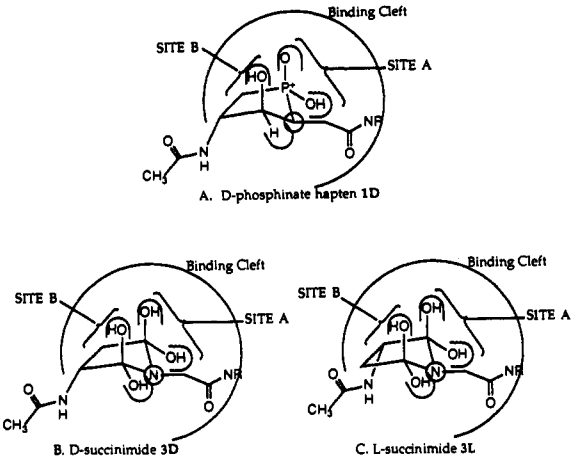

Figure 1. Binding of D- and L-succinimide into the catalytic pocket of antibody generated to bifunctional phosphinate **1**.

antibodies produced in response to racemic mixtures usually show enantiomeric preferences.[6] Once the D isomer is bound, the energetics of hydrolysis at both carbonyls are equivalent since $k_2 \approx k_4$. In the case of the L isomer, however, hydrolysis to the isoaspartate product **4** is 30 times more likely than hydrolysis to the aspartate product **5**.

One possible explanation for these results is illustrated in Figure 1. Provided the D isomer of **1** is the antigen leading to RG2-23C7 (Figure 1A), then the antibody binding site should stabilize both tetrahedral intermediates of the D-succinimide **3D** leading to **4** and **5** (Figure 1B). Since the phosphinate more closely resembles the hydrolysis intermediate than the secondary alcohol, one presumes that site A in Figure 1 might be more active than site B. Thus the aspartate product **5** would be the preferred product. However, this preference is offset by the intrinsically 4-fold greater rate of isoaspartate formation owing to the electronic effect of the α-*N*-acetyl substituent leading to similar values for k_2 and k_4.[7] On the other hand, when the L-succinimide **3L** is fit into this same pocket it must be flipped 180° in order to accommodate the *N*-acetyl group (Figure 1C). Since the α-amide of **1** serves to link the hapten to the carrier protein, it is likely that the *N*-acetyl protrudes from the binding site owing to the insensitivity of many catalytic antibodies to changes in the linkage region of their haptens.[8] The carbonyl adjacent to the *N*-acetyl now occupies site A (the more active of the two sites). The 30-fold faster catalytic rate for the formation of isoaspartate **4** (k_2) as compared to aspartate **5** (k_4) reflects the electronic effects of the *N*-acetyl augmenting the better catalytic site. The ratio of k_2 for the L isomer to k_4 for the D isomer of **3** (rate constants for attack at the carbonyl center associated with the phosphinate mimic) is 6-fold, principally reflecting the 4-fold difference arising from the electronic effect of the α-*N*-acetyl group.

Although other interpretations are plausible, the variations in k_2 and k_4 between the L- and D-succinimides and their increased magnitudes relative to k_6 and k_7 (Table I) are in accord with both tetrahedral mimics acting to generate catalytically active binding pockets. For the conversion of the L-succinimide to the isoaspartate product, the value of $k_2/(k_{-1}/k_1)$ is 7×10^5 M^{-1} s^{-1} as compared to values of ca. 10^7 M^{-1} s^{-1} for diffusion-controlled enzymatic processes, the upper limit on enzyme-catalyzed turnovers. Thus this antibody is within a factor of 10^2 of maximal efficiency.

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Supplementary Material Available: Details of the synthesis of **3L** and **3D**, listings of physical and spectral data for **3–5**, and kinetic analyses and plots for the hydrolyses of the succinimides (5 pages). Ordering information is given on any current masthead page.

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Model Compounds Can Mimic Spectroscopic Properties of Bovine Rhodopsin

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Visual pigments (rhodopsins) consist of 11-*cis*-retinal chromophore bound to a protein (opsin) via a protonated Schiff base linkage with a lysine ϵ-amino group.[1-4] The absorption maxima of the various rhodopsins are characterized by a wide range of wavelengths (440–620 nm) despite the fact that all of them consist of a similar chromophore. The red shift observed in these pigments relative to a model retinal protonated Schiff base ($RSBH^+$) in a methanol solution which absorbs at 440 nm was defined as the opsin shift (OS).[5] The mechanism through which the protein regulates the absorption maxima has been studied extensively, and various models have been suggested.[6,7]

One of the striking spectroscopic characteristics of bovine rhodopsin (λ_{max} = 498 nm) is its C=N stretching frequency (1656 cm^{-1}), which resembles that of retinal protonated Schiff base in methanol solution.[8] It was shown previously that weakening the interaction between the positively charged Schiff base linkage and its counteranion in model compounds leads to a lower C=N frequency accompanying the observed red shift.[9] Thus, one of the major difficulties in explaining the red shift observed in bovine rhodopsin (498 nm) by only separating its positively charged Schiff base linkage from its counteranion is the above explained contradiction of the high C=N stretching frequency observed for bovine rhodopsin despite its red-shifted absorption.

In the present study, we demonstrate experimentally by model compound studies that it is possible to red shift the absorption maximum of a retinal protonated Schiff base by weakening the

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Table I

chromophore	counterion	λ_{max} (nm)[a]	chromophore	counterion	λ_{max} (nm)[a]
$RSBH^+$	Cl^-	455	**2**	IO_4^-	496
1	COO^-	510	**3**	COO^-	522
1	Cl^-	502	**3**	Cl^-	510
1	IO_4^-	508	**3**	IO_4^-	514
2	COO^-	512	**4**	COO^-	480
2	Cl^-	490			

[a] Absorption in methylene chloride, 2.5×10^{-5} M.

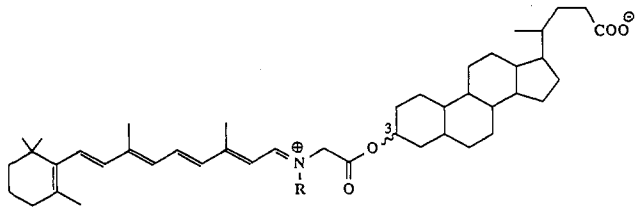
Table II

compound	λ_{max} (nm)[a]	$\nu(C{=}N^+{-}H)$ (cm^{-1})	$\nu(C{=}N^+{-}D)$ (cm^{-1})
rhodopsin[b]	498	1656	1623
1[c]	484	1654	1626
2[c]	480	1652	1627
4[c]	480	1653	1627
$RSBH^+$[c]	455	1653	1630

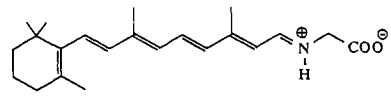
[a] In methylene chloride, 2.5×10^{-3} M. [b] Data taken from ref 8c. [c] Counterion is Cl^-.

Schiff base–counterion electrostatic interaction while maintaining a high C=N stretching frequency.

Compound **1** was prepared by condensation of *all-trans*-retinal with the corresponding amino steroid[10] in trifluoroethanol.[11]



1, R = H; 3α configuration
2, R = H; 3β configuration
3, R = CH_3; 3α configuration



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The absorption maximum of the zwitterion was remarkably red shifted to 510 nm (in CH_2Cl_2 solution, 10^{-5} M) relative to protonated retinal Schiff base ($RSBH^+$) derived from *n*-butylamine (455 nm). Furthermore, protonation of the carboxylate group with HCl or HIO_4 shifts the absorption to 502 or 508 nm, respectively (Table I). The observed red-shifted absorption maximum of **1** may be explained by weak electrostatic interaction between the positively charged Schiff base linkage and the remote negative charge in the zwitterion. However, lithocholic acid tends to form organized structures in the solid state and in solution.[12] Thus, an intermolecular interaction between the carboxyl group and protonated Schiff base of another molecule cannot be excluded, and it is further supported by the observation that the absorption maximum is concentration-dependent. When the carboxylate counterion is replaced by either Cl^- or IO_4^-, a remarkable red shift is still exhibited, probably due to steric hindrance of the neighboring carbonyl group which prevents intimate ion-pairing between the Schiff base linkage and its counterion.

A similar absorption maximum was obtained with compound **2**, bearing an axial substituent with the β configuration at the 3 position of the steroid skeleton. In this case, protonation of the carboxylate group with HCl or HIO_4 blue shifted the absorption to 490 or 496 nm, respectively. The axial configuration probably allows closer distance and stronger electrostatic interaction between the ions. The iminium salt **3**, derived from sarcosine, further demonstrated the unusual shift observed in these chromophores. The importance of the carbonyl group in the vicinity of the Schiff base linkage for inducing a red shifted absorption maximum is demonstrated by condensation of retinal with glycine, which leads to compound **4**, absorbing at 480 nm (relative to 455 nm for $RSBH^+$). The steroid skeleton in compound **1** imposes further weakening of the electrostatic interactions and exhibits a further red shift.

FTIR measurements for compounds **1**,**2**, and $RSBH^+$ demonstrate that they all exhibit very similar and high C=N stretching frequencies despite their different absorption maxima (Table II). The C=N stretching frequencies of these chromophores are very close to the value observed in rhodopsin or isorhodopsin, in both H_2O and D_2O (1656 and 1623 cm^{-1}).[8]

The absorption maxima of the chromophores are controlled by the electrostatic interaction between the positively charged Schiff base linkage and its counterion.[13] The presence of the carbonyl group in the Schiff base vicinity, in addition to the steroid skeleton, enforces an increased distance between the two charges and induces a red shift in the spectrum. In a simple protonated retinal Schiff base, such a weak interaction will lower the N—H rock frequency, thereby reducing the C=N stretching frequency due to weak C=N and N—H rock coupling. Such a phenomenon was predicted by theoretical calculations[14] and was demonstrated experimentally by studies with model compounds.[9] However, in the molecules described above, effective hydrogen bonding probably prevails between the carbonyl group and the Schiff base proton (forming a five-membered ring). The involvement of a water molecule in the hydrogen-bonding bridge (connecting the carbonyl and the N–H moiety) cannot be excluded. Effective hydrogen bonding maintains a high C=N stretching frequency despite the fact that a weak electrostatic interaction prevails between the positively charged Schiff base linkage and its counterion, reflected in the red-shifted absorption maximum of the protonated retinal Schiff base.

The present studies demonstrate experimentally the possibility of red shifting the absorption maximum of the retinal protonated Schiff base chromophore in bovine rhodopsin while maintaining a high C=N stretching frequency just by weak electrostatic interaction between the Schiff base linkage and the counterion, while keeping effective hydrogen bonding to the N—H with a neighboring residue. These results are in keeping with two photon studies, indicating a neutral binding site.[7] In this respect, we note that substitutions of all the potential negatively charged groups in bovine rhodopsin by neutral residues did not significantly affect the absorption maxima (besides Glu 113, which is believed to be the Schiff base counterion).[15] Recent resonance Raman studies[16] on Glu 113 mutant indicated a lower C=N stretching frequency, probably due to Schiff base environment perturbation and Schiff base interaction with exogenous counterion.

^{13}C NMR studies are also in keeping with separation between the positively charged Schiff base linkage and its counterion. Model studies indicated that the odd-numbered carbons of the retinal polyene are affected by π-electron delocalization induced by weak electrostatic interaction with the Schiff base linkage.[17]

(10) The steroid was prepared by esterification of lithocholic benzyl ester with *N*-Cbz-glycine, followed by hydrogenation over Pd/C in EtOH at 25 °C.

(11) Zwitterion **1** was obtained in methylene chloride (following evaporation of trifluoroethanol) and stabilized by addition of 0.1% trifluoroethanol, which forms effective hydrogen bonding mainly with negative charges.

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In addition, the ^{13}C NMR chemical shifts of the odd carbons are affected by close dipoles and downfield shifted by polar solvents. C_{13} is a very sensitive carbon (to dipoles and charge delocalization), and its ^{13}C chemical shift in bovine rhodopsin (168.9 ppm)[18] is closely mimicked in model compounds[17] adopting positive-negative charge separation in the Schiff base vicinity (reflected in a red-shifted absorption maximum) and measured in polar solvents (trifluoroethanol). The chemical shifts of other odd-numbered carbons (C_{11}, 141.6; C_9, 148.5; and C_7 132.3)[18] are closely mimicked as well in model compounds, by weakening of the electrostatic interaction in the Schiff base vicinity, however, through introduction of a relatively nonpolar (chloroform) solvent. Thus, it is tempting to suggest that in bovine rhodopsin a weak electrostatic interaction prevails between the positively charged Schiff base linkage and its counterion red shifting the absorption maximum. A relatively polar environment prevails around the C_{13}-N retinal moiety, formed by bound water and/or by protein residues. The negatively charged counterion, which might be close to C_{13}, can be a part of this environment and induces π-electron delocalization due to its large distance from the positively charged Schiff base linkage. A nonpolar environment prevails in the vicinity of the other section of the retinal chromophore (especially around the C_5-C_9 moiety).[7]

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A Mechanistic Study of the Oxidative Addition of H_2 to $W(PMe_3)_4I_2$: Observation of an Inverse Equilibrium Isotope Effect

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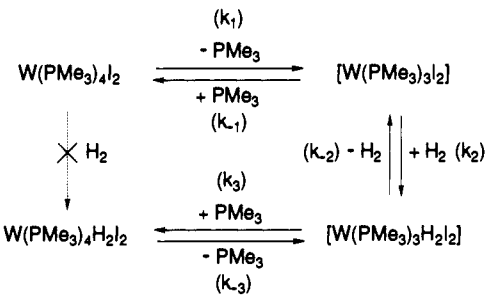
Oxidative addition and its reverse, reductive elimination, are two of the most elementary transformations in organometallic chemistry.[1] In particular, the oxidative addition of dihydrogen represents an important step in many catalytic hydrogenation and hydroformylation processes,[2] and a knowledge of the factors that influence metal-hydrogen bond dissociation energies (BDEs) would provide key information central to understanding, and predicting, reactivity pathways.[3,4] Unfortunately, relatively few metal-hydrogen BDEs have been reported in the literature, in part due to the difficulty of such measurements. Here we report kinetic and thermodynamic studies of the reversible oxidative addition of dihydrogen to six-coordinate *trans*-$W(PMe_3)_4I_2$, which have allowed both (i) the determination of the W-H BDE in $W(PMe_3)_4H_2I_2$ and (ii) the elucidation of the mechanism of the oxidative addition/reductive elimination transformation.

Scheme I. Proposed Mechanism for Oxidative Addition of H_2 to $W(PMe_3)_4I_2$



Sattelberger was the first to report the oxidative addition of H_2 to six-coordinate complexes of the type $M(PR_3)_4Cl_2$ (M = Nb, Ta).[5] Sharp subsequently extended this method for the synthesis of the tungsten analogue $W(PMe_3)_4H_2Cl_2$.[6] Although $W(PMe_3)_4H_2Cl_2$ has been reported to be thermally stable to reductive elimination of dihydrogen,[6] we have found that the iodide analogue $W(PMe_3)_4H_2I_2$[7] undergoes facile reductive elimination of H_2 at 60 °C to give *trans*-$W(PMe_3)_4I_2$.[8] The molecular structures of both six- and eight-coordinate iodide complexes *trans*-$W(PMe_3)_4I_2$ and $W(PMe_3)_4H_2I_2$ have been determined by X-ray diffraction.[9]

Significantly, under 1 atm of H_2, *trans*-$W(PMe_3)_4I_2$ and $W(PMe_3)_4H_2I_2$ exist in equilibrium (eq 1) with comparable concentrations so that the equilibrium constant (K) can readily be measured by using ^1H NMR spectroscopy. The temperature

$$W(PMe_3)_4I_2 + H_2 \overset{K}{\rightleftharpoons} W(PMe_3)_4H_2I_2 \qquad (1)$$

dependence of the equilibrium constant has established the values of $\Delta H° = -19.7$ (6) kcal mol^{-1} and $\Delta S° = -45$ (2) eu for the oxidative addition reaction. Since we may define $\Delta H° = D(H-H) - 2D(W-H)$ for this reaction,[10] a value of 62.0 (6) kcal mol^{-1} is obtained for the average W-H BDE in $W(PMe_3)_4H_2I_2$.[11] W-H BDEs have previously been reported for only a few complexes, for which a range of 64-73 kcal mol^{-1} has been observed.[12]

Investigation of the corresponding oxidative addition of D_2 to *trans*-$W(PMe_3)_4I_2$ reveals a substantial *inverse* equilibrium deuterium isotope effect, with $K_H/K_D = 0.63$ (5) at 60 °C. The

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