Pure & Appl. Chem., Vol. 58, No. 4, pp. 575–584, 1986.
Printed in Great Britain.

Free radical mechanisms in organometallic and bioorganometallic chemistry

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Abstract - The characteristic weakness of many metal-metal, metal-alkyl and metal-hydrogen bonds contributes to the facile occurrence of a variety of homolytic bond dissociation and displacement reactions that result in the formation of metal-centered and/or carbon-centered free radicals. The thermodynamic, kinetic and mechanistic aspects of such processes are discussed. Particular emphasis is accorded to recent studies relating to (a) free radical mechanisms of catalytic hydrogenation and hydroformylation, (b) free radical processes in bioorganometallic chemistry, notably coenzyme B_{12}-dependent rearrangements, and (c) free radical chain mechanisms of oxidative addition, reductive elimination and insertion.

INTRODUCTION

Recent studies have accorded increasing emphasis to the roles of free radical processes in organometallic chemistry and catalysis (ref. 1-3). Such processes now are recognized as being much more widespread than previously suspected not only in new contexts but also in the context of familiar reactions, for example certain hydrogenation, hydroformylation, insertion and reductive elimination reactions that had previously been interpreted in terms of alternative non-radical mechanisms.

Eqs. 1-3 depict schematically several general reactions in which metal-centered radicals ($L_nM^\bullet$) are generated and eqs. 4-8 some processes that generate carbon-centered radicals ($R^\bullet$).

$$L_{2n}M_2 \longrightarrow 2L_nM^\bullet \quad (1)$$

$$L_{2n}M_2 + X^\bullet \longrightarrow L_nM\text{-}X + L_nM^\bullet \quad (2)$$

$$L_nM\text{-}H + R^\bullet \longrightarrow L_nM^\bullet + RH \quad (3)$$

$$L_nM\text{-}H + \text{>C=C<} \longrightarrow L_nM^\bullet + H\text{-}\overset{|}{C}\text{-}\overset{|}{C}^\bullet \quad (4)$$

$$L_nM^\bullet + R\text{-}X \longrightarrow L_nM\text{-}X + R^\bullet \quad (5)$$

$$L_nM\text{-}R \longrightarrow L_nM^\bullet + R^\bullet \quad (6)$$

$$L_nM\text{-}R \xrightarrow{e^-} [L_nM\text{-}R]^- \longrightarrow [L_nM]^- + R^\bullet \quad (7)$$

$$L_nM\text{-}R \xrightarrow{-e^-} [L_nM\text{-}R]^+ \longrightarrow [L_nM]^+ + R^\bullet \quad (8)$$

The facile occurrence of the reactions depicted by eqs. 1 and 2 reflect the characteristic weakness of many transition metal-metal single bonds, while eqs. 3-4 and eq. 6 reflect the characteristic weakness of transition metal-alkyl and -hydrogen bonds, respectively. The metal-alkyl bond dissociation that frequently follows electron transfer (eqs. 7 and 8) reflects the stability typically associated with the closed shell (usually 18 electron) configurations of organotransition metal compounds.

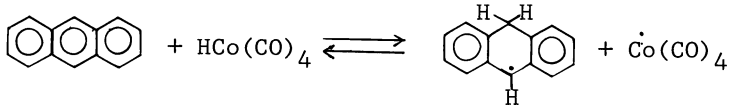
This paper is concerned with the thermodynamic, kinetic and mechanistic aspects of the reactions depicted by eqs. 1-8 and with recent studies relating to the roles of such free radical processes in the mechanisms of catalytic hydrogenation and hydroformylation reactions, of coenzyme B_{12}-dependent rear-

rangement and of certain oxidative addition, reductive elimination and insertion reactions.

CATALYTIC HYDROGENATION AND HYDROFORMYLATION

In 1975, Feder and Halpern (ref. 4) proposed that the $HCo(CO)_4$-catalyzed hydrogenation of arenes, which previously had been interpreted in terms of a concerted insertion of the arene into the Co-H bond of $HCo(CO)_4$ (ref. 5), proceeds instead by a free radical mechanism, depicted for the case of anthracene by eqs. 9-12, in which the rate-determining step (eq. 10) involves the transfer of an H atom from $HCo(CO)_4$ to the substrate. Coordination of the substrate or formation of organometallic intermediates does not play a role in this mechanism.

$$Co_2(CO)_8 + H_2 \rightleftharpoons 2HCo(CO)_4 \qquad (9)$$



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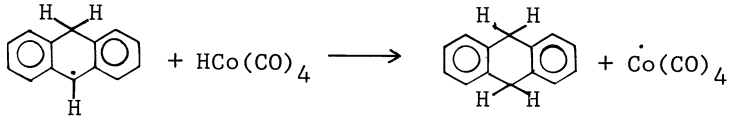

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$$2\dot{C}o(CO)_4 \rightleftharpoons Co_2(CO)_8 \qquad (12)$$

In view of evidence that the reverse of reaction (10) and related H atom transfers from organic radicals to metal-centered radicals are characterized by very low activation barriers (exhibiting near diffusion-controlled kinetics) it was concluded that $\Delta H^{\ddagger}$ for a reaction such as (10) could be approximated by the endothermicity of the reaction. On this basis it was predicted that the mechanistic scheme of eq. 10-11 should be widespread and should extend particularly to the catalysis by metal hydrides of the hydrogenation of conjugated olefins such as styrene which yield stabilized radicals after H atom transfer (ref. 1). Indeed, analogous mechanisms had been advanced earlier for the $Co(CN)_5^{3-}$-catalyzed hydrogenation of cinnamate and related substrates (ref. 6-8).

Convincing evidence for such a mechanism (eqs. 13-15) has been provided by the results of a study of the stoichiometric hdyrogenation of α-methylstyrene by $HMn(CO)_5$ (eq. 16) (ref. 9). Supporting this mechanistic scheme are (a) the kinetics of the reactions ($-d[PhC(CH_3)=CH_2]/dt = k[PhC(CH_3)=CH_2][HMn(CO)_5]$, $\Delta H^{\ddagger}$ = 21.4 kcal mol^{-1}, $\Delta S^{\ddagger}$ = -12 cal mol^{-1} K^{-1}, no inverse CO dependence), (b) an inverse kinetic isotope effect [$k(HMn(CO)_5)/k(DMn(CO)_4) \sim 0.4$, interpreted in terms of the increase in the stretching frequency when H(D) is transferred from Mn to C [$\nu_{Mn-H} \sim 1800$ cm^{-1}, $\nu_{C-H} \sim 3000$ cm^{-1}], (c) formation of $HMn(CO)_5$ accompanying the reaction of $DMn(CO)_5$ with α-methylstyrene, consistent with the reversibility of the first step of eq. 13 and, most convincingly, (d) the observation of CIDNP signals that could be quantitatively interpreted in terms of this mechanistic scheme.

$$PhC(CH_3)=CH_2 + HMn(CO)_5 \rightleftharpoons \overline{Ph\dot{C}(CH_3)_2, \dot{M}n(CO)_5}$$

$$\longrightarrow Ph\dot{C}(CH_3)_2 + \dot{M}n(CO)_5 \quad \text{(rate-determining)} \qquad (13)$$

$$Ph\dot{C}(CH_3)_2 + HMn(CO)_5 \longrightarrow PhCH(CH_3)_2 + \dot{M}n(CO)_5 \qquad (14)$$

$$2\dot{M}n(CO)_5 \longrightarrow Mn_2(CO)_{10} \qquad (15)$$

$$PhC(CH_3)=CH_2 + 2HMn(CO)_5 \longrightarrow PhCH(CH_3)_2 + Mn_2(CO)_{10} \qquad (16)$$

Based on these considerations we also proposed (ref. 1) that the $HCo(CO)_4$-catalyzed hydroformylation of styrene and related conjugated substrates (but not of simple alkenes such as propylene) proceeds by an analogous free radical mechanism depicted by Fig. 1 rather than by the conventional Heck-Breslow mechanism involving coordination and migratory insertion of the olefin (i.e., $HCo(CO)_4 \xrightarrow{RCH=CH_2} HCo(CO)_3(RCH=CH_2) \longrightarrow RCH_2CH_2Co(CO)_3$, etc.). Convincing support for this proposal now has been advanced through a number of studies on

the stoichiometric and catalytic hydrogenation and hydroformylation reactions of styrene and related substrates with $HCo(CO)_4$. Paralleling the results of our study of reaction (16), evidence for the free radical mechanism of the $HCo(CO)_4$-catalyzed hydroformylation of styrene encompasses the following observations.

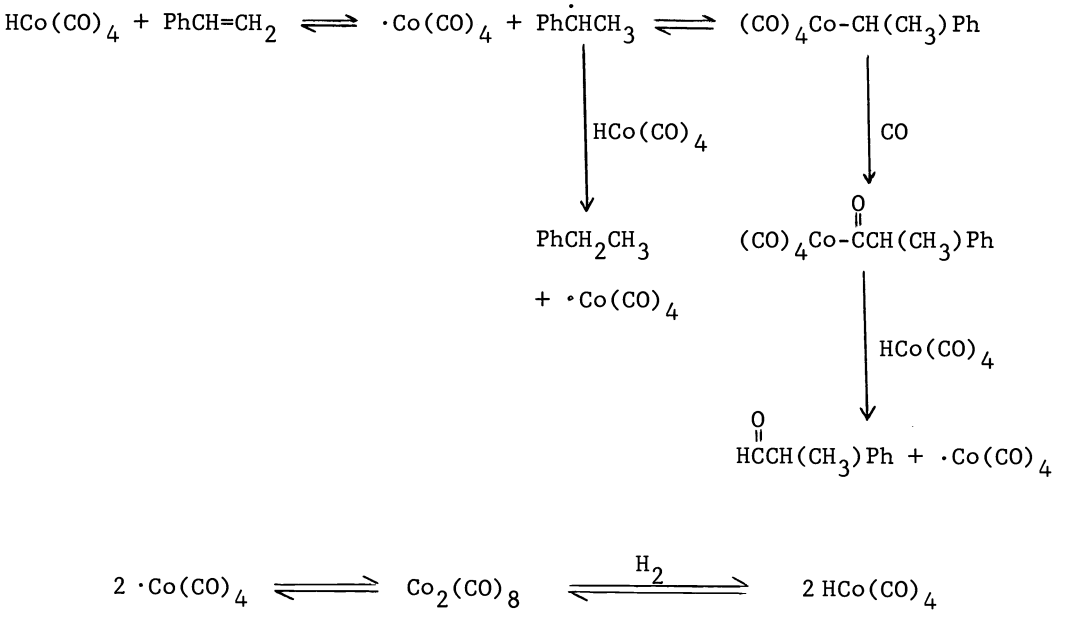
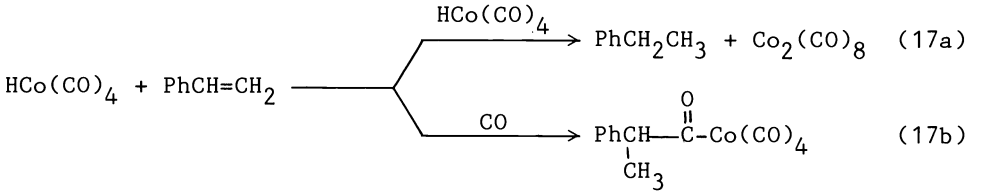


Fig. 1. Proposed mechanism of the hydroformylation of styrene

(a) In contrast to the hydroformylation of simple alkenes, the reaction of $HCo(CO)_4$ with styrene exhibits a simple second order rate-law ($k[PhCH=CH_2][HCo(CO)_4]$) with no inverse CO dependence (ref. 10).

(b) Both the stoichiometric hydrogenation and acyl-forming reactions of $HCo(CO)_4$ with styrene (eqs. 17(a) and 17(b)), exhibit inverse H/D kinetic isotope effects; $k(HCo(CO)_4)/k(DCo(CO)_4) = 0.45$ and 0.49, respectively (ref. 10). Similar inverse isotope effects now have been observed for the reactions of $HCo(CO)_4$ and $HMn(CO)_5$ with a variety of other olefins and arenes. Indeed, such inverse kinetic isotope effects, which we originally reported for the reaction of $HMn(CO)_5$ with α-methylstyrene (ref. 9) now have come to be considered as diagnostic for this type of mechanism (ref. 11-14).



(c) The acyl product of the stoichiometric reaction of $HCo(CO)_4$ with styrene (eq. 17b) is the α- rather than β-adduct (ref. 10).

(d) The yields of $PhCH(CH_3)C(=O)Co(CO)_4$ (eq. 17b) relative to that of $PhCH_2CH_3$ (eq. 17a) is favored by solvents of high viscosity which are expected to impede the cage escape of the $PhĊH(CH_3)$ and $·Co(CO)_4$ radicals in the scheme of Fig. 1 and hence favor their combination to form $PhCH(CH_3)$-$Co(CO)_4$ (ref. 15).

(e) CIDNP signals, attributable to the mechanistic scheme of Fig. 1, now

have been reported for the stoichiometric reaction of $HCo(CO)_4$ with $Ph_2C{=}CH_2$ (paralleling reaction 16) as well as for the hydroformylation of styrene (ref. 16-17).

A scheme analogous to that of Fig. 1, but involving $HFe(CO)_4^-$ instead of $HCo(CO)_4$, has been invoked for the hydroformylation and hydrogenation of styrene by CO and water, catalyzed by iron carbonyls (ref. 18).

We also have proposed that the product-forming step in the cobalt carbonyl-catalyzed hydroformylation reaction (eq. 21) may proceed through a free radical mechanism, namely that depicted by eqs. 18-21 (ref. 1).

$$R\overset{O}{\overset{\|}{C}}Co(CO)_4 \longrightarrow R\dot{C}O + {}^{\cdot}Co(CO)_4 \qquad (18)$$

$$R\dot{C}O + HCo(CO)_4 \longrightarrow RCHO + {}^{\cdot}Co(CO)_4 \qquad (19)$$

$$2\,{}^{\cdot}Co(CO)_4 \longrightarrow Co_2(CO)_8 \qquad (20)$$

$$R\overset{O}{\overset{\|}{C}}Co(CO)_4 + HCo(CO)_4 \longrightarrow RCHO + Co_2(CO)_8 \qquad (21)$$

A recent study has supported this proposal (ref. 19). In addition we have provided evidence for analogous free radical mechanisms in the related C-H bond-forming reductive elimination reactions involving manganese carbonyls depicted by eqs. 22 and 23 (R = $p\text{-}CH_3OC_6H_4CH_2$; L = $(p\text{-}CH_3OC_6H_4)_3P$)(ref. 20-21).

$$\underline{cis}\text{-}RMn(CO)_4L + HMn(CO)_5 \longrightarrow RH + Mn_2(CO)_9L \qquad (22)$$

$$\underline{cis}\text{-}RMn(CO)_4L + HMn(CO)_5 + CO \longrightarrow RCHO + Mn_2(CO)_9L \qquad (23)$$

MECHANISMS OF COENZYME B_{12}-DEPENDENT REARRANGEMENTS

Coenzyme B_{12} (5'-deoxyadenosylcobalamin, abbreviated $AdCH_2\text{-}B_{12}$), whose structure is depicted in Fig. 2, serves as a cofactor for a variety of enzymatic reactions, a common feature of which involves the 1,2-interchange of a hydrogen atom and another substituent on adjacent carbon atoms of the substrate according to eq. 24 (X = OH, NH_2, $CH(NH_2)COOH$, C(=O)SCoenzyme A or $C(=CH_2)COOH$) (ref. 22, 23). Specific examples of such reactions are the deamination of ethanolamine catalyzed by ethanolamine ammonia lyase (eq. 25) and the methylmalonyl-CoA mutase rearrangement (eq. 26).

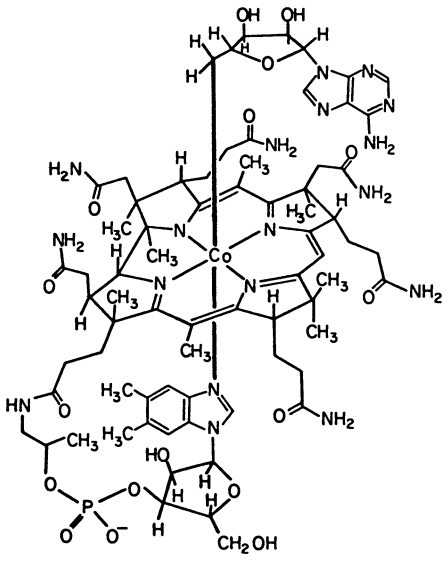
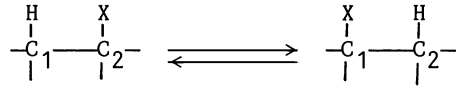


Fig. 1. Coenzyme B_{12}



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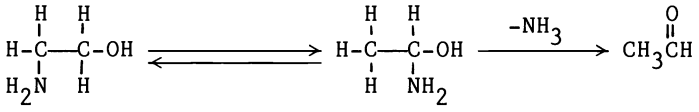

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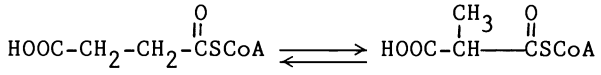

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A variety of enzymatic studies have provided evidence for the essential features of the mechanistic scheme depicted by Fig. 3, which encompasses the following sequence of steps (ref. 22, 23): (i) enzyme-induced homolytic dissociation of the cobalt-carbon bond of coenzyme B_{12} to generate cob(II)alamin (vitamin B_{12r}) and a 5'-deoxyadenosyl radical ($AdCH_2^{\cdot}$), (ii) H-atom abstraction from the substrate to generate a substrate radical and 5'-deoxyadenosine ($AdCH_3$), (iii) rearrangement of the resulting substrate radical (either directly or through additional intermediate steps) to the corresponding product radical, and (iv) abstraction of an H-atom from $AdCH_3$ by the rearranged product radical to complete the rearrangement reaction.

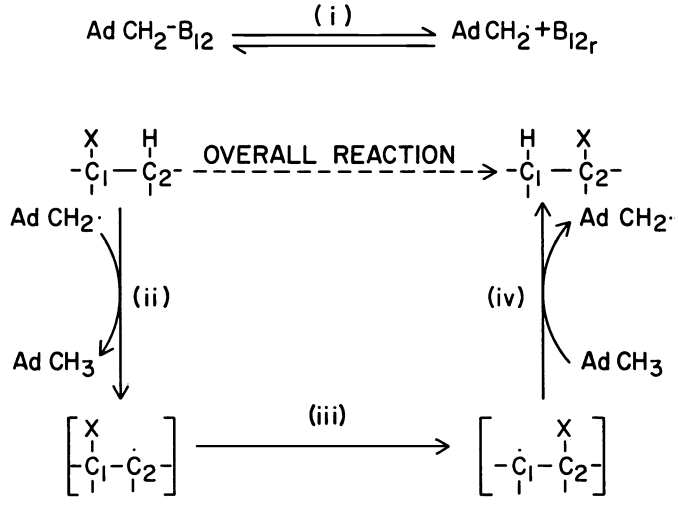


Fig. 3. Schematic mechanism of coenzyme B_{12}-dependent rearrangements

The role of coenzyme B_{12} encompassed by the mechanistic scheme of Fig. 3 implies a very weak cobalt-carbon bond. A troublesome feature of this interpretation has been the absence, at least until recently, of precedents for such weak transition metal bonds or, indeed, of information about transition metal bond dissociation energies in general (ref. 24).

We have recently accomplished the measurement of the cobalt-carbon bond dissociation energy ($D_{Co\text{-}CH_2Ad}$) of coenzyme B_{12} by determining the kinetics of the bond dissociation process in aqueous solution using $[Co^{II}(DH)_2(H_2O)]$ (where DH_2 = dimethylglyoxime), which forms a stronger Co-C bond than B_{12r}, to trap the $AdCH_2^{\cdot}$ radical in accord with eqs. 27-29 (ref. 25).

$$AdCH_2\text{-}B_{12} \underset{k_{-27}}{\overset{k_{27}}{\rightleftharpoons}} AdCH_2^{\cdot} + B_{12r} \qquad (27)$$

$$AdCH_2^{\cdot} + [Co^{II}(DH)_2(H_2O)] \xrightarrow{k_{28}} [AdCH_2\text{-}Co(DH)_2(H_2O)] \qquad (28)$$

$$AdCH_2\text{-}B_{12} + [Co^{II}(DH)_2(H_2O)] \longrightarrow [AdCH_2\text{-}Co(DH)_2(H_2O)] + B_{12r} \qquad (29)$$

From the measured activation enthalpy of reaction 27 ($\Delta H_{27}^{\ddagger}$ = 28.6 kcal/mol, $\Delta S_{27}^{\ddagger}$ = 2 cal mol^{-1} k^{-1}) the value of $D_{Co\text{-}CH_2Ad}$ was deduced to be 26 ± 2 kcal/mol. (A related measuremnt in ethylene glycol yields a somewhat higher value of ca 31 kcal/mol) (ref. 26).

Although the Co-C bond of coenzyme B_{12} is weak compared with typical covalent bonds in organic molecules, the value of k_{27} calculated from the above activation parameters is only ca 10^{-7} sec^{-1}. This is some 10^9 times smaller ($\Delta\Delta G^{\ddagger} \sim$ 13 kcal/mol) than the values of the catalytic rate constants (ca 10^2 sec^{-1}) that have been estimated for several coenzyme B_{12}-dependent enzymatic reactions. Thus, it would appear that considerable further weakening of the Co-C bond, by interaction with the enzyme, is required to achieve dissociation rates that are comparable with the enzymatic rates.

To identify the factors that might be responsible for this bond weakening and dissociation, we have examined the influence of various electronic and steric parameters on the Co-C bond dissociation energies of some coenzyme B_{12} model compounds including bis(dimethylglyoxime)cobalt alkyl and Schiff base cobalt alkyl compounds (ref. 24, 27, 28). The bond dissociation energies yielded by these measurements encompass the range 17 to 25 kcal/mol, i.e., of the same order as the value for coenzyme B_{12} itself. While these measurements reveal some influence of electronic factors on the Co-C bond dissociation energy, by far the most important influences are exerted by steric factors, the Co-C bond dissociation energy exhibiting a marked inverse dependence on the size of either the alkyl group or the trans-axial ligand. Structural determinations of bis(dimethylglyoxime)cobalt alkyl compounds also reveal marked steric influences on the Co-C bond length (ref. 29). In the light of these considerations it seems highly likely that the enzyme-induced weakening of the coenzyme Co-C bond is due to steric influences, namely an upward conformational distortion of the corrin ring that increases the steric repulsion of the 5'-deoxyadenosyl substituent and induces dissociation of the Co-C bond (ref. 22, 23).

The least well understood and most controversial aspect of the mechanism of coenzyme B_{12}-dependent rearrangements is the mechanism of the rearrangement step itself (i.e., step iii in Fig. 3). The mechanism depicted in Fig. 3 implies that the rearrangement is triggered by H-atom abstraction from the substrate radical but does not require that the rearrangement involving the 1,2-migration of X (to yield, ultimately, the product radical) actually occur at the free radical stage. Suggested possible alternatives to such a direct rearrangement include rearrangement via intermediate carbonium ions or carbanions (generated by oxidation or reduction of the substrate radical by vitamin B_{12r}) or via an organocobalt intermediate, formed by combination of the substrate radical with B_{12r} (ref. 22, 23).

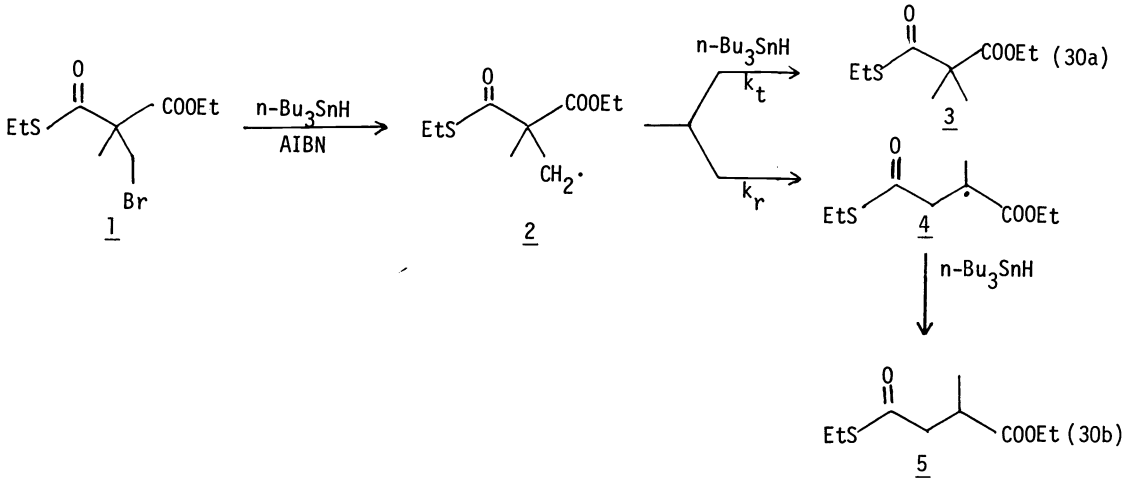
One problem with the proposal of rearrangement at the initially-formed free radical stage is that such 1,2-migration in a free radical is precedented for only one of the coenzyme B_{12} substrates, namely α-methyleneglutarte, involving the migration of a substituted vinyl group, i.e., $-C(=CH_2)COOH$. For the other B_{12} substrates, migration of X (i.e., of OH, NH_2, $C(=O)SCoA$ or $CH(NH_2)COOH$) had not previously been observed in model free radicals. However, it should be noted that those coenzyme B_{12}-dependent rearrangements whose rates have thus far been reported are fairly slow ($k_{cat} \sim 10^2$ sec^{-1}). Radical rearrangement processes compatible with this time scale may well have escaped detection in earlier studies of free radical rearrangements, most of which were restricted to much shorter time scales.

To test whether a given substrate radical (or appropriate model thereof) would rearrange spontaneously on the time scale of coenzyme B_{12}-dependent reactions, it was necessary to generate the free radical unambiguously under conditions

where its lifetime was fairly long ($\gtrsim 10^2$ sec) and, preferably, susceptible to measurement and systematic variation. Furthemore, to eliminate the issue of possible cobalt participation in such rearrangements it was preferable to accomplish this in the absence of any cobalt complexes.

We have recently accomplished this for a radical, EtSC(=O)C(CH_3)($\dot{C}H_2$)COOEt (2) that models the substrate radical of the methylmalonyl-CoA mutase reaction (i.e., CoASC(=O)CH($\dot{C}H_2$)COOH (ref. 30). The model radical 2 was generated from the corresponding bromide 1 by reaction of n-$Bu_3\dot{S}n$ (generated by reaction of n-Bu_3SnH with 2,2'-azobisisobutylronitrile, AIBN) and the competiton between direct trapping (k_t) with n-Bu_3SnH to yield 3, and rearrangement (k_r) followed by trapping of the rearranged radical 4 to yield 5 in accord with eq. 30, was monitored as a function of the initial n-Bu_3SnH concentration. These measurements yielded values of k_t/k_r.

In combination with literature date for k_t, the following values were deduced: k_r (60.5°C) = 24 sec^{-1}, $\Delta H_r^{\ddagger}$ = 13.8 kcal/mol, $\Delta S_r^{\ddagger}$ = -11 cal mol^{-1} K^{-1}. The value of k_r at 30°C, calculated from these activation parameters, is 2.5 sec^{-1}. This is ca 40 times lower than the estimated value of k_{rat} for the methylmalonyl-CoA mutase reaction. However, this relatively modest difference ($\Delta\Delta G^{\ddagger}$ = 2.2 kcal/mol) could well be accommodated by chemical and structural differences between the model radical 2 and the methylmalonyl-CoA radical, as well as by effects of interaction of the (enzyme-bound) substrate with the enzyme, e.g., hydrogen bonding to the sulfur atom or conformational influences. Thus, we conclude that, while contributions from other pathways involving carbonium ions, carbanions or organocobalt intermediates cannot be definitively excluded, there is no plausible rationale at this stage for invoking such additional intermediates at least for the α-methyleneglutarate mutase and methylmalonyl-CoA mutase reactions. The rearrangement pathways for other coenzyme B_{12}-dependent substrates remain to be elucidated.



Thus, it appears that the principal, if not only, role of coenzyme B_{12} in these enzymatic processes is that of a free radical precursor, a role that utilizes the distinctive weakness of the cobalt-carbon bond. The use of an organometallic molecule for this purpose seems entirely appropriate since it is difficult to conceive of a stable organic molecule that would undergo thermal dissociation under such mild conditions to generate a highly reactive primary radical. At this stage there is no convincing evidence that the coenzyme or the cobalt atom plays any other role, for example in mediating the rearrangement step itself.

FREE RADICAL CHAIN MECHANISMS OF INSERTION AND OXIDATIVE ADDITION

We have recently found that styrene and certain other olefins undergo facile insertion reactions into the Rh-Rh and Rh-H bonds of Rh_2(OEP)$_2$ and (OEP)RhH, respectively, in accord with eqs. 31 and 32 (OEP = octaethylporphyrin) (ref. 31).

$$Rh_2(OEP)_2 + PhCH{=}CH_2 \longrightarrow (OEP)RhCH_2CH(Ph)Rh(OEP) \qquad (31)$$

$$(OEP)RhH + PhCH{=}CH_2 \longrightarrow (OEP)RHCH_2CH_2Ph \qquad (32)$$

The facile occurrence of reaction (32) was somewhat unexpected since it is not apparent that (OEP)RhH possesses an accessible cis-coordination site that is generally considered to be necessary for olefin migratory insertion.

On the basis of kinetic evidence it was concluded that reaction (31) proceeds through the free radical chain mechanism depicted by eqs. 33-35 (ref. 31).

$$\text{Initiation/Termination:} \quad Rh_2(OEP)_2 \rightleftharpoons 2(OEP)Rh^{\cdot} \qquad (33)$$

$$\text{Propogation:} \begin{cases} (OEP)Rh^{\cdot} + PhCH{=}CH_2 \rightleftharpoons (OEP)RhCH_2\dot{C}HPh & (34) \\ (OEP)RhCH_2\dot{C}HPh + Rh_2(OEP)_2 \longrightarrow \\ \qquad (OEP)RhCH_2CH(Ph)Rh(OEP) + (OEP)Rh^{\cdot} & (35) \end{cases}$$

Further evidence for this mechanism and, particularly, for the intermediacy of the $(OEP)RhCH_2\dot{C}HPh$ radical was provided by trapping of the latter. Efficient trapping by (OEP)RhH was manifested in catalysis by $Rh_2(OEP)_2$ of reaction (32) in accord with the mechanistic scheme of eqs. 33, 34 and 36.

$$\text{Initiation/Termination:} \quad Rh_2(OEP)_2 \rightleftharpoons 2(OEP)Rh^{\cdot} \qquad (33)$$

$$\text{Propogation:} \begin{cases} (OEP)Rh^{\cdot} + PhCH{=}CH_2 \rightleftharpoons (OEP)RhCH_2\dot{C}HPh & (34) \\ (OEP)RhCH_2\dot{C}HPh + (OEP)RhH \longrightarrow \\ \qquad (OEP)RhCH_2CH_2Ph + (OEP)Rh^{\cdot} & (36) \end{cases}$$

A free radical mechanism, analogous to that of eqs. 33, 34 and 36, also accommodates the previously reported (ref. 32) insertion of CO into the Rh-H bond of (OEP)RhH (eqs. 33, 37-39).

$$\text{Initiation/Termination:} \quad Rh_2(OEP)_2 \rightleftharpoons 2(OEP)Rh^{\cdot} \qquad (33)$$

$$\text{Propogation:} \begin{cases} (OEP)Rh^{\cdot} + CO \rightleftharpoons 2(OEP)Rh\dot{C}O & (37) \\ (OEP)Rh\dot{C}O + (OEP)RhH \longrightarrow \\ \qquad (OEP)RhCHO + (OEP)Rh^{\cdot} & (38) \end{cases}$$

$$\text{Overall Reaction:} \quad (OEP)RhH + CO \longrightarrow (OEP)RhCHO \qquad (39)$$

This mechanism parallels the microscopic reverse of mechanisms previously postulated for the decarbonylation of metal formyl complexes (ref. 33). Reaction (38) also finds a parallel in the mechanism recently proposed for the generation of metal formyl complexes by H-atom transfer to electrochemically generated metal carbonyl radicals (ref. 34).

The oxidative addition of benzyl bromide to $Rh_2(OEP)_2$ (eq. 42) also can be accommodated by a free radical chain mechanism, depicted by eqs. 33, 40 and 41 (ref. 31).

$$\text{Initiation/Termination:} \quad Rh_2(OEP)_2 \rightleftharpoons 2(OEP)Rh^{\cdot} \qquad (33)$$

$$\text{Propogation:} \begin{cases} (OEP)Rh^{\cdot} + C_6H_5CH_2Br \longrightarrow \\ \qquad (OEP)RhBr + C_6H_5CH_2^{\cdot} & (40) \\ C_6H_5CH_2^{\cdot} + Rh_2(OEP)_2 \longrightarrow \\ \qquad (OEP)RhCH_2C_6H_5 + (OEP)Rh^{\cdot} & (41) \end{cases}$$

$$\text{Overall Reaction:} \quad Rh_2(OEP)_2 + C_6H_5CH_2Br \longrightarrow (OEP)RhCH_2C_6H_5 + (OEP)RhBr \qquad (42)$$

We have found that $Rh_2(OEP)_2$ also undergoes oxidative addition reactions with

$HSnBu_3$ and with 9,10-dihydroanthracene (AH_2) (eqs. 43 and 44) (ref. 31). While analogous free radical chain mechanisms seem likely for these reactions their kinetics remain to be elucidated.

$$Rh_2(OEP)_2 + HSnBu_3 \longrightarrow (OEP)RhH + (OEP)RhSnBu_3 \quad (43)$$

$$Rh_2(OEP)_2 + AH_2 \longrightarrow (OEP)RhH + (OEP)Rh\text{-}AH \quad (44)$$

Several factors may be identified as contributing to the distinctive free radical chain processes that we have identified in these systems, namely (a) the weak Rh-Rh bond in $Rh_2(OEP)_2$ which is responsible for the accessibility of the (OEP)Rh$^{\bullet}$ radical (eq. 33), (b) unusually strong Rh-C bonds which contribute to the driving force for the chain propogating steps (34) and (37) and (c) the absence of axial ligands in $Rh_2(OEP)_2$ which renders feasible the homolytic displacement steps (35) and (41). Several features of these free radical chain mechanisms, notably the chain propogation sequences involving addition of metal free radicals to olefins and to CO and the S_H2 displacement of metal radicals at metal-metal bonds, are without direct precedent in transition metal chemistry.

Acknowledgement This research was supported by grants from the National Science Foundation (CHE82-07950) and the National Institutes of Health (AM-13339).

REFERENCES

1. J. Halpern, Pure Appl. Chem., 51, 2171-2182 (1979) and references therein.
2. M. F. Lappert and P. W. Lednov, Adv. Organomet. Chem., 14, 345-399 (1976).
3. J. K. Kochi, Acc. Chem. Res., 7, 351-360 (1974).
4. H. M. Feder and J. Halpern, J. Amer. Chem. Soc., 97, 7186-7188 (1975).
5. P. D. Taylor and M. Orchin, J. Org. Chem., 37, 3913-3915 (1972).
6. J. Kwiatek, Catalysis Revs., 1, 37-72 (1968). J. Kwiatek and J. K. Seyler, Adv. Chem. Ser., 70, 207-232 (1968).
7. J. Halpern and L. Y. Wong, J. Amer. Chem. Soc., 90, 6665-6669 (1968).
8. L. Simandi and F. Nagy, Acta Chim. Hung., 46, 137-148 (1968).
9. R. L. Sweany and J. Halpern, J. Amer. Chem. Soc., 99, 8335-8337 (1977).
10. F. Ungvary and L. Marko, Organometallics, 1, 1120-1125 (1982).
11. R. L. Sweany, S. C. Butler and J. Halpern, J. Organomet. Chem., 213, 487-492 (1981).
12. J. A. Roth and M. Orchin, J. Organomet. Chem., 182, 299-311 (1979).
13. T. E. Nalesnik and M. Orchin, J. Organomet. Chem., 199, 265-269 (1980); T. E. Nalesnik, J. H. Frendenberger and M. Orchin, J. Molec. Cat., 16, 43-49 (1982).
14. J. A. Roth, P. Wiseman and L. Ruzzala, J. Organomet. Chem., 240, 271-275 (1983).
15. F. Ungvary and L. Marko, J. Organomet. Chem., 249, 411-414 (1983).
16. M. Orchin, Ann. N.Y. Acad. Sci., 415, 129-134 (1983).
17. T. M. Bockman, J. F. Garst, R. B. King, L. Marko and F. Ungvary, J. Organomet. Chem., 279, 165-169 (1985).
18. J. Palagyi and L. Marko, J. Organomet. Chem., 236, 343-347 (1982).
19. J. Azran and M. Orchin, Organometallics, 3, 197-199 (1984).
20. M. J. Nappa, R. Santi, S. P. Diefenbach and J. Halpern, J. Amer. Chem. Soc.,104, 619-621 (1982).
21. M. J. Nappa, R. Santi and J. Halpern, Organometallics, 4, 34-41 (1985).
22. J. Halpern, in B_{12}, Vol. 1, D. Dolphin, Ed., Wiley, New York (1982), pp. 501-541 and references therein.
23. J. Halpern, Science, 227, 869-875 (1985) and references therein.
24. J. Halpern, Accts. Chem. Res., 15, 238-244 (1982) and references therein.
25. J. Halpern, S. H. Kim and T. W. Leung, J. Amer. Chem. Soc., 106, 8317-8319 (1984).
26. R. G. Finke and B. P. Hay, Inorg. Chem., 23, 3041-3043 (1884).
27. F. T. T. Ng, G. L. Rempel and J. Halpern, J. Amer. Chem. Soc., 101, 7124-7126 (1979); ibid., 104, 621-623 (1982); Inorg. Chim. Acta, 77, L165-L166 (1983).
28. T. T. Tsou, M. Loots and J. Halpern, J. Amer. Chem. Soc., 104, 623-624 (1982).
29. L. Randaccio, N. Bresciani-Pahor, P. J. Toscano and L. G. Marzilli, J. Amer. Chem. Soc., 103, 6347-6351 (1981); N. Bresciani-Pahor, M. Calligaris, G. Nardin and L. Randaccio, J. Chem. Soc. Dalton Trans.,

2549-2551 (1982); M. F. Summers, P. J. Toscano, N. Bresciani-Pahor, G. Nordin, L. Randaccio and L. G. Marzilli, J. Amer. Chem. Soc., 105, 6259-6263 (1983).
30. S. Wollowitz and J. Halpern, J. Amer. Chem. Soc., 106, 8319-8321 (1984).
31. R. S. Paonessa, N. C. Thomas and J. Halpern, J Amer. Chem. Soc., 107, 4333-4335 (1985).
32. B. B. Wayland and B. A. Woods, J. Chem. Soc. Chem. Comm., 700-701 (1981); B. B. Wayland, B. A. Woods and R. Pierce, J. Amer. Chem. Soc., 104, 302-303 (1982).
33. B. A. Naraganan, C. Amatore, C. P. Casey and J. K. Kochi, J. Amer. Chem. Soc., 105, 6351-6352 (1983); C. E. Sumner and G. O. Nelson, J. Amer. Chem. Soc., 106, 432-433 (1984).
34. B. A. Narayanan and J. K. Kochi, J. Organomet. Chem., 272, C-49-C53 (1984).