Specific Poisoning of Brønsted Acid Sites on Solid Isomerization and Metathesis Catalysts

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Summary Gaseous hexamethyldisilazane can quantitatively and irreversibly poison Brønsted acid sites on silica gel and on tungsten oxide–silica without affecting the Lewis acid sites

THE usual poisoning agents for solid, acidic catalysts are ammonia and pyridine[1] These agents, however, produce surface complexes that are unstable above 500 K and do not discriminate significantly between Lewis and Brønsted acid sites Hexamethyldisilazane (HMDS) reacts quantitatively with Brønsted-acidic hydroxyl-groups[2,3] As HMDS is a nitrogen base, it may also react with Lewis acid sites, but the co-ordination complexes thus formed are probably less stable than the interaction products of HMDS with hydroxyl-groups, silylated silica does not decompose below 900 K[4] Here we demonstrate that HMDS can serve as a specific poison for Brønsted acid sites and that silylation can be a useful tool to discriminate between the proposed models for active centres on solid catalysts Two reactions in which Brønsted acid sites have been proposed as active centres were investigated, *viz* , the double bond isomerization of *trans*-but-2-ene on silica gel and the metathesis of propene on tungsten oxide-silica

The isomerization of linear butenes on silica gel proceeds *via* the same mechanism and on the same active sites as for silica–alumina[5] Finch and Clark[6] stated that the activity of silica–alumina is brought about by the simultaneous action of polymeric complexes (butene oligomers chemisorbed on Lewis acid sites, *i e* , exposed aluminium ions) and surface water (weak Brønsted acid sites, *i e* , the surface silanols) Other authors believe that the activity is caused by aluminium-induced, strongly Brønsted-acidic hydroxyl-groups only[1]

The metathesis of alkenes probably involves metal–carbene complexes[7] The generation of the initial carbenes on oxidic metathesis catalysts, such as tungsten oxide–silica, is still not fully understood Laverty *et al*[8] suggested that transition metal hydrides, obtained from the interaction of reduced promotor ions with hydroxyl-groups from the support, initiate carbene formation *via* alkylation of the transition metal. The presence of Brønsted-acidic hydroxyl-groups, therefore, would be necessary for the formation of active centres.

The experiments were carried out in a closed circulation system at 700 K and 0·1—0·2 MPa. The silica gel (*ca.* 0·03 wt% Al_2O_3; 360 $m^2 g^{-1}$) and the tungsten oxide–silica (4·2 wt% WO_3; 360 $m^2 g^{-1}$) were calcined in air at 825 K and activated *in vacuo* at 750 K. After introduction of the reactant (*trans*-but-2-ene or propene) into the system, the catalytic activity was measured by following the conversion *via* g.c. analysis of the reaction mixture. The system was then cooled *in vacuo* to 525 K. The appropriate amount of HMDS, diluted with helium, was admitted and allowed to react for 15—20 min.† Finally, the system was heated *in vacuo* to 700 K and the activity was measured again.‡ Some typical results of the silylation experiments are summarized in the Table.

The isomerization on the silica gel is almost completely suppressed by adding $2{\cdot}0 \times 10^{-6}$ mol m^{-2} of HMDS. This amount is less than the surface hydroxyl-group concentration ($3{\cdot}1 \times 10^{-6}$ mol m^{-2}, calculated from the weight increase on exhaustive silylation with HMDS),[5] in accordance with the fact that one molecule of HMDS can react with two hydroxyl-groups. The residual activity can be ascribed to hydroxyl-groups in narrow pores not reached by the large HMDS molecules. Three times as much HMDS as the maximum surface aluminium ion concentration ($0{\cdot}02 \times 10^{-6}$ mol m^{-2}, assuming that all aluminium ions are present on the surface) hardly affected the catalytic activity. Hence, it appears that the isomerization is not brought about by aluminium-induced, strongly Brønsted-acidic hydroxyl-groups[1] and that Finch and Clark's model gives a better description of the active centres. Given the latter model, it can be concluded that HMDS does not affect Lewis acid sites (surface aluminium ions) under the applied conditions.

TABLE. Influence of silylation on the catalytic activity of silica gel and tungsten oxide–silica.[a]

Amount of HMDS added (10^{-6} mol m^{-2})	Isomerization rate on SiO_2 (10^{-9} mol $m^{-2} s^{-1}$)	Metathesis rate on WO_3–SiO_2 (10^{-9} mol $m^{-2} s^{-1}$)
0	110	4·9
0.065	103	—
2·0	7·4	—
3·0	—	702

[a] See the text for the reaction conditions and silylation procedure.

The large activity increase of the tungsten oxide–silica on silylation shows that Brønsted-acidic hydroxyl-groups are not likely to be precursors for the active sites in propene metathesis, as suggested by Laverty *et al.*[8] A model which can account for the observed activity increase will be presented elsewhere.

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† Kinetic, gravimetric, and i.r. spectroscopic experiments showed that the silylation is almost complete after this reaction time.

‡ Applying the same procedure, but deleting the HMDS, did not affect the activity of our catalysts. Treatment with HMDS before any contact with alkene gave results closely similar to those in the Table.

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