REVIEW

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Protonation mechanisms of nickel complexes relevant to industrial and biological catalysis

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The sites of protonation and the subsequent rearrangement reactions of simple nickel complexes containing hydride, thiolate and alkyl ligands are reviewed, and the relevance of these reactions to the action of certain nickel-based catalysts are discussed.

Keywords: Nickel, protonation, mechanism, hydrocyanation, hydrogenase

Introduction

The protonation of nickel complexes is a fundamental reaction which has direct relevance to the action of certain industrial and biological catalysts, yet the mechanistic chemistry of proton-transfer to simple nickel complexes is largely unexplored. The reasons for this are simple: the basic pathways of the catalyses (particularly for the well-established industrial processes) have been known for several years and the intimate mechanisms are presumed to be known. In reality the mechanisms are only based on reasonable chemical intuition. Whilst these presumed mechanisms are often operationally satisfactory, allowing successful prediction of products *etc*, gaps or errors in our understanding of the elementary reactions can lead to inefficiencies in the industrial applications and misconceptions in our understanding of biological processes. This is always a serious problem, but it becomes acute if we intend to use chemical principles learnt in studying metalloenzymes to improve or develop new abiological catalysts.

In the last few years we have started to investigate the mechanisms of proton-transfer to selected nickel complexes. So far the types of complexes we have studied are far removed from those found in the industrial or biological catalysts. Our choice of coligands has been made on what renders the system amenable to mechanistic study rather than a rational selection of a coordination environment mimicking that found in the catalysts. The bottom line is that so little is known about the rates and mechanisms of proton-transfer to and from nickel complexes that any study will improve our understanding of the reactivity of nickel complexes towards acids.

The presentation that follows will encompass the action of nickel in industrial homogeneous catalysts and metalloenzymes which involves protonation reactions, and will thus cover the protonation mechanisms of Ni-C, Ni-H and Ni-thiolate complexes. However, before discussing the specifics relating to nickel complexes it is worth emphasising some of the entirely general characteristics of proton-transfer mechanisms of transition metal complexes.

In general understanding the factors which control how rapidly transition metal complexes are protonated is relatively simple. Only two rules need to be remembered.[1,2] (1) In ligands, thermodynamically-favourable proton-transfer reactions to stereochemical lone pairs on atoms are diffusion-controlled ($k_{diff} = 1 \times 10^{10}$ dm^3 mol^{-1} s^{-1}).[3] (2) However, even thermodynamically-favourable proton transfer reactions to carbon and metal sites can be appreciably slower than the diffusion-controlled limit. The slowness of these reactions has been attributed to the necessary change in hybridisation for protonation at carbon, and the reorganisation of the ligands when a proton binds to a metal site. A consequence of these rules is that whereas it is often easy to predict where a proton will preferentially bind on complexes with most ligands, it is often difficult to predict in organometallic compounds whether metal or carbon will be protonated most rapidly.

Of course a variety of examples of slow proton transfer reactions involving protonation of lone-pairs of electrons on ligands are known, but here invariably special circumstances lead to the departure from diffusion-controlled rates. However, it is now emerging that there are other general areas (such as with clusters) where slow proton transfer reactions are common.[4]

Finally, it worth emphasising that although kinetics control where the proton will bind initially, the final residence of the proton is controlled by thermodynamics. Proton-transfer *from* complexes can also be facile and thus reorganisation from the kinetically-favoured to the thermodynamically-favoured product is easily accomplished.

Protonation and industrial catalysts

Many oligomerisation and isomerisation catalysts are based on nickel, and this area has been reviewed extensively.[5,6] Thus the oligomerisation of ethylene to α-olefins is catalysed by $[NiPh(PPh_3)(Ph_2PCHCPhO)]$ and the analogous allyl complexes oligomerise butadiene. Furthermore, the dimerisation of propene by nickel-allyl complexes is the basis of the "Dimersol" process for the synthesis of octane enhancers. Whilst the oligomerisation and isomerisation catalysts are often Ni-H species, such species are rarely prepared by protonation reactions. Two exceptions are: (i) the isomerisation of alkenes by $[Ni\{P(OEt)_3\}_4]$ where H_2SO_4 or CF_3CO_2H produce the catalyst $[NiH\{P(OEt)_3\}_3]^+$, and (ii) the hydrocyanation of alkenes and alkynes.

Hydrocyanation reactions are industrially and commercially very important, with alkenes being converted into alkyl nitriles[7] as shown in Figure 1, and alkynes form α, β-unsaturated nitriles. Complexes of Cu, Ni and Pd (especially Ni-phosphite complexes such as $[Ni\{P(OC_6H_4R\text{-}4)_3\}_4]$) are active catalysts in adding HCN across alkenes or alkynes. Using chiral phosphite ligands has resulted in enantioselective hydrocyanation.

Economically, the hydrocyanation of butadiene to adiponitrile $\{NC(CH_2)_4CN\}$ is important because adiponitrile is a precursor for 1,6-diaminohexane, essential for the synthesis of nylon. The first stage of the reaction involves an intermediate η^3-1-methylallyl-species which protonates with poor stereoselectivity to a mixture of 3- and 4-pentenenitriles as shown in Figure 2. The second stage involves the addition of the second HCN. This stage is also catalysed by a nickel catalyst promoted by Lewis acids.

* To receive any correspondence.

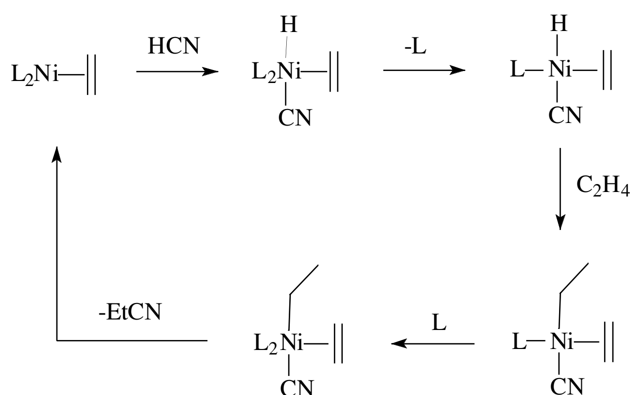


Fig. 1

Studies on the protonation of Ni-C species are rare, and systematic kinetic studies are even rarer. We have started to investigate the protonation of complexes based on the $\{Ni(triphos)\}^{2+}$ (triphos = $\{Ph_2PCH_2CH_2\}_2PPh$) site. The kinetics of the protonation of $[NiMe(triphos)]^+$ have been studied in MeCN[8] and shows three key mechanistic features: (i) initial protonation is at the metal; (ii) the hydrido-species thus formed $[NiH(Me)(triphos)]^{2+}$ does not undergo a migration reaction to produce methane, but rather, (iii) further protonation directly at the methyl-group produces methane. These features are summarised by the mechanism in Figure 3. Analysis of the kinetics reveals that the pK_a of $[NiH(Me)(triphos)]^{2+}$ is 8.8 in MeCN.

$[NiH(Me)(triphos)]^{2+}$ has been detected in solution using ^1H NMR spectroscopy. This is an important point because in many analogous systems the detection of an intermediate hydrido, alkyl-species is often taken as evidence that formation of alkane involves migration of hydride to the alkyl.[9] However, few detailed kinetic studies have been performed on these systems and it is only the kinetics that reveals the hydrido, alkyl species is not a kinetically competent intermediate on the pathway to alkane.

In an extension to the work on $[NiMe(triphos)]^+$ we have also studied the protonation chemistry of the analogous $[Ni(\eta^3\text{-}C_3H_5)(triphos)]^+$. The kinetics of the protonation reaction[10] in MeCN demonstrate that the reaction involves two coupled equilibria as shown in Figure 4. Unfortunately the kinetics cannot distinguish whether the nickel or the allyl site is protonated preferentially. Earlier studies on a variety of different complexes[1,2] show that it is impossible to reach a decision based on precedent: preferential protonation at metal or ligand can dominate depending on the complex being studied. Irrespective of where the initial protonation occurs, the important point is that the proton rapidly equilibrates between the allyl and nickel sites. If protonation is initially at the allyl-group as shown in Figure 4, then we can analyse the kinetics and obtain the pK_a's in MeCN for $[Ni(\eta^2\text{- }MeCHCH_2)(triphos)]^{2+}$ (16.7) and $[Ni(H)(\eta^3\text{-}C_3H_5)(triphos)]^{2+}$ (15.2). Clearly the difference in the basicities of the nickel and carbon sites is small. This seems intuitively reasonable if the proton is to equilibrate between the two positions. Interestingly, we can estimate that when bound to nickel, propene is greater than 10^{33} times stronger acid than free propene.

Comparison with the kinetic data obtained with $[NiMe(triphos)]^+$ shows that the nickel in $[Ni(\eta^3\text{-}C_3H_5)(triphos)]^+$ is more than a million times more basic than in $[NiMe(triphos)]^+$. This large difference is in stark contrast to what is observed in organic chemistry where the difference in pK_a's of a substituent bound to methyl and allyl groups is small (*eg* for $MeNH_3^+$, pK_a = 10.7; for $H_2CCHCH_2NH_3^+$, pK_a = 9.5).[11] It has been speculated that the reason for the large increase in the basicity of the nickel in the allyl complex is due to the electron-releasing effect of the η^3-allyl ligand. It would be interesting to measure the basicity of the nickel in the analogous complex $[Ni(\eta^1\text{-}C_3H_5)(triphos)]^+$. It might be anticipated that the basicity of the nickel in $[Ni(\eta^1\text{-}C_3H_5)(triphos)]^+$ would be similar to that of nickel in $[NiMe(triphos)]^+$.

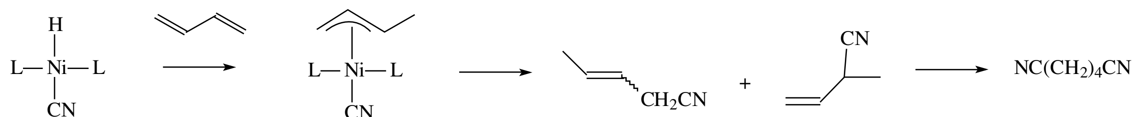


Fig. 2

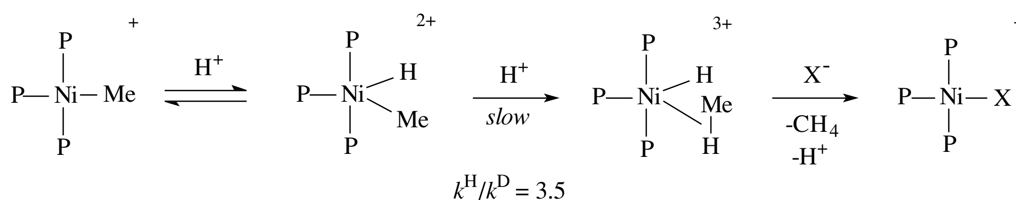


Fig. 3

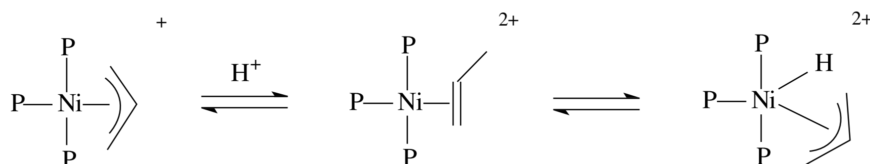


Fig. 4

Protonation and metalloenzymes

There is a structural diversity in the active sites for nickel-based enzymes which is mirrored in the diversity of the catalytic roles performed by the enzymes.[12] Thus, urease is a binuclear hydrolytic enzyme which hydrolyses urea to ammonia and carbon dioxide. The role of the nickel atoms in this enzyme is that of Lewis acids. In contrast, methyl coenzyme M reductase catalyses the final step of the production of methane in methanogenic bacteria and contains the F-430 prosthetic group in which a nickel atom is contained within a tetrahydrocorphine (a tetrapyrrole macrocycle but with many saturated bonds, hence making it more flexible and puckered than either a corrin or porphyrin). The mechanism of the catalysis probably involves radical reactions and hydrogen atom transfer rather than proton transfer to produce methane. Acetyl coenzyme A / carbon monoxide dehydrogenase are enzymes which transform carbon monoxide. The active site of carbon monoxide dehydrogenase is a Ni-Fe-S cluster.[13] However, it is only in the hydrogenases that proton-transfer reactions are central to the enzyme's action and thus of all the nickel-based enzymes, only the NiFe-based hydrogenases will be discussed in detail.

The hydrogenases[12,14–16] are a group of enzymes which accomplish the reaction shown in Equation (1), and are classified according to their composition. Three distinct classes of metal-containing hydrogenases have been identified: Fe-only hydrogenases; NiFe-hydrogenases and NiFeSe-hydrogenases. In general, NiFe-based hydrogenases involve the reduction of protons.

$$2H^+ + 2e \rightleftharpoons H_2 \qquad (1)$$

All NiFe-hydrogenases comprise at least two subunits of molecular weights *ca* 60 and 30kDa respectively. The metal content comprises: a NiFe centre and at least two Fe_4S_4 clusters. The structure of the oxidised NiFe binuclear active site from *Desulfovibrio gigas* is shown in Figure 5. The binuclear active site comprises a nickel atom which is predominantly coordinated by cysteinate ligands and a hydroxide in a distorted square-based pyramidal arrangement. Two of the cysteinate ligands and the hydroxide act as bridges to an adjacent iron atom. The iron atom has a distorted octahedral geometry. In addition, from at least one source a 1,3-dithiopropane ligand also bridges the nickel and iron. The ligation of the iron atom is analogous to that found in the Fe-only hydrogenase binuclear active site. It comprises three diatomic non-protein ligands: CO and CN^-. Presumably these ligands are beneficial for the chemistry that the site performs, nonetheless they seem rather bizarre choices, since both CO and CN^- are commonly associated with toxic effects on biological systems. A subclass of the NiFe-hydrogenases are the NiFeSe-hydrogenases where a selenocysteinate is a ligand to nickel.

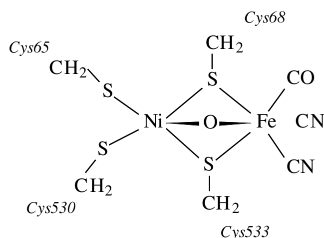


Fig. 5

The NiFe-site is buried deep in the larger subunit, whilst the Fe-S clusters are contained within the small subunit and form an approximate linear arrangement. This arrangement supports the proposal that these clusters form an electron transfer pathway between the external reductant and the active site.

Since Fe-only hydrogenases are known, and operate perfectly adequately, it has been proposed that the nickel in the NiFe-hydrogenases facilitate substrate binding. Thus, although the Fe-only hydrogenases have a higher hydrogenase activity (V_V = 9000–50000 μmol min^{-1} mg^{-1}) they have lower dihydrogen affinity (K_M = 7μmol dm^{-3}) than the NiFe hydrogenase (V_V = 700 μmol min^{-1} mg^{-1}; K_M = 0.07μmol dm^{-3}).

Studies on the NiFe-hydrogenase indicate that at least six states of the binuclear NiFe site are detectable, with at least three states characterised by EPR spectroscopy.[17–19] The EPR detectable states have been labelled Ni-SR (EPR silent); Ni-C (only paramagnetic state; formed from reduction of Ni-SR) and Ni-R (EPR silent; formed by reduction of Ni-C). The EPR signals have been attributed to the nickel atom in the binuclear active site, indicating that the iron is low spin Fe^{II} throughout the catalysis. The oxidation state of the nickel is unknown but Ni^{I}, Ni^{II} or Ni^{III} have been proposed.

Comparison of EPR and ESEEM spectra[20] of the NiFe-hydrogenases in H_2O and D_2O indicates the presence of exchangable protons in the vicinity of the nickel in the Ni-C state. Q-band ENDOR spectroscopy[21] indicates two types of exchangable protons. These exchangable protons only interact weakly with nickel and so it is concluded that they are outside the first coordination sphere and probably represent water or acidic amino acid side chains. It is unlikely that the protons correspond to Ni-H or Ni-H_2 species because of the weak coupling constants (4.4 and 16.6MHz) observed with these species. In addition, flushing the system with argon did not remove these signals (*ie* H_2 was not flushed away).

A mechanism for the action of the Ni-based hydrogenase, consistent with all the currently known experimental observations[12] is shown in Figure 6. The initial "activation" of the binuclear site by reduction, protonation and consequent

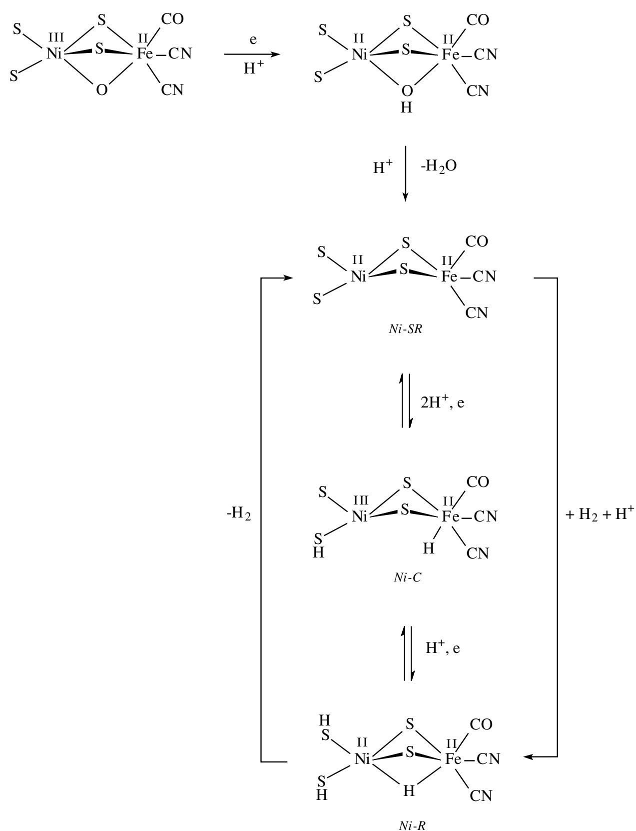


Fig. 6

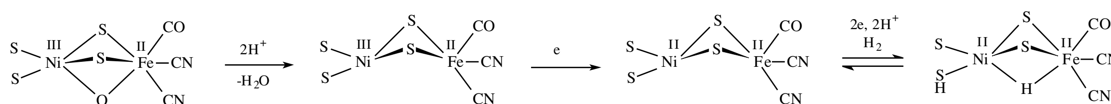


Fig. 7

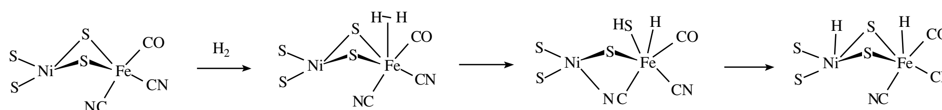


Fig. 8

dissociation of water primes the site for binding dihydrogen. The activation of dihydrogen involves the formation of a hydride (proposed to bridge between the iron and nickel) and a coordinated thiol. In this manner dihydrogen is formally cleaved heterolytically into H^- and H^+.

More recently, mechanisms have been developed from quantum mechanical calculations. Figure 7 shows such a mechanism proposed[22] by Dole et al. In this mechanism dihydrogen binds to the active site and is heterolytically cleaved to produce a bridging hydride and protonated cysteinate ligand.

In an analogous study, Pavlov et al[23] have probed the mechanism of the NiFe hydrogenase using DFT calculations. The proposed mechanism shown in Figure 8 involves initial binding of dihydrogen to iron to form a η^2-H_2 species, which then undergoes heterolytic splitting. In the key step hydride transfer to iron and proton transfer to an adjacent cysteinate sulfur, is accompanied by ligand dissociation of the thiol cysteine from nickel while remaining bound to iron. Simultaneously, the cyanide ligand on iron binds to nickel in a bridging mode. After dihydrogen dissociation, the hydride bound to iron can be transferred to nickel which should be a necessary preliminary for subsequent hydrogen atom or electron transport.

One elementary reaction which features in all of the mechanisms shown in Figures 6-8 is the transfer of protons between sulfur and metal atoms. The movement of protons between metal and sulfur ligands has also been proposed in the action of the nitrogenases.[24] The structures of the active sites of both nitrogenases and hydrogenases have been determined. The hydrogenase site has been discussed above, and for the Mo-based nitrogenase is an Fe-S-based cluster whose core comprises $MoFe_7S_9$. The predominant sulfur ligation in both active sites has led to the reasonable conclusion that sulfur plays a key role in the transfer and reduction of protons by nitrogenases and hydrogenases.

Although thiol and hydride/thiolate complexes are known, there are few studies which show that the hydrogen can move between metal and sulfur.[25] Consequently we have only a poor understanding of the electronic factors which facilitate this transfer, and no direct evidence that the reaction is truly *intramolecular*. Whilst the intramolecular migration of protons between metal and ligand is a reaction which is widespread with carbon-based ligands (as in the well-known formal insertion of alkenes into M-H bonds) this pathway is less evident with ligands containing electronegative donor atoms, where acid-base-catalysed mechanisms can be energetically more favourable.

Recently, we have studied the protonation of $[Ni(SR)(triphos)]^+$ (R = aryl or alkyl) by $[lutH]^+$ (lut = 2,6-dimethylpyridine)[26] in MeCN. With $[Ni(SPh)(triphos)]^+$ the kinetics indicate a simple equilibrium proton-transfer reaction in which the proton is transferred to and from the complex, presumably at one of the lone pairs of electrons on the sulfur atom. The rate constants for proton transfer are appreciably slower than the diffusion-controlled limit ($k_{forward}$ = 20 $dm^3\ mol^{-1}\ s^{-1}$, $k_{reverse}$ = 5 $dm^3\ mol^{-1}\ s^{-1}$). It seems likely that the slowness of this reaction is, at least in part, because of unfavourable steric interactions when the $[lutH]^+$ or lut approaches the sulfur atom which is buried between the phenyl-groups of the triphos co-ligand. We have now extended our studies to $[Ni(SC_6H_4R\text{-}4)(triphos)]^+$ (R = NO_2, Cl, MeO or Me).[27] Interestingly, there are marked and unexpected differences in reactivity to that observed with $[Ni(SPh)(triphos)]^+$: (i) the rates of proton transfer are slower with the 4-substituted thiolates; (ii) the rates are very insensitive to the nature of R and (iii) the kinetics are different. In particular, the kinetics are consistent with a mechanism in which there is initial formation of a hydrogen bonded species which precedes the transfer of the proton. It appears that the major effect of the 4-substituent is to restrict the access of $[lutH]^+$ to the sulfur atom, possibly by perturbing the positions of the phenyl-groups on the triphos ligand, thus crowding the sulfur atom.

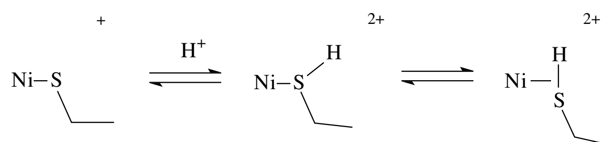


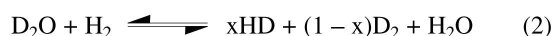
Fig 9

The kinetics of the protonation of $[Ni(SEt)(triphos)]^+$ by $[lutH]^+$ are more complicated than those observed with the aryl thiolate derivatives, and are consistent with the mechanism shown in Figure 9. Thus, initial protonation occurs at the sulfur but this is followed by an intramolecular equilibration which most likely involves the formation of an η^2-thiol ligand. We cannot entirely rule out the possibility that this intramolecular reaction involves complete transfer of the proton to the nickel and formation of $[Ni(H)(SEt)(triphos)]^{2+}$, but this seems unlikely since such five-coordinate, d^6 Ni^{IV} species are unknown.

The studies on simple nickel-thiolate complexes are pertinent to discussions on the mechanisms of action of hydrogenases. Consider the pathway for reduction of protons at a nickel site. Our studies on $[Ni(SR)(triphos)]^+$ show that initial protonation of metal-thiolate species will always occur at the lone pair of electrons on sulfur, since this is the most basic site. Protonation at the metal is thermodynamically less favourable, and usually kinetically slower than protonation of a stereochemical lone pair of electrons.[1,2] If it is essential during the enzyme's action that proton transfer to the metal occur,

then this is more favourable with alkyl thiolates than aryl thiolates. Analysis of the kinetic data for the reactions of $[Ni(SR)(triphos)]^+$ shows that the acidities of the corresponding coordinated PhSH and EtSH vary by less than a factor of 40. This is in contrast to the behaviour of the free thiols, where PhSH is 10^4 times stronger acid than EtSH. Thus, coordination of RSH to the $\{Ni(triphos)\}^{2+}$ site has a levelling effect on the acidities of the thiols, and this must have a complementary effect on the electron-richness of the $\{Ni(triphos)\}^{2+}$ site. It seems reasonable that with the more electron-releasing alkyl thiolate ligands the $\{Ni(triphos)\}^{2+}$ site is more electron-rich. Thus, in $[Ni(SEt)(triphos)]^+$, the nickel and sulfur are sufficiently similar in basicity so that in $[Ni(SHEt)(triphos)]^{2+}$ the proton effectively bridges the two sites.

The involvement of metal hydrides in the action of hydrogenases is indicated by the ability of these enzymes to catalyse the hydrogen isotope exchange shown in Equation (2) and the equilibration of *ortho*- and *para*-hydrogen shown in Equation (3).



$$D_2O + H_2 \rightleftharpoons xHD + (1 - x)D_2 + H_2O \quad (2)$$

$$ortho\text{-}H_2 \rightleftharpoons para\text{-}H_2 \quad (3)$$

These reactions are well known in simple polyhydrido-complexes of all metals and are not specific for nickel. The reactions are merely a consequence of rapid proton/hydride exchange at the metal site.[28] The selective formation of HD or D_2 has been achieved in the reaction of $[Ni(Ph_2PCH_2CH_2PPh_2)_2]$ with DCl: HD being the exclusive product at low acid concentrations and D_2 at high acid concentrations. Both pathways involve the formation of the detected intermediate, $[NiD(Ph_2PCH_2CH_2PPh_2)_2]^+$ as shown in Figure 10. The formation of HD occurs by the pathway[29] shown at the top of this Figure.

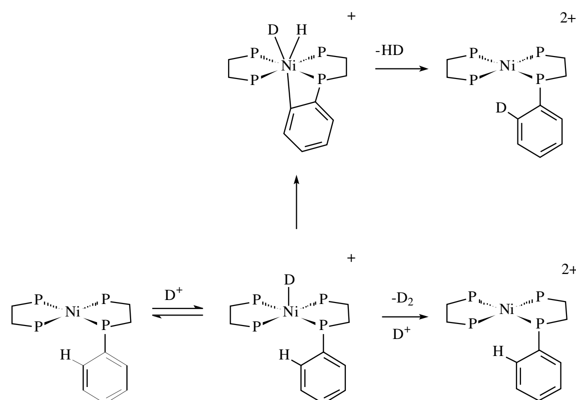


Fig. 10

Deuteration of the metal labilises the Ni-P bond to dissociation and at low concentration of DCl this reaction is faster than D^+ attack at the Ni-D bond, thus allowing *ortho*-metallation to occur. Subsequent release of HD and further attack of D^+ results in deuteration of the *ortho*-site on the phenyl group. At high concentrations of DCl direct attack at the Ni-D (or transient formation of $[NiD_2(Ph_2PCH_2CH_2PPh_2)_2]^{2+}$ is faster than *ortho*-metallation and results in the selective release of D_2.

Summary

Protonation at the metal and ligand is central to the understanding of how both enzymes and industrial catalysts operate at the molecular level. The recurring theme in studies on the protonation of all metal complexes is that the ultimate residence of the proton is not necessarily the initial binding site, and the movement of proton between sites can occur by a variety of mechanisms. These features are also evident in the reactions of simple nickel complexes and mechanistic studies are revealing the subtle interplay between ligand and metal which are the basis of the kinetic and thermodynamic control of protonation reactions at these sites.

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