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Kinetics of Deactivation of a Reforming Catalyst during Methylcyclohexane Dehydrogenation in a Diffusion Reactor

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The deactivation of a commercial reforming catalyst having 0.6% Pt supported on γ-alumina was studied in a single pellet diffusion reactor using the dehydrogenation of methylcyclohexane to toluene reaction as a model of the reforming process. The reaction was studied in the temperature range 350–400°C and methylcyclohexane and hydrogen partial pressure from 15–60 and 0–800 Torr, respectively. In this range of concentrations, the reaction is approximately first order with respect to MCH and H_2.

Results of this work establish the sensitivity of the poisoning rate to hydrogen partial pressure in agreement with previous work. It is shown that only a part of the intermediate poison structures can be removed by hydrogenation under reaction conditions.

A model of the poisoning process is developed which explains the data quantitatively, and agrees with previous work in important aspects.

NOMENCLATURE

$\mathfrak{D}_{eff}$	effective diffusivity
h_1	Thiele modulus, main reaction
k_i, k_{-i}	forward and backward rate constant of step i of the reaction sequence
k'	pseudo-homogeneous first order rate constant, liters/sec
K_1, K_2, K_3, K_4	equilibrium constants of step i of the reaction sequence
L	thickness of catalyst pellet, cm
[MCH·S]	concentration of sites occupied by the adsorbed methylcyclohexane, N^0 sites/cm^3
[MCH]	gas phase methylcyclohexane concentration, mol/cm^3
[P]	concentration of sites occupied by reversible poisons, N^0 sites/cm^3
[P·S]	concentration of sites occupied by poison precursor
R	reaction rate, mol/cm^2 sec
$[S]_0$	initial site concentration, N^0 sites/cm^3
$[S]_{t_0}$	initial site concentration after induction period, N^0 sites/cm^2
[S]	concentration of empty sites, N^0 sites/cm^3
Sg	specific active catalyst area
t	time, sec
t_0	induction time, sec
[TOL·S]	concentration of sites occupied by adsorbed toluene, N^0 sites/cm^3
[TOL]	gas phase toluene concentration, mol/cm^3
[W]	concentration of sites occupied by irreversible poison, N^0 sites/cm^3
x	distance coordinate, cm
η	dimensionless distance coordinate
θ	fraction of catalytic surface remaining unpoisoned

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θ_{irr} fraction of irreversible poisons
ψ dimensionless reactant concentration
τ dimensionless time
ρ_p pellet density

INTRODUCTION

Catalyst deactivation rates dictate the temperature and hydrogen pressure at which catalytic reformers operate. The long-term operation of naphtha reformers for months or even years without regeneration would appear to contradict the above statement, but in fact it does not. The known general pattern of reformer operation is that high hydrogen pressures suppresses coke formation on the catalyst, but at the same time makes it necessary to use higher temperatures to shift the equilibrium constant and composition to favor the dehydrogenation of naphthenes and obtain the desired aromatic products. Higher temperatures, however, also favor undesirable side reactions, principally hydrocracking and coke formation. From this simplified analysis, it is clear that the optimal reformer operating conditions are arrived at by making a series of compromises and tradeoffs between the severity of the treatment and the catalyst regeneration cycle time for a given stock.

Catalysts which are more resistant to coking are desirable in order to permit longer periods between regenerations, or to operate for the same time on stream at higher temperatures to give greater conversion, or to operate at lower pressures, thereby favoring the dehydrogenation equilibrium. Thus the catalyst performance is limited in a direct way by the deactivation characteristics of the catalyst.

It was the purpose of this work to ascertain the mechanism of deactivation of a reforming catalysts with the belief that information on mechanism should be important in designing catalysts which are more resistant to coking and interpreting deactivation kinetics for use in reactor design.

The deactivation mechanism and kinetics of the poisoning reaction were obtained using the single pellet diffusion reactor (SPDR). This technique has proven to be advantageous to the diagnosis of the mechanism of poisoning reactions. Balder and Petersen (*1*) and Hegedus and Petersen (*3*) have used it to study the poisoning mechanism occurring during hydrogenolysis of cyclopropane. The single pellet diffusion re-

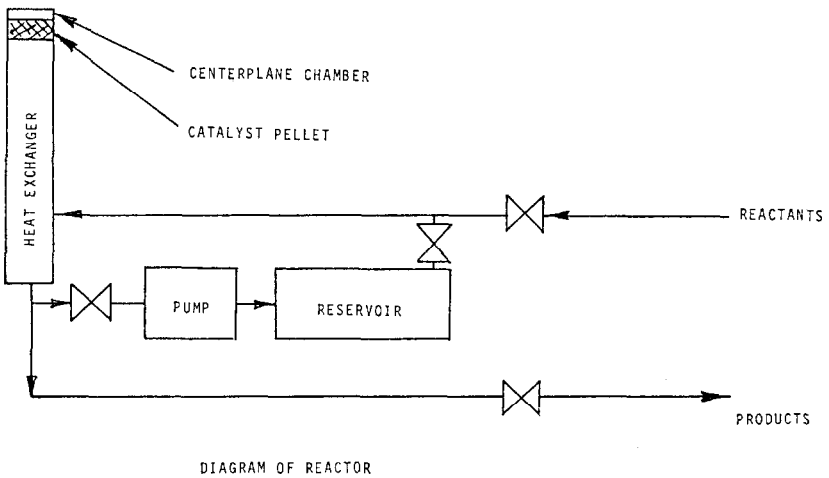


FIG. 1. Schematic diagram of the equipment.

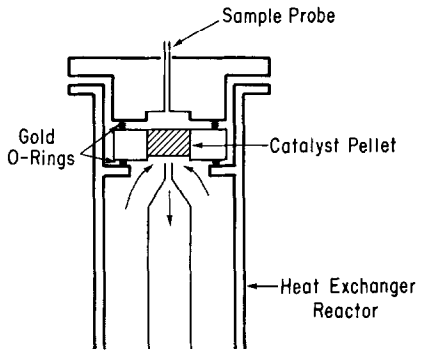


FIG. 2. Enlarged view of the reactor showing the catalyst pellet.

actor theory and operation has been described elsewhere (*2*, *3*).

EXPERIMENTAL EQUIPMENT

The naphtha reforming reactions are carried out at high pressures and high temperatures. Typically, temperatures between 350 and 500°C and pressures up to 600 psi are used. An experimental apparatus built especially to meet these pressure and temperature requirements is schematically presented in Fig. 1. The SPDR consists basically of two chambers, the bulk chamber and the centerplane chamber, which are connected by the catalyst pellet. The bulk chamber consists of a heat exchanger reactor, recirculation pump and a well-mixed reservoir. The centerplane chamber is formed by a floating head that closes one of the ends of the heat exchanger. Figure 2 shows an enlarged view of the centerplane chamber. The catalyst powder is pressed into a special holder to form the pellet. The holder is made of stainless steel and is 0.5 in. i.d., 1.375 in. o.d. and 0.25 in. thick. The holder is placed inside the heat exchanger between an annular support and the floating head. A gas seal is provided by gold O-rings at each face of the holder. The centerplane chamber has a volume of 3.6 cm^3 and contains a thermowell and a 0.0625 in. o.d. connection to the sampling valve.

The main component of the bulk chamber is the heat exchanger that allows heat exchange between inlet and outlet streams. The heat exchanger is made of two 304-ss concentric tubes. The outer being 1.5 in. o.d. and the inner being 0.5 in. o.d. The heat exchanger is 45 in. long and has two electric heaters in the upper section. The upper heater (Lindberg 8 in., 430 W) is controlled by a proportional-temperature controller which maintains the pellet at constant temperature within ±1°C at 400°C. The reactants are recirculated using a bellows pump which is enclosed in a stainless steel box maintained approximately at the reactor pressure. A pressure control system protects the bellows from sudden pressure changes.

Another component of the bulk chamber is a well-mixed reservoir that adds volume to the system to store reactants. The reservoir is 14 in. long, 3.75 in. i.d. and has a porous concentric cylinder of 1.5 in. diameter. Landau and Petersen (*4*) have shown that this system gives the same residence-time distribution as a well-mixed tank.

The reactor can be operated either in a batch-recirculation or in a differential flow mode. Concentrations are measured at the reactor inlet and outlet and at the centerplane. Balder and Petersen (*1*) used gas chromatography (GC) to measure centerplane concentrations by withdrawing a small sample volume from the centerplane chamber. In the work of Hegedus and Petersen (*3*) the centerplane chamber was an infrared cell whereby the centerplane concentrations were measured without withdrawing sample. Both sets of results agreed, indicating that a small GC sample has no effect on the results, therefore in this work GC was used to measure both centerplane and bulk concentrations. Special care was taken to avoid dead spaces and incomplete sampling. Microvolume gas sampling valves (Carle 2018) suitable for the pressures and temperatures of this work were used. A 5 ft, 0.12 in. o.d. TCEP column was used

for the GC analysis. The column was operated at 55°C and with a carrier gas (He) flow of 60 ml/min.

The same precautions described by Petersen (*5*) were taken to minimize nonuniformities in the pellet. The catalyst was a commercial 0.6% Pt on γ-alumina catalyst (PHF-4 American Cyanamid). All the experiments were made with the same catalyst pellet of 1.25 cm diameter and 0.4 cm thick weighting 0.464 g. Before pelletizing, the catalyst powder was treated in a water-saturated N_2 stream at 350°C for 48 hr (*6*) to eliminate residual acidity (Cl_2).

The methylcyclohexane and toluene were Matheson spectroquality 99% purity. Nitrogen and helium were ultrahigh purity (Matheson). Hydrogen was high purity (99.99% purity, Liquid Carbonic).

Prior to each run the catalyst was pretreated as follows: First the catalyst was regenerated using a 6% O_2–N_2 mixture at 400°C for 2 hr to burn off any carbon residues. Second, after evacuation for 3 hr, the catalyst was activated in H_2, flowing at 100 ml/min, for 12 hr at 500°C. After hydrogen pretreatment the reactor is cooled to reaction temperature in hydrogen. Before beginning each experiment, the reactor is by-passed until the reactants and helium are well mixed.

Batch experiments were carried out by recirculating the reactants during the run, whereas in flow experiments the reactants were continuously fed into and removed from the reactor without recirculation. Therefore, the batch experiments are inherently transient in pellet surface concentration and in catalyst activity, whereas the flow experiments are transient only in catalyst activity.

The dehydrogenation of methylcyclohexane was studied at atmospheric pressure and at temperatures between 350–375°C to give toluene and hydrogen as main reaction products plus a small amount of benzene (<0.1%) and paraffins (<0.1%).

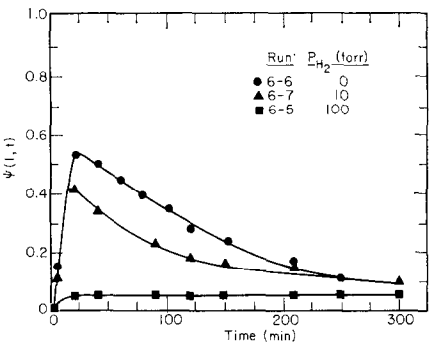


FIG. 3. Centerplane concentration vs time. Batch recycle experiments; oxygen regeneration and hydrogen activation; T = 355°C.

EXPERIMENTAL RESULTS AND THEORETICAL ANALYSIS

Preliminary experiments demonstrated that the catalyst poisoned rapidly in the absence of hydrogen and that part of this lost activity could be recovered by increasing the hydrogen pressure during the reaction. At first, the conditions for reactivation of catalysts were studied. Subsequent experiments were carried out to study the kinetics of poisoning. On the basis of the results from reactivation and poisoning experiments, a single reaction model is proposed which explains both processes qualitatively and quantitatively.

a. Catalyst Reactivation Studies

The centerplane concentration versus time results of Fig. 3, obtained during initial catalyst batch experiments, show two distinctive results. First the centerplane concentration at P_{H_2} = 100 Torr remained almost constant at a low value indicating constant, high activities and also it demonstrates that the reactor operated in the diffusion influenced regime. Second, quite different results are obtained when P_{H_2} = 0 at the beginning of the experiments. In this case the centerplane concentration rises sharply at first, reaches a maximum and then decreases. The steep initial rise of $\psi(1, t)$ can be interpreted as the combina-

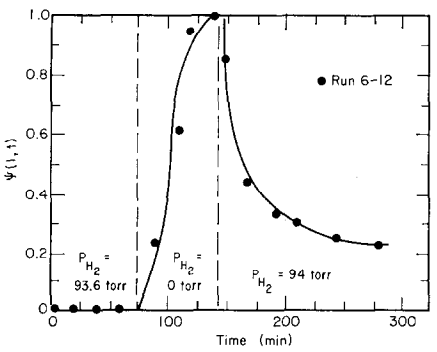


FIG. 4. Centerplane concentration vs time. Flow experiment; T = 355°C.

tion of poisoning and a transient response of the equipment. The slow decrease in the centerplane concentration after the maximum can be interpreted as a process of catalyst reactivation. This self-reactivation process is believed to occur because the hydrogen produced by the reaction accumulates during the batch recycle operation. As the reaction proceeds, the hydrogen pressure increases until it is large enough to hydrogenate the initial poisons.

The batch experiments do not lead to a unique interpretation because $\psi(1, t)$ changes with both the bulk concentration and activity. Accordingly, flow experiments at constant reactant and hydrogen concentration were carried out in order to study only the activation–deactivation process found in the batch experiments.

During flow experiments the reactants, H_2, methylcyclohexane and He as a diluent are fed continuously into the reactor. Bulk concentrations are analyzed at the reactor inlet and outlet and the reaction rate is computed from the conversion to toluene.

Figure 4 shows the variation in centerplane concentration with time obtained in a flow experiment. The figure is divided into three time periods: 70 min at a constant hydrogen partial pressure of 93.6 Torr; then 35 min at zero hydrogen partial pressure followed by about 100 min at a hydrogen partial pressure of 94 Torr. During the initial period the centerplane concentration is low and constant indicating a high activity similar to the one exhibited during the batch experiments at P_{H_2} = 100 Torr. During the second period without hydrogen the centerplane concentration increases rapidly to unity indicating a rapid poisoning occurring under these conditions. The centerplane response during this period parallels the initial steep rise obtained in the batch experiments. When H_2 is again fed into the reactor during the third period, the centerplane concentration decreases rapidly and levels off at a value corresponding to a lower activity than in the first period but higher than in the second period. These results show that hydrogen partially reactivates a completely poisoned catalyst; a result consistent with the interpretation of the batch experiments. This means that a fraction of the poisons are not removed from the surface by hydrogen at 94 Torr partial pressure.

To explore the kinetics of reactivation, experiments were carried out by increasing the time of exposure of the catalyst to the reactants at zero hydrogen partial pressure. The results of these experiments are shown in Fig. 5. For each curve the centerplane concentration decreases fast initially and levels off to a plateau. The platcau height increases for a catalyst having been exposed longer to zero hydrogen partial pressures. These results suggest that there are

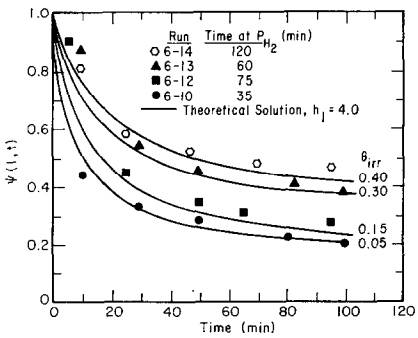


FIG. 5. Centerplane concentration vs time. Reactivation experiments; T = 355°C.

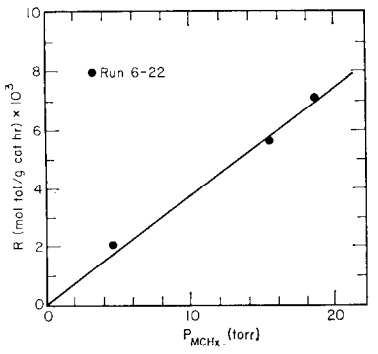


FIG. 6. Rate of toluene formation vs methylcyclohexane partial pressure. No poisoning; T = 355°C; P_{H_2} = 266 Torr.

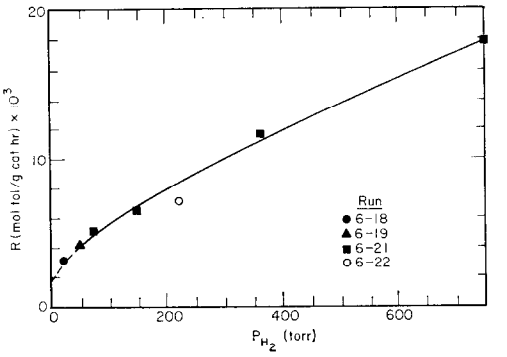


FIG. 7. Rate of toluene formation vs hydrogen partial pressure. T = 355°C; methylcyclohexane partial pressure, 18.6 Torr.

two types of poison structures: A reversible poison structure which is readily hydrogenated and a residue which cannot be hydrogenated at the partial pressures of hydrogen used to reactivate the catalyst. The relative amount of each type of poison depends on the duration of the no-H_2 period.

To model the poisoning–reactivation process the following additional experiments were carried out to determine the poisoning mechanism.

b. Catalyst Poisoning Studies

During the previous experiments the rate of poisoning was too fast at P_{H_2} = 0 and too slow at P_{H_2} = 100 Torr. It follows that at hydrogen partial pressures between 0 and 100 Torr it is possible to study the poisoning mechanism. The dependence of the rate of poisoning on the hydrogen pressure indicates that a self-poisoning mechanism is occurring, thus impurity type mechanism can be discarded.

The catalyst can deactivate through three self-poisoning mechanisms (*3*). Parallel and series self-poisoning correspond to a case wherein the poison precursor is the reactant or product, respectively. A combination of series and parallel poisoning is the triangular mechanism. The rates of the poisoning reactions are assumed to be proportional to the precursor concentration. The theoretical relative rate vs centerplane curves are obtained from the simultaneous solution of the continuity equation for the single pellet and an equation describing the poisoning kinetics. The simpler poisoning kinetics used previously are appropriate to diagnose the poisoning mechanism because the theoretical solutions depend only on the initial value of the centerplane concentration and can be used to diagnose more complex cases.

To discriminate among different self-poisoning mechanisms, the data plotted in terms of relative rate vs normalized center-

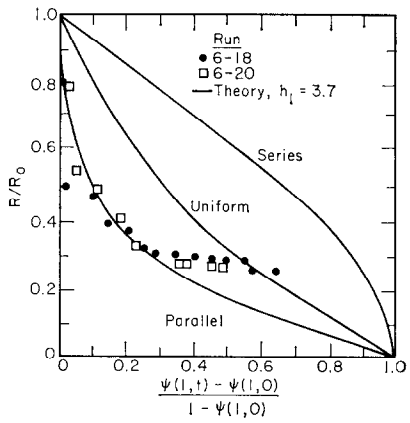


FIG. 8. Relative rate vs normalized centerplane concentration. Effect of product in the feed; T = 355°C; P_{H_2} = 55 Torr.

plane concentration are compared with the theoretical solutions of different poisoning mechanisms. The reader is referred to the literature (*3*, *7*) for a discussion of the diagnosis of poisoning mechanisms using the relative rate versus normalized centerplane concentration plots.

To diagnose the poisoning mechanism, it is necessary to know the order of the main reaction with respect to reactant concentration. Figure 6 shows data of reaction rates versus methylcyclohexane concentration. The results indicate that the reaction is first order with respect to methylcyclohexane. The effect of hydrogen on the rate is shown in Fig. 7. These results are discussed below.

The theoretical relative rate versus normalized centerplane concentration results for series, uniform and parallel poisoning and for a first order main and poisoning reaction are shown in Fig. 8. These results were obtained for an initial Thiele parameter $h_1 = 3.7$ calculated from the initial value of the centerplane concentration. Figure 8 also shows the experimental results of runs 6-18 and 6-20 carried out at 55 Torr of H_2 and 355°C.

At low values of the normalized centerplane concentration, the experimental results fall in a region accessible to parallel or triangular poisoning, but not to the series poisoning mechanism which can be discarded. To discriminate between parallel

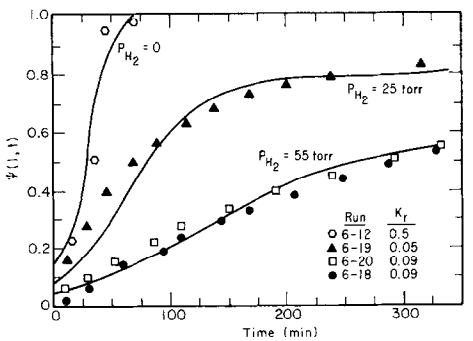


FIG. 9. Centerplane concentration vs time. Effect of hydrogen pressure on poisoning; T = 355°C.

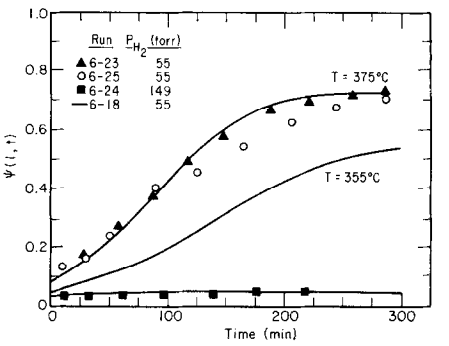


FIG. 10. Centerplane concentration vs time. Effect of temperature on poisoning at constant hydrogen pressure.

and triangular poisoning, during run 6-20, toluene was added to the feed. The results shown in Fig. 8 indicate that increasing product concentration has no effect on the relative rate versus centerplane curves. This result indicates that there is no series contributor to the poisoning mechanism ruling out triangular poisoning.

At higher values of the normalized centerplane concentration, the experimental results deviate from the parallel poisoning solution toward the uniform poisoning solution. In this region both the rate and the centerplane concentration changes very little. The transition from the poisoning regime to one having constant activity is more clearly shown in Fig. 9. The results in Fig. 9 show that after a rapid initial change, the centerplane reaches a plateau where the change in activity occurs in a longer time scale. The results in Figs. 8 and 9 lead to the conclusion that poisoning starts as a parallel mechanism and then tends to a regime of constant activity.

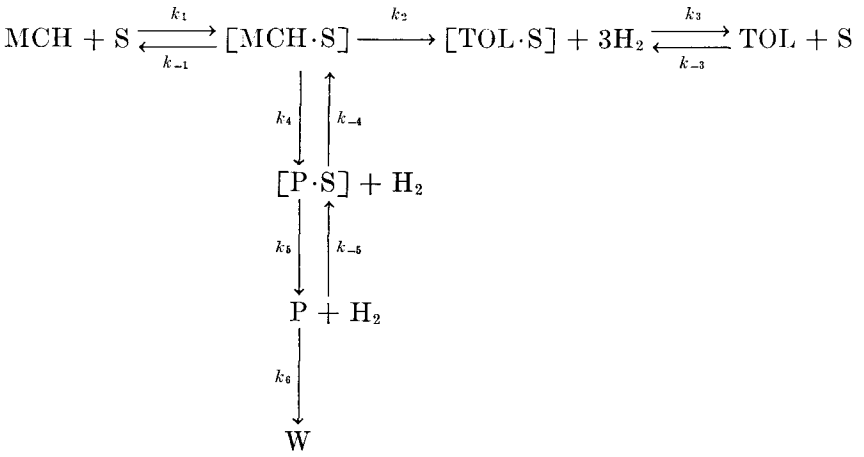
Additional experiments were carried out to investigate the effect of P_{H_2} and temperature on the initial and final activity. Figure 9 shows the centerplane vs time curves obtained at different hydrogen pressures. The results show that the rate of poisoning increases when the H_2 pressure decreases. Consequently, the initial and

final activity decrease when decreasing the H_2 pressure. The centerplane vs time curves obtained at different temperatures but same H_2 pressure are shown in Fig. 10. The rate of poisoning also increases when increasing the temperature. The initial and final activities are lower at the higher temperature.

Summarizing the work up to this point leads to the following conclusions.

1. The catalysts can be partially reactivated in hydrogen indicating that there are two types of poison structures. Reversible poisons are removed by hydrogen while irreversible poisons are not.

2. The poison precursor appears to be the adsorbed reactant. These results can be interpreted by the following reaction mechanism which accounts for both reactivation and poisoning experiments.



According to the above mechanism the reactant MCH is adsorbed in the catalyst active sites S to form MCH·S which undergoes reaction to form adsorbed toluene, TOL·S, or a poison precursor, P·S. Further dehydrogenations lead to the formation of reversible poisons P and irreversible poisons W. Adsorbed toluene is desorbed freeing an active center. According to run 6-20 the rate of toluene formation is not affected by the gas phase toluene concentration but the rate increases with the H_2 pressure. Consequently, the surface reaction is considered as the rate limiting step. At a given H_2 pressure the concentration of reversible poisons reaches an equilibrium which determines the final activity. During the experiments with no H_2 in the feed the poison precursor equilibrium is completely displaced toward the formation of poisons giving rise to the rapid poisoning observed. As the reactivation runs indicate, the rate of formation of irreversible poisons W is slow and determines the residual activity.

c. Quantitative Evaluation of Reactivation and Poisoning Kinetics

A single rate equation describing both the kinetics of poisoning and reactivation can be obtained from a Langmuir–Hinshelwood analysis of the reaction mechanism. In order to evaluate these experimental results we must look closely at the time scales on which the various reactions occur. The main reaction MCH to TOL is envisioned as a rapid process for which the usual steady-state assumption is assumed valid, thereby establishing an initial concentration of [MCH·S] on a time scale very short compared with the experimental runs. On a longer time scale, [MCH·S] begins to produce [P·S] and removes sites from the main reaction and begins to deactivate

the catalyst. The time scale on which this equilibrium occurs is assumed to be still short compared with the time of an experiment. Hence, the initial measured reaction rates are really determined on poisoned catalysts in which a surface balance is given by

$$[S] + [MCH\cdot S] + [TOL\cdot S] = [S]_0 - [P\cdot S]_0 = [S]_{t_0}, \quad (1)$$

where $[S]_0$ is the total initial site concentration per unit volume of catalyst, $[S]$ is the concentration of empty sites, and $[P\cdot S]_0$ is the concentration of poison precursors at some time t which is small compared to the experimental runs. Consequently, $[S]_{t_0}$, the initial measured activity is predetermined by $[P\cdot S]_0$ which in turn is a function of the operating conditions.

The second time scale is of the order of the experimental observations. During this period reversible poisons and to a lesser extent irreversible poisons start forming. Finally in the longer time scale the reversible poisons reach an equilibrium and only irreversible poisoning occurs.

A surface balance at $t > t_0$ gives

$$[S] + [MCH\cdot S] + [TOL\cdot S] + [P\cdot S] = [S]_{t_0} - ([P] + [W]), \quad (2)$$

where $[P]$ and $[W]$ are the reversible and irreversible poison concentrations, respectively. These two terms were excluded from the initial balance because these reactivation processes are much slower than the other reactions in the proposed mechanism.

On the basis of the results of run 6-20 (carried out with toluene in the feed), we assume that the rate determining step among the fast reactions is the surface reaction, i.e.,

$$k_2 < k_1, k_{-1}, k_3, k_{-3},$$

but

$$k_4, k_{-4} < k_2.$$

Consequently, we can write

$$[MCH\cdot S] = K_1[MCH][S], \quad (3)$$

$$[TOL\cdot S] = K_3[TOL][S], \quad (4)$$

and also

$$[P\cdot S] = K_4[MCH\cdot S]/[H_2], \quad (5)$$

where $[MCH]$ and $[TOL]$ are the gas phase reactant and product concentration and K_1, K_3, K_4 are the equilibrium constant of steps 1, 3 and 4. Combining Eqs. (2) to (5) one can obtain the total number of empty sites

$$[S] = \frac{[S]_{t_0} - ([P] + [W])}{1 + K_1[MCH] + K_1K_4[MCH]/[H_2] + K_3[TOL]}. \quad (6)$$

In accord with the assumption that the surface reaction is the rate determining step the rate of the main reaction is given by

$$R = k_2[MCH\cdot S] = \frac{k_2K_1\{[S]_{t_0} - ([P] + [W])\}[MCH]}{1 + K_1[MCH] + K_1K_4[MCH]/[H_2] + K_3[TOL]}. \quad (7)$$

At $P_{H_2} > 0$, the denominator of Eq. (7) is assumed to be unity and accordingly gives rise to a rate first order in MCH in agreement with the experimental results. Therefore, Eq. (7) reduces to

$$R = k_2K_1[S]_{t_0}\left(1 - \frac{[P] + [W]}{[S]_{t_0}}\right) \times [MCH]. \quad (8)$$

The rate of formation of reversible

poisons, P, is given by

$$\frac{d[\mathrm{P}]}{dt} = k_5[\mathrm{P}\cdot\mathrm{S}] - k_{-5}[\mathrm{P}][\mathrm{H_2}] - k_6[\mathrm{P}]. \tag{9}$$

Substituting [P·S] from Eqs. (5) and (3) into Eq. (9), we obtain

$$\frac{d[\mathrm{P}]}{dt} = \frac{k_5K_1K_4}{[\mathrm{H_2}]}\left(1 - \frac{[\mathrm{P}] + [\mathrm{W}]}{[\mathrm{S}]_{t_0}}\right)[\mathrm{S}]_{t_0} \times[\mathrm{MCH}] - k_5[\mathrm{P}][\mathrm{H_2}] - k_6[\mathrm{P}]. \tag{10}$$

Equation (10) is similar to a parallel poisoning rate equation except for the last two terms on the right side. The rate equations of the main and poisoning reactions obtained above can be combined with a differential mass balance in the gas phase. The catalyst pellet can be considered as an infinite flat porous slab of finite thickness (*3*). Diffusion of reactants is assumed to be one-dimensional and at quasi-steady state because the time scale of diffusion is smaller than the time constant of the poisoning reactions. Furthermore, the effective diffusivity is assumed to be independent of the amount of carbon on the surface, a good assumption for this kind of reaction. The concentration of H_2 is assumed to be constant throughout the pellet. The resulting dimensionless equations are:

$$\frac{d^2\psi}{d\eta^2} - h_1^2\theta\psi = 0, \tag{11}$$

$$-\frac{d\theta}{d\tau} = \theta(\psi + K_r) - (1 - \theta_i)K_r, \tag{12}$$

$$\frac{d\theta_i}{d\tau} = K_i(1 - \theta - \theta_i), \tag{13}$$

where

$$\psi = \frac{[\mathrm{MCH}]}{[\mathrm{MCH}]_0} \quad \theta = 1 - \frac{[\mathrm{P}] + [\mathrm{W}]}{[\mathrm{S}]_{t_0}} \quad \theta_i = \frac{[\mathrm{W}]}{[\mathrm{S}]_{t_0}},$$

$$\eta = \frac{x}{L} \quad \tau = k_5K_1K_4[\mathrm{MCH}]_0t/[\mathrm{H_2}],$$

$$h_1 = \mathrm{L}\left(\frac{k'}{\mathfrak{D}_{\mathrm{eff}}}\right)^{\frac{1}{2}} \quad k' = k_2K_1[\mathrm{S}]_{t_0}\,\mathrm{Sg}\,\rho_p,$$

$$K_r = \frac{k_{-5}[\mathrm{H_2}]^2}{k_5K_1K_4[\mathrm{MCH}]_0}$$

and

$$K_i = \frac{k_6[\mathrm{H_2}]}{k_5K_1K_4[\mathrm{MCH}]_0}.$$

In the above equations, $[\mathrm{MCH}]_0$ is the bulk reactant concentration, η is a dimensionless distance into a pellet of thickness $2L$, τ is a dimensionless time of the order of the time constant of the formation of reversible poisons, h_1 is the Thiele modulus of the main reaction referred to the initial activity after the induction time, k' is a pseudo-first order rate constant which depends on $[\mathrm{S}]_{t_0}$, k_2, and K_1, K_r and K_i are the dimensionless rate constants characteristic of the reversible poison formation and of the irreversible poisoning reaction, respectively.

The boundary conditions on Eq. (11) are:

$$\psi(0, \tau) = 1, \tag{14}$$

$$\frac{\partial\psi}{\partial\eta}(1, \tau) = 0. \tag{15}$$

The initial conditions depend upon whether the surface is initially poisoned (reactivation experiments) or clean (poisoning experiments). Accordingly, for reactivation experiments, the active area is initially zero and the reactant concentration inside the pellet is equal to the bulk reactant concentration, that is

$$\theta(\eta, 0) = 0, \tag{16}$$

and

$$\psi(\eta, 0) = 1. \tag{17}$$

Furthermore, the amount of irreversible poisons initially present will depend upon the conditions of the poisoning period prior

to reaction, i.e.,

$$\theta_i(\eta, 0) = \theta_{irr}, \qquad (18)$$

where θ_{irr} is a constant which depends on time of exposure to the no-H_2 feed.

For poisoning experiments, the total number of active sites is assumed to be $[S]_{/0}$. Accordingly the initial conditions for poisoning experiments is

$$\theta(\eta, 0) = 1. \qquad (19)$$

After a short initial period the reactant concentration is fully developed inside the pellet, and it is assumed to be equal to the steady-state Thiele solution for the pellet after the induction period, i.e.,

$$\psi(\eta, 0) = \frac{\cosh\{h_1(1 - \eta)\}}{\cosh(h_1)}. \qquad (20)$$

Equations (11)–(13) with boundary conditions given by Eqs. (14) and (15) and initial conditions dictated by the type of experiment were solved numerically in a CDC 6400 computer (*7*) using the quasi-linearization technique described by Lee (*8*).

DISCUSSION

The proposed reaction model can be tested by comparing experimental and theoretical results.

The reactivation experiments are discussed first. The theoretical and experimental centerplane concentration versus time curves of the reactivation runs are shown in Fig. 5. The dimensionless rate constants obtained from the best fit of experimental and theoretical results are given in Table 1. The theoretical results indicate that the centerplace concentration decreases with time at a rate which depends on the value of K_r. This agrees with the experimental observation that reactivation follows exposure to increased hydrogen pressure. Theoretical solutions obtained for $K_r < 0.05$ indicate that reactivation does not occur at this condition. As K_r increases, the activity recovers faster, but levels off at a value which depends on θ_{irr}, while θ_{irr} is determined by the severity of the poisoning period prior to the reactivation. The results of Table 1 show that K_r varies slightly depending on whether or not fresh catalyst is contacted directly with feed containing no hydrogen (runs 6-10 and 6-12 vs 6-13 and 6-14). Apparently the strength and extent of bonding of W species increases when no H_2 is present. The value of K_i has only a minimal effect on the results and was mainly determined from the poisoning experiments which are discussed next.

During the poisoning experiments, the hydrogen pressure was adjusted so that a steady decrease of activity can be observed. The theoretical and experimental centerplane concentration versus time results obtained at different hydrogen pressures and temperatures, are presented in Figs. 9 and 10. The dimensionless rate constants obtained from the best fit of experimental and theoretical results are given in Table 1.

TABLE 1

Run No.:	Reactivation runs				Poisoning runs		
	6-10	6-12	6-13	6-14	6-18/6-20	6-19	6-23/6-25
P_{H_2} (Torr)	94/0/94	93.5/0/93.5	0/96	0/90.5	55	25	55
T (°C)	355	355	354	355	355	355	375
K_r	0.25	0.25	0.2	0.2	0.09	0.05	0.07
h_1	4.	4.	4.	4.	3.7	3.2	3.5
θ_{irr}	0.05	0.15	0.3	0.4	0.	0.	0.
K_i		8×10^{-3}				16×10^{-3}	

The two important constants to analyze in this case are K_r and the initial rate which appears in the Thiele modulus. Poisoning occurs only if K_r is small enough so that the formation of P species overtake the hydrogenation reactions. This effect is clearly demonstrated in the centerplane concentration versus time curves (Figs. 9 and 10). As K_r increases (i.e., P_{H_2} increases) the rate of poisoning decreases as indicated by the slopes of the centerplane versus time curves. When K_r, i.e., P_{H_2} are high enough as in run 6-24, no poisoning occurs. The opposite limiting case corresponds to $P_{H_2} = 0$ or $K_r = 0$ where no reactivation component exists and poisoning proceeds to completion as demonstrated by run 6-12. K_r for the poisoning runs was about 2.5 times lower than in the reactivation runs which would be expected because of the lower hydrogen pressures used in the poisoning experiments. However, K_r for poisoning and reactivation experiments are not inversely proportional to the ratio of P_{H_2} as expected. The effect of the temperature on the rate of poisoning is shown in Fig. 10. At the same hydrogen pressure the rate of poisoning increases with temperature as indicated by a larger slope of the centerplane concentration versus time curve.

The initial rates were affected by the conditions of operation in that they increase with the hydrogen pressure and decrease with temperature. The initial rate constants are included in the Thiele modulus. Increasing the hydrogen pressure increases $[S]_{t_0}$ the number of sites available for reaction due to a decrease in the rate of formation of initial poisons. The same reasoning applies to explain the temperature effect. At the higher temperature the rate of formation of initial poisons [P·S] to decrease $[S]_{t_0}$ has a greater effect than the activation energy, hence, the rate decreases with increase in temperature.

This, then, was the reason for including the species [P·S] in the model. However,

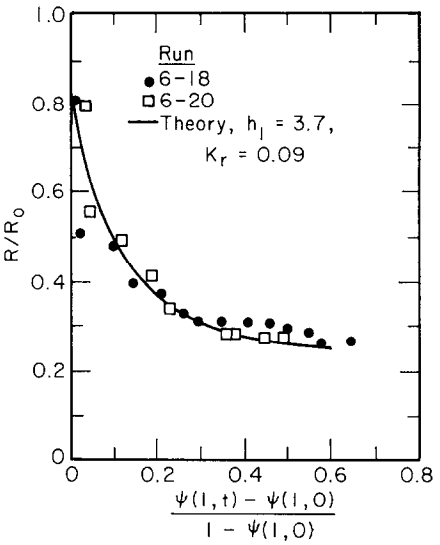


FIG. 11. Relative rate vs centerplane concentration. $T = 355°C$; $P_{H_2} = 55$ Torr.

this effect would make the initial rate depend upon a nonuniform distribution of activities in the pellet which was not accounted for in the results. Although the discrepancy due to this effect does not change the general results, more experiments must be done to explore this point.

The relative rate plotted versus the normalized centerplane concentration is shown in Fig. 11 along with the corresponding theoretical solutions. It can be seen that the new model accounts for the deviation of the data from the pure parallel poisoning model shown in Fig. 8.

The rate constant for the irreversible reaction was obtained from the best fit of the theory to the long-term variation of the centerplane concentration. As shown in Table 1, K_i is about five times smaller than K_r.

The reaction model presented here is a general one. It can be applied to a number of cases wherein both the main and side reactions are important. Care must be exercised in recognizing the relative importance of each reaction step as it applies to particular cases. In our work, for ex-

ample, if the contribution of side reactions had been small, as in the work of Sinfelt and Rohrer (*9*), then our initial rate results would have been different. Multistep reaction mechanisms have been proposed in the literature for other hydrocarbon reactions and other catalysts (*10, 12*). Tetenyi and Paal (*10, 11*) found that during the reactions of *n*-hexane, its isomers and olefins on Pt black at atmospheric pressure, decreasing the hydrogen partial pressure and increasing the temperature increases the yield of 1,3,5 hexatrienes while the yield of benzene formation decreases rapidly. Inami *et al.* (*12*) reported that removing H_2 from the reactor during the isomerization of 1-butene over a Ag–Pd membrane completely poisoned the catalyst. These authors found that the addition of H_2 to the reactor did not restore the activity. However, the catalyst was regenerated by diffusing H_2 onto the catalyst surface through the membrane. Thus it appears that in the absence of H_2, unsaturated species are so strongly attached to the surface that they prevent chemisorption of H_2 from the gas phase.

Sinfelt and Rohrer (*13*) reported that a fast poisoning occurred when pulses of methylcyclohexane were injected into a helium stream flowing over a Pt catalyst. However, after passing hydrogen over the catalyst for 48 hr, 75% of the activity was recovered. Galwey and Kemball (*14*) have shown that the chemisorption of hydrocarbons on metals is accompanied by extensive decomposition to form hydrogen-deficient surface residues. Evidence of removal of surface residues from a Pt catalyst has also been reported by Pitkethly and Goble (*15*). These authors concluded that hydrogen maintains a steady-state concentration of hydrocarbon from Pt sites, which are active for the main reaction. Myers *et al.* (*16*) studied poisoning of a Pt reforming catalyst by different hydrocarbons including methylcyclohexane. These authors found that cyclo-diene components are the most damaging intermediates leading to catalyst poisoning. The catalyst activity was recovered by flowing H_2, hydrogen and naphtha. They also found that an equilibrium activity can be approached starting from an overly poisoned or fresh catalyst. All of the above independent evidence further supports the mechanism presented here.

CONCLUSIONS

The single pellet reactor proved to be a sensitive tool to investigate the poisoning mechanism of an industrially important reaction at low hydrogen pressures. It was found that poisoning occurs as a parallel reaction of a multiple step reaction sequence where hydrogen abstraction or addition determines the predominant reaction path. The reaction model interprets the experimental results well both qualitatively and quantitatively. These results are in agreement with other independent studies at conditions where poisoning plays a role and show the critical role played by hydrogen in hydrocarbon reactions on Pt.

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