B. Delmon and G.F. Froment (Editors), *Catalyst Deactivation*

CATALYST POISONING AND CHEMICAL PROCESS DYNAMICS

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ABSTRACT

The purpose of this review is to link the more fundamental aspects of catalyst deactivation by poisoning to those related problems which plague industrial practitioners. As a more or less central theme, the topic of transient and unsteady state behavior in catalytic systems as it is affected by, or induced by, poisoning has been chosen.

INTRODUCTION

Unsteady state behavior of catalytic processes can be caused by all types of catalyst decay; however there are certain features of poisoning processes, particularly in how we can model their kinetics, that simplify the task of sorting out the transients we are interested in from the vast array of other complicating factors that normally exist. The following review of unsteady behavior in catalyst poisoning process is selective rather than comprehensive; it is not a general survey of literature.

KINETICS AND RELATED TOPICS

The best place to begin is to define what is meant by poisoning, which in essence is the removal of active sites from a catalytic surface via the strong chemisorption (either reversible or irreversible) of some impurity substance contained within a reaction mixture. In general, catalyst poisoning is relatively well characterized in a chemical sense, at least by comparison with coke formation. Also we may refer to a nonselective poison as one for which every active site of the surface looks like every other active site; conversely, in selective poisoning there is a nonuniform deactivation which often appears as an exponential relationship between activity and amount of poison chemisorbed. Adsorption inhibition terms in the denominator of Langmuir Hinshelwood (LH) rate equations ("self poisoning") are excluded from our definition here.

Pervasive in the literature is the concept that one may separate the deactivation rate from the rate of the main reaction, and thus decouple the kinetic analysis to the extent that only two parallel rate expressions must be treated [1]. Thus:

$$(-r)_T = r_1(C)\ r_2(T)\ r_3(s) \tag{1}$$

$$(-r)_s = r_4(C)\ r_5(T)\ r_6(s) \tag{2}$$

Where $(-r)_T$ is the rate of a main reaction, $r_1(C)$, $r_4(C)$, $r_2(T)$ and $r_5(T)$ are concentration and temperature factors, $r_3(s)$ and $r_6(s)$ activity variables, and $(-r)_s$ the resultant rate of change of activity. In most applications, $r_3(s)$ and $r_6(s)$ assume the form of a scaled variable $0 \leq s \leq 1$. This approach seems to work reasonably well for coking; extensive examples have been presented for catalytic cracking [2-7] and dehydrogenation [8]. The concept of separability seems a little more vulnerable for deactivation by poisoning. While Szepe and Levenspiel [1] make a convincing case for the examples they present, recent studies of the poisoning of the dehydration of alcohols on SiO_2/Al_2O_3 [9] and of benzene hydrogenation on Ni/Kieselguhr [10] indicate substantial deviation from separability. In both cases, it was shown that the adsorption constants in the denominator of LH correlations varied substantially with the extent of poisoning. In [10] it was further shown that this variation was due to decrease in the "heat of adsorption" parameter, suggesting that energetically more favorable sites were preferentially poisoned. Interestingly, the activation energy was independent of the level of poisoning.

In contrast, Barbier, et al. [11] have investigated the problem using the hydrogenolysis of cyclopentane on a series of Pt/Al_2O_3 catalysts poisoned by lead, zinc or sulfur. The kinetics were correlated by:

$$(-r)_4 = \frac{kS\ (K_C/K_H)^{M/2}\ P_{C5}\ P_H}{P_H^N + (K_C/K_H)^{M/2}\ P_{C5}} \tag{3}$$

where k is the rate constant, S the specific surface of P available, and K_C and K_H adsorption constants. Some typical results obtained for a 16% Pt catalyst of average crystallite size 11.8 nm showed that kS varied proportionately to Pb^{2+} (0.04 -0.08%) or Zn^{+} (0.025 - 0.05%), while both $(K_C/K_H)^{M/2}$ and N remained constant. This, of course, is precisely the result to be expected on the basis of separability.

Hence, on the basis of these results [9, 10, 11] and others not illustrated here, the question of whether a separable formulation for poisoning kinetics is proper or not is moot. Probably we have a situation corresponding to that of structure sensitive catalytic reactions, which can be resolved as to classification

only by experiment. However, it can be demonstrated unequivocally that the assumption of separability fails for surfaces other than those ideal in the Langmuir sense [12]. If we consider the nonideal surface to consist of the summation of a subassembly of ideal surfaces distributed according to the heat of chemisorption, then for nonseparable kinetics:

$$(-r)_T = \Sigma\, s_q\, n_q\, (-r)_q \simeq \int_o^{q_m} s_q\, n_q\, (-r)_q\, dq \qquad (4)$$

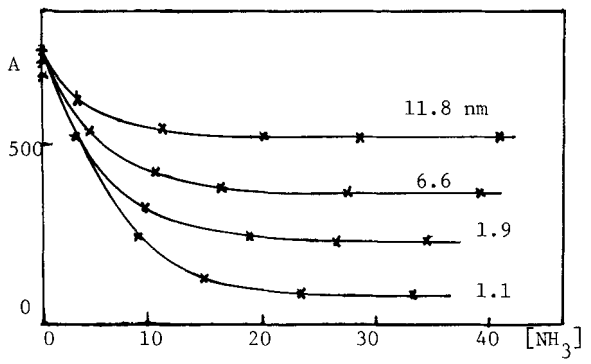
Where $\underline{s}$ is the activity of the subunit, $\underline{n}$ a distribution function, and $\underline{q}$ representative of the distribution in terms of heat of chemisorption. For separable kinetics:

$$(-r)_T = \langle s \rangle = \frac{1}{q_m} \int_o^{q_m} n_q\, (-r)_q\, dq \qquad (5)$$

$$\text{with:} \quad \langle s \rangle = \frac{1}{q_m} \int_o^{q_m} s_q\, dq \qquad (6)$$

Mathematically the differences between eq. (4) and (5) arise because the sum of the averages is not the average of the sum; agreement between the two is obtained only for fortuitous combinations of parameters [12].

A new area concerned with "structure sensitive" poisoning is just now beginning to be studied. Early work, although somewhat tangential to direct investigation of structure sensitivity, was reported by Maurel, et al. [13] for S poisoning of cyclopentane hydrogenolysis and deuterium exchange with benzene on Pt/Al_2O_3, and by Fuentes and Figueras [14] for metals such as Fe on Pd/SiO_2 and Pd/Al_2O_3. More to the point are the data of Barbier, et al. [11] shown in Figure 1 for poisoning of benzene hydrogenation on Pt/Al_2O_3 by NH_3 at 85°C. Clearly there is a dependence upon crystallite size, which is all the more interesting since this reaction on Pt has been reported to be structure insensitive [15].



A = molecules /h-Pt_s

$[NH_3]$ = molecules/Pt_s

Fig. 1. Structure sensitive poisoning of Pt/Al_2O_3 by NH_3: $C_6H_6 + H_2$

Another recent example of structure sensitive poisoning is provided by Ostermaier, et al. [16] for Pt/Al_2O_3 and Pt black in ammonia oxidation, where the temperature dependence of poisoning was a function of metal particle size. Poisoning was more severe with smaller crystallites, but it was always possible to reactivate with H_2, 673°K, suggesting that O_2 was the poison and PtO the deactivated surface.

These demonstrations of structure sensitive poisoning are most significant to future advances in catalytic reaction engineering. Eventually it could be hoped that one would be able to design a priori catalysts with maximum resistance to poisoning as well as for maximum activity/selectivity. However, the conjunction of transport processes with reaction and poisoning rates has a lot to say about this matter.

One type of catalyst poisoning which we have neglected in prior surveys [17, 18] is the interaction of metals with various types of metal oxide catalysts. The problem is most often encountered in hydrotreating processes, where metals deposition is accompanied by coke formation. The action of metals on cracking catalysts has been reported by Connor and coworkers [19, 20] and Cimbalo, et al. [21], and recently the problem of metals poisoning in resid hydrotreating has been addressed by Habib, et al.[22]. Equilibrium catalysts were impregnated with Ni and V naphthenate to 510, 870 and 2080 ppm Ni equivalent. Cracking was carried out with hydrotreated resid of high reactivitiy at 1000°F and 3-5 s contact time. Total conversion dropped only slightly, while C_5^+ gasoline yield was about 3% lower but independent of metals loading. Coke formation nearly doubled up to the 900 ppm level, then remained constant. Hydrogen production was also substantially increased up to 900 pm, then also remained constant. Gasoline octane numbers were about 1.5 units higher for cracking over poisoned catalysts, due to higher olefin and lower paraffin content. Most of these results can be explained in terms of the residual dehydrogenation activity of the metals [19, 21], in additionto their action as poisons by physically blocking active surface. The increase in hydrogen yield is a particularly important problem, since it would significantly increase gas handling requirements in commercial operation.

Finally, the large amount of recent work on sulfur poisoning of metallic catalysts should be mentioned. Again, we must be selective in citation; the matter is treated extensively in a forthcoming review [23]. Particular mention should be made of studies of poisoning on model Pt surfaces [24], and the work of Bartholomew and coworkers on Ni and Ru catalysts [25, 26]. The latter report the effects of S on H_2, O_2 and CO chemisorption; effects of S for CO on Ni have also been reported by Erley and Wagner [27], and H_2S for H_2, CO, C_6H_6 and C_2H_2 on Ni/SiO_2 by Ng and Martin [28]. Certainly this is a lively topic of investigation at the present time, and we can hope that the sum total of such efforts will lead us to an enhanced understanding of poisoning in general.

In sum, there is a simple but extremely important concept that can be gleaned from these examples and many others [1, 17, 18]. It is that, in general, rather elementary kinetic models normally can serve as adequate representations of deactivation rates; we have already stated the case in eq. (1) and (2). As for the simplicity of poisoning kinetics, so also for the reaction networks we might write to represent overall schemes. For poisoning this assumes a classical parallel structure, here written for a monofunctional academic reaction as:

$$A + S \rightleftharpoons B + S$$

$$L + S \rightleftharpoons L \cdot S$$

Where L is the poison and S is some active site. The impact of this particular parallel structure on the transient behavior of reactions and reactors is enormous. Reversibility is shown above, but the essential features of transient problems are adequately represented by the irreversible network. An analogous scheme exists for coke formation, with L replaced by A, and there are many studies in the literature couched in the terminology of coking that have direct parallelism to poisoning. We will be concerned with both here.

CATALYST POISONING AND THE DYNAMICS OF INDIVIDUAL PARTICLES

Any discussion of poisoning must begin with a citation of the work of Wheeler [29]. His concerns were twofold: (a) what is the nature of the interaction between diffusion and poisoning, and (b) to what extent is this interaction influenced by the strength of the poisoning? The results of the analysis define analytically selective and nonselective poisoning, and can be summarized by what has come to be known as a "Wheeler plot," Figure 2. The two lower curves here represent increasing degrees of diffusional limitation and correspond to pore mouth poisoning. Now what we are interested in here is not the locus of a particular point on the diagram, but the trajectory along these curves as denoted by the arrows. That is, we wish to investigate dynamics, in which Figure 2 might represent a phase-plane plot with time implicit.

Over the years most workers have chosen to work with overall particle models rather than the individual pore, hence the early study of Masamune and Smith [30] can be viewed as an extension of the normal steady state effectiveness factor analysis. For the poisoning reaction network:

$$D_A \nabla^2 C_A - \varepsilon \frac{\partial C_A}{\partial t} - \rho k_A C_A s = 0 \qquad (7)$$

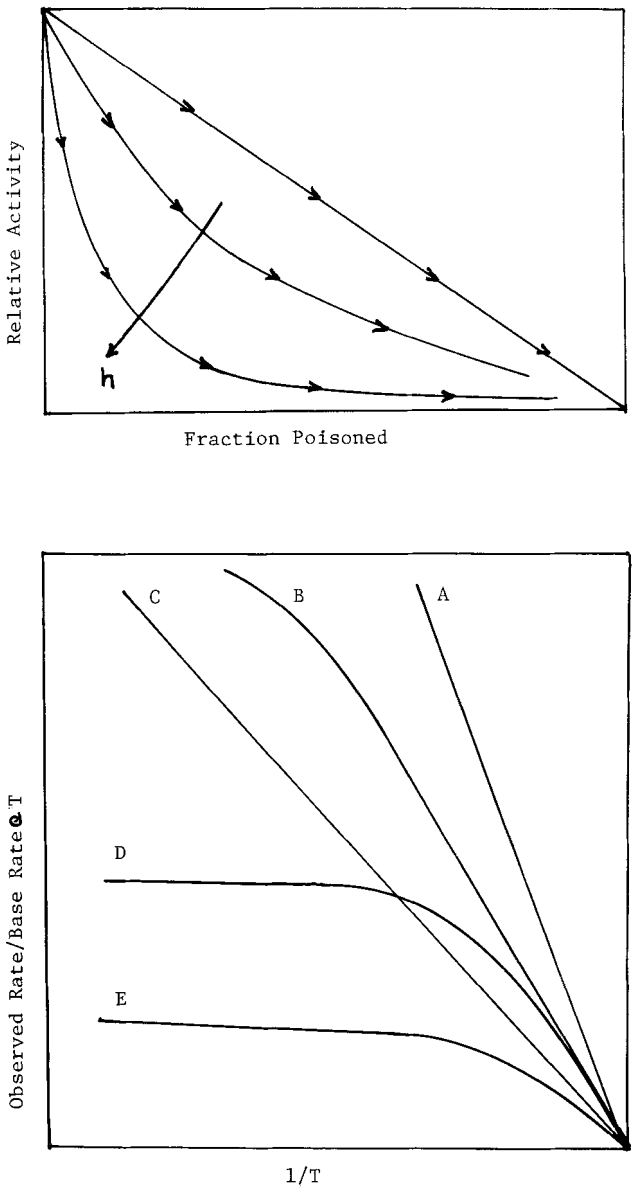


Fig. 2 (Top) Activity - poisoning relationship: The Wheeler Plot.
(Bottom) Poison - diffusion interaction versus temperature level.
A. Fresh catalyst, intrinsic kinetics; B. Small influence of diffusion, highly poisoned; C. Fresh catalyst, diffusion-limited; D. Moderate influence of diffusion, half-poisoned; E. Strong influence of diffusion, half-poisoned.

$$D_L \nabla^2 C_L - \varepsilon \frac{\partial C_L}{\partial t} - \rho k_L C_L s = 0 \tag{8}$$

The poisoning problem, appended to normal diffusion and reaction, complicates the initial conditions, since internal gradients exist at the start of deactivation:

$$\begin{aligned}
&s = 1, \quad t = 0 && \text{all } r \\
&C_A = C_{A_0}, \; C_L = C_{L_0} && t > 0, \; r = R \\
&\frac{\partial C_A}{\partial r} = \frac{\partial C_L}{\partial r} = 0 && t > 0, \; r = 0 \\
&D_A \nabla^2 C_A - \rho k_A C_A = 0 && t = 0, \text{ all } r \\
&D_L \nabla^2 C_L - \rho k_L C_L = 0 && t = 0, \text{ all } r
\end{aligned} \tag{9}$$

where r is the radial coordinate, R the radius, ρ particle density, k_A and k_L volumetric rate constants, ε porsity and D_A and D_L appropriate effective diffusivities. The rate of activity loss is given by:

$$\frac{ds}{dt} = -k_{L,d} C_L s \tag{10}$$

Some assumption as to the time scale of deactivation must be made. In general we are concerned with three characteristic times: diffusion, reaction and poisoning. Most analysis has been carried out when the last is small in comparison with others and quasi steady-state results are presented. Dynamics due to progressive poisoning are shown qualitatively in Figure 3. [30].

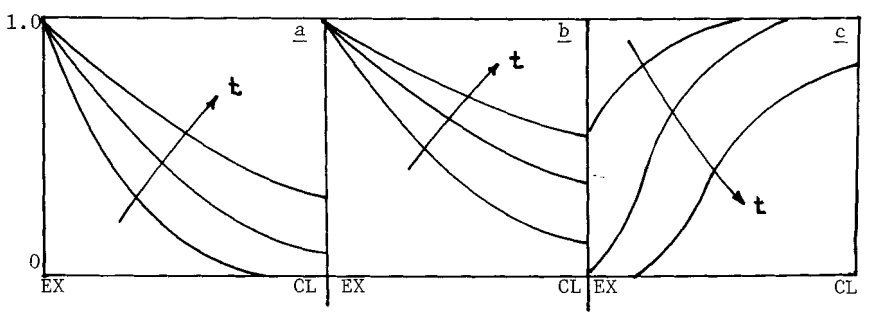


Fig 3. Concentration and activity profiles for intraparticle isothermal poisoning. EX = external surface; CL = centerline. Profiles are: a) poison, b) reactant, c) activity.

As poisoning progresses the reaction becomes less diffusion-limited, since the net rate constant $k_A s$ decreases while the diffusivity is unchanged. The general shape of the activity profile is of interest, since due to the parallel poisoning mechanism the particle must be poisoned from outside to inside. In the limit of rapid poisoning, these profiles become very sharp and the process is referred to as "shell progressive" poisoning. The parallel structure also provides the basis for some creative reaction engineering, since if the poison molecule is of comparable or smaller effective diffusivity one may control the extent of deactivation by control of the pore structure. Such an effect is shown qualitatively in Figure 4, where as one increases the Thiele modulus, h , the effectiveness of the fresh catalyst is diminished but the penetration of poison is also restricted and poisoning occurs more slowly. We are interested, of course, in the compromise between activity and total life.

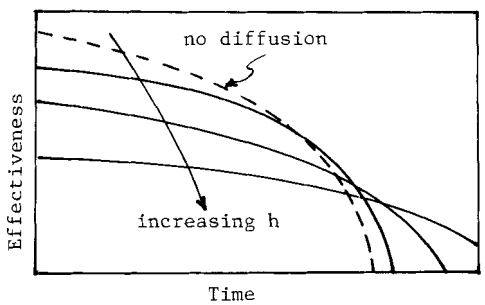


Fig. 4. Effect of pore structure upon poisoning. Molecules of similar D.

A respectable body of literature has grown up extending these basic concepts of intraparticle poisoning. Ozawa and Bischoff [31] presented a similar analysis for parallel coking, and Murkami et al.[32] and Gioia and Greco [34] considered diffusion-limited poison adsorption with Langmuir isotherm for water poisoning of ethylene hydrogenation over copper-magnesia. Haynes [35] reported a solution for both poisoning and main reaction diffusion-limited using a shell model analysis, and Kam, et al. [36] have considered the case for LH kinetics of the main reaction.

Corresponding nonisothermal problems have also been considered by a number of workers. Sagara, et al. [37] extended the basic analysis of [30] and demonstrated the development of very complex intraparticle profiles for the parallel network. It will be recalled that nonisothermal conditions require the definition of a thermicity parameter, $\beta = (-\Delta H) C_{A_o} D_A / k_e T_o$, and an activation energy parameter $\gamma_A = E/RT_o$. For poisoning, γ_L must also be defined, and the behavior of nonisothermal systems is extremely dependent upon the relative magnitudes of γ_A and γ_L. Catastrophic deactivation can occur when $\gamma_L > \gamma_A$ [37, 38] . Interactions

between poisoning and multiple steady state phenomena in nonisothermal systems have been investigated by Ray [39] and Ramachandran, et al. [38]. The latter demonstrate transitions from upper to lower steady states induced by poisoning.

Recently there has been considerable interest in the influence of nonuniform distributions of active ingredient on activity and selectivity under poisoning conditions. The simple thought is to maximize active sites where they will do the most good. For diffusionally influenced systems, this would normally mean near the external surface to provide additional capacity for poison uptake. Corbett and Luss [40] considered a combined activity/selectivity problem with continuous distributions, discussed in [18], and Becker and Wei [41] analyzed activity maintenance with discontinuous distributions. The activity distribution problem has also been addressed by Shadman-Yazdi and Petersen [42] for coke formation via a series reaction, Minhas and Carberry [43] for SO_2 oxidation, and by Delancey [44] for A to products, diffusion limited, with uniform poisoning. In most cases some advantage has been claimed for preferential loading near the outer surface. The discontinuous distributions examined in [41] are of interest in pointing out limiting cases. Four distributions, outer shell, inner core, annular ring, or uniform were considered and catalyst performance evaluated in terms of the relative Thiele moduli for the main and poisoning reactions, $\underline{h}_A$ and $\underline{h}_L$. Computation of total operating time to a final limiting effectiveness was presented for a specific case, but it seems that the results can reasonably be generalized. The product is a catalyst selection chart, Figure 5. The shaded area at the top represents a region in which $\underline{h}_A$ is sufficiently large that the effectiveness specification is violated even for fresh catalyst. The idea of distributed quantities has been further extended by Wolf [45] and Polonski and Wolf [46] to a composite catalyst in which both pore structure and active ingredient is varied with position. An inert support structure is placed external to an active core and in general the effective diffusivities of both reactant and poison will differ in both zones. Suitable control of the inert zone pore structure will result in limitation of access of poison to the active core.

In reviewing the presentation to this point, it is disheartening to discover the scarcity of relevant experimental data. Results have been reported for parallel coking [31, 32, 47] where the kinetic structure is similar to poisoning, and Petersen and coworkers have worked extensively with a single pellet reactor [18], but only for coke formation. In this laboratory we have developed an analagous single pellet reactor, in which we measure radial temperature profiles rather than centerline concentrations [48]. Experimental data on the effect of poisoning on various types of transient behavior have been reported [49, 50] for the thiophene-poisoned hydrogenation of benzene on Ni/kieselguhr. Differing ranges of poison concentration, metals loading and effective thermal conductivity

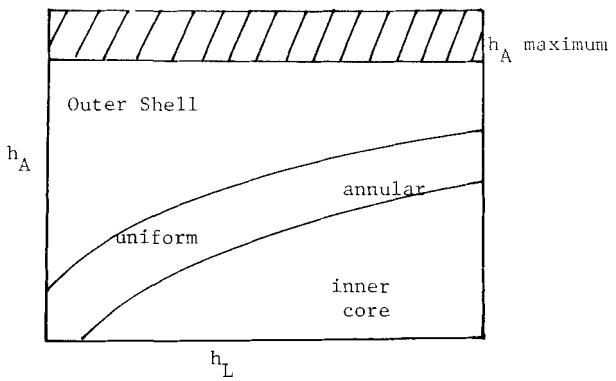


Fig. 5. Catalyst selection chart for distribution of active ingredient as a function of $\underline{h}_A$ and $\underline{h}_L$. (log-log scale).

have been investigated. Considerable effort has been devoted to the a priori simulation of such transients, using mathematical models in which all parameters are determined in separate experimentation. An example is the ignition experiment shown in Figure 6. Both experiment and theoretical calculation are shown for a typical experiment, in which the pellet has been poisoned under reaction conditions to the specified fraction of initial activity, s, at which point the reaction is stopped, hydrogen introduced into the system followed by a step input of benzene. Important results are that the time scale of response is not severely affected [49], although the magnitude of the intraparticle exotherm is decreased by a factor of three. However, under conditions of severe poisoning the steady state intraparticle temperature gradient exceeds the boundary layer gradient. The simulation results shown in Figure 6 are representative; qualitative trends, including maxima, are well predicted but quantitative agreement is difficultly attained. The mathematical description of these experiments is roughly the same as that of eq. (7)-(10) although without the quasi-stationary assumption and with LH kinetics for the main reaction. While it is impossible to give here a full description of the simulation, we invite attention to the somewhat pessimistic conclusion [50] that we are victims of the accumulation of uncertainties in parameter values which exacerbate problems of parametric sensitivity. Hence, with reference to the many theoretical studies cited above, it is fair to question how well we know the parameters required for description of intraparticle poisoning. Certainly this is a plea for more experimentation; we must avoid the fate of the steady state effectiveness factor problem where an armada of theoretical models pursues each datum.

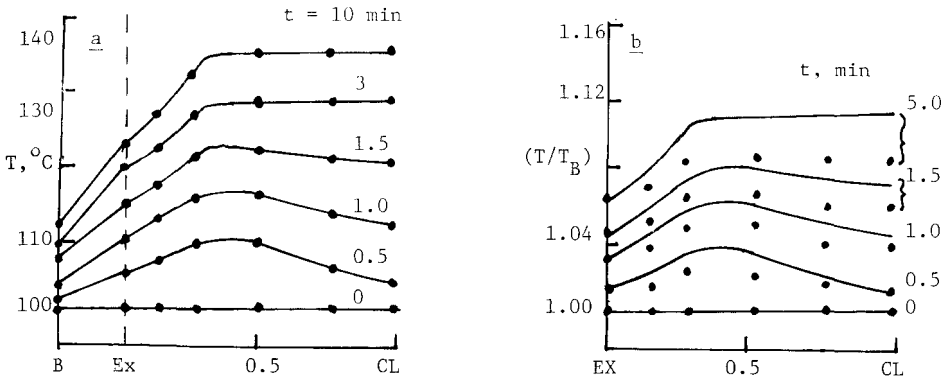


Fig. 6. Experiment and simulation for ignition of poisoned catalyst pellet, 16% original activity; $C_6H_6 + H_2$ $x_B \simeq 0.16$). Thiophene poisoning of Ni/kieselguhr. (a) experiment, (b) simulation.

CATALYST POISONING AND THE DYNAMICS OF FIXED BED REACTORS

We begin with an apology to those devotees of fluidized beds, slurry reactors, trickle beds and the like if they feel their interests are slighted in the following. Yet it can be argued that there is no other single example setting forth so starkly the dynamics of deactivation as a fixed bed.

Before exploring dynamics however, let us first look at activity distributions in fixed beds and what they tell us. Anderson and Whitehouse [51] first studied the influence of various internal activity distributions on the overall performance of fixed beds. Two balances were written, a distribution of poison within the bed and a relationship between poison loading and activity. In general notation:

$$C_p = f_1(Z) \tag{11}$$

$$s = f_2(C_p) \tag{12}$$

and:

$$\bar{s} = \frac{1}{L}\int_0^L f_2\,[f_1(z)]\,dz \tag{13}$$

Various combinations of $f_1(Z)$ and $f_2(C_p)$, where C_p is poison concentration, were explored. An interesting result of this exercise was that log-log plots of activity loss, $1 - \bar{s}$, vs. average cumulative poison on catalyst (an indirect measure of time on stream) were linear over large ranges of parameters. This corresponds to a kind of Vorrhies correlation for the fixed bed reactor. Thus

all combinations tend to look the same overall and one can infer that a fixed bed is not going to be an optimum device for inferring kinetic data or building kinetic models in the face of deactivation.

A first proposal for a poisoning wave model in fixed beds was made by Wheeler and Robell [52]. Again the parallel network for poisoning has much to do with the reasoning involved. When poisoning is rapid and irreversible, an activity profile develops such that bed inlet is completely deactivated and a central portion will display a sharp change from no activity to full activity. The situation is analagous to breakthrough waves encountered in fixed bed ion exchange, and indeed the Wheeler-Robell analysis is based on the fixed bed ion exchange (adsorption) theory of Bohart and Adams [53]. Thus we have:

$$s = 1 - C_p/C_{p\infty} \tag{14}$$

$$\frac{C_p}{C_{p_\infty}} = \frac{1 - \exp(-N_t\,\theta/\theta_\infty)}{1 + \exp(-N_t\theta/\theta_\infty)\,[\exp(N_t\,z/L - 1]} \tag{15}$$

in which $N_t = k_p L/v$ where k_p is the poison adsorption rate constant, L the reactor length, θ time and θ_∞ the ratio of total poison capacity to the rate of introduction of poison. For first order reaction we may define a conversion as

$$\ln (C_L/C_o) = \frac{1}{L}\int_o^L k_o s \, dz \tag{16a}$$

and; upon integration using the results of eq. (14) and (15):

$$\ln (C_L/C_o) = -(k_o/k_p)\ln\{1 - \exp(-N_t\theta/\theta_\infty) + \exp[N_t(1-\theta/\theta_\infty)]\} \tag{16b}$$

Typical profiles are shown in Figure 7. The wavefront can be diffuse or sharp, largely depending on the value of (k_o/k_p). Sharp profiles lead to very abrupt

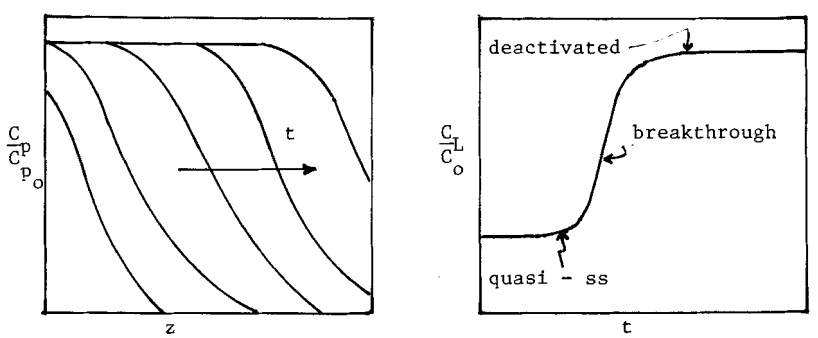


Fig. 7. Conversion - time for a sharp poisoning wave.

changes in exit conversion, also shown in Figure 7. An extension of this analysis where the main reaction is diffusion controlled, but not the poisoning step, is also given in [52], and Haynes [35] extends the analysis further to diffusion-controlled poisoning.

Interesting as these approaches are, they are not fundamental in the sense that they represent only a type of superposition of theories for analagous problems. Froment and Bischoff [54] were the first to approach the problem quantitatively; their primary concern was with coking but the parallel kinetic network was considered. A model for fixed bed poisoning, akin to that of Froment and Bischoff is:

$$\varepsilon \frac{\partial C_A}{\partial t} = -\varepsilon \frac{\partial (vC_A)}{\partial z} + s \rho_c (-r_A) \tag{17}$$

Where ρ_c is catalyst density, $(-r_A)$ rate/wt., and v interstitial velocity. For the poison:

$$\varepsilon \frac{\partial C_L}{\partial t} = -\varepsilon \frac{\partial (vC_L)}{\partial z} + \rho_c (-r_L) \tag{18}$$

It is shown [54] that for separable kinetics the nature of the $C_L - s$ relationship does not alter essential nature of the results obtained. Qualitative agreement with wave theory is demonstrated with preferential deposition of poison at the bed entrance and decreasing concentration of poison with bed length. A more profound result is shown in Figure 8, wherein is plotted the evolution of the

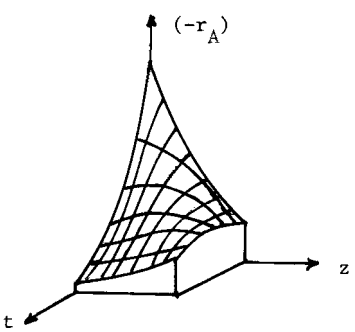


Fig. 8. Activity waves in an isothermal reactor.

volumetric rate of reaction with time and distance. Clearly a locus of maximum rate develops and passes through the reactor with the final activity profile the inverse of the initial one. If the reactor were nonisothermal, this would manifest itself as a thermal front passing through the bed, as shall be seen

later. Some alternative methods for treating the mathematics of moving zones in isothermal, fixed bed deactivation have been reported by Bischoff [55], Ozawa [56] and Wentrcek, et al. [57].

An experimental study of isothermal fixed bed poisoning has been reported by Richardson [58] for various sulfur compounds on commercial Ni/kieselguhr. The bed was prepoisoned and the resultant sulfur profile determined as a function of time using a magnetic technique. Excellent agreement of the resultant profiles with Bohart-Adams theory was obtained, although the parameter corresponding to $\underline{k}_p$ in eq.(15) exhibited a more complex dependence upon velocity than would be expected for an adsorption rate constant. It was concluded that the $\underline{N}_t$ quantity could be written in terms of a more general rate constant $\underline{\bar{k}}$ determined from the inverse reciprocal additive of contributions from mass transfer, pore diffusion, solid state diffusion, adsorption and chemical reaction. Any one of these could contribute in different ways to $\underline{\bar{k}}$ for various conditions.

A final comment on isothermal reactor poisoning concerns averaging disguises both of kinetics and selectivity by deactivation. The problem has been discussed extensively by Weekman and coworkers [2-7, 59], Froment and Bischoff [54] and by Krishnaswamy and Kittrell [60]. The standard integral reactor analysis presumes the rate constant independent of position, which it cannot be if there are activity profiles within the ractor. Hence we measure an average value (cf. eq. (11-13)):

$$\bar{k}_A = \frac{1}{L}\int_0^L k_A(z, t)\, dz \tag{19}$$

which is then related to poison concentration, which is also an averaged value. However the implied relationship that:

$$\bar{k}_A = k_A(\bar{C}_L) \tag{20}$$

is not true in general. This is indicated indirectly by Anderson and Whitehouse [51] and Froment and Bischoff [54] discuss a detailed example. Other studies of relevance are those of Corma, et al. [61] (methylcyclohexene dehydrogenation on Pt/NaY), Snyder and Matthews [62] (bifunctional catalysts) and work previously reviewed in [18].

Nowhere is the effect of poisoning on reactor dynamics more pronounced than in nonisothermal operation. Activity waves in the isothermal case now become temperature waves, and there has been a fair amount of both simulation and experiment for both coking and poisoning deactivation. Computer studies of poisoning in adiabatic reactors have been reputed by Ervin and Luss [63] and Kam and Hughes [64], and Blaum [65] gives extensive results for nonisothermal-

nonadiabatic (NINA) operation. Menon and Sreeramamurthy [66] and later Menon et al. [67] reported temperature profile measurements for air oxidation of H_2S on activated carbon in a NINA reactor. Rate maxima corresponding roughly to temperature maxima were observed which passed through the bed at constant velocity. Pexidr, et al. [68] studied hydrogenation of benzene over Ni/Al_2O_3 after pre-poisoning the catalyst with the injection of various amounts of CS_2. Both temperature and composition profiles could be measured experimentally and the results are shown in Figure 9. Progression of the activity wave is evident from

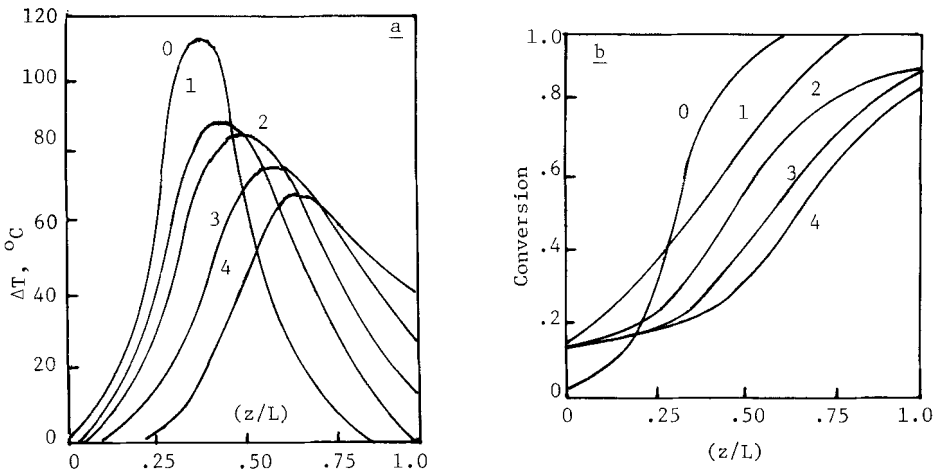


Fig. 9. Temperature and conversion profiles, $C_6H_6 + H_2$ on Ni/Al_2O_3, CS_2 poisoned. 0 - Fresh bed; 1 - 10 unit dose CS_2; 2-30; 3-50; 4-80 [68]. T_o = 100 °C.

both temperature and conversion profiles. The measurements shown were carried out under considerable intraparticle diffusional resistance (activation energy ca. 4 kcal/mol), yet there remains a very strong influence of poisoning on the magnitude of the hot spot.

These data, and other for $C_6H_6 + H_2$ on Ni/kieselguhr poisoned by thiophene [69], indicate a decrease in the magnitude of the hot spot as poisoning progresses. The point has been explored conputationally by Blaum [65] using a two phase model and the parameters for CO oxidation on NiO. For deactivation by a parallel mechanism he found the primary factors dictating reactor dynamics were the time scale of poisoning compared to that of the main reaction and the relative values of the activation energies for reaction and poisoning. For slow deactivation and $\underline{E}_L = 0$, the temperature profile travels with the same shape at constant velocity through the bed. For $\underline{E}_L < \underline{E}_A$ an activity minimum develops which remains localized near bed inlet and the thermal wave travels with diminishing magnitude through the bed. For $E_L > E_A$ the activity minimum becomes very sharp, still

remaining near the bed entrance, and shoulders appear on the leading edge of the temperature profiles. When deactivation is rapid, qualitatively different behavior is encountered. For example, when $\underline{E}_L < \underline{E}_A$ (the least sensitive case) the magnitude of the hot spot increases as the reaction zone moves down the bed and eventually exceeds the adiabatic temperature rise. Corresponding activity minima are very broad. Growth of the hot spot can be explained in terms of the relative heat capacities of solid and fluid, in ratio about 1000:1. Subsequently, Mikus, et al. [70] investigated such effects experimentally for CO oxidation on Pt/Al_2O_3 with continuous CS_2 poisoning. At an inlet temperature of 150°C, they were able to obtain general experimental trends in agreement with the rapid and slow poisoning results of Blaum by variation of inlet CO (1-3%) and CS_2 (0.006-0.03%) concentrations in various combinations. No mathematical interpretation of their results was given, however.

A large amount of work has been carried out in our own laboratories on the dynamics of fixed beds subject to rapid irreversible poisoning, using the same model reaction system shown in Figure 6. Both nonisothermal [69, 71] and adiabatic [72, 73] operation have been investigated. In a typical configuration the active portion of the bed is contained between inert fore and aft sections; steady state hydrogenation is established with poison-free feed, then poison introduced continuously and the migration of the temperature profiles is monitored as a function of time. Two typical sets of profiles are shown in Figure 10. As in the case of the intraparticle profiles of Figure 6, considerable effort has been devoted to a priori simulation. A conventional one dimensional dispersion model has been employed for simulation, with mass balances for benzene and thiophene and an overall energy balance; the details of solution have been given by Eigenberger and Butt [74] who report a variable space step Crank-Nicolson method. In view of recent literature it would appear that collocation methods are also suitable and perhaps more rapid [64, 75]. As in the case of

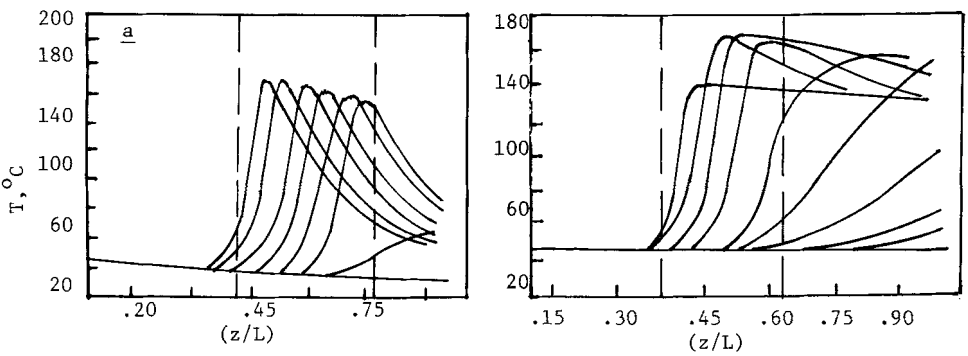


Fig. 10. Temperature profiles for NINA and adiabatic bed poisoning; $C_6H_6 + H_2$, thiophene-Ni/kieselguhr. a) 30 min intervals, $x_B = 0.035$, $x_T = 0.0019$, $^{6}_{640}$ min^{-1}; b) 15 min intervals, $x_B = 0.014$, $x_T = 0.00064$, 460 min^{-1}.

the simulation of intraparticle poisoning, there are a large number of parameters (16 this time for NINA operation) with an uneven distribution of parametric sensitivity. While effective diffusivity and boundary layer heat coefficient were troublesome for the intraparticle problem [50], the quantity corresponding to $\underline{C}_{p\infty}$ in eq. (14), poison adsorption capacity, is the difficult one in reactor simulation. It is the quantity which essentially dictates the rate of propagation of the reaction front through the bed. For this system, $\underline{E}_L < \underline{E}_A$, and the profiles are of the same shape and travel at essentially constant velocity, as one would expect from the results of Blaum. Note also, that the magnitude of the exotherm grows in the adiabatic case but not for NINA operation. The results of computational simulation of the run depicted in Figure 10b are shown in Figure 11. The quality of fit here is considerably better than for the intraparticle problem, and we have recently conducted simulations of ignition experiments on fresh and partially deactivated beds which demonstrate excellent agreement for the most part [73].

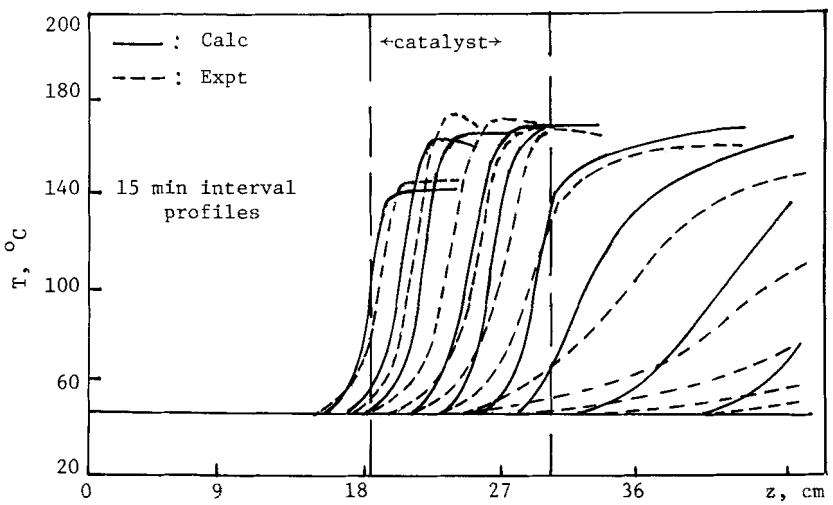


Fig. 11. Computational simulation of adiabatic bed poisoning, (Fig. 10b).

Other encouraging simulation results are reported by Christainsen and Andersen [75] for sulfur poisoning of nickel catalyst in a steam-reforming reactor. Both intraparticle and intrareactor gradients were considered, with a Langmuir isotherm for adsorption of S on Ni. An interesting consequence of the latter is that at higher inlet S concentrations there was no development of a moving sulfur front through the bed; profiles demonstrated gradually decreasing concentration with bed length. A comparison with understandably scanty data on profiles from a commercial unit led to the conclusion that the simulation provided "....good qualitative agreement with industrial experience," a substantial accomplishment.

A final problem in nonisothermal reactor analysis that deserves mention is that of constant conversion operation under deactivating conditions. Temperature is varied to maintain conversion constant and one obtains temperature-time histories (sometimes referred to as temperature increase requirements). Early work on the topic was published by Butt and Rohan [76] for various models of nonselective poisoning using a mixing cells representation of the reactor. This and subsequent work has been reviewed earlier [17]. Normally one is interested in the time-temperature history required for constant conversion. Particularly simple results pertain for first order reaction and parallel poisoning. From the series mixing cell model we have:

$$C_{A_n} = \prod^{n} (1 + k_{A_i} s_i \Theta)^{-1} C_{A_0} \tag{21}$$

$$C_{L_n} = \prod^{n} (1 + k_{L_i} s_i \Theta)^{-1} C_{L_0} \tag{22}$$

Where $\underline{s_i}$ is the activity variable and $\underline{\Theta}$ the holding time per cell. It is easy to derive that a required temperature schedule is

$$\frac{dT}{dt} = \frac{\sum^{n} \frac{k_A}{s_i} \frac{(ds_i/dt)}{(k_A/RT^2)}}{n} \tag{23}$$

In eq. (23) it has been assumed that the temperature at any instant is uniform throughout the reactor. The term (ds_i/dt) refers to any form of poisoning kinetic law that may be applicable.

Recently Krishnaswamy and Kittrel [77] have shown that for concentration-independent decay, that is:

$$\frac{ds}{dt} = -k_L s^n \tag{24}$$

and Arrhenius dependence of both main and deactivation rate constants, the following relationship between time and temperature may be derived:

$$t = \frac{\exp (E_L/RT_o)}{A^o_L (E_L/E_A)^{-n+1}} \left\{ 1 - \exp \left[\left(\frac{E_L - n E_A + E_A}{R} \right) \left(\frac{1}{T} - \frac{1}{T_o} \right) \right] \right\} \tag{25}$$

where $\underline{A_L^o}$ is the preexponential factor in $\underline{k_L}$, $\underline{T_o}$ initial temperature, and $\underline{E_L}$ and $\underline{E_A}$ the poisoning and reaction activation energies. A convenient method for

the manipulation of eq. (25) for analysis of constant conversion deactivation data is given in [77] and illustrated for the coking of hydrocracking and reforming catalysts (though in principle the method is applicable for poisoning as well). Recognize that the equation is of the form:

$$t = C\,(1-e^{AY}) \tag{26}$$

If plots of t vs. $(1/T)$ are made and the $(1/T)$ axis subdivided into four equal segments as $(1/T_o - 1/T_1)$, $(1/T_1 - 1/T_2)$, etc., then it turns out that:

$$C = (t_1 t_4 - t_2 t_3)/(t_1+t_4) - (t_2+t_3) \tag{27}$$

Where t_1, t_2 ... are the times corresponding to $(1/T_1)$, $(1/T_2)$ .... Once an estimate of C is obtained, then write eq. (26) as:

$$\ln (1-t/C) - AY \tag{28}$$

Thus, a semilog plot of $(1-t/C)$ vs. Y should be linear and yield the value for the constant A. Various procedures for estimating the individual components of C and A are discussed in [77].

The merits of using any type of temperature forcing experiment to determine deactivation kinetics, by whatever mechanism, are debatable at best. It is a particularly dubious procedure when $E_L \geq E_A$, and even more so when bifunctional or other catalysts involving product selectivity are involved 78 . However, such procedures are firmly entrenched in industrial practice, so we must try to cope.

ACKNOWLEDGMENT

Portions of the research at Northwestern University described herein were supported by the National Science Foundation, Grants NSF CPE 7915234 and ENG77-20177, by the Mobil Foundation, Inc., and by a Dow USA Central Research Fellowship in Catalysis.

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