Catal Lett (2009) 129:50–60
DOI 10.1007/s10562-008-9777-x

Deep HDS of Diesel Fuel: Inhibiting Effect of Nitrogen Compounds on the Transformation of the Refractory 4,6-Dimethyldibenzothiophene Over a $NiMoP/Al_2O_3$ Catalyst

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Received: 27 August 2008 / Accepted: 11 November 2008 / Published online: 13 December 2008

Abstract The effect of nitrogen-containing compounds (acridine and 1,4-dimethylcarbazole: 14DMCARB) on the hydrodesulfurization of 4,6-dimethyldibenzothiophene (46DMDBT) was studied on a sulfided $NiMoP/Al_2O_3$ catalyst in a fixed bed microreactor (340 °C, 40 bar). Both nitrogen-containing compounds inhibited the hydrodesulfurization of 46DMDBT. However, the effect of acridine (a basic compound) and mainly its hydrogenated product (presumably 1,2,3,4,5,6,7,8-octahydroacridine: OHA1) was much more significant than the effect of 14DMCARB (a non-basic compound). In the presence of acridine, the so-called direct desulfurization pathway (DDS) of the HDS of 46DMDBT was less affected than the hydrogenation pathway (HYD) and was even slightly promoted when the partial pressure of acridine increased (after a strong initial inhibition). This was ascribed to a cocatalytic contribution of the nitrogen-containing compound to the C–S bond cleavage. 14DMCARB had the same inhibiting effect on both pathways (DDS and HYD). We also demonstrated that acridine inhibited the transformation of 14DMCARB and can explain why carbazoles are generally the main nitrogen impurities present in gasole after hydrotreatment.

Keywords Hydrodesulfurization · Gasole · Acridine · Carbazole · $NiMoP/Al_2O_3$ catalyst · 4,6-Dimethyldibenzothiophene

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1 Introduction

It is well known that emissions (NOx and SOx) from motor vehicles widely contribute to air pollution. To address this environmental problem, new restrictive regulations were adopted by the European community [1]. For instance, the sulphur level in diesel fuel and gasoline will have to be lowered to 10 ppm by 2009. Moreover, sulphur impurities contained in diesel fuels lead to sulphur oxides and then sulphates formation after combustion in the engine. Sulphates are known to be irreversible poisons for the catalytic converters used for exhaust emission treatment, especially for the NOx trap systems. It is now well established that main sulphur compounds remaining in hydrotreated diesel fuels at sulphur levels of less than 250 wt ppm are almost exclusively substituted dibenzothiophenes [2–5]. The HDS of such compounds which are among the most refractory sulphur impurities is very sensitive to the presence of other molecules in the feed (even in small amounts), especially heterocyclic nitrogen compounds [6–17]. Despite the fact that deeply desulphurised diesel oils mostly contain carbazole and its alkylated derivatives, it was found that these compounds did not have a major effect on the rate of HDS [10]. According to previous studies, the more powerful nitrogen containing compound inhibitor would be acridine, whereas anilines, quinolines and benzoquinolines would have only a moderate inhibiting effect [6, 8]. In order to increase the performances of hydrotreating catalysts for the HDS of the most refractory sulphur compounds, it can be useful to identify the nitrogen containing compounds that are really inhibitors and to better understand the mechanism of the inhibition.

In the present work, the effect of nitrogen compounds (acridine as a basic compound and 1,4-dimethylcarbazole (14DMCARB) as a non basic one) on

the hydrodesulfurization of 4,6-dimethyldibenzothiophene (46DMDBT) was studied under conditions close to those used in the deep hydrotreating of diesel fuels on a NiMoP catalyst.

2 Experimental

2.1 Catalyst and Chemicals

The catalyst was a commercial $NiMoP/Al_2O_3$ catalyst containing 3 wt% NiO, 16 wt% MoO_3 and 6% P_2O_5. The extrudates were crushed, sieved to a 250–315 μm size range and sulfided in situ in a dynamic flow reactor using a sulfiding feed made of 4.75% by volume of dimethyldisulfide (DMDS) in *n*-heptane, under a 4 MPa total pressure; this corresponded to a hydrogen pressure of 3 MPa, H_2S and methane pressures of 0.125 MPa and *n*-heptane pressure of 0.75 MPa. The sulfiding mixture was injected at a starting temperature of 150 °C. After 1 h the temperature was raised to 350 °C at a 5 °C/min rate and was maintained for 14 h to complete the sulfidation at this temperature. The temperature was then lowered to the reaction temperature (340 °C).

Dimethyldisulfide (>98% purity) and *n*-heptane (>99% purity) were purchased from Aldrich Chemicals; 46DMDBT (>95% purity), from Eburon organics and acridine (>98% purity), from Lancaster synthesis. They were used without further purification. 14DMCARB was prepared as described by Dalton and et al. [18].

2.2 Reaction Conditions

The HDS of 46DMDBT with or without a spiked nitrogen compound (acridine or 14DMCARB) was carried out in a fixed bed microreactor at 340 °C, under 4 MPa of total pressure after in situ sulfidation of the catalyst using a sulfiding feed made of 4.75% by volume of dimethyldisulfide (DMDS) in *n*-heptane.

46DMDBT (400–520 wt ppm S) and the nitrogen compound (0–200 wt ppm N) were dissolved in *n*-heptane to which dimethyldisulfide (DMDS) (8,500–11,000 wt ppm S) was added to generate H_2S.

The transformations of 46DMDBT and of the nitrogen compounds were studied separately or in mixture. To examine the effect of the nitrogen compounds on the 46DMDBT transformation, the partial pressure of the latter was maintained at the standard 0.014 MPa (corresponding to 500 ppm of sulphur), while the pressure of the added nitrogen compound varied from 0 to 0.0022 MPa (corresponding of 0 from 200 ppm of nitrogen). All the other partial pressures were kept constant by changing the partial pressure of the solvent. In all the experiments the ratio between the hydrogen flow rate (volume) and the reactant liquid flow rate (volume) over the total pressure (MPa) $((H_2/HC)/P_{total})$ was kept constant and equal to 117.5. For a better precision in the activity measurements, the contact time was chosen such as to keep the overall conversion of 46DMDBT nearly constant around 25% (24–27%) whatever the nitrogen content. Under these conditions the conversion through the DDS pathway varied between 5 and 12%.

2.3 Analysis

Owing to the high boiling point of the reactants, on-line analysis of the reaction products was not convenient. Consequently, the reactor effluents were condensed and liquid samples were periodically collected and analyzed by gas chromatography. Gaseous products were not found except for methane which was produced by dimethyldisulfide decomposition. The analyses were carried out with a Varian 3400 chromatograph equipped with a 25 m BP5 (SGE) capillary column (inside diameter: 0.32 mm; film thickness: 1 μm) with a temperature program from 80 to 150 °C (10 °C/min) then from 150 to 175 °C (4 °C/min) and from 175 to 280 °C (10 °C/min). Unknown products were identified by GC–MS (Finnigan INCOS 500).

3 Results

3.1 Transformation of 46DMDBT, Acridine and 14DMCARB Separately

3.1.1 Transformation of 46DMDBT

Before performing the simultaneous reaction of 46DMDBT and of acridine or 14DMCARB, the transformation of the sulphur compound alone was investigated. 46DMDBT underwent direct desulfurization (DDS) by cleavage of the C–S bonds yielding dimethylbiphenyl (DMBPh), as well as desulfurization through hydrogenation of one of the benzenic rings (HYD) leading to 4,6-dimethyltetrahydrodibenzothiophene (46DMTHDBT) and eventually to methylcyclohexyltoluene (MCHT) (Fig. 1). MCHT was the main desulfurization product. The formation of dimethylbiphenyl (DMBPh) was very limited. Actually, whatever the conversion, HYD was the main way of transformation of the sulphur compound and the selectivity measured by the ratio HYD/DDS was nearly constant and equal to about three (Fig. 2). These results which are in accordance with previous studies [4, 19–30] fit with the generally accepted reaction scheme of the transformation of 46DMDBT (Scheme 1).

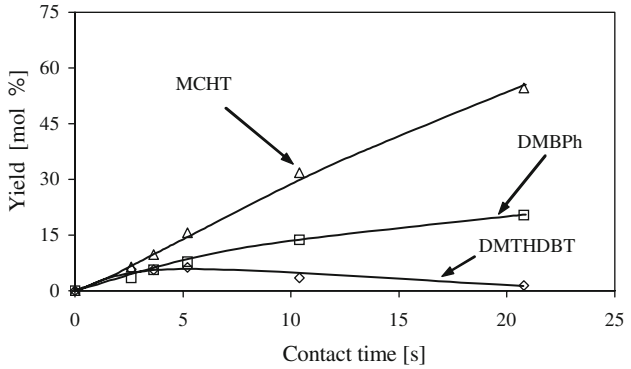


Fig. 1 Transformation of 46DMDBT on NiMoP/Al_2O_3 (T = 340 °C, P = 4 MPa, H_2/HC = 470 L/L). Products yield versus contact time. MCHT, methylcyclohexyltoluene; DMBPh, dimethylbiphenyl

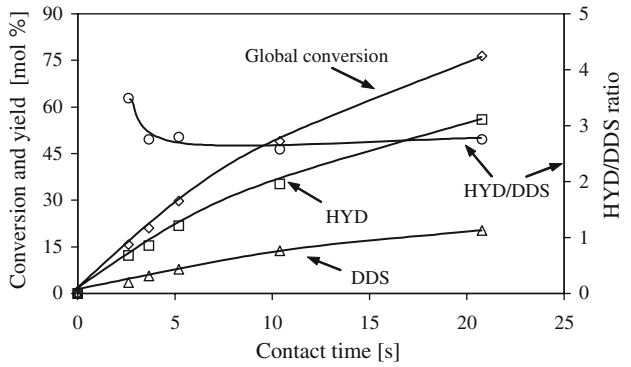
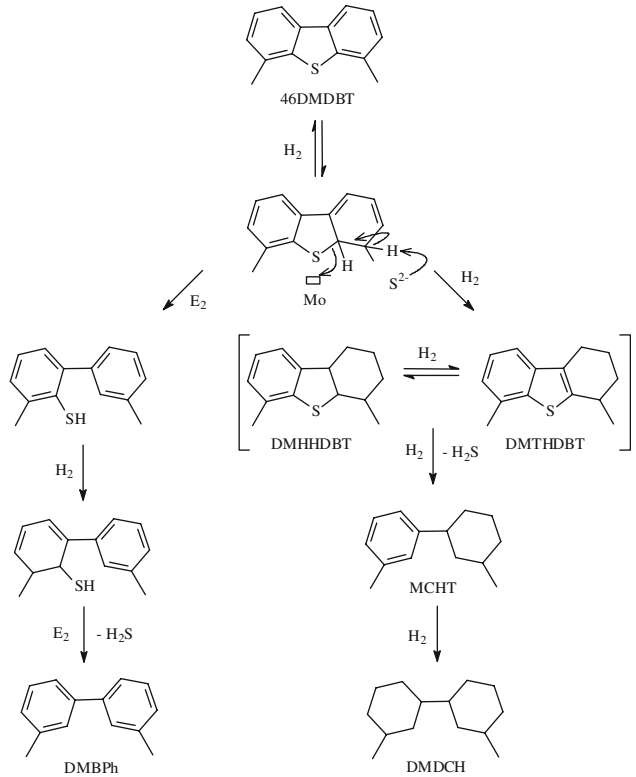


Fig. 2 Transformation of 46DMDBT on NiMoP/Al_2O_3 (T = 340 °C, P = 4 MPa, H_2/HC = 470 L/L). Contribution of the DDS and HYD pathways to the overall conversion. HYD, hydrogenation pathway (MCHT and DMTHDBT); DDS, direct desulfurization pathway (DMBPh)

3.1.2 Transformation of Acridine

As reported previously [28], the transformation of acridine was separately studied in order to identify its products of decomposition under our experimental conditions. The transformation of acridine into hydrogenation products (HYD) was very fast and complete initially, then it was transformed into hydrodenitrogenation products (HDN) when the contact time increased (Fig. 3). For the sake of clarity the distributions of the hydrogenation products and of the denitrogenation products are separately presented. Figure 4 shows the distribution versus contact time of the various products due to a partial or to a total hydrogenation of acridine:

- 9,10-Dihydroacridine (DHA), which comes from the hydrogenation of the pyridinic ring;
- 1,2,3,4-Tetrahydroacridine (THA), resulting from the hydrogenation of one of the benzenic cycles;



Scheme 1 Transformation of 46DMDBT on NiMoP/Al_2O_3 (T = 340 °C, P = 4 MPa, H_2/HC = 470 L/L)

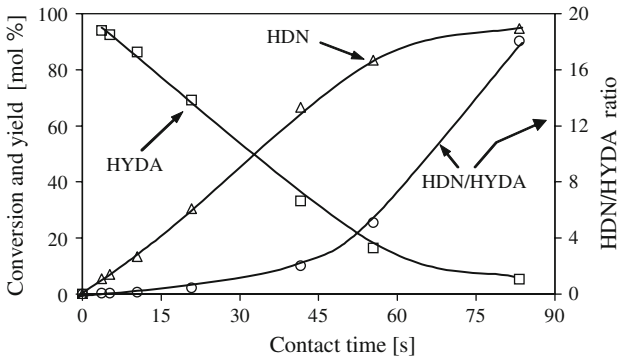


Fig. 3 Transformation of acridine on NiMoP/Al_2O_3 (T = 340 °C, P = 4 MPa, H_2/HC = 470 L/L). Yield in hydrogenation (HYDA) and hydrodenitrogenation (HDN) products versus contact time

- 1,2,3,4,5,6,7,8-Octahydroacridine (OHA1), the main hydrogenation product, resulting from the hydrogenation of the two benzenic rings adjacent to the heterocycle;
- 1,2,3,4,4a,9,9a,10-Octahydroacridine (OHA2), in which the pyridinic ring and one of the benzenic cycles are hydrogenated;
- Orthocyclohexylmethylaniline (OCHMAn), which comes from the hydrogenolysis of a C–N bond in OHA2;
- Perhydroacridine (PHA), the totally hydrogenated product.

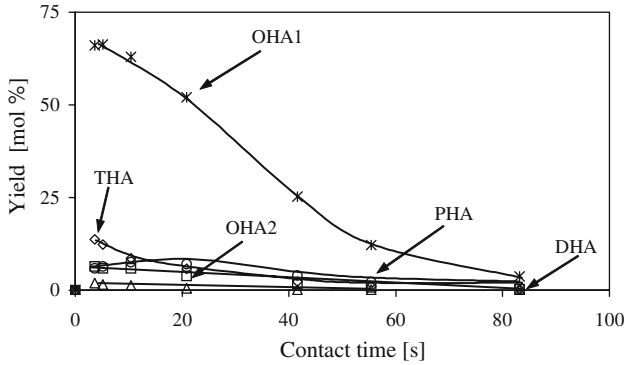


Fig. 4 Transformation of acridine on $NiMoP/Al_2O_3$ ($T = 340$ °C, $P = 4$ MPa, $H_2/HC = 470$ L/L)—Yield in hydrogenation products versus contact time. OHA1 and OHA2, octahydroacridine1 (*) and octahydroacridine 2 (□); THA, tetrahydroacridine (◇); DHA, dihydroacridine (Δ); PHA, perhydroacridine (○)

All the partially hydrogenated products appeared as primary products while perhydroacridine appeared as a secondary product which apparently reacted very readily to give the denitrogenation products.

Figure 5 shows the distribution versus contact time of the denitrogenated products resulting from the cleavage of C–N bonds in the intermediate hydrogenated compounds. Three denitrogenated products were detected:

- Cyclohexenylcyclohexylmethane (CHCHM), resulting from the denitrogenation of perhydroacridine (PHA);
- Benzylcyclohexane (BCH), which is supposed to come from the denitrogenation of orthocyclohexylmethylaniline (OCHMAn);
- Dicyclohexylmethane (DCHM), the final and major product of denitrogenation, resulting from the hydrogenation of CHCHM or of BCH.

It can actually be considered as it was the case for quinoline [30] that the main primary product of

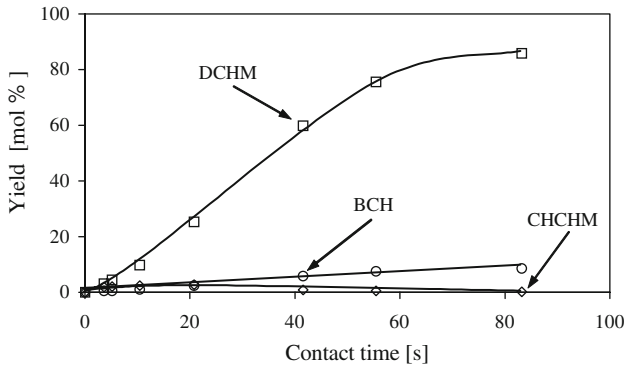
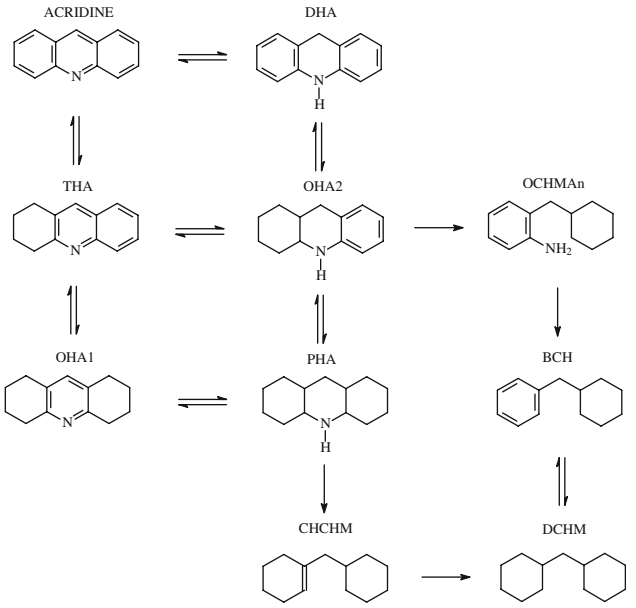


Fig. 5 Transformation of acridine on $NiMoP/Al_2O_3$ ($T = 340$ °C, $P = 4$ MPa, $H_2/HC = 470$ L/L). Yield in hydrodenitrogenation products versus contact time. DCHM, dicyclohexylmethane (□); BCH, benzylcyclohexane (○); CHCHM, cyclohexenylcyclohexylmethane (◇)



Scheme 2 Transformation of acridine on $NiMoP/Al_2O_3$ ($T = 340$ °C, $P = 4$ MPa, $H_2/HC = 470$ L/L)

denitrogenation was the alkene (here, CHCHM) which, in turn, was hydrogenated very readily into DCHM.

The reaction network of the transformation of acridine is reported in Scheme 2. We considered to be consistent with the HDN of quinoline [31], that the hydrogenation steps were reversible under the conditions of acridine transformation.

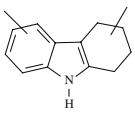
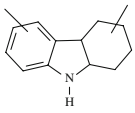
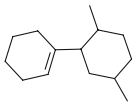
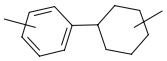
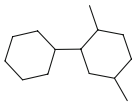
3.1.3 Transformation of 14DMCARB

Under the same conditions as those of the acridine transformation, the conversion of 14DMCARB was not complete. It gave both hydrogenation (HYDcarb) and HDN products (Table 1; Fig. 6) and it was more reactive than acridine towards HDN. Indeed, the main products were the hydrocarbons resulting from HDN. Alkylation products were also observed in low amounts.

Figure 7 shows the distribution versus conversion of the various products due to a partial or to a total hydrogenation of 14DMCARB: the main hydrogenated products are the dimethyltetrahydrocarbazoles (DMTHCARB) which amount decreased when the conversion increased strongly. The denitrogenated products observed are: dimethyldicyclohexyl-1-enes (DMDCHene), dimethylcyclohexylbenzenes ((DM)(CH)B) and the dimethyldicyclohexane (DMDCH). The formation of these two last products remained low whatever the conversion (Fig. 8).

The reaction network of the 14DMCARB transformation is reported in Scheme 3. By analogy with quinoline [30] and acridine [27, 28], we considered that the hydrogenation steps were reversible under the conditions of 14DMCARB transformation.

Table 1 Transformation of 14DMCARB

HYDcarb	DMTHCARB		DMHHCARB
			
HDN	DMDCHene	(DM)(CH)B	DMDCH
			
Alkylation	Trimethylcarbazoles		

($T = 340$ °C, $P = 4$ MPa, H_2/HC = 470 L/L, $NiMoP/Al_2O_3$). Hydrogenation (HYDcarb), hydrodenitrogenation (HDN) and alkylation products

DMTHCARB, dimethyltetrahydrocarbazoles; DMHHCARB, dimethylhexahydrocarbazoles; DMDCH, dimethyldicyclohexane; DMDCHene, dimethyldicyclohexyl-1-enes; (DM)(CH)B, dimethylcyclohexylbenzenes

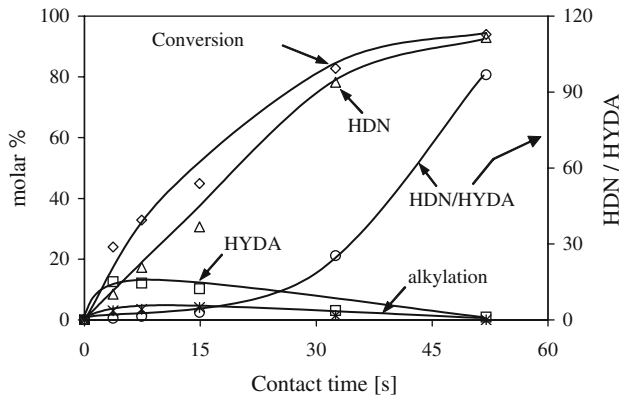


Fig. 6 Transformation of 14DMCARB on $NiMoP/Al_2O_3$ ($T = 340$ °C, $P = 4$ MPa, H_2/HC = 470 L/L). Change in conversion of 14DMCARB, formation of HDN, HYD and alkylation products and in HDN/HYD ratio versus residence time

3.2 Competitive Experiments—Effect of Acridine on the Transformation of 14DMCARB

The simultaneous reaction of both nitrogen compounds indicated that the acridine transformation was not modified by the presence of 14DMCARB. On the contrary, the presence of acridine inhibited the transformation of 14DMCARB greatly (Table 2).

Indeed, the total conversion of 14DMCARB decreased and the HDN reaction was completely inhibited under the conditions of the experiments. The main product became DMHHCARB. This was due to a stronger adsorption of acridine or of its products than of 14DMCARB. This

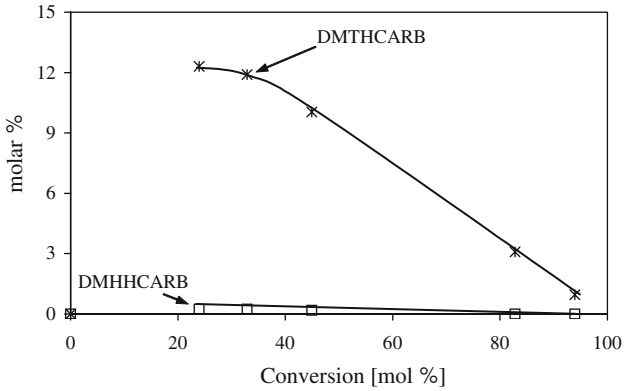


Fig. 7 Transformation of 14DMCARB on $NiMoP/Al_2O_3$ ($T = 340$ °C, $P = 4$ MPa, H_2/HC = 470 L/L). Yield in hydrogenation products versus conversion. DMTHCARB, dimethyltetrahydrocarbazoles; DMHHCARB, dimethylhexahydrocarbazoles

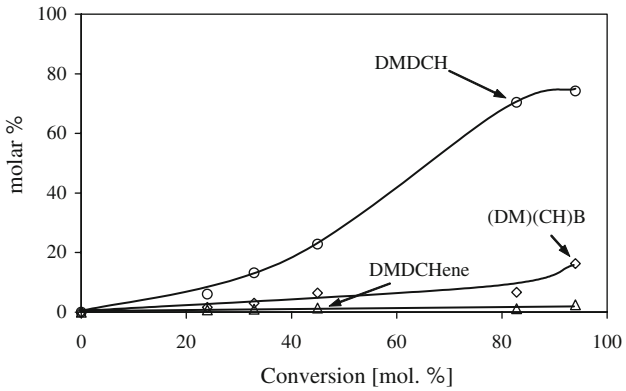


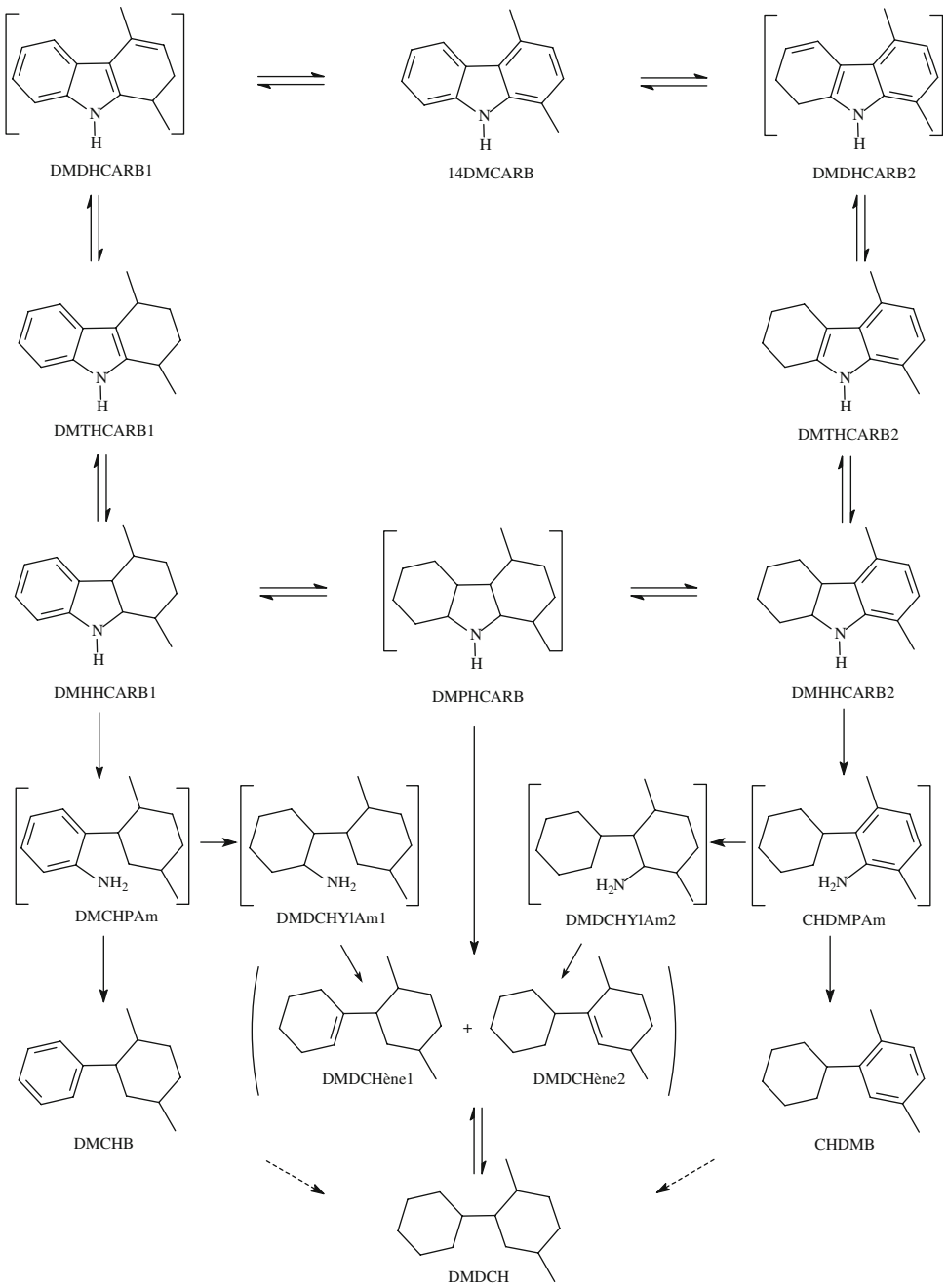
Fig. 8 Transformation of 14DMCARB on $NiMoP/Al_2O_3$ ($T = 340$ °C, $P = 4$ MPa, H_2/HC = 470 L/L). Yield in denitrogenation products versus conversion. DMDCH, dimethyldiclyclohexane; DNDCHene, dimethyldiclyclohexyl-1-enes; (DM)(CH)B, dimethylcyclohexylbenzenes

inhibition phenomenon can explain why carbazoles are generally the main nitrogen impurities present in hydrotreated gasoils [10].

3.3 Competitive Experiments—Effect of Acridine and 14DMCARB on the Transformation of 46DMDBT

The transformation of 46DMDBT in the presence of various amounts of nitrogen compounds (acridine and 14DMCARB) showed a strong inhibiting effect of the latter. This effect was more significant with acridine than with 14DMCARB (Fig. 9). In a previous work, we showed that acridine was also totally transformed in the presence of the sulphur compound and that the real inhibitor was probably OHA1 [27, 28]. The decrease of the global HDS activity corresponded to a decrease of both the HYD and

Scheme 3 Transformation of 14DMCARB on NiMoP/Al_2O_3 ($T = 340$ °C, $P = 4$ MPa, H_2/HC = 470 L/L)



DDS contributions. However, beyond 20 N-ppm of acridine ($P_N = 0.0016$ bar), the activity through the DDS pathway was partly recovered and slightly increased when the amount of acridine increased (Fig. 10). Indeed, at a nearly constant global conversion of about 25%, the DDS yield increased from 5 to 12%. It is suggested that this promotion effect on the DDS pathway could be due to a cocatalytic contribution of acridine or of its hydrogenation products to the process leading to C–S bond cleavage as reported in a previous work [27, 28].

The transformation of 14DMCARB was not modified by the presence of 46DMDBT and the contributions of HYD and DDS pathways to the transformation of the latter were not both modified.

3.4 Kinetic Modelling of the Inhibiting Effect of Nitrogen Compounds

3.4.1 Equations

The inhibiting effect of acridine and of 14DMCARB on the HDS of 46DMDBT was calculated with a Langmuir–Hinshelwood kinetic model. We considered that all the reactants and products were in gas phase and that we had a

Table 2 Transformation of 14DMCARB in the absence or in the presence of acridine ($T = 340$ °C, $P = 4$ MPa, $P_N = 0.0008$ MPa, residence time = 3.7 s, H_2/HC = 470 L/L, NiMoP/Al_2O_3)

Nitrogen compounds	14DMCARB alone	14DMCARB + acridine
14DMCARB conversion (%)	24	16
HYDcarb (%)		
DMTHCARB	12	6
DMHHCARB	0	10
Total HYDcarb	12	16
HDN (%)		
DMDCHene	1	0
(DM)(CH)B	2	0
DMDCH	6	0
Total HDN	9	0
Alkylation (%)		
TMCARB	3	0

Hydrogenation (HYDcarb), hydrodenitrogenation (HDN) and alkylation products

DMTHCARB, dimethyltetrahydrocarbazoles; DMHHCARB, dimethylhexahydrocarbazoles; DMDCH, dimethyldicyclohexane; DMDCHene, dimethyldicyclohexyl-1-enes; (DM)(CH)B, dimethylcyclohexylbenzenes; TMCARB, trimethylcarbazoles

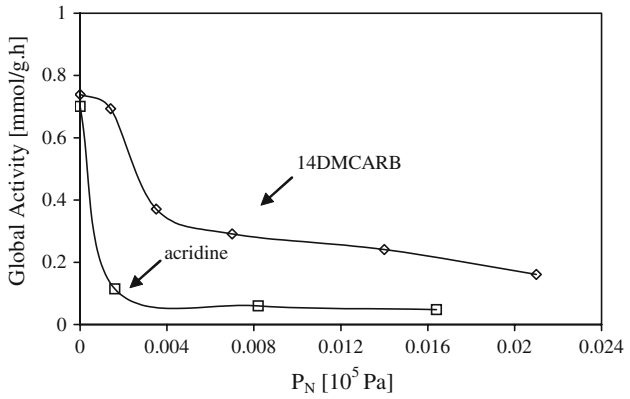


Fig. 9 Transformation of 46DMDBT in the presence of nitrogen compounds (acridine and 1,4-dimethylcarbazole: 14DMCARB) on NiMoP/Al_2O_3 ($T = 340$ °C, $P = 4$ MPa, H_2/HC = 470 L/L, conversion ≈25%). Change in global activity versus nitrogen partial pressure

first order for the transformation of 46DMDBT and for hydrogen.

Under these experimental conditions, hydrogen was in large excess and the partial pressures of 46DMDBT and H_2S were constant and equal to 0.0014 and 0.058 MPa, respectively.

As described by Whitehurst et al. [4], we can consider two different sites for the adsorption of 46DMDBT and of H_2. The HDS rate (r_{HDS}) can be expressed as:

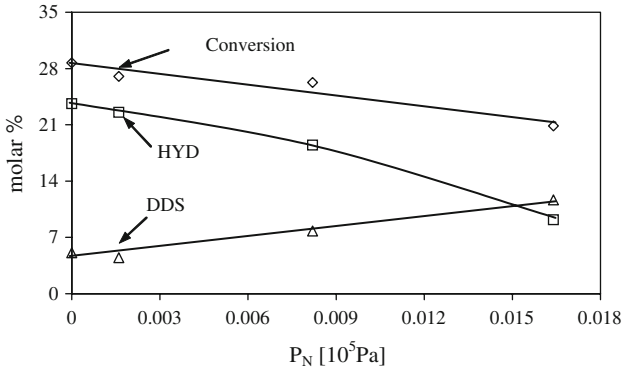


Fig. 10 Transformation of 46DMDBT in the presence of acridine on NiMoP/Al_2O_3 ($T = 340$ °C, $P = 4$ MPa, H_2/HC = 470 L/L, conversion ≈25%). Changes in DDS and HYD activity versus acridine partial pressure

$$r_{HDS} = k\frac{K_S C_S}{1 + K_S C_S + K_{H_2S} C_{H_2S}} \times \frac{K_H C_H}{1 + K_H C_H} \quad (1)$$

where k was the kinetic constant of the rate; K_S, K_H and K_{H_2S} the adsorption constant of 46DMDBT, of H_2 and of H_2S; C_S, C_H and C_{H_2S} the concentration of 46DMDBT, of H_2 and of H_2S.

Under our experimental conditions (under H_2 pression), $K_H C_H >> 1$, the Eq. 1 became:

$$r_{HDS} = k\frac{K_S C_S}{1 + K_S C_S + K_{H_2S} C_{H_2S}} \quad (2)$$

If we considered that H_2S is more strongly adsorbed than 46DMDBT, $(1 + K_{H_2S} C_{H_2S}) >> K_S C_S$. So, the Eq. 2 became:

$$r_{HDS} = k\frac{K_S C_S}{1 + K_{H_2S} C_{H_2S}} \quad (3)$$

Under these conditions, $(1 + K_{H_2S} C_{H_2S})$ was constant:

$$k_{HDS} = k\frac{K_S}{1 + K_{H_2S} C_{H_2S}} \quad (4)$$

The rate of HDS could be expressed as:

$$r_{HDS} = k_{HDS} C_S \quad (5)$$

where k_{HDS} was the apparent rate constant.

In the presence of a nitrogen compound, this rate constant was modified and was transformed as followed:

$$r'_{HDS} = k'_{HDS} C_S \quad (6)$$

In addition, if we consider the effect of a given compound (I) as being due to a competition in the adsorption type "Langmuir–Hinshelwood and exponent n, the apparent rate constant of the reaction of HDS is given by the modified expression of Langmuir–Hinshelwood [32, 33].

$$k'_{HDS} = \frac{k_{HDS}}{1 + K_I^n C_I^n} \quad (7)$$

with K_I as the apparent adsorption constant of the nitrogen compound (I), C_I, the concentration and n, the exponent from I compound.

Under these conditions, we can consider the partial pressures of the various compounds and the Eq. 7 became:

$$\frac{k_{HDS}}{k'_{HDS}} = 1 + K_I^n P_I^n \quad (8)$$

From the Eq. 5 and 6, $k_{HDS}/k'_{HDS} = r_{HDS}/r'_{HDS}$ and from the Eq. 8,

$$\frac{k_{HDS}}{k'_{HDS}} = \frac{r_{HDS}}{r'_{HDS}} = 1 + K_I^n P_I^n \quad (9)$$

3.4.2 Results

From the Eq. 9, n and K were calculated with the experimental activities (global, DDS, HYD) of the transformation of 46DMDBT in the presence of acridine and 14DMCARB as nitrogen compounds (Table 3). Excepted for the DDS pathway involved in the transformation of 46DMDBT in the presence of acridine, the correlation coefficients indicated well the good correlation between the experimental results and the modified Langmuir–Hinshelwood kinetic model. Indeed, the inhibiting effect of acridine was due to its very strong adsorption on the active sites corresponding to a very high global K_I. The global K_I in the presence of 14DMCARB was lower corresponding also to a lower inhibiting effect in the 46DMDBT transformation. Calculated k_{HDS}/k'_{HDS} ratios from the model were compared to the experimental results in order to show the good correlation between experimental and calculated values, respectively, showed Fig. 11 for the global activity, Fig. 12 for the activity via the HYD pathway and Fig. 13 for the activity via the DDS pathway. In general, the inhibiting effect increased with the amount of acridine and of 14DMCARB. Indeed, the k_{global}/k'_{global} ratios increased with the partial pressure of the inhibitor (Fig. 11). This

Table 3 Exponent (n) and apparent constant of adsorption (K_I) of inhibitors (acridine and 14DMCARB) for the HDS of 46DMDBT ($T = 340$ °C, $P = 4$ MPa, H_2/HC = 470 L/L, NiMoP/Al_2O_3) ($|R^2|$, correlation coefficient)

Nitrogen compounds	HDS routes	n	K_I (kPa^{-1})	$\|R^2\|$
Acridine	Global	0.43	283	1
	HYD	0.63	71	1
	DDS	–	–	0
14DMCARB	Global	1.33	1.5	1
	HYD	0.69	2.4	1
	DDS	0.87	2.4	1

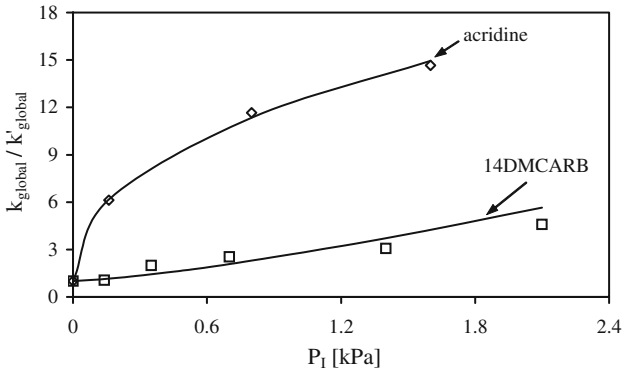


Fig. 11 Transformation of 46DMDBT. k_{global}/k'_{global} ratio (global activity): comparison of experimental results in the presence of acridine (◇), 14DMCARB (□) and calculated results obtained with a Langmuir–Hinshelwood model (in *continuous line*)

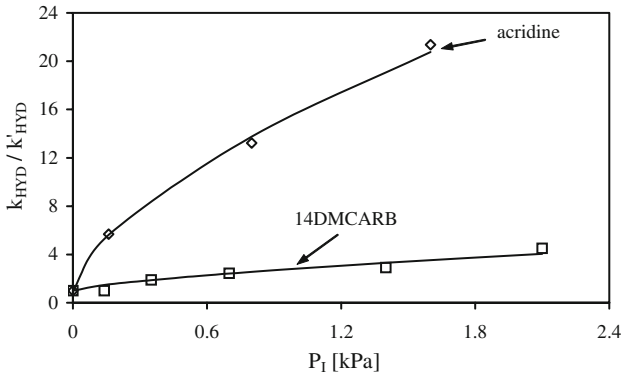


Fig. 12 Transformation of 46DMDBT. k_{global}/k'_{global} ratio (HYD activity): comparison of experimental results in the presence of acridine (◇), 14DMCARB (□) and calculated results obtained with a Langmuir–Hinshelwood model (in *continuous line*)

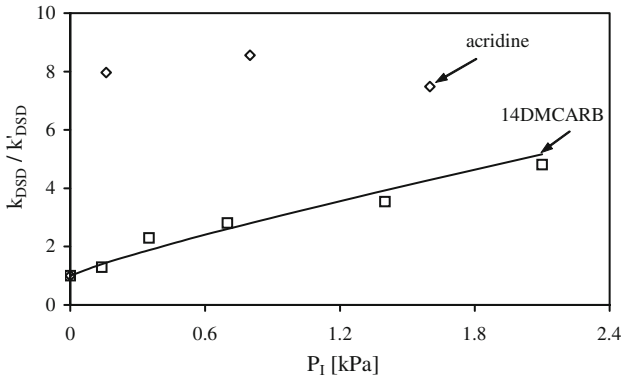


Fig. 13 Transformation of 46DMDBT. k_{global}/k'_{global} ratio (DDS activity): comparison of experimental results in the presence of acridine (◇), 14DMCARB (□) and calculated results obtained using Langmuir–Hinshelwood model (in *continuous line*)

effect was more significant with acridine than with 14DMCARB and was also similar in the main hydrogenation pathway of the transformation of 46DMDBT (Fig. 12). Thus, the global conversion and the HYD pathway followed the modified "Langmuir–Hinselwood" model. The results indicated that the sulphur compound and the inhibitor were in competition on the active sites on the catalyst surface for their adsorption. The nitrogen compounds—and mainly acridine which was more basic—were in competition with 46DMDBT for their adsorption on the active sites involved in the HYD pathway. However, no correlation was obtained for the inhibiting effect of acridine on the transformation of 46DMDBT involving the DDS pathway. Consequently, the modified "Langmuir–Hinselwood" model was not followed in the presence of acridine on the contrary to what is observed in the presence of 14DMCARB.

4 Discussion

In the present work, very low amounts of N-containing compounds were used to be representative of real feeds. The reaction scheme of the 46DMDBT HDS remained in agreement with previous studies [1–8] and 46DMDBT decomposed according to two parallel and independent routes. The prevailing route was the HYD pathway (75%) leading to a partially hydrogenated intermediate (46DMTHDBT), then to a partially hydrogenated and desulphurized product (MCHT). The second route was the DDS pathway yielding DMBPh.

Full conversion of acridine, in hydrogenated products mainly, was observed under the present operating conditions. The hydrogenation steps were followed by the cleavage of C–N bonds which mainly yielded DCHM. On the other hand, the non-basic 14DMCARB was not fully converted under identical experimental conditions and the major denitrogenated product was DMDCH.

The addition of small amounts of nitrogen-containing compounds (20 ppm N) to the feed inhibited the transformation of 46DMDBT. The effect of acridine, the basic nitrogen compound, is the more pronounced with a decrease in the HDS activity of about 80% on both HDS pathways. This result is in agreement with previous works [4, 6–16]. However, as also mentioned by others [14, 28], it must be pointed out that, regarding specifically the inhibition, it seems very likely that acridine itself is not the actual inhibitor of the HDS of 46DMDBT (since it is totally converted under the experimental conditions) but one of its hydrogenation products. As demonstrated in a previous paper [27], the real inhibitor was one of the octahydroacridines (OHA1). This could be due to a stronger adsorption or to a higher proton affinity as proposed in the literature to explain the inhibition effects of quinoline and of tetrahydroquinoline on the HDS of dibenzothiophene (DBT) and 46DMDBT [17, 34].

An interesting finding as reported in the previous work [27, 28] was that, when the concentration of acridine was increased, its effect was not the same on both pathways (HYD and DDS) of the 46DMDBT HDS. That was not observed in the presence of 14DMCARB. As described before [27], it was due to a strong inhibition of the activity through the HYD pathway on the one hand and to a slight promotion (or recovery) of the activity through the DDS pathway on the other hand. This promoting effect on the DDS pathway was rather unexpected although it has already been reported [6, 9] that the inhibiting effect of N-compounds was more pronounced on the HYD pathway than on the DDS one. Several explanations can be proposed to account for the discrimination in the effect of acridine on both pathways of the HDS of 46DMDBT. One is that HYD and DDS may occur on discrete catalytic centres and that acridine or its hydrogenation products would adsorb preferentially on the centres involved in the HYD pathway. Another explanation could be that the inhibition may depend on the way the nitrogen compound is adsorbed on a given centre: since acridine and its partially hydrogenated products undergo essentially hydrogenation, it can be assumed that they are adsorbed "flat" and that it will particularly inhibit the HYD pathway of HDS for which 46DMDBT has also to be adsorbed "flat" [6]. However, none of these explanations can account for any promotion effect on the DDS pathway. Therefore another hypothesis must be considered. Various authors [3, 6, 8, 35] proposed that the HDS of DBT-type compounds through both the HYD and DDS pathways occurs via a common dihydro-intermediate (Scheme 1); they also suggested that the C–S bond cleavages occur by an elimination process involving a basic site (presumably a sulphur anion) and a Lewis acid site (a coordinatively unsaturated molybdenum ion). Actually C–S bond cleavage is the first step subsequent to the formation of the dihydrointermediate in the DDS pathway (Scheme 1) and, in the case of 46DMDBT, it is supposed to be hindered by the methyl groups and possibly to be rate-limiting (it is particularly inhibited in comparison with DBT). Therefore, we can assume that the N-compound which is basic in character could possibly act as a cocatalyst in this reaction [27] and hence promote the DDS pathway. Indeed, this positive effect after a high decrease could be explained with the "Rideal–Eley" model corresponding of a reaction between the hydrogenated intermediate sulphur compound adsorbed on the catalyst surface and the nitrogen compound on the gas phase, this effect increasing with the amount of nitrogen.

In fact, we have shown that the observed inhibiting effect depends on the properties of the nitrogen molecules. Acridine, a basic nitrogen compound, inhibited more strongly the transformation of 46DMDBT than 14DMCARB, a non basic one. Experimental results were compared to those calculated considering the modified Langmuir–Hinshelwood kinetic model. They confirmed the inhibition, corresponding to a competitive adsorption between the nitrogen compound and the sulphur compound, depended on the n exponent of the inhibitor but mainly on its apparent constant of adsorption (k_I). Indeed, acridine has an apparent constant of adsorption more significant than 14 DMCARB and it explains the higher inhibiting effect. A good correlation was obtained for the global activity and for the HYD activity for both molecules. Differences were noticed for the DDS pathway in the presence of acridine resulting, as reported before, to a reaction between an adsorbed sulphur intermediate and nitrogen in gas phase.

As reported by authors [8, 12], the alkylcarbazoles would not be the most nitrogen compounds inhibitors of the HDS alkyldibenzothiophenes, even if they are the main nitrogen compounds found in hydrotreated cuts. Actually, their transformation was inhibited by traces of basic nitrogen compounds such as acridine as shown in this article. The basic compounds would be by far the most inhibitors and would then be the most appropriate model molecules to estimate the effect of nitrogen compounds on the dibenzothiophenes HDS.

Several authors [8, 36–38] indicate that the non-basic nitrogen compounds could be transformed into hydrogenated basic compounds which, then, would inhibit the reaction of HDS more strongly than the initial nitrogen compounds. Under our operating conditions, acridine is fast transformed into OHA1 ($pKa = 7.37 \pm 0.20$) which would be more basic than acridine ($pKa = 5.50 \pm 0.20$). That would mean, as described above, that OHA1 could be the real inhibitor of the HDS of 46DMDBT.

Regarding the 14DMCARB impact, the pKa of 14DMCARB, DMTHCARB and DMHHCARB (the hydrogenated products) are, respectively, 1.28 (±0.20), 3.97 (±0.20) and 6.30 (±0.20). We can expect that the hydrogenated products of 14DMCARB are stronger inhibitors than 14DMCARB itself. In fact, in the presence of 46DMDBT, hydrogenated products are mainly DMTHCARB which amounts increase with the partial pressure of 14DMCARB. The DMTHCARB would be potential inhibitors of the reaction of HDS, by their presence and their basic properties. Therefore, it appears that the inhibition of the reaction of HDS by nitrogen compounds is strongly linked, not only to the basicity of the initial nitrogen molecules, but also to their ability to be hydrogenated in products that are often more basic than the initial compounds.

5 Conclusion

Nitrogen compounds inhibited the transformation of 46DMDBT, one of the most refractory sulphur compounds. However, the effect of a basic compound such as acridine or its products (OHA1 for example) is more significant than this one observed in the presence of a non basic compound such as 14DMCARB. In the presence of acridine, the DDS pathway was less affected than the HYD pathway and could be slightly promoted after a strong inhibition due to a cocatalytic effect of the nitrogen compound. In the presence of 14DMCARB, a same inhibitor effect was determined in both pathways (DDS and HYD).

We also demonstrated that acridine inhibited the transformation of 14DMCARB and can explain why carbazoles are generally the main nitrogen impurities present in hydrotreated gasoles.

Finally, the performances of various catalysts, under deep HDS of diesel fuel could be evaluated using a model feed composed of 46DMDBT, the most common refractory sulphur compound, and of acridine, the most representative of inhibiting molecules.

Acknowledgments V. Rabarihoela-Rakotovao thanks I.F.P. for a PhD grant.

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