these parameters, it is always possible to determine the fourth. We thus calculated a decomposition constant of the BP at 70°C near to $1.18 \times 10^{-5}\ s^{-1}$, and it was found to be independent of the hydroquinone content. We also estimated an initial inhibitor content of 0.01% in the commercial resin.

Using the adjustment $\ln t = A + E/RT$, we found that the activation energy varied not only with the inhibitor content but also with the degree of conversion. The variation in the activation energy during the curing process is attributed to the phenomena of autoacceleration and vitrification, and the increase in viscosity of the medium. The decrease in the activation energy associated with the autoacceleration is lower when the inhibitor content is greater, since the inhibitor hinders the autoacceleration. The increase in activation energy observed when the hydroquinone content increases must be attributed to the decrease in curing rate caused by the HQ.

The use of the adjustment $\ln t = A + E/RT$ at the time of induction when $\alpha = 0$ (equation (12)) can determine the activation energy and the frequency factor for the decomposition of the initiator, since up to this moment the only process that has taken place is the decomposition of the initiator and its reaction with the inhibitor. We therefore found that the activation energy and the frequency factor associated with the decomposition of the BP are not influenced greatly by the inhibitor content.

The adjustment $\ln t = A + E/RT$ obtained over a given interval of temperatures may be applied inside and outside this interval provided that the mechanism remains invariant and the $\ln t$ vs. $1/T$ curves can be extrapolated. The simulation made with this kinetic model shows an excellent agreement both inside the temperature interval used to deduce the adjustments, and outside it, where it was necessary to extrapolate the curves $\ln t = A + E/RT$.

The rate constants calculated by autocatalytic adjustment decrease when the inhibitor content increases and when the temperature decreases. The reaction orders show practically no variation with the inhibitor content or with the temperature, which indicates that the reaction mechanism is invariant. The activation energies calculated by this procedure hardly vary with the inhibitor content, whereas the frequency factors decrease slightly when the HQ content increases.

The simulation made with the autocatalytic model shows an acceptable agreement both inside the interval of temperatures used to deduce the adjustments, and outside it, where it was necessary to extrapolate the Arrhenius equation and assume constant reaction orders.

Though the activation energies determined by the autocatalytic model differ from those calculated at different degrees of curing by adjustment $\ln t = A + E/RT$, both methods serve to describe the curing and the inhibitor effect. The differences between the activation energies obtained must be attributed to the fact that through the autocatalytic model we assume a single reaction mechanism that does not vary with the degree of conversion, whereas in reality we have seen that it can vary. In general, the isoconversional adjustment $\ln t = A + E/RT$ is the one that gives the best results and the most complete description of the curing, since besides showing the effect that the inhibitor exerts on the curing process, it provides a description of what happens during the induction time and shows how the curing evolves. Furthermore, in this method no assumptions are made on the rate equation that governs the curing process.

ACKNOWLEDGEMENT

The research was supported by CICYT under Grant No. MAT93-0343-C02-02.

REFERENCES

1 Bamford, C. H. 'Radical polymerization' in 'Encyclopedia of Polymer Science and Engineering', Vol. 13, Wiley-Interscience, New York, 1988, p. 708
2 Billmeyer, F. W. 'Textbook of Polymer Science', 3rd Edn., Wiley-Interscience, New York, 1984
3 Cuadrado, T. R., Borrajo, J. and Williams, R. J. J. *J. Appl. Polym. Sci.* 1983, **28**, 485
4 Stevenson, J. F. *Polym. Eng. Sci.* 1986, **26**, 746
5 Ng, H. and Manas-Zloczower, I. *Polym. Eng. Sci.* 1989, **29**, 1097
6 Odian, G. 'Principles of Polymerization', 3rd Edn., Wiley-Interscience, New York, 1991, p. 262
7 Novák, J., Wagenknechtová, M. and Mleziva, J. *Angew. Makromol. Chem.* 1984, **126**, 27
8 Novák, J. *Prog. Org. Coatings* 1988, **16**, 231
9 Prime, R. B. 'Thermosets' in 'Thermal Characterization of Polymeric Materials' (Ed. E. A. Turi), Academic Press, New York, 1981, p. 441
10 Salla, J. M. and Ramis, X. *J. Appl. Polym. Sci.* 1994, **51**, 453
11 Barton, J. M. *Br. Polym. J.* 1979, **11**, 115
12 Wisanrakkit, G. and Gillham, J. K. *Adv. Chem. Ser.* 1990, **227**, 143
13 Sesták, J. and Berggren, G. *Thermochim. Acta* 1971, **3**, 1
14 Tollens, F. R. and Lee, L. J. *Polymer* 1993, **34**, 29
15 Hsu, C. P. and Lee, L. J. *Polymer* 1993, **34**, 4516
16 Barnett, B. and Vaughan, W. E. *J. Phys. Colloid Chem.* 1947, **51**, 942
17 Cohen, S. G. *J. Am. Chem. Soc.* 1947, **69**, 1057
18 Masson, J. C. 'Polymer Handbook' (Eds. J. Brandrup and E. H. Immergut), 2nd Edn., Wiley, New York, 1975, p. II-1
19 Fava, R. A. *Polymer* 1968, **9**, 137
20 Cernee, F., Osredkar, U., Moze, A., Vizivisek, I. and Lapanje, S. *Makromol. Chem.* 1977, **178**, 2197
21 Muzumdar, S. V. and Lee, L. J. *Polym. Eng. Sci.* 1991, **31**, 1647