Heterogeneous Hydrogenation of Fish Oils: Kinetic Determination of Catalyst Poisoning[1]

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ABSTRACT

Mackerel oil was hydrogenated with Nysel Ni catalyst at 170 C and atmospheric pressure. The dependence of the differential rates of hydrogenation on iodine value and catalyst concentration indicated that severe poisoning of the catalyst took place. Further experiments with addition of catalyst during the run as well as with prehydrogenation of the oil to various iodine values revealed the existence of three kinetically distinguishable poisoning effects: a rapid initial poisoning, probably due to the presence of sulfur compounds; a strong primary poisoning completed within the first few minutes of the hydrogenation; and a comparatively slow secondary poisoning reaching a state of equilibrium after 25-30 min of hydrogenation. Similar results were obtained with capelin oil as well as with other commercial catalysts. With soybean oil, no such poisoning effects were observed.

INTRODUCTION

The complexity of the Ni-catalyzed heterogeneous hydrogenation of fatty oils is well known. Kinetic and mechanistic investigations of the process are difficult, not only due to the chemical reactions which involve a series of simultaneous and consecutive steps, but also because of the capital importance of various mass transfer steps. Several papers concerning the effect of the operating variables on hydrogenation rate, mechanism and selectivity have been published during the last decade, for vegetable oils (1-6) as well as for model systems (7-10). The most important mechanisms invoked in the hydrogenation of mono- and diolefins were recently reviewed by Frankel and Dutton (11).

Similiar attention has not been paid to the hydrogenation of fish oils. This may partly be due to the more complex fatty acid composition of these oils. Furthermore the presence of more or less unknown catalyst poisons may obscure the interpretation of the experimental results.

Although various sulfur compounds are known to be powerful poisons for Ni catalysts, relatively little information is available about the exact structure of the sulfur compounds present in triglyceride oils or about their influence on the rates and mechanisms of adsorption and hydrogenolysis. Coenen and Linsen (12) have estimated that one sulfur atom occupies about two surface nickel atoms, hence 1 mg sulfur corresponds to a sulfur-poisoned surface area of 2.38 m^2. The poisoning effects of various sulfur and phosphorus compounds in the hydrogenation of rapeseed oil (13) and fish oils (14) have recently been reported. The deactivating effect of various model substances on the Ni-catalyzed hydrogenation of fish oils has recently been determined by Ottesen (15).

The intention of the present work was initially that of investigating the effect of the experimental variables on the rates and products of the hydrogenation of mackerel oil. However it was soon realized that the observed kinetics were influenced strongly by catalyst poisons. A closer investigation of these effects was therefore undertaken.

EXPERIMENTAL PROCEDURES

Hydrogenation apparatus

The external hydrogenation system, allowing a semi-continuous registration of the hydrogen consumption at a constant hydrogen pressure, was quite similar to that described by Coenen (3). The design of the all-glass reactor is shown in Figure 1. Heat was supplied to the reactor by a 300 watt heat element inserted in a finger in the bottom of the reactor. The oil temperature was kept constant by an Ether "Mini" temperature controller (Ether Ltd., Herts, England) connected to the heating element and to a thermocouple. The temperature was continuously registered on a Honeywell recorder. The effect on the element could be adjusted by regulating the voltage or by means of a variable resistance connected to the temperature controller. This system allowed the temperature to be kept at 170 C ± 1.5 C.

Hydrogen supplied by a circulation pump passed through a glass sinter (G3) ring, situated close to the bottom of the reactor, at a rate of 2.8 liters/min. The unreacted hydrogen was recirculated over an activated carbon column. The turbulence caused by the hydrogen bubbles emerging from the glass sinter made mechanical stirring superfluous. Catalyst was added by lowering the stainless steel rod carrying the catalyst beaker. The bottom of this beaker was made of a thin paraffin film (mp 50-60 C) in order to assure rapid and quantitative transfer of the catalyst from the beaker to the oil. If desired, samples could be drawn through a capillary tube by means of a special sampling device (not shown on the figure).

Procedure

With the gas burettes closed the circulation pump was turned on, and hydrogen from a pressure flask was passed through the purifying system to the reactor for ca. 20 min to remove air. With the hydrogen current still flowing, 30 g oil and the weighed amount of catalyst were placed in the reactor, and the hydrogen flushing was continued for another 30 min. All outside connections were then closed. The circulation rate and the hydrogen pressure were adjusted, and the burettes and the pressure control system were connected. The heating system was turned on and the system was allowed to equilibrate at 170 C. The hydrogen pressure was then adjusted and the hydrogenation was started by lowering the rod with the catalyst beaker. In the initial period, the burettes were read every 30 sec, later at 1 min intervals. In the runs where catalyst was added twice, two rods were applied. In the prehydrogenation experiments, the hydrogen supply was cut off when the desired iodine value was reached. The oil was filtered under nitrogen blanket and the hydrogenation was continued as described.

Analysis

Gas liquid chromatography analyses of the oils were performed on a Pye Argon chromatograph equipped with ionization detector and a 4 ft x 0.25 in. glass column packed with 15% PEGA on silanized Gas Chrom P, operated at 190 C. The content of conjugatable polyenes in

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the hydrogenated samples was determined by UV absorption spectrophotometry after alkali isomerization (16).

The sulfur content of the oils was determined by Baltes' (17) method of adsorption on Raney Ni, modified to a hydrogenation temperature of 170 C. For comparision, the total sulfur content of the mackerel oil was also determined by a combustion method described by E. Bladh (unpublished work).

Materials

The refined and bleached mackerel oil (IV = 149, FFA = 0.1%) was stored in 50 ml brown glass bottles under nitrogen at -20 C. The oil contained 10.6% hexaenoic-, 6.8% pentaenoic-, 4.8% tetraenoic-, 2.6% trienoic-, 1.9% dienoic- and 45.7% monoenoic acids. The sulfur content according to Baltes' method was 2.9 ppm, while a value of 4.2 ppm was obtained by the combustion method. The content of bromine, as determined by activation analysis, was 3.6 ppm. The refined and bleached capelin oil (IV = 138) contained 4.4 ppm sulfur (Baltes). The refined and bleached soybean oil (IV = 132) contained 8.3% trienoic-, 51.8% dienoic and 23.6% monoenoic acids. Methyl docosahexaenoate was obtained from Nu-Chek-Prep, Elysian, Minn. Estimated purity was 95%, the major contaminant being C-22:5. Electrolytic hydrogen was used. The Nysel catalyst (Harshaw Chemical Co.) contained 25.3% Ni with a specific Ni surface of 70 m^2/g Ni. Catalyst G-70B, obtained from Girdler Südchemie, W. Germany, contained 21.2% Ni. Germania P (20.1% Ni) was delivered by Oelwerke Germania and the formiate catalyst KE/FR II (25.5% Ni) was obtained from Koenigswarter & Ebell.

RESULTS

Effect of Catalyst Concentration

Unless otherwise stated, all runs are carried out with 30 g mackerel oil and Nysel catalyst at 170 C and atmospheric pressure. Some experimental curves giving ml H_2/g oil consumed as a function of the time of hydrogenation with various catalyst concentrations (% Ni = g Ni/100 g oil) are shown in Figure 2. The experimental points (100-150 for each run) are omitted as they are too numerous to be shown on the small scale of Figure 2. In all cases the hydrogenation reaction started within 30 sec of the addition of the catalyst. The rate of hydrogen consumption increased during the first 30-60 sec. This is not observable on Figure 2, but was seen on a large scale plot from which the differential rates of hydrogenation (ml H_2/g oil/min) at various degrees of saturation were obtained as the slope of the curves. Day to day variations in room (H_2 reservoir) temperature and atmospheric pressure were eliminated by converting the rates to iodine value units (IVU) per minute by means of the equation, IVU/min = (ml H_2/g oil/min) x 0.407(P/T), where P is the atmospheric pressure (mm Hg) and T is the room temperature (K). The corresponding iodine values were calculated from ml H_2/g oil consumed. Occasional analyses of the hydrogenated oil gave excellent agreement with the calculated iodine values.

Figure 3 gives the differential rates of hydrogenation as a function of the iodine value for various catalyst concentrations. The two sets of points on each curve refer to two separate runs. A marked feature of these curves is the strong decrease in the rate during the first minutes of the hydrogenation, followed by a more or less sharp bend in the rate curves around iodine values of 140. This may be taken to indicate that severe poisoning of the catalyst takes place. A hydrogenation experiment with 0.06% Ni was also carried out in a reactor equipped with both a 1500 rpm stirrer and a glass sinter for H_2 dispersion. The rate curve with 0.06% Ni in Figure 3 was reproduced nicely, so the strong decrease in the rate cannot be ascribed to the absence of mechanical stirring. Activation analysis revealed that the oil contained ca. 3.6 ppm bromine, most of which was found to disappear during the early stages of the hydrogenation. Possibly bromine may contribute to the above poisoning.

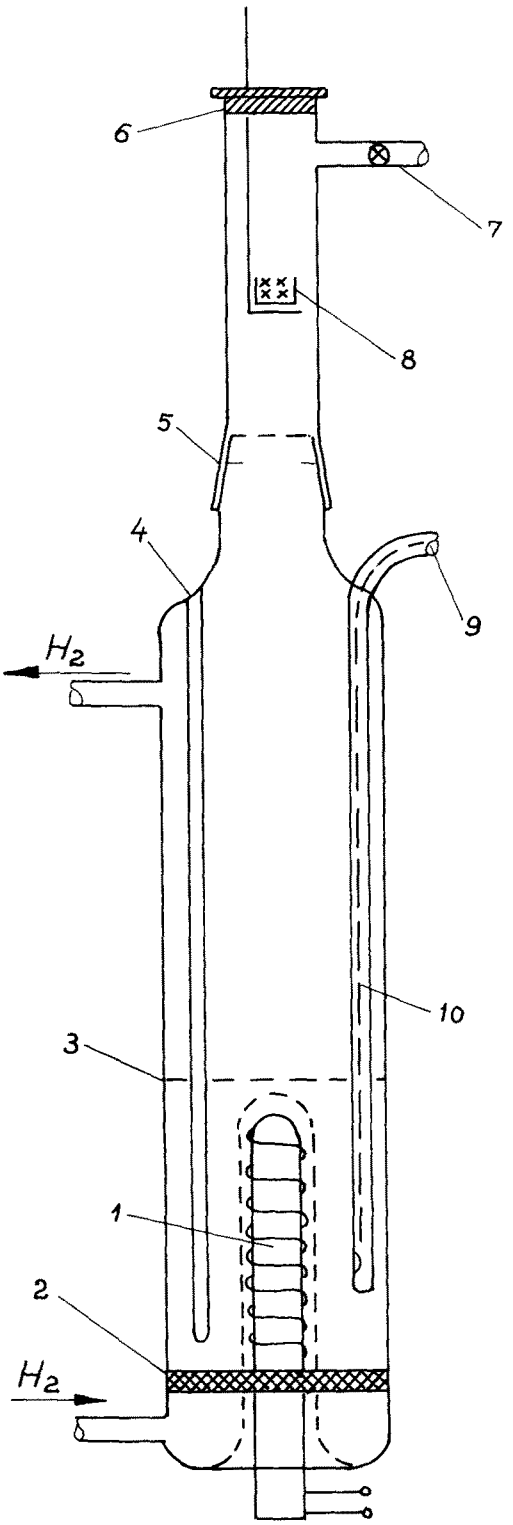

FIG. 1. Hydrogenation reactor. 1. Heat element, 2. glass sinter ring, 3. oil level, 4. pockets for thermocouples, 5. glass joint, 6. self-sealing cap, 7. H_2 rinsing outlet, 8. catalyst beaker, 9. connection to sampling device, 10. capillary tube.

In Figure 4 the differential rates of hydrogenation are plotted versus % Ni for various iodine values. At the lowest iodine values, straight lines intercepting the abcissa at 0.009 % Ni, are obtained. The intercept may be taken to indicate the decrease in the catalyst activity (expressed as the

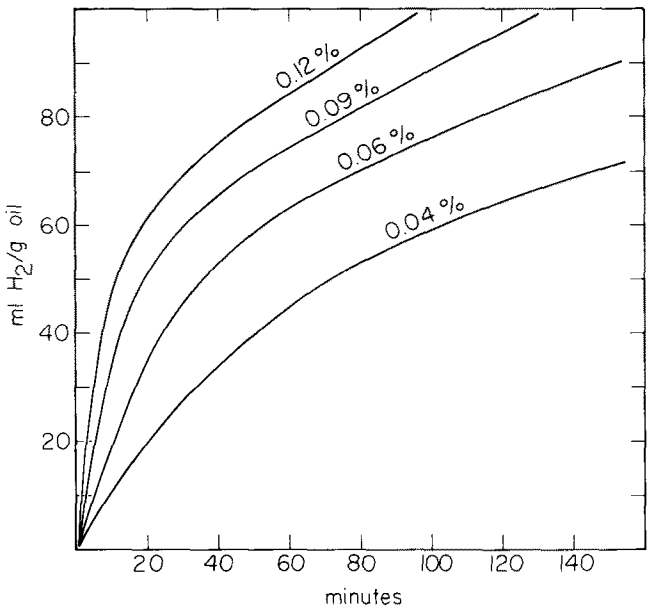

FIG. 2. Hydrogen consumption as a function of time. Catalyst concentrations as indicated. Mackerel oil, Nysel Ni, 170 C, 1 atm.

equivalent % Ni) caused by an initial, rapid adsorption of catalyst poison. This is supported by an experiment carried out with a catalyst concentration of just 0.009 % Ni. As is seen on Figure 4, the rate was approximately zero with this amount of catalyst.

The mackerel oil was found to contain 2.9 ppm sulfur according to Baltes' method, as compared to 4.2 ppm sulfur obtained by the combustion method. The difference of 1.3 ppm may perhaps be explained if the oil contains sulfur compounds that either do not adsorb on the Raney Ni or, alternatively, do not hydrogenolyze on adsorption. Such compounds will probably not be detected by Baltes' method. Analysis (Baltes) of filtered samples of the hydrogenated oil, taken at various iodine values, showed that on the average 0.7 ppm sulfur remained in the oil at iodine values below 130. It would therefore appear that the amount of sulfur adsorbed by the Nysel catalyst is in the region of 2.2-3.5 ppm, the lower value being perhaps the most probable. According to the results reported by Ottesen (15), the above intercept of 0.009% Ni seems to be a reasonable figure for the deactivation due to 2.2-3.5 ppm sulfur. Further, Coenen and Linsen (12) have estimated that 1 mg sulfur deactivates 2.38 m^2 Ni surface. With a specific Ni surface of 70 m^2/g Ni for the Nysel catalyst, a rough calculation again supports the assumption that the intercept of 0.009% Ni is mainly due to a deactivation by sulfur.

A marked feature of Figure 4 is the upward curvature of several of the curves. As the iodine value decreases the curves seem to straighten out; at a higher iodine value, the lower the catalyst concentration. This is quite contrary to what is usually observed in the hydrogenation of triglyceride oils. If the hydrogen mass transport resistance is negligible, a linear relationship between rate and catalyst concentration is expected. If not, the rate should increase less than proportional to the catalyst concentration, as demonstrated by Coenen (3).

The unusual dependence of the rate of hydrogenation on catalyst concentration and iodine value may perhaps be explained in terms of a time-dependent poisoning effect. Obviously the time of contact between the oil and the catalyst at any iodine value is longer the less the catalyst concentration. The degree of catalyst poisoning at a given iodine value will therefore be less the larger the catalyst concentration, thus leading to an upward curvature instead of a straight line. As the equilibrium surface coverage of

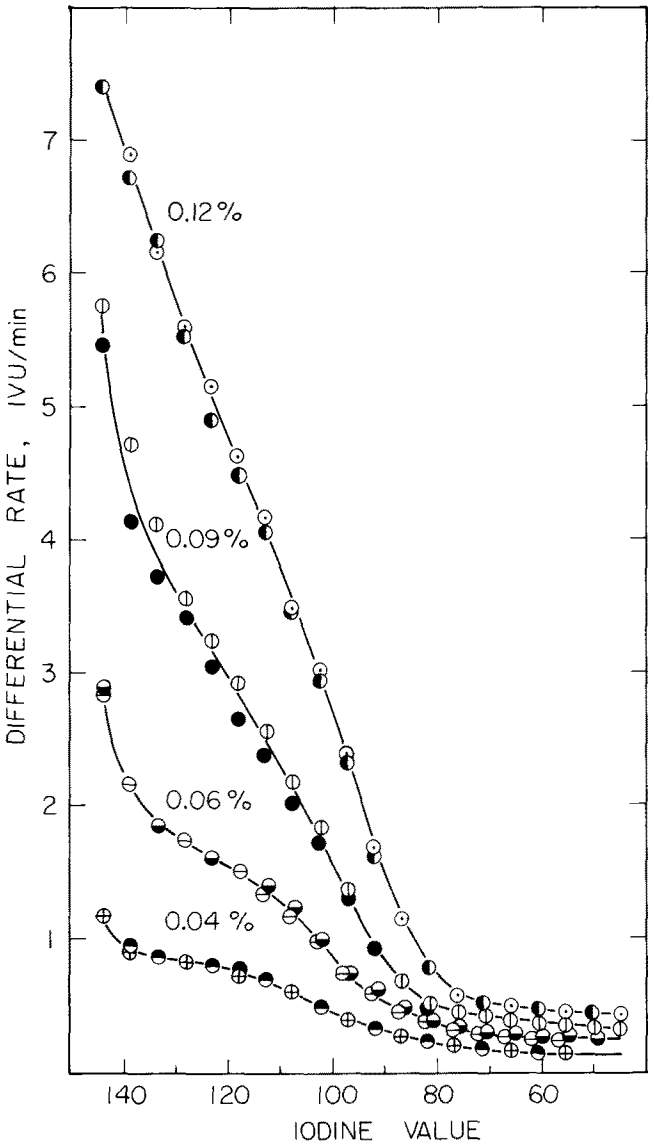

FIG. 3. Differential rates of hydrogenation (iodine value units per min) v. IV. Mackerel oil, Nysel Ni, 170 C, 1 atm. Catalyst concentrations as indicated.

poison is reached, the rate obtained with a given % Ni conforms to a straight line through the points representing the lower catalyst concentrations at the same iodine value. It is seen from Figure 4 that with 0.06% Ni this is the case at an iodine value of ca. 100, with 0.09% Ni at ca. 90, and with 0.12% Ni at ca. 80. In all cases these iodine values are reached after reaction times of 25-30 min, indicating that this is the time necessary to reach the equilibrium coverage.

In connection with Figure 3, a strong decrease in the rate during the first minutes of hydrogenation was noted. Thus, with 0.09% Ni the rate falls from ca. 5.8 IVU/min at IV = 145 to ca. 4.7 IVU/min at IV = 140 in the course of ca. 1 min. This effect is too large to be ascribed to the initial sulfur poisoning which was found to deactivate only 0.009% Ni, and can probably not be accounted for by the relatively slow poisoning effect suggested above. It might seem as if yet another poisoning effect is present. In order to investigate this a little closer, some other experiments were carried out.

Post-Addition Of Catalyst

In the preceding section it was found that 0.009% Ni was deactivated initially, probably by sulfur. If one therefore adds 0.06-0.009 = 0.051% Ni at any point during a run with 0.06% Ni, a doubling of the rate of hydrogena-

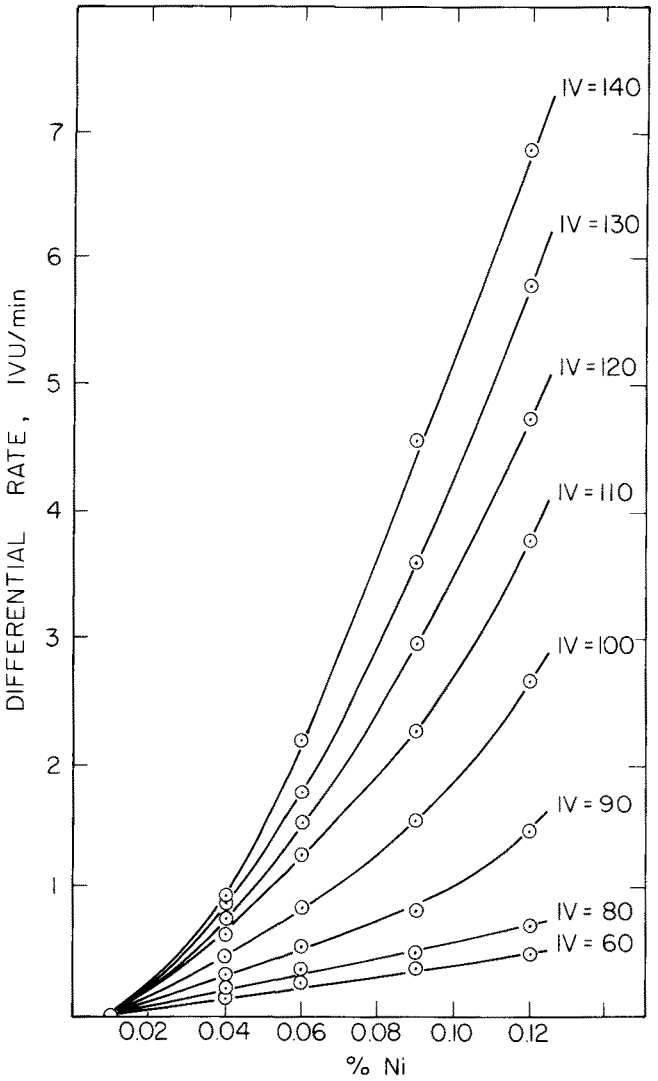

FIG. 4. Differential rates of hydrogenation v. catalyst concentration at various iodine values. Mackerel oil, Nysel Ni, 170 C, 1 atm.

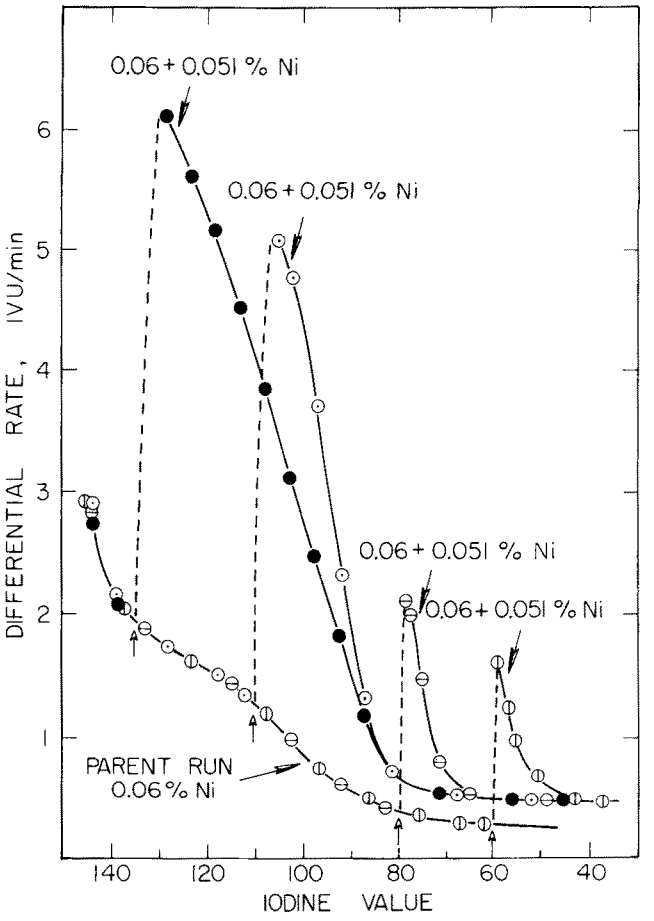

FIG. 5. Post-addition of 0.051% Ni to parent run with 0.06% Ni. In four separate runs, catalyst was added at IV = 130 (●), 110 (⊙), 80 (⊖) and 60 (Φ), respectively. Mackerel oil, Nysel Ni, 170 C, 1 atm.

tion would be expected, provided no other change in the catalyst activity has taken place during the run (poisoning, agglomeration, etc.).

Figure 5 shows four runs in which 0.051% Ni was added to a parent run with 0.06% Ni at iodine values of 135, 110, 80 and 60, respectively. Within ca. 1 min of the post-addition of catalyst, the rate in all cases increased to a maximum value which was from 3.5-6 times larger than that of the parent run, compared at the same iodine value. The rates then decreased to a value about twice that of the parent run. In all four cases the time necessary to reach a rate ratio of two was 25-30 min. Again indications are that the catalyst of the parent run, even at an iodine value of 135, was strongly deactivated and further that the post-added catalyst was exposed to a comparatively slow poisoning which lowered its activity to a level equal to that of the parent run catalyst, i.e., a rate ratio of two was obtained. Similiar results were obtained with higher catalyst concentrations.

Prehydrogenation Experiments

The term prehydrogenation denotes a hydrogenation carried out to a certain predetermined iodine value. The catalyst is then removed by filtration and the hydrogenation is continued with a new portion of catalyst. Again, 0.06% Ni was chosen for the parent run, and for the same reasons as those mentioned in the preceeding section, hydrogenation was continued with 0.051% Ni. If no further deactivation of the parent run catalyst takes place, one would expect that the rate curve obtained after prehydrogenation should coincide with that of the parent run. The results of prehydrogenation to three different iodine values are given in Figure 6. A substantial increase in the rate was obtained on the addition of new catalyst, thus supporting the assumption that the catalyst of the parent run was strongly poisoned. If no poisons other than sulfur were present, the initial rate of the parent run with 0.06% Ni should at least be equal to the maximum rate of 7.3 IVU/min obtained on prehydrogenation to IV = 128.

After a time interval of 25-30 min the curves representing the prehydrogenated runs coincide with the parent run. Evidently the new catalyst has then been poisoned to the same extent as the parent run catalyst, and furthermore the reaction time necessary to reach the assumed equilibrium coverage of poison is the same as that found on the two previous occasions. Some data from Figures 5 and 6 which are of interest in the forthcoming discussion are collected in Table I.

Other Catalysts

Other commercial catalysts were also investigated in order to see if the observed effects were specific for the Nysel catalyst. Experiments similiar to those previously described were carried out with Girdler G-70B, Germania P and the formiate catalyst KE/FR II. Although somewhat less extensive than with the Nysel catalyst, the experiments with these catalysts clearly revealed the existence of the same poisoning effects.

Capelin Oil

A point of major interest would be whether the observed

poisoning effects are specific for the mackerel oil used in the present investigation. Similiar experiments were there fore carried out with capelin oil and Nysel catalyst at 170 C and atmospheric pressure. The results obtained with capelin oil were qualitatively consistent with those found with mackerel oil.

Soybean Oil

For comparison, soybean oil was also investigated along the same lines with Nysel catalyst at 170 C and atmospheric pressure. Figure 7 shows the rate curves obtained with various catalyst concentrations. The shape of the curves is quite similiar to those reported by Coenen (3). A plot of the differential rate v. % Ni would reveal that the rate increases less than proportionally to the catalyst concentration in contrast with the results obtained with mackerel oil (Fig. 3). Prehydrogenations were carried out to IV = 110 and 60, respectively, with 0.02% Ni. No increase in the rate as compared to the parent run, was observed. Some runs were also carried out with the addition of up to 5.5% methyl docosahexaenoate to the soybean oil in order to see if the presence of hexaenoate would bring about any specific poisoning effect. As expected, somewhat higher rates were obtained in the first stage of the hydrogenation due to the increased content of polyunsaturates. At iodine values of ca. 100, however, the rate v. iodine value curves coincided with those obtained with pure soybean oil.

DISCUSSION

The experimental results have established that a substantial loss of catalyst activity is encountered in the hydrogenation of mackerel and capelin oil. With soybean oil no such effect was observed. The decrease in the catalyst activity must therefore be ascribed to the presence of one or more catalyst poisons in the fish oils.

The total catalyst deactivation observed with mackerel oil may *a priori* be regarded as composed of three kinetically distinguishable poisoning effects, namely: (a) initial poisoning, defined as the intercept on the abcissa of the rate v. % Ni plot; (b) primary poisoning, a strong poisoning responsible for a major part of the rapid decrease in the rate above iodine values of ca. 140; (c) secondary poisoning, a comparatively slow poisoning which appears to reach a state of equilibrium in the course of 25-30 min. The initial poisoning is thought to be due mainly to the sulfur compounds present in the oil, in accordance with the results given in a previous section. Possibly bromine may also contribute to this effect. It takes place rapidly and deactivates a certain amount of catalyst (with mackerel oil and Nysel equal to 0.009% Ni), independent of the catalyst concentration. It should be stressed that any poison that acts similar to sulfur (12-15), i.e., that deactivates a certain

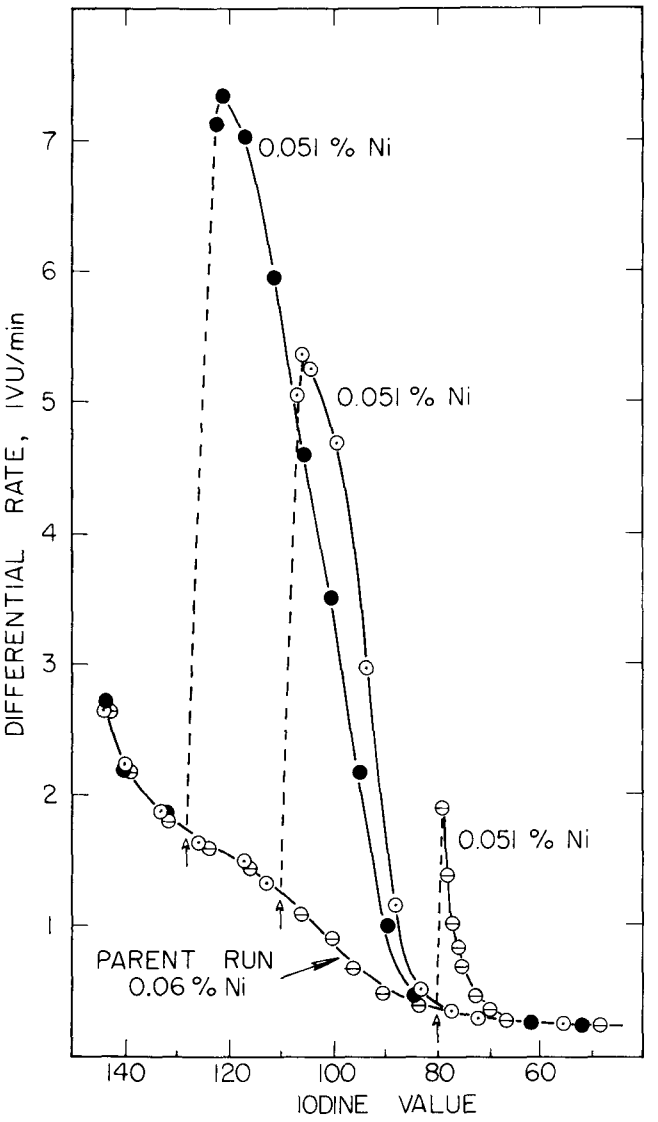

FIG. 6. Effect of prehydrogenation with 0.06% Ni to IV = 128 (●), 110 (⊙) and 80 (⊖), respectively. Hydrogenation continued with 0.051% Ni. Mackerel oil, Nysel Ni, 170 C, 1 atm.

% Ni (g Ni/100 g oil) should increase the intercept on Figure 4, whereas an equilibrium poisoning or a specific poisoning of a certain type of crystal faces or active sites would affect the apparent rate constant (slope of lines in Fig. 4), and only in special cases increase the intercept. As is evident from Figure 4, the intercept does not increase in the course of the hydrogenation and further, the rate

TABLE I

Post-Addition and Prehydrogenation Experiments. Experimental and Calculated Data

Experiment	Prehydrogenation[b]			Post-addition			
0.051 % Ni added at IV[a]	128	110	80	135	110	80	60
Maximum rate obtained at IV	121	104	79	130	107	79	58
Maximum rate (R_M)[c]	7.3	5.45	1.9	6.2	5.1	2.1	1.55
Corresponding rate of parent run (Rp)	1.6	1.05	0.35	1.75	1.2	0.35	0.25
Relative rate increase (R_M/Rp)	4.6	5.2	5.4				
Activity of parent run catalyst (a_P)[d]	$0.22a_o$	$0.19a_o$	$0.185a_o$				
Net relative rate increase, (R_M-Rp)/Rp				2.55	3.25	5.0	5.2
Activity of post-added catalyst (a_A)[e]				$0.55a_o$	$0.62a_o$	$0.92a_o$	

[a]Parent run with 0.06 % Ni.

[b]Parent run catalyst removed before addition.

[c]Rate in IVU/min.

[d]Calculated from equation [1], a_o = activity of unpoisoned catalyst.

[e]Calculated from equation [2].

obtained with 0.009% Ni is approximately zero. Thus the poison(s) responsible for the initial poisoning must be adsorbed in an early stage of the hydrogenation. This is also supported by the prehydrogenation experiments in which the runs with 0.051% Ni coincide with the parent run (0.06% Ni) after 25-30 min.

The first indication of the primary poisoning effect was the sharp drop in the rate during the first 2-3 min of the hydrogenation (Fig. 3). Further support for this effect was obtained from the post-addition and the prehydrogenation experiments.

An approximate calculation of the extent to which the parent run catalyst is poisoned at various iodine values may be performed, based on the maximum rates (R_M) obtained after prehydrogenation. These rates were obtained within ca. 1 min of the addition of new catalyst, and are thus assumed to be representative for the activity of fresh, unpoisoned catalyst (a_O). The activity of the parent run catalyst (a_P) at IV = 121, 104 and 79, respectively, may then be calculated from the equation,

$$R_M/R_P = a_O/a_P \qquad [1]$$

where R_P is the rate of the parent run at the iodine value where R_M is obtained. The values calculated for a_P are given in Table I. It is seen that the activity of the 0.051% Ni remaining after the initial poisoning of the parent run catalyst has decreased to 22% of its original value (a_O) when an iodine value of 121 is reached. Although the secondary poisoning also contributes to this decrease, the greater part is thought to be caused by the primary poisoning during the first 2-3 min of the hydrogenation. The comparatively slight decrease in activity from IV = 121 to IV = 79 represents the last phase of the secondary poisoning. As mentioned in connection with Figure 4, the secondary poisoning seems to reach an equilibrium coverage at approximately IV = 100.

Figure 5 shows that the rate obtained after prehydrogenation to IV = 128 decreases from a maximum value of 7.3 IVU/min to 0.35 IVU/min at IV = 79. At this point the rate curve coincides with the parent curve. Consequently the catalyst activity is equal to $0.185a_O$, as calculated above. In this case the greater part of the decrease in activity from a_O to $0.185a_O$ must be ascribed to the secondary poisoning for the following reasons: most of the primary poison is probably removed with the parent run catalyst; the sharp initial decrease in the rate observed in the parent run (ascribed to the primary poisoning) is not present; and the rate curve coincides with the parent curve 25-30 min after the addition of catalyst, in accordance with the previously established time necessary to complete the secondary poisoning.

As can be seen from Table I, the effect of the catalyst added after prehydrogenation to IV = 128 (R_M/R_P) is about twice that of the catalyst added in the corresponding post-addition experiment ($[R_M-R_P]/R_P$). At IV = 110 this difference is somewhat less, and at IV = 80 the effect is about the same in both cases. Disregarding for the moment the last result, it is obvious that the mere presence of the parent run catalyst leads to a considerable reduction of the activity of the post-added catalyst. As the parent run catalyst has been shown to be strongly poisoned, it seems reasonable to ascribe this result to a rapid redistribution of primary poison from the parent run catalyst to the post-added catalyst. This explanation necessitates the assumption that certain Ni crystal faces (or active sites) have a substantially higher catalytic activity than others, and furthermore that the primary poison is present in excess of these sites. Such a nonuniform distribution of the catalytic activity of metal surfaces has been observed experimentally for several reactions, including the hydrogenation of ethylene on nickel (18). On post-addition of catalyst, primary

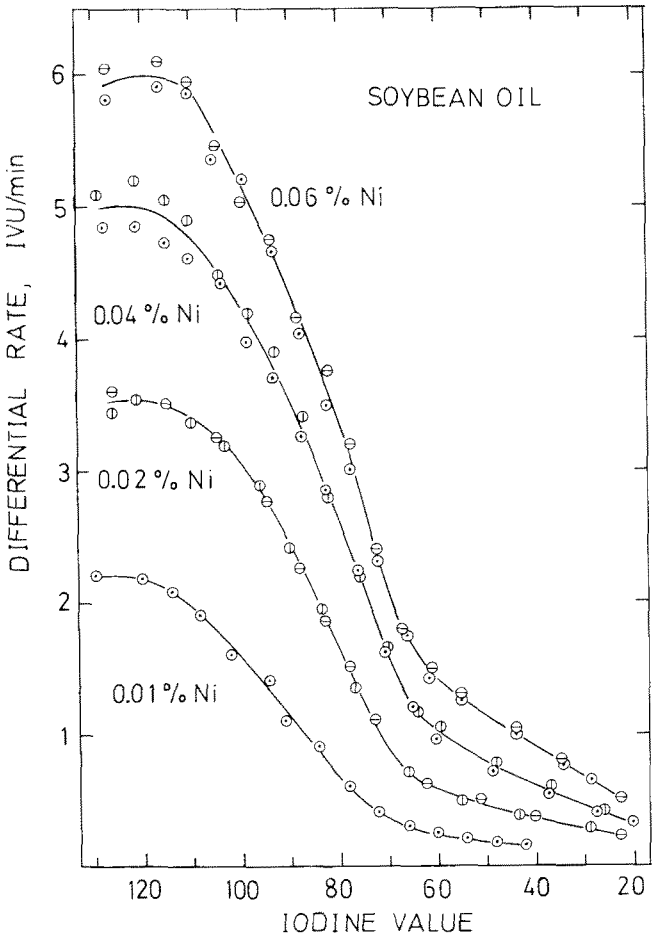

FIG. 7. Differential rates of hydrogenation as a function of IV. Catalyst concentrations as indicated. Soybean oil, Nysel Ni, 170 C, 1 atm.

poison may transfer from the less active sites on the original catalyst to the sites of high activity on the added catalyst, perhaps due to a higher heat of adsorption on the most active sites.

The magnitude of this "instantaneous" deactivation may be estimated from the equation,

$$(R_M-R_P)/R_P = a_A/a_P \qquad [2]$$

where a_A is the activity of the post-added catalyst at the iodine value where the maximum rate is obtained, and a_P is the corresponding activity of the parent run catalyst, previously calculated from the prehydrogenation experiments at almost the same iodine values. From Table I it is seen that the "instantaneous" deactivation of the post-added catalyst decreases from ca. 45% at IV = 130 to ca. 8% of the fresh catalyst activity at IV = 79. This was surprising, as it seemed unlikely that the effect of the redistribution should depend on the iodine value. A possible explanation may be found by considering the fatty acid composition at the various iodine values. While the content of conjugatable polyunsaturates (dienoic through hexaenoic) at the two highest iodine values was ca. 25 and 15%, respectively, the corresponding figure at IV = 79 was less than 2%, most of which was dienes. In accordance with the previous assumption of an energetically nonuniform catalyst surface, the active sites subject to poisoning by redistribution may be far more reactive towards conjugatable polyenes than towards monoenes or nonconjugatable polyenes. According to current views (11) on the mechanisms of hydrogenation, conjugated species may well be important intermediates. The above explanation thus involves that certain crystal faces or active sites are

especially suited for the adsorption and formation of the pentadiene structures or butadienyl complexes invoked in the suggested mechanisms.

The "instantaneous" deactivation observed on the post-addition of catalyst seems to justify the distinction between a primary and a secondary poisoning effect. It would appear that in the post-addition run, some catalyst poison must be present that was not there during the prehydrogenation experiment in which the poisoned parent run catalyst was removed. The primary and the secondary poisoning effects can therefore hardly be ascribed to only one kind of poison, adsorbing with different rates at various locations in the catalyst pores due to pore diffusion effects.

As a conclusion, however, it may be said that although three poisoning effects can be distinguished kinetically, the poisoning mechanisms advanced above are suggestive rather than conclusive. Consequently other mechanisms may well be possible.

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