# Promotion effect of 2-methylpiperidine on the direct desulfurization of dibenzothiophene over NiMo/$\gamma$-$Al_2O_3$

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The hydrodesulfurization (HDS) of dibenzothiophene (DBT) was studied at 300 °C, 4.8 MPa $H_2$ and 35 kPa $H_2S$ in the presence of 0 to 6 kPa 2-methylpyridine and 2-methylpiperidine. 2-Methylpyridine suppressed the hydrogenation pathway by a factor of 6 to 30, depending on its partial pressure, and moderately inhibited the direct desulfurization pathway of the HDS of DBT (by a factor of 2 or less). 2-Methylpiperidine suppressed the hydrogenation by a factor of 10 to 50 but promoted the direct desulfurization of DBT at low partial pressures of 2-methylpiperidine. Both pathways were inhibited at high concentrations of 2-methylpiperidine. Structural and electronic factors may account for the promoting effect of 2-methylpiperidine.

**KEY WORDS:** hydrotreating; hydrodesulfurization; HDS; dibenzothiophene; DBT; hydrodenitrogenation; HDN; 2-methylpyridine; 2-methylpiperidine; promoting effect of N-compound.

## 1. Introduction

The long-term trend in the petroleum industry is to process heavier feedstocks that contain a high percentage of sulfur and nitrogen. Therefore, knowledge of the mutual influence of hydrodesulfurization (HDS) and hydrodenitrogenation (HDN) is becoming more important. Studies on competitive HDS and HDN have shown that N-containing molecules strongly inhibit HDS reactions [1–12], because of their high adsorption constants on the catalyst surface. Moreover, the inhibitory effect of N-compounds depends on the nature of the molecule. In some cases, molecules with a different number or location of substituents have a different inhibitory influence. Thus, 2,6-dimethylpyridine was a weaker inhibitor in the HDS of thiophene than 4-methylpyridine and 3,5-dimethylpyridine [6,13]. This led to the conclusion that the inhibiting effect of pyridines on HDS occurs when these molecules are adsorbed perpendicular to the catalyst surface, since in this conformation the adsorption of 2,6-dimethylpyridine would be the weakest because of the steric hindrance of the methyl groups [13]. However, pyridines may undergo hydrogenation under HDS reaction conditions. As a consequence, if 2,6-dimethylpyridine were hydrogenated faster than the other substituted pyridines, its inhibitory influence would be the weakest, because of the weaker adsorption of 2,6-dimethylpiperidine in a one-point mode with steric hindrance due to the two methyl groups on the $\alpha$ and $\alpha'$ carbon atoms. The inhibition of HDS has also been correlated with the basicity of N-containing molecules [6], and it was observed that the more basic molecules have the strongest inhibitory influence. Nagai *et al.* found a correlation with gas-phase proton affinities rather than with solution basicities [4].

Dibenzothiophene (DBT) is often used as a model compound for HDS, since it is a good representative of the S-containing molecules in middle and heavy distillates. The HDS of DBT consists of two reaction pathways: (i) direct desulfurization (DDS) leading to the formation of biphenyl, and (ii) hydrogenation (HYD) of DBT to tetrahydrodibenzothiophene followed by desulfurization to cyclohexylbenzene. Several research groups reported that N-containing molecules inhibit the DDS and the HYD pathways of the HDS of DBT to different extents: the HYD route was strongly suppressed, whereas the DDS route was less affected [4,14–17]. In some cases, even a promotion of the DDS pathway was observed, whereas the total conversion of the S-compound decreased [4,14,15]. Nagai reported a real promotion of the DDS of DBT over NiMo/$Al_2O_3$ and NiW/$Al_2O_3$ catalysts, i.e., the enhancement of the overall conversion of DBT in the presence of acridine, but gave no explanation [16,17].

At 340 °C, 4.8 MPa $H_2$ and 35 kPa $H_2S$, we observed a strong inhibition of the HYD pathway of the HDS of DBT at different partial pressures of 2-MPy and 2-MPiper and an enhancement of the biphenyl formation in the presence of 2 kPa 2-MPy and 2-MPiper [18]. This promotion was explained by the higher amount of DBT available for the DDS because of suppression of the HYD pathway. We proved this with calculations in which it was assumed that the rate constant of the DDS route was not affected at low concentration of N-

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compounds and that the HYD pathway was totally blocked. In our previous study [18], we found that 2-MPiper has a somewhat stronger inhibitory influence on the HYD route than 2-MPy. The DDS pathway was hardly affected at 2 kPa and only slightly retarded at 6 and 10 kPa 2-MPy and 2-MPiper. Moreover, the inhibitory effect of 2-MPy on the DDS was stronger than that of 2-MPiper. Since the HDS of DBT goes mainly via the DDS pathway, 2-MPy was also the stronger poison for the overall HDS of DBT.

In the present work, we studied the effect of 2-MPy and 2-MPiper on the HDS of DBT in more detail. We extended the partial pressure range of the N-containing molecules in the feed to clarify the mechanism of the poisoning influence and also decreased the temperature to 300 °C. At the lower temperature the inhibitory effect of N-containing molecules should be more pronounced and can therefore be studied better. While at 340 °C, 2-MPy reacts to 2-MPiper and 2-MPiper reacts further to acyclic amines and hydrocarbons, at 300 °C, 2-MPy is only hydrogenated to 2-Mpiper, and the reaction is irreversible because of thermodynamics. The cleavage of the C–N bond does not take place at 300 °C under our conditions.

## 2. Experimental

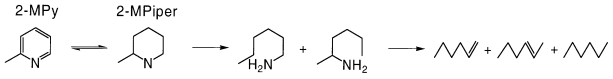
The NiMo/$\gamma$-$Al_2O_3$ catalyst used in this work contained 8 wt% Mo and 3 wt% Ni and was prepared by successive incipient wetness impregnation of $\gamma$-$Al_2O_3$ (Condea, pore volume 0.5 $cm^3g^{-1}$, specific surface area 230 $m^2g^{-1}$) with aqueous solutions of $(NH_4)_6Mo_7O_{24}\cdot 4H_2O$ and $Ni(NO_3)_2\cdot 6H_2O$ (both Aldrich). After each impregnation step the catalyst was dried in air at ambient temperature for 4 h, then dried in an oven at 120 °C for 15 h and finally calcined at 500 °C for 4 h.

Reactions were carried out in a continuous mode in a fixed-bed inconel reactor as described previously [18]. The experiments were carried out at 300 °C. The feed consisted of 130 kPa toluene (as solvent for the DBT and amine), 8 kPa dodecane (as reference for DBT and its derivatives in the gas chromatography (GC) analysis), 11 kPa heptane (as reference for the N-compounds in the GC analysis), 1 kPa dibenzothiophene, 0.1–6 kPa amine reactant (2-MPy or 2-MPiper), 35 kPa $H_2S$ and 4.8 MPa $H_2$.

The reaction products were analyzed by on- and off-line gas chromatography, as described in [18]. Weight time was defined as $\tau = w_{cat}/n_{feed}$, where $w_{cat}$ denotes the catalyst weight and $n_{feed}$ the total molar flow to the reactor. The weight time ($\tau$) was changed by varying the flow rates of the liquid and the gaseous reactants while keeping their ratio constant. The reaction was stable after 3 to 4 h, and during two weeks of operation almost no catalyst deactivation was observed. The HDS of DBT alone and in the presence of the N-containing molecules could be well described with a first-order kinetic model with respect to DBT. This fact is in good agreement with literature [8].

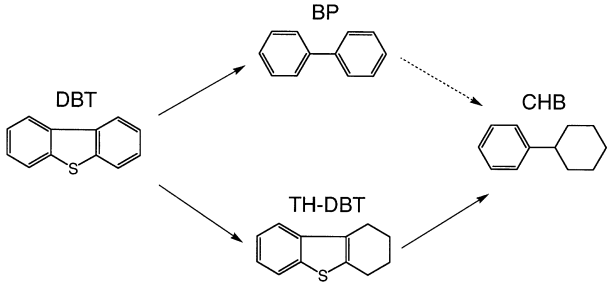
## 3. Results

2-MPy and 2-MPiper were chosen as N-containing molecules because the methyl group on the $\alpha$-carbon atom of pyridine strongly suppresses the disproportionation reaction of two molecules of piperidine, the first intermediate in the HDN of pyridine, to N-pentylpiperidine and ammonia [19]. Thus, the overall reaction network of 2-MPy and 2-MPiper is less complicated than that of pyridine and piperidine, and is schematically presented in scheme 1.




Scheme 1. Reaction network of the HDN of 2-methylpyridine and 2-methylpiperidine.

As mentioned in the introduction, DBT undergoes HDS via two reaction pathways: DDS and HYD. At 300 °C, the selectivity toward biphenyl formation is about 85% at low-weight time and 80% at high-weight time [18]. These results indicate that the DDS route is much easier than the HYD one, and that slow hydrogenation of biphenyl to cyclohexylbenzene takes place in the presence of a NiMo/$\gamma$-$Al_2O_3$ catalyst. Therefore, the overall reaction network of the HDS of DBT is as shown in scheme 2.




Scheme 2. Reaction network of the HDS of dibenzothiophene.

The results of the HDS of 1 kPa DBT in the presence of 0.1, 0.5, 1, 2 and 6 kPa of 2-MPy are presented in figure 1. The overall conversion of DBT was slightly decreased at the lowest partial pressure of 0.1 kPa 2-MPy and it decreased further with increasing 2-MPy partial pressure. The HYD pathway of the HDS of DBT was already strongly suppressed at 0.1 kPa 2-MPy. Its selectivity had decreased from 15% in the absence of 2-MPy to only 4% at 0.1 kPa 2-MPy and 2% at 6 kPa 2-MPy. Thus, the HYD route is greatly inhibited at all 2-MPy concentrations. The selectivity toward biphenyl

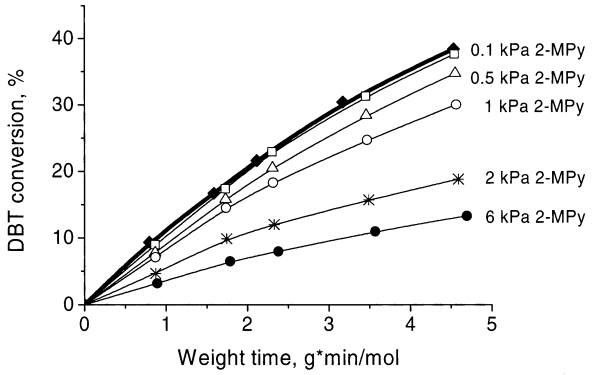



Figure 1. Total conversion of 1 kPa DBT at 300 °C and different 2-methylpyridine partial pressures. The heavy line represents the results for DBT alone.

formation stayed constant in the course of one single competitive experiment, showing that the hydrogenation of biphenyl to cyclohexylbenzene is also inhibited in the presence of 2-MPy.

The conversion to biphenyl in the presence and absence of 2-MPy is shown in figure 2. It is clear that the biphenyl production was higher in the presence of 0.1, 0.5 and 1 kPa 2-MPy than in its absence. A direct comparison of the conversion to biphenyl in the presence of 2-MPy with the conversion to biphenyl in the absence of 2-MPy is not meaningful; however, since in the first case biphenyl is the final product, whereas in the second case it is partly converted further to cyclohexylbenzene. Furthermore, a higher conversion to biphenyl (eventually 96–98%) is possible in the presence of 2-MPy because the HYD route is almost completely suppressed in the presence of 2-MPy. We calculated the theoretical conversion to biphenyl, assuming that the rate constant of the DDS pathway is not affected by the presence of 2-MPy and that the HYD pathway is fully suppressed (dashed bold line in figure 2). If we now compare the observed conversions

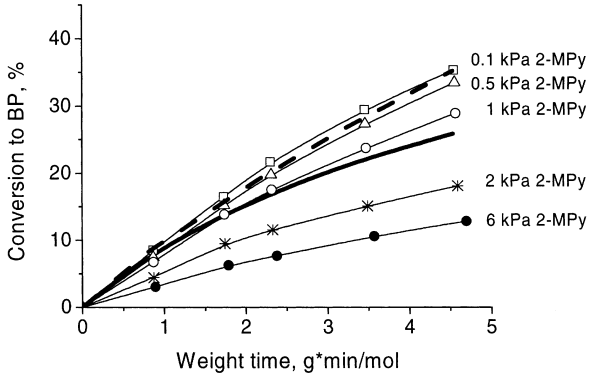



Figure 2. Conversion to biphenyl in the HDS of DBT at 300 °C and different 2-methylpyridine partial pressures. The heavy line represents the conversion to biphenyl in the absence of 2-MPy. The heavy dashed line represents the conversion to biphenyl in the absence of 2-MPy corrected for further hydrogenation of biphenyl and for the absence of the HYD pathway.

to biphenyl with this theoretical estimate, we see that the DDS activity is hardly affected at 0.1 kPa 2-MPy and decreases at higher 2-MPy partial pressures. Therefore, after correction of these "trivial" effects, it is clear that 2-MPy does not promote the formation of biphenyl. 2-MPy suppresses not only the HYD pathway but also the DDS pathway, be it to a smaller extent. The inhibitory influence of 2-MPy on the DDS of DBT could not be described with a Langmuir–Hinshelwood model with one adsorption constant. The inhibiting effect increased with the partial pressure of 2-MPy, as if its adsorption constant depended on its partial pressure. We will return to this problem in the following text.

Also, in the HDS of 1 kPa DBT in the presence of 0.1, 0.5, 1, 2 and 6 kPa 2-Mpiper, the HYD pathway was strongly suppressed. The selectivity to HYD products decreased from 15% in the absence of 2-MPiper to 3% at 0.1 kPa and to 1% at 6 kPa 2-MPiper. These values show that 2-MPiper has a somewhat stronger inhibitory influence on the HYD pathway than 2-MPy. As was observed in the case of 2-MPy, 2-MPiper also inhibited the hydrogenation of biphenyl to cyclohexylbenzene.

The conversion of DBT was enhanced in the presence of 0.1, 0.5 and 1 kPa 2-MPiper and decreased in the presence of 2 and 6 kPa 2-MPiper (figure 3). The formation of biphenyl was higher at all partial pressures of 2-MPiper than in the absence of 2-MPiper (figure 4). After correcting for the further hydrogenation of biphenyl and for the suppression of the HYD pathway (see above), the DDS conversion was still enhanced at 0.1, 0.5 and 1 kPa of 2-MPiper (figure 4). These results show that below 2 kPa 2-MPiper the DDS route of the HDS of DBT is promoted, since the total conversion of DBT is enhanced and the yield of biphenyl is higher than theoretically possible. Therefore, the influence of 2-MPiper on the HDS of DBT cannot be described with a Langmuir–Hinshelwood model either, since we deal with a promotion at low and inhibition at high partial pressures of 2-MPiper.

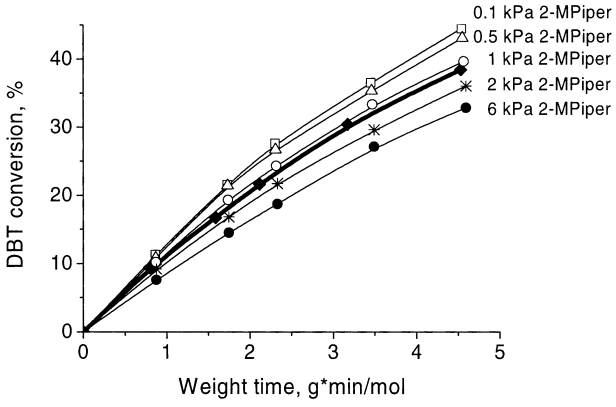



Figure 3. Total conversion of 1 kPa DBT at 300 °C and different 2-methylpiperidine partial pressures. The heavy line represents the results for DBT alone.

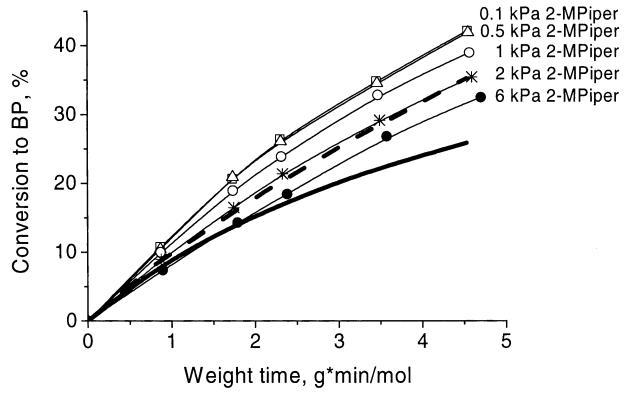



Figure 4. Conversion to biphenyl in the HDS of DBT at 300 °C and different 2-methylpiperidine partial pressures. The heavy line represents the conversion to biphenyl in the absence of 2-MPiper. The heavy dashed line represents the conversion to biphenyl in the absence of 2-MPiper corrected for further hydrogenation of biphenyl and for the absence of the HYD pathway.

2-MPiper is not converted under our reaction conditions (300 °C, 4.8 MPa $H_2$ and 35 kPa $H_2S$), whereas the conversion of 2-MPy to 2-MPiper varied from 85 to 30% at the highest weight time ($\tau = 4.5$ g min/mol) when increasing the 2-MPy partial pressure in the feed from 0.1 to 6 kPa. This may explain the nonlinear inhibitory influence of 2-MPy on the DDS pathway, since we actually deal with inhibition and promotion at the same time. At the lowest partial pressure of 0.1 kPa 2-Mpy, the inhibitory effect of 2-MPy is compensated by the promotion because of the 2-MPiper formed. Above 1 kPa, not only 2-MPy but also 2-MPiper acts as a poison for the DDS of DBT. Thus, 2-MPy would have almost no inhibitory effect at low partial pressure and a strong inhibitory effect at high partial pressure.

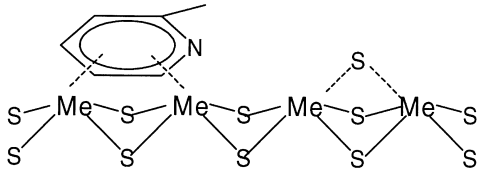
## 4. Discussion

The enhancement of the DDS pathway in the HDS of DBT at low partial pressures of 2-MPiper can only be explained if we assume that the catalyst surface is changed and that an increased number of active sites are created. This means that either the active catalyst surface becomes rougher due to the adsorption of small amounts of 2-MPiper, or that the number of DDS sites is increased by a transformation of HYD sites to DDS sites. How can such a transformation take place and what are the active sites for the DDS and HYD pathways?

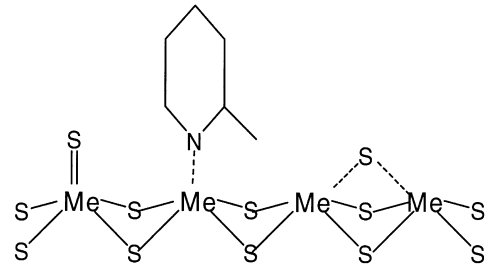
It is generally assumed that the catalytically active sites in Mo/$\gamma$-$Al_2O_3$ hydrotreating catalysts are the molybdenum atoms at the edges and corners of the $MoS_2$ crystallites, which have at least one sulfur vacancy to allow chemical adsorption of the reacting molecule on the molybdenum atom [20–22]. Upon addition of nickel and cobalt, the HDS and HDN activities of a $MoS_2/\gamma$-$Al_2O_3$ catalyst increase substantially. Density functional theory (DFT) calculations showed that the most stable position for the promoter atom (Co or Ni) is at the edge, substituting the molybdenum atom [23], forming the so-called Co-Mo-S phase (Ni-Mo-S phase for Ni-Mo catalysts) [22]. DFT calculations suggest that a combined action of the promoter (Ni or Co) and molybdenum atom is responsible for the catalysis [23–25]. It was shown that a sulfur atom between a nickel (or cobalt) and a molybdenum atom is less strongly bonded than a sulfur atom between two molybdenum atoms and can be more easily removed, creating a vacancy.

Therefore, the catalytically active sites in our system are the nickel or molybdenum atoms present at the molybdenum edge. One vacancy at a metal atom could be enough to perform the DDS of DBT, in which case the molecule is adsorbed in a one-point way perpendicular to the catalyst surface. However, when DBT is $\pi$-adsorbed and follows the HYD pathway, it needs more space and at least two neighboring vacancies must be available.

Most likely, 2-MPy is adsorbed in a flat conformation through its aromatic $\pi$-system and is coordinated by two metal atoms, as shown in scheme 3. 2-MPiper has no $\pi$ electrons and can, therefore, only adsorb through the nitrogen atom in a one-point manner (scheme 4). When




Scheme 3. Flat adsorption of 2-methylpyridine on the HYD active site.




Scheme 4. One-point adsorption of 2-methylpiperidine on the DDS active site.

2-MPiper is adsorbed on a HYD site, consisting of several S-free metal atoms, it leaves the neighboring metal atom free. This metal atom is not available for hydrogenation of DBT, because hydrogenation needs at least two neighboring free sites, but it is available for the one-point adsorption of DBT and, thus, for the DDS pathway of the HDS of DBT. In this way, a HYD active site can transform into a DDS site at low partial pressures of 2-MPiper. This would explain the promo-

tion of the direct sulfur removal route in the HDS of DBT. At higher concentration, 2-MPiper blocks both metal centers on the HYD active site, thereby also decreasing the DDS rate of the HDS of DBT.

A similar explanation can be given in terms of edge- and corner-active sites. Let us assume that edge centers are responsible for the DDS pathway or for the perpendicular adsorption of the reactant, whereas the corner sites situated between two edges have more room for adsorption and can therefore coordinate the molecule in a flat mode and perform the HYD pathway. When 2-MPy is present in the feed, both DDS and HYD sites are poisoned. On the other hand, when 2-MPiper adsorbs on an HYD site, it leaves room available for the DDS.

Another explanation for the DDS improvement could be an electronic modification of the catalyst surface. The adsorption of 2-MPiper may lead to an increase in the electron density on the metal atoms, which, in turn, can cause an increase in the number of sulfur vacancies on the Ni-Mo-S surface or an increase in the intrinsic catalytic activity of the site. In other words, low partial pressures of 2-MPiper result in an increased number of DDS sites or in an increase in their activity. At higher concentrations of 2-MPiper, the N-compound adsorbs on more and more sites, inhibiting the total conversion of DBT.

Two other explanations seem less likely. One is that the improvement of the DDS of DBT in the presence of 2-MPiper could result from an acid–base interaction between 2-MPiper and DBT. This does not seem likely because the lone pair of the 2-MPiper is bonded to the catalyst surface and is not available for an interaction with DBT. Another explanation could have been that the N-containing molecule acts a mediator, as in electrocatalytic promotion [26]. Whereas such a type of promotion would have been possible for a positive influence of 2-MPy on the HYD pathway of the HDS of DBT, it is not possible for 2-MPiper in the DDS pathway.

## 5. Conclusion

Both 2-MPy and 2M-Piper poisoned the HYD pathway of the HDS of DBT greatly, but the inhibitory influence of 2-MPiper was somewhat stronger than that of 2-MPy. The DDS route of the HDS of DBT was suppressed in the presence of 2-MPy and promoted at low partial pressures of 2-MPiper. The total conversion of DBT was also enhanced in the presence of 0.1, 0.5 and 1 kPa 2-MPiper.

The enhancement of the DDS pathway at low partial pressures of 2-MPiper can be explained in three ways: (i) transformation of HYD sites into DDS sites because the HYD site consists of several metal centers and is not completely covered after adsorption of 2-MPiper in the one-point mode; (ii) electronic modification of the catalyst surface, resulting in an increase in the electron density on the metal centers due to interaction with the 2-MPiper molecules that leads to an increased number of sulfur vacancies or to an increased intrinsic activity of the active site; (iii) interaction between 2-MPiper and DBT (acid–base interaction) when they are both adsorbed perpendicular to the catalyst surface.

## References

[1] F.W. Kirsch, H. Shalit and H. Heinemann, Ind. Eng. Chem. 51 (1959) 1379.
[2] C.N. Satterfield, M. Modell and J.F. Mayer, AICHE J. 21 (1975) 1100.
[3] C.N. Satterfield, M. Modell and J.A. Wilkens, Ind. Eng. Chem. Proc. Des. Dev. 19 (1980) 154.
[4] M. Nagai, T. Sato and A. Aiba, J. Catal. 97 (1986) 52.
[5] J. Yang and F.E. Massoth, Appl. Catal. 34 (1987) 215.
[6] V. La Vopa and C.N. Satterfield, J. Catal. 110 (1988) 375.
[7] F. Gioia and F. Murena, J. Hazard. Mater. 57 (1998) 177.
[8] G.C. Laredo, J.A. De los Reyes, J.L. Cano and J.J. Castillo, Appl. Catal., A 207 (2001) 103.
[9] C. Kwak, J.J. Lee, J.S. Bae and S.H. Moon, Appl. Catal., B 35 (2001) 59.
[10] P. Zeuthen, K.G. Knudsen and D.D. Whitehurst, Catal. Today 65 (2001) 307.
[11] T. Koltai, M. Macaud, A. Guevara, E. Schulz, M. Lemaire, R. Bacaud and M. Vrinat, Appl. Catal., A 231 (2002) 253.
[12] G.C. Laredo, E. Altamirano and J.A. De los Reyes, Appl. Catal., A 243 (2003) 207.
[13] H. Kwart, J. Katzer and J. Horgan, J. Phys. Chem. 86 (1982) 2641.
[14] M. Nagai and T. Kabe, J. Catal. 81 (1983) 440.
[15] V. La Vopa and C.N. Satterfield, Chem. Eng. Commun. 70 (1988) 171.
[16] M. Nagai, Ind. Eng. Chem. Prod. Res. Dev. 24 (1985) 489.
[17] M. Nagai, Chem. Lett. 6 (1987) 1023.
[18] M. Egorova and R. Prins, J. Catal. (2003) to be published.
[19] M. Egorova, Y. Zhao, P. Kukula and R. Prins, J. Catal. 206 (2002) 263.
[20] R. Prins, V.H.J. de Beer and G.A. Somorjai, Catal. Rev.-Sci. Eng. 31 (1989) 1.
[21] R.R. Chianelli, M. Daage and M.J. Ledoux, Adv. Catal. 40 (1994) 177.
[22] H. Topsøe, B.S. Clausen and F.E. Massoth, in *Catalysis: Science and Technology*, Vol. 11 (Springer, Berlin, 1996).
[23] P. Raybaud, J. Hafner, G. Kresse, S. Kasztelan and H. Toulhoat, J. Catal. 190 (2000) 128.
[24] L.S. Byskov, J.K. Nørskov, B.S. Clausen and H. Topsøe, J. Catal. 187 (1999) 109.
[25] P. Raybaud, J. Hafner, G. Kresse, S. Kasztelan and H. Toulhoat, J. Catal. 189 (2000) 129.
[26] M.M. Baizer, H. Lund, in *Organic Electrochemistry* (Marcel Dekker Journals, New York, 1983).