(IV)/Mo(V) in $[Mo^{VI}O_2(bdt)_2]^{2-}$ in DMF.

$$[W^{IV}O(bdt)_2]^{2-} + Me_3NO \rightarrow [W^{VI}O_2(bdt)_2]^{2-} + Me_3N$$

The X-ray crystal structures of monooxotungsten(V) and -(IV) complexes **1** and **2b** and dioxotungsten(VI) complex **3a** are shown in Figure 1.[18] **1** and **2b** have square pyramidal structures that are isomorphous and isostructural with the reported structures of $(PPh_4)[Mo^{V}O(bdt)_2]$ and $(NEt_4)_2[Mo^{IV}O(bdt)_2]$.[19] The difference between W^{V}=O and Mo^{V}=O[20] distances shows the same trend as that between $(AsPh_4)[W^{V}OCl_4]$[21] and (PPh_4)-$[Mo^{V}OCl_4]$.[22] The mean W^{V}-S distance is 2.366 (3) Å,[23] which is shorter than that (2.377 (2) Å) of the corresponding Mo(V) complex.

2a and **2b** can be considered to be model complexes of reduced W oxidoreductase, which has been reported not to exhibit any ESR signal for a W(V) species.[5,6] The shorter W^{IV}=O (1.727 (9) Å) and the longer W^{IV}—S distances (mean 2.372 (4) Å) of **2b**[24] suggest the presence of a stronger π-interaction between W(IV) and sulfur.

On the other hand, **3a** is a structural model complex of the oxidized W enzymes which have been studied by the EXAFS analysis.[5,6] The X-ray analysis indicates that W^{VI}=O distances in **3a** are 1.727 (9) and 1.737 (6) Å. The W^{VI}-S distances trans to W^{VI}=O and cis to W^{VI}=O are 2.597 (4) Å (mean) and 2.425 (4) Å (mean), respectively. A trans influence is observed with the elongation of the bond distance of W^{VI}—S trans to W^{VI}=O. Similar elongation has been found also for $(NEt_4)_2[Mo^{VI}O_2$-$(bdt)_2]$.[25]

A DMF solution of **3b** exhibits significantly blue-shifted UV-visible absorption maxima at 323 nm (sh, 15000 M^{-1} cm^{-1}), 419 nm (2300), and 483 nm (1300) compared with those at 335 nm (7000), 430 nm (sh, 2400), and 533 nm (1400) for $(NEt_4)_2$-$[Mo^{VI}O_2(bdt)_2]$ in DMF. The excitation profile in the resonance Raman spectra indicates that the absorption maxima at 419 and 483 nm are due to the ligand-to-metal charge-transfer bands of the W^{VI}—S or W^{VI}=O bond.

3b exhibits a reduction peak at −1.34 V vs SCE whereas the corresponding $(NEt_4)_2[Mo^{VI}O_2(bdt)_2]$ complex shows a reduction peak at −0.97 V vs SCE in DMF. Under stoichiometric conditions, **3b** reacts with benzoin at room temperature to give $(NEt_4)_2$-$[W^{IV}O(bdt)_2]$ and benzil without side reaction as in the following equation but reacts extremely slowly with triphenylphosphine. The observed second-order rate (10×10^{-6} M^{-1} s^{-1}) at the initial stage of the stoichiometric reaction between **3b** and benzoin is similar to that (5×10^{-6} M^{-1} s^{-1}) of the corresponding dioxomolybdenum(VI) complex. The addition of an excess of benzoin (10 equiv) to $(NEt_4)_2[W^{VI}O_2(bdt)_2]$ results in formatioin of benzil in 90% yield.

$$[W^{VI}O_2(bdt)_2]^{2-} + PhCH(OH)COPh \rightarrow [W^{IV}O(bdt)_2]^{2-} + PhCOCOPh + H_2O$$

The formation of $(NEt_4)_2[W^{IV}O(bdt)_2]$ is ascribed to the lack of a comproportionation reaction between $[W^{VI}O_2(bdt)_2]^{2-}$ and $[W^{IV}O(bdt)_2]^{2-}$ to a binuclear W(V) complex probably due to inertness of $[W^{IV}O(bdt)_2]^{2-}$, different from the formation of $W_2O_3(pipdtc)_4$ (pipdtc = *N*-piperidinecarbodithioate) reported in the reaction between $WO_2(pipdtc)_2$ and trimethyl phosphite.[26]

cis-Dioxotungsten(VI) thiolate complex **3b** is thermodynamically stable. However, **2b** and **3b** show almost the same reductive and oxidative reactivities as the corresponding molybdenum complexes. Although each oxidation state of tungsten ion has an ionic radius similar to that of the corresponding molybdenum ion, the stronger π-interaction between W and S leads to significant differences in the chemical properties of the molybdenum complexes. The negative value of the redox potential of monooxotungsten(IV) thiolate complex probably contributes to the observed higher reductive reactivity in W oxidoreductase.

No participation of O-atom transfer in a W enzyme system has been considered owing to the difficulty of formation of the W(IV) species from dioxotungsten(VI) complexes.[26,27] Our results suggest that the chelating coordination of the dithiolene part of pterin cofactor shifts the W(IV)/W(V) redox potential to the positive side and the W(IV) state is involved in the catalytic cycle of W enzymes.

Supplementary Material Available: Tables of atomic positional and thermal parameters, bond distances, and bond angles for **1**, **2b**, and **3a** (43 pages); tables of observed and calculated structure factor amplitudes for **1**, **2b**, and **3a** (98 pages). Ordering information is given on any current masthead page.

(18) Crystal data for **1**: *Cc* (monoclinic), a = 12.719 (3) Å, b = 15.601 (3) Å, c = 16.529 (2) Å, β = 93.53 (1)°, Z = 4, V = 3273.7 (7) Å^3, μ = 39.24 cm^{-1}; 4959 unique reflections were measured, and 3574 with $I_o > 3\sigma(I)$ were used in the refinement to R = 0.041, R_w = 0.043. Crystal data for **2b**: $P2/n$ (monoclinic), a = 18.53 (2) Å, b = 9.231 (2) Å, c = 18.899 (3) Å, β = 93.45 (3)°, Z = 4, V = 3226 (3) Å^3, μ = 39.27 cm^{-1}; 7384 unique reflections were measured, and 5239 with $I_o > 3\sigma(I)$ were used in the refinement to R = 0.052, R_w = 0.075. For **3a**: $P2_1/c$ (monoclinic), a = 13.92 (1) Å, b = 16.672 (9) Å, c = 22.618 (7) Å, β = 103.66 (5)°, Z = 4, V = 5101 (6) Å^3, μ = 25.74 cm^{-1}; 12221 unique reflections were measured, and 5414 with $I_o > 3\sigma(I)$ were used in the refinement to R = 0.044, R_w = 0.045.

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Inhibition of Steroid Biosynthesis by Steroid Sulfonates

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Steroid sulfation plays a major role in the metabolism of steroids.[1] Steroid sulfates such as estrone sulfate and dehydroisoandrosterone sulfate are the predominant form of these steroids found in plasma[2] and are important intermediates in the biosynthesis of estrone in breast tumor cells.[3] Another abundant sulfated steroid, cholesterol sulfate, plays a number of crucial biochemical roles, including the stabilization of cell membranes.[4] This communication reveals a facile method for the synthesis of steroid sulfonates, which are nondegradable analogues of sulfated

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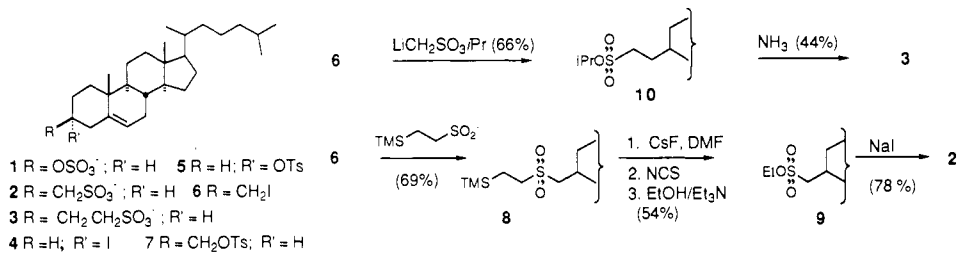
Scheme I



steroids. We further demonstrate that sulfonate analogues of cholesterol sulfate can replace cholesterol sulfate in an important and specific biological role—the inhibition of the side-chain cleavage of cholesterol.[5] These results demonstrate for the first time that sulfonate salts are excellent isosteres of sulfate monoesters.

Only recently have *isosteric* sulfonate analogues of even some of the simplest biologically occurring sulfate monoesters (i.e., glucose 6-sulfate) been reported.[6-8] Although steroid sulfates have been studied for many years, a complete examination of their biological roles, activities, and metabolism is hampered by the fact that these steroid conjugates are readily degraded by the action of ubiquitous sulfatases.[9] It occurred to us that a sulfonate analogue of a sulfated steroid, such as cholesterol sulfate (**1**), would be stable toward sulfatase-mediated hydrolysis and might mimic some of the important biological properties of this steroid, such as the ability to inhibit cholesterol side-chain cleavage[5] or the fusion of viruses to cell membranes.[10]

We envisioned that the steroid sulfonates (**2** and **3**) might be synthesized by application of the α-sulfonate anion chemistry that was recently used for the synthesis of carbohydrate sulfonates.[6] Deprotonation of isopropyl methanesulfonate with BuLi followed by addition of the iodide (**6**) (synthesized from the corresponding alcohol[11] by treatment with PPh_3/I_2) gave **10**, the sulfonate homologue of cholesterol sulfate, as its isopropyl ester. Ammonolysis of this material gave the sulfonate **3** in acceptable yield (Scheme I). However, reaction of the mesylate anion with either cholesteryl iodide (**4**) or cholesterol tosylate (**5**) led only to elimination, not substitution. Horner–Emmons reaction of cholestenone with a phosphonate-stabilized sulfonate anion[7,12] led to base-catalyzed migration of the double bond. We also attempted to displace the iodide of **6** or the tosyl group of **7** with sulfite or bisulfite ion (the Strecker reaction).[13] However, this reaction also proved unsatisfactory.

The steroid sulfonate **2** was successfully synthesized by the method diagramed above (Scheme I). The iodide **6** was converted to the sulfone **8** by reaction with the 2-(trimethylsilyl)ethanesulfinate.[14] Treatment of this sulfone with fluoride ion elicited the expected fragmentation[15,16] to give the sulfinate, which (as its free acid) was oxidized with NCS to the sulfonyl chloride. The sulfonyl chloride was then converted to the ethyl ester (**9**) for purposes of purification and characterization. Cleavage of the ester with NaI gave cholesterol sulfonate **2** as its sodium salt.[17] This strategy provides an expedient method for the synthesis of both sulfonyl halides and sulfonate derivatives of steroids. The sulfonyl halides are potential active site directed inactivators of enzymes that synthesize or hydrolyze cholesterol esters, such as acylcoenzyme A:cholesterol acyltransferase.[18]

Table I. Effects of Cholesterol Sulfonates on the Side-Chain Cleavage of [^{3}H]Cholesterol in Rat Adrenal Mitochondria[a]

compound added	relative activity (%) exp 1	exp 2	exp 3
vehicle alone	100	100	100
cholesterol sulfate (**1**) (100 μM)	22	33	45
cholesterol sulfonate (**2**) (100 μM)	21	29	43
cholesterol homosulfonate (**3**) (100 μM)	48	29	42

[a] Mitochondrial preparations, incubations, and analyses were carried out essentially as detailed in ref 19.

Since it has never been demonstrated that sulfonate salts are effective isosteres for sulfate esters, we chose to examine whether the steroid sulfonates **2** and **3** could effectively replace cholesterol sulfate in a specific biological role. Thus, the sulfonates were tested for their ability to inhibit steroid side-chain cleavage in rat adrenal mitochondria. Cholesterol sulfate has been demonstrated to be a naturally occurring, noncompetitive inhibitor[19] of the transport of cholesterol into the mitochondria, which is the rate-limiting step in steroidogenesis.[5,20,21] The structural requirements for this kind of inhibition have been demonstrated to be quite demanding, with regard to the steroid nucleus, side chain, and the charged moiety at the 3-position.[20] For example, a variety of sulfated steroids, SDS, and cholesterol 3-phosphate are much less effective inhibitors than cholesterol sulfate.[5,20] As shown in Table I,[22] the sulfonate which is isosteric to cholesterol sulfate (**2**) is as effective an inhibitor of steroidogenesis as cholesterol sulfate itself, thus demonstrating that sulfonates are effective isosteres of sulfate esters. The homologous sulfonate, **3**, also appears to be an active inhibitor of side-chain cleavage.

These experiments demonstrate the capacity of steroid sulfonates to substitute for steroid sulfates in a functioning biological system and open the way for additional applications of this methodology. Interestingly, at least in this one example, sulfonates

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appear to be much better isoelectronic isosteres of sulfate esters than phosphonates are of phosphate esters. This suggests that, in contrast to phosphonates which are generally poor phosphatase inhibitors, sulfonates should be potent sulfatase inhibitors. In addition, because of the resistance of the sulfonate group toward hydrolysis, sulfonated steroids should be very useful agents for probing the metabolism, transport, and biological function of the analogous sulfated steroids. Such studies are currently being pursued.

Supplementary Material Available: Experimental details and ^1H and ^{13}C NMR spectra for the compounds mentioned in the text (22 pages). Ordering information is given on any current masthead page.

Model Studies of DNA Photorepair: Energetic Requirements for the Radical Anion Mechanism Determined by Fluorescence Quenching

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The most significant form of photochemical damage to DNA is the [2 + 2] cyclodimerization of pyrimidine bases.[1] Direct reversal of this damage is mediated by photolyase enzymes, which use a photon to initiate the catalytic step.[2] The successful cloning and overexpression of *Escherichia coli* photolyase by Sancar[3] has renewed interest in the mechanism of these photochemical enzymes. Studies of model repair reactions have suggested a reductive SET (single electron transfer) mechanism.[4] In this mechanism, the enzyme excited state transfers a single electron to the pyrimidine dimer. The dimer anion radical then fragments to yield a monomer and a monomer anion radical. The latter re-reduces the enzyme, and the DNA bases are reverted to their normal form. Recently, we demonstrated that thymine dimer anion radicals cleave very rapidly (2×10^6 s^{-1}) in free solution.[5] Evidence for the corresponding cation radical mechanism also exists.[6] The experiments reported here were undertaken in order to evaluate the energetic requirements for the anion radical mechanism. The results support that mechanism.

$$S + Q \xrightarrow{h\nu} S^* + Q \underset{}{\overset{k_{dif}}{\rightleftharpoons}} [S\cdots Q]^* \overset{k_\alpha}{\rightleftharpoons} S^{\cdot+} + Q^{\cdot-} \quad (1)$$

The most common test for photoinitiated single electron transfer (SET) mechanisms is to calculate the free energy change for the charge-transfer step (ΔG_{ct}).[7] This is usually done by measuring the oxidation potential of the donor (E_{ox}) and the reduction potential of the acceptor (E_{red}) and applying eq 2. Generally, if

$$\Delta G_{ct} = 23.06\left(E_{ox} - E_{red} - \frac{e^2}{R\epsilon}\right) - E_{oo} \quad (2)$$

the charge transfer is more than ca. 5 kcal/mol endergonic, it does not compete effectively with relaxation of the sensitizer excited state. On the other hand, if the charge-transfer step is exergonic, then SET can be considered a feasible pathway. Despite the growing interest in the DNA photorepair mechanism and the large number of model reactions studied, no reduction potential for any pyrimidine dimer is known.[8]

In order to assess the proposed mechanism, we sought to determine E_{red} for thymine dimers. Because our attempts to measure this quantity by cyclic voltammetry were unsuccessful, an alternative technique was used. This approach is based on the principle that the rate constant for electron transfer (k_{ct}) is related to ΔG_{ct}. For the bimolecular systems illustrated in eq 1, k_{ct} can be determined from the rate constant for fluorescence quenching, k_q. In the present case, the sensitizer (S) is an excited-state electron donor and the quencher (Q) is the pyrimidine dimer.

A series of sensitizers was examined where the excited-state oxidation potential, E_{ox}^* (= $E_{ox} - E_{oo}/23.06$), was systematically

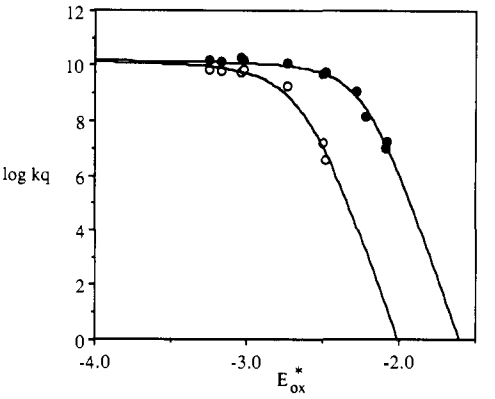

Figure 1. Effect of sensitizer excited-state oxidation potential, E_{ox}^*, on the rate constant for fluorescence quenching (k_q). The substrates were dimethylthymine (●) and its cis,syn dimer (○). The solid lines are calculated on the basis of the diffusion-limited Rehm-Weller relationship using $E_{red} = -2.60$ V, $\lambda = 15.7$ kcal/mol (dimer) and $E_{red} = -2.21$ V, $\lambda = 13.7$ kcal/mol (monomer). The solvent is CH_3CN.

Table I. Sensitizer Excited-State Oxidation Potentials (E_{ox}^*) and Quenching Rate Constants (k_q) for Dimethylthymine and Dimethylthymine Dimer

sensitizer	E_{ox}^*, V	k_q, M^{-1} s^{-1} (monomer)	k_q, M^{-1} s^{-1} (dimer)
N,N,N',N'-tetramethylphenylenediamine	−3.25	1.54×10^{10}	6.54×10^9
N,N,N',N'-tetramethylbenzidine	−3.17	1.39×10^{10}	5.91×10^9
N,N-dimethylaniline	−3.04	1.79×10^{10}	5.47×10^9
aniline	−3.02	1.55×10^{10}	7.06×10^9
p-dimethoxybenzene	−2.73	1.17×10^{10}	1.80×10^9
acenaphthene	−2.50	5.04×10^9	1.52×10^7
naphthalene	−2.48	5.53×10^9	3.65×10^6
2-methoxynaphthalene	−2.28	1.09×10^9	$<1 \times 10^6$ [a]
anthracene	−2.22	1.42×19^8	$<1 \times 10^6$ [a]
phenanthrene	−2.09	9.84×10^6	$<1 \times 10^6$ [a]
chrysene	−2.08	1.72×10^7	$<1 \times 10^6$ [a]

[a] No quenching was measurable at 0.2 M quencher concentration.

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