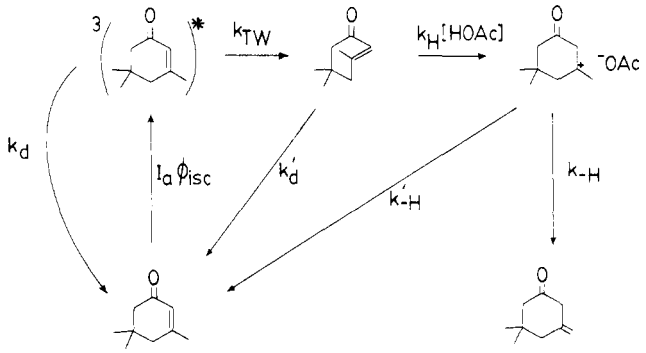
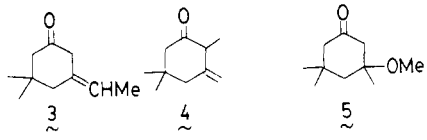
Scheme I



The quantum yield of deconjugation of isophorone solutions containing 4×10^{-3} M acetic acid showed a systematic decline from 0.044 to 0.035 when the isophorone concentration was gradually increased from 0.1 to 1 M. This is inconsistent with a mechanism of deconjugation which is bimolecular in isophorone but is attributable to self quenching of isophorone triplets by ground-state isophorone.[8]

The photochemical deconjugation reaction is reported to fail for 3-alkylcyclohexenones if a 3-methyl group is replaced by larger alkyl groups.[4b] However, we find that if 4×10^{-3} M acetic acid is present, then the reaction does proceed with 5,5-dimethyl-3-ethylcyclohexenone to give a mixture of (*E*)- and (**Z**)-**3**; **4** is also formed as a secondary (Norrish Type I) photoproduct at high conversions.



3 **4** **5**

The simplest explanation of our observations is that the carbon–carbon double bond of the triplet excited state of isophorone is protonated by the acid with Markovnikov regiochemistry to give a carbocation which eliminates to isophorone and the deconjugated product. However, this would require that interception of the triplet with 4×10^{-3} M acetic acid be much more efficient than triplet decay which would require a rate constant for protonation of the order of 10^{12} M^{-1} s^{-1} by acetic acid in benzene. Clearly a longer lived intermediate formed from the triplet is required for reaction with the acid. By analogy with the acid-catalyzed photochemical reactions of cyclohexenes[9] and the results of flash photolysis studies of the reaction of 4,4-dimethylcyclohexenone with alkenes[10] we suggest that the intermediate is a ground-state twisted cyclohexenone (a *trans*-cyclohexenone). This gives rise to the mechanism shown in Scheme I which in turn leads to eq 3 for the dependency of the deconjugation quantum yield, Φ_p, upon

$$\frac{1}{\Phi_p} = K\left\{1 + \frac{k_d'}{k_H[\mathrm{HOAc}]}\right\} \qquad (3)$$

$$\text{where } \frac{1}{K} = \Phi_{isc}\left\{\frac{k_{-H}}{k_{-H} + k_{-H'}}\right\}\left\{\frac{k_{TW}}{k_d + k_{TW}}\right\}$$

acid concentration.[11] The fit of the data from Figure 1 to eq 3 is shown in Figure 2. From the slope and the intercept of the plot the value of k_d'/k_H can be determined and is 1.9×10^{-4} M. The fastest value k_H can adopt is the rate of diffusion (1.5×10^{10} M^{-1} s^{-1} in benzene); and hence k_d' must be $\lesssim 10^6$ s^{-1}. This corresponds to an intermediate with a lifetime greater than a microsecond which compares with 9 μs for *trans*-1-phenylcyclohexene.[9a]

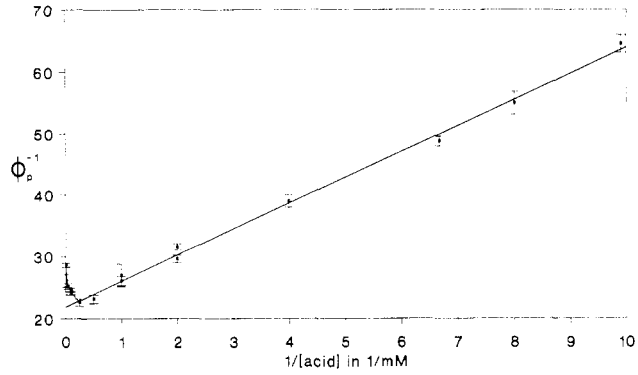


Figure 2. Photochemical isophorone deconjugation at various concentrations of acetic acid: [isophorone] = 0.145 M.

If a highly strained twisted enone or a carbocation are intermediates in the reaction, then they should be trappable by nucleophiles.[12] Indeed, irradiation of isophorone in benzene containing 10% methanol gave a mixture of deconjugated product **2** and the anticipated methyl ether **5** in the ratio 3:1.

Our observation of acid catalysis suggests that the previously reported[4,5] results for the photochemical deconjugation reaction of cyclohexenones need to be reexamined. In addition, our evidence for a twisted intermediate may have consequence for the other reactions of cyclohexenones such as rearrangement and cycloaddition where a similar intermediate has also been proposed;[10] the observation of acid catalysis may also be of relevance to the understanding of the mechanism of photochemical addition of nucleophiles to cyclohexenones and larger ring cycloenones.[12]

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Long-Range Electron Transfer in Structurally Engineered Pentaammineruthenium (Histidine-62)cytochrome *c*

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In many biological processes, long-range electron transfer (ET) plays a key role.[1] When the three-dimensional structures of proteins are accurately known, use of modified proteins[1a,2] and protein–protein complexes[3,4] provides an experimental approach

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to study ET rates between two metal centers. For Ru(His)-modified proteins, the introduction of histidine residues at any desired surface location by site-directed mutagenesis opens the way for systematic investigations of ET pathways.[2b]

Some studies suggest that long-range donor–acceptor electronic coupling can be enhanced by aromatic groups or sulfur atoms in the intervening protein medium.[5,6] To examine this possibility, we created a yeast cytochrome *c* variant with a surface histidine at residue 62 by preparing the N62H mutant.[7] Computer modeling of the ruthenated mutant, whose structure should be virtually identical[8] with that of the native protein determined by X-ray diffraction,[9] shows that the putative pathway for electron transfer to the heme iron contains both an aromatic residue (Trp59) and a sulfur of methionine (Met62).[10]

The N62H mutant was prepared by oligonucleotide-directed mutagenesis[11] on the yeast iso-1-cytochrome *c* gene (CYC1) cloned into M13mp8 viral DNA.[12,13] The 17-mer d(ACATGTT-*ATG*TTCGTCC) was used to direct the change of the AAT codon for asparagine to the CAT codon for histidine at position 62 in the iso-1-cytochrome *c* gene. Individual viral colonies were screened for the gene mutation by the dot blot method,[14] and the base substitution was confirmed by dideoxy sequencing.[15] The mutant CYC1 gene was prepared for expression in yeast by the method of Smith and co-workers.[12b] The N62H mutant was excised from the M13RF DNA and inserted into the yeast expression vector, YEp213 (contains ampicillin and tetracycline resistance genes and the Leu2+ gene from *Saccharomyces cerevisiae* as markers),[16] to produce the YEp213/CYC1(N62H) hybrid plasmid.[17] This plasmid was then transformed into the *S. cerevisiae* strain GM-3C-2[18] (a yeast strain deficient in both production of cytochrome *c* and biosynthesis of leucine) by the LiCl method.[19,20] A 170-L fermentation of transformed GM-3C-2 cells producing the N62H mutant yielded 330 mg of pure protein after isolation by standard methods.[13] SDS–polyacrylamide gel electrophoresis after FPLC purification gave a single protein band with mobility corresponding to 13.4 kD, as expected for cytochrome *c*.

The N62H cytochrome *c* mutant[21] was modified with [a_5Ru-

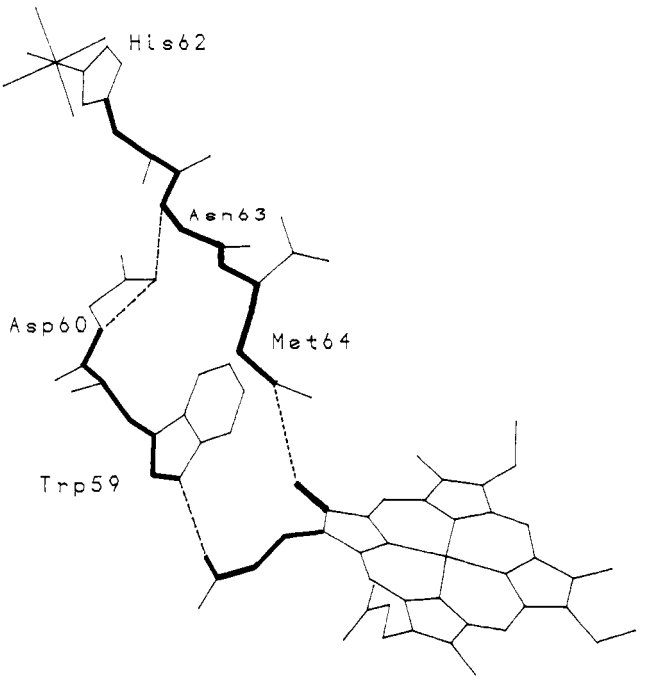

Figure 1. The ET medium between ruthenated His62 and the heme, derived from side-chain replacement and molecular mechanics minimization of the native yeast iso-1-cytochrome *c* structure.[10] For clarity, the iron axial ligands, His18 and Met80, have been omitted, as have the Asn63 and Asp60 side chains. The two best Beratan–Onuchic pathways[30] are shown in bold. Long dashed lines (---) denote hydrogen bonds, and short dashed lines (- - -) are through-space interactions. Calculations according to ref 29 give $[H_{ab(His62-Met64)}/H_{ab(His33-Pro30)}]^2$ = 0.013 and $[H_{ab(His62-Trp59)}/H_{ab(His33-Pro30)}]^2$ = 0.0092.

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(7) The N62H (tuna numbering) derivative is a triple mutant built on a base protein with Cys102Ser (prevents protein dimerization by disulfide formation) and His39Gln (removes the native surface histidine) mutations (relative to the wild-type protein): Mayo, S. L. Ph.D. Thesis, California Institute of Technology, 1988.

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(10) The computer model of the ruthenated N62H mutant was generated by side-chain substitution using the Biograf/III software package (version 1.34, Biograf was designed and written by S. L. Mayo, B. D. Olafson, and W. A. Goddard III). The three-dimensional structure of yeast iso-1-cytochrome *c* was derived from the tuna and rice structures by side-chain substitution and molecular mechanics minimization (see: Mayo, ref 7). An 18 × 18 conformational search about the α–β, β–γ dihedral angles of the ruthenated histidine 62 side chain was performed. The 10 lowest energy conformations of that side chain were then further minimized by molecular mechanics methods (Biograf/III), holding the remainder of the protein rigid. The lowest energy conformation was taken as the most likely position of the His62-a_5Ru side chain.

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(21) The UV–vis spectrum of N62H cytochrome *c* is identical with that of the wild-type protein; the heme($Fe^{3+/2+}$) reduction potential is 268 mV vs NHE (differential pulse polarography; 7.5 mM 4,4′-bipyridine mediator: 50 mM sodium phosphate, pH 7.0, 0.4 M NaCl).

$(H_2O)](PF_6)_2$ (a = NH_3);[22] the products were separated by FPLC.[23] The singly modified product was characterized by differential pulse polarography; the heme($Fe^{3+/2+}$) reduction potential is 275 mV (NHE); the reduction potential of the $a_5Ru(His62)^{3+/2+}$ moiety is 75 mV (NHE), as expected.[24] The $Ru^{2+} \rightarrow Fe^{3+}$ ($-\Delta G° = 0.2$ eV) ET rate was measured by using the $[Ru(bpy)_3]^{2+}$/EDTA flash photolysis technique.[24] Electron transfer was monitored by the increase in absorbance at 550 nm, attributable to reduction of the heme iron. The kinetics were first order over three half-lives ($\sigma = 0.98$),[25] with $k_{obsd} = 1.7$ (1) s^{-1}.

A simple exponential edge–edge distance dependence [$\exp[-\beta(d - d_0)]$, with $\beta = 0.9$ Å^{-1} and no correction for difference in reorganization energy or driving force][6a] predicts a $Ru^{2+} \rightarrow Fe^{3+}$ ET rate for the N62H mutant of 0.4–2.0 s^{-1} relative to the 30 s^{-1} observed[24] for a_5Ru(His33)cytochrome c.[26] In the a_5Ru-(His33) derivative of horse heart cytochrome c, the ET pathway consists only of aliphatic residues.[2b,27] The finding that the rate for the ruthenated N62H mutant agrees strikingly with that calculated relative to horse heart cytochrome c suggests that the mere presence of aromatic residues and/or polarizable sulfur atoms along the pathway for electron transfer does not necessarily create conditions for significantly stronger donor–acceptor electronic coupling through the protein medium.[28]

Beratan and Onuchic have proposed a theoretical framework for long-range donor–acceptor coupling involving pathways that are combinations of covalent-bond, hydrogen-bond, and through-space interactions.[29] Using this approach, we have estimated the pathways of strongest coupling between the ruthenated histidine and the heme for N62H (mutant, see Figure 1) and for His33 (horse heart cytochrome c).[30] A comparison of the best pathways gives $k_{ET(His62)} = k_{ET(His33)}[H_{ab(His62-Met64)}/H_{ab(His33-Pro30)}]^2 = 0.4$ s^{-1} relative to 30 s^{-1} for His33 (horse heart cytochrome c).[31] This value also agrees well with the observed rate for the ruthenated N62H mutant.

Acknowledgment. We thank Professor Judith L. Campbell, Dr. Guy Guillemette, Dr. Alfred Gartner, and Professor A. G. Mauk for helpful discussions and Dr. Adrienne Raphael for assistance with the electrochemical (differential pulse polarography) measurements. Large-scale fermentations were done with the aid of Dr. Tom Sutherland at the UCLA Molecular Biology Institute. B.E.B. acknowledges the Medical Research Council (Canada) for a postdoctoral fellowship. This research was supported by National Science Foundation Grant CHE88-14222.

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(26) The range for k_{His62} was obtained by using $d_{His62} = 14.7$–15.6 Å (ref 10); $d_{His33} = 10.8$–11.7 Å (ref 6a).

(27) Meade, T. J.; Gray, H. B.; Winkler, J. R. *J. Am. Chem. Soc.* **1989**, *111*, 4353–4356.

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(31) No range is reported for the rate calculation based on the pathway model, since the best pathway (not a range of pathways) from His62 of the N62H mutant is being compared with the best pathway from His33 (horse heart cytochrome c).

Radical Cation Cope Rearrangement of 1,5-Hexadiyne to 1,2,4,5-Hexatetraene (Bis(allene)) at 77 K

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Although the formation of chair cyclohexane-1,4-diyl radical cation intermediates in the oxidation of 1,5-hexadienes[1,2] can be regarded as the first step in a Cope-like reaction, the subsequent retrocyclization required to complete the rearrangement is quite unlikely to occur in degenerate or nearly degenerate systems in view of the manifestly greater stability of the cyclic intermediate.[1] Indeed, this return step was previously calculated to be endothermic by 34 kcal mol^{-1} for the parent 1,5-hexadiene.[3] The identification of cycloolefinic and aromatic products in these oxidations[1,4] also clearly points to this irreversibility. It therefore seems likely that some,[2] if not all,[5] of the previously reported radical cation induced Cope rearrangements of aryl-substituted 1,5-hexadienes[2,5] actually proceed through back electron transfer[2] to form neutral cyclohexane-1,4-diyl precursors which can easily undergo the necessary cleavage to the rearranged 1,5-hexadienes.[4] At any rate, a Cope-type rearrangement has not hitherto been demonstrated exclusively at the radical cation stage, and here we report the first direct observation of such a reaction.

Acetylenic Cope processes leading to allenes[6] provide examples of extremely nondegenerate systems with an appreciable net driving force that should be augmented in the radical cation because of the higher ionization potentials associated with acetylenes.[7] In studying the radiolytic oxidation of 1,5-hexadiyne (**1**) in Freon matrices, we observed an intense and well-defined ESR pattern (Figure 1a) in several haloethanes (CF_3CCl_3, CF_2ClCCl_3, $CF_2ClCFCl_2$, and $CFCl_2CFCl_2$) which is readily analyzed as a quintet (a(4H) = 28.6 G) of triplets (a(2H) = 3.8 G) with a g factor of 2.0024. This is clearly the spectrum of a symmetrically delocalized species, and since the corresponding spectrum (b) from 1,6-dideuterio-1,5-hexadiyne is a simple quintet[8]

† Undergraduate research participant from Indiana University Southeast, New Albany, IN 47150.

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