Table II. Cyclization of the Nitrones **4**

entry	4	R_1	R_2	R_3	TFA (equiv)	condition	5 (%)	6 (%)	6α:6β
1	**a**	H	COOMe	Me	2	−78 °C/1 h		**a** (97)	1:41
2	**b**	H	TROC	CBZ	5	−78 °C/2 h		**b** (82)	1:8
3	**c**	H	BOC	TROC	5	−78 °C/2 h		**c** (94)	1:10
4	**d**	H	TROC	Me	5	−78 °C/2 h		**d** (96)	1:21
5	**e**	H	BOC	Me	2	−78 °C/1 h		**e** (96)	1:10
6	**g**	H	COOMe	TROC	2	−78 °C/1 h		**g** (100)	1:12
7	**h**	H	CBZ	TROC	2	−78 °C/1 h		**h** (97)	1:12
8	**a**	H	COOMe	Me	1	rt/5 min	**a** (75)	**a** (24)	1:7
9	**e**	H	BOC	Me	1	rt/5 min	**e** (70)	**e** (21)	1:5
10	**g**	H	COOMe	TROC	1	rt/5 min	**g** (33)	**g** (56)	1:6
11	**h**	H	CBZ	TROC	2	rt/1 h	**h** (28)	**h** (67)	1:4
12	**f**	Me	COOMe	TROC	1	rt/5 min	**f** (90)		
13	**i**	Me	COOMe	Me	1	rt/5 min	**i** (90)		
14	**f**	Me	COOMe	TROC	5	−78 °C/1 h	**f** (93)		
15	**i**	Me	COOMe	Me	5	−78 °C/1 h	**i** (90)		

spiroindolenine intermediates 7α and 7β (Scheme III). The isomer 7α could convert either to **5** via trapping by intramolecular cyclization or to 6α by a competitive rearrangement. On the other hand, 7β could only rearrange to 6β due to the instability of the cis diastereoisomer of **5**. These results provide evidence for the involvement of a rapid equilibrium between **4**, **5**, and **6** in the PS reaction.[7] The reaction at low temperature suggests direct electrophilic attack via a favored 6-endo-trig pathway[8] at the indole 2-position under kinetically controlled conditions.

With the optically active key intermediates **5** and **6**[5] in hand, the intramolecular Pummerer cyclization of **6d**β was examined. Treatment of the sulfoxide of **6d**β with acetic anhydride gave the diacetate **8a** which was selectively O-deacetylated to yield **8b**. However, **8b** does not undergo the desired cyclization.

In order to prevent the acetylation of the hydroxyl group and to favor the desired cyclization via a more reactive intermediate, chlorination[9] of **6e**β was carried out. Thus, when **6e**β was treated with NCS (1.2 equiv, CCl_4, 0 °C, 12 h), the desired cyclization occurred to give the oxathiazepine **9a** {$[\alpha]_D$ +93.2° (21 °C, *c* 0.25, MeOH), 4%}. Deprotection of the BOC group (50% TFA-CH_2Cl_2, room temperature, 20 min; IRA-400) afforded the enantiomeric debromoeudistomin L (+)-**1e** {$[\alpha]_D$ +105.8° (21 °C, *c* 0.19, MeOH), quantitative} (Scheme III).

Likewise, the condensation of **10**, prepared from D-cysteine, with **2a** gave (−)-**4e**[5] {$[\alpha]_D$ −68.6° (23 °C, *c* 0.50, MeOH), mp 135.5–136.5 °C, 90%} which was cyclized (TFA, −78 °C) to give **12a** (enantiomer of **6e**β, 90%) and the enantiomer of **6e**α (4%). Treatment of **12a** with NCS (1.2 equiv, CCl_4, 5–10 °C, 1.5 h, 8%) afforded **14a** {$[\alpha]_D$ −99.0° (22 °C, *c* 0.10, MeOH), mp 197–198 °C}. Deprotection of **14a** provided debromoeudistomin L (**1e**) as a TFA salt which on treatment with IRA-400 gave debromoeudistomin L (1e) {$[\alpha]_D$ −96.3° (22 °C, *c* 0.08, MeOH), 94%; lit.[3c] $[\alpha]_D$ −58.3° (*c* 0.06, MeOH)}.

After an extensive survey of this final oxidative cyclization,[10] the *p*-TsOH-catalyzed[11] cyclization (*p*-TsOH, 2.0 equiv; PPTS, 1.0 equiv, room temperature, 12 h) of the sulfoxide **13** (mCPBA, 94–99%) increased the yield of **9** {**9a**, 10%; **9b**, $[\alpha]_D$ +87.3° (23 °C, *c* 0.51, MeOH), 17%}.

The unexpected formation of **5** from **4** could now be used to modify the benzene ring for the synthesis of eudistomins carrying substituents on the benzene ring. Thus, the PS reaction of (−)-**4e** at room temperature gave **11a** {$[\alpha]_D$ +138.3° (24 °C, *c* 0.47, MeOH), 69.8%}. Bromination of **11b**, obtained by selective acetylation of **11a**, with NBS (1.2 equiv, room temperature, 20 min) proceeded regioselectively to give **11c** which afforded **11d** {$[\alpha]_D$ +171.3° (23 °C, *c* 0.40, MeOH), 3 steps from **11a**, 75%} on O-deacetylation. Rearrangement of **11d** with TFA (3 equiv, room temperature, 40 h, 33%) yields the desired β-carboline **12b**. Final cyclization of **12b** with NCS (CH_2Cl_2, −78 °C, 2 h, 4%) provides eudistomine L (**1a**) {$[\alpha]_D$ −58.3° (22 °C, *c* 0.06, MeOH), 76%; lit.[1] $[\alpha]_D$ −77° (25 °C, *c* 0.2, MeoH)}, via **14b** {$[\alpha]_D$ −24.0° (24° C, *c* 0.10, MeOH)}.

Synthetic eudistomins, **1a** and **1e**, exhibited identical spectroscopic data (HRMS, ^{13}C and ^1H NMR) to reported spectra.[1,3c] The synthesis also provides direct evidence for the absolute configuration of eudistomins. Further efforts for the improvement of the final cyclization step are currently underway in our laboratory.

Acknowledgment. We are grateful for support of this research by a Grant-in Aid for Scientific Research (62470134 and 63105005) from the Ministry of Education, Science, and Culture, Japan and Uehara Memorial Foundation. We are also grateful to Professors Blunt and Munro for the spectral data of **1e** and for invaluable information. We also thank Dr. K. Hirayama and S. Akashi, Central Research Laboratories, Ajinomoto Co. Inc., for high resolution mass spectral analysis.

Supplementary Material Available: Spectral and physical data (mp, $[\alpha]_D$, λ_{max}, ν_{max}, *m/z*, and δ ppm) for **1a**, **1e**, (+)- and (−)-**4e**, (+)-**4f**, **5e**, **6e**β, **11a**, **11d**, **12a**, **12b**, **14a**, and **14b** (5 pages). Ordering information is given on any current masthead page.

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Electrochemical Activation of Oxygenated Fe·Bleomycin

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Oxygen activation in metal ion-based biochemical systems has received considerable attention in recent years.[1] Intensively studied ligands include bleomycin (BLM),[2] an antitumor antibiotic whose conversion to one or more reactive intermediates involves

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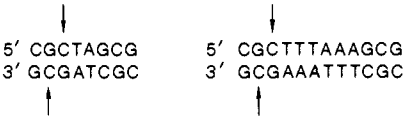
Table I. Catalytic Degradation of DNA Oligonucleotides by Electrochemically Activated Fe·Bleomycin[a]

Fe·BLM (μM)	oligonucleotide	treatment	total DNA lesions (μM)	DNA lesions/ Fe·BLM	$C_3 + C_{7(11)}$ specificity (%)	$C_3/C_7(C_{11})$
45	d(CGCTAGCG)	−0.22 V, 6 h	580	12.9	100	12/88
45	d($CGCT_3A_3GCG$)	−0.22 V, 22 h	371	8.2	91	8/92
20	d($CGCT_3A_3GCG$)	Na ascorbate, 0.25 h	199	10.0	89	7/93

[a] A 200-μL solution containing 45 μM Fe^{III}·BLM and either 0.9 mM d(CGCTAGCG) or 0.4 mM d($CGCT_3A_3GCG$) in 50 mM Na cacodylate, pH 7.0, was electrolyzed at −0.22 V in a cell containing a 1.1 cm^2 polished glassy carbon plate electrode. The electrolysis mixture was saturated continuously with O_2; oligonucleotides were added in 4 aliquots over a period of 2–3 h. Product analysis was carried out by reverse-phase HPLC.[8] Also shown for comparison are the products resulting from treating 0.33 mM d($CGCT_3A_3GCG$) with 20 μM Fe·BLM + 2 mM Na ascorbate + O_2.



reductive activation of dioxygen in the presence of metal ions such as Fe(II)[3] or Cu(I).[4,5] Activated metallobleomycins mediate oxidative DNA damage[2] and can also oxidize and oxygenate small substrates.[5,6]

Oxygen activation by Fe(II)·bleomycin apparently requires an additional electron;[7] this can be provided by the disproportionation of two Fe(II)·bleomycins or by reductants such as ascorbate[3,8] or alkyl thiols.[3] Oxygen activation by Fe·BLM can also occur catalytically,[8] a facet of BLM chemistry that may be important therapeutically; however, all catalytic systems described to date require excess reducing agents[8,9] or oxygen surrogates such as iodosobenzene.[5,6,10]

To facilitate the study of Fe·BLM activation and characterization of the resulting activated species, we have developed a highly efficient electrochemical system for reductive activation of dioxygen + Fe^{III}·BLM. The electrochemical potential of Fe^{III}·BLM A_2 was determined by cyclic voltammetry at a glassy carbon electrode[11] (Figure 1, lower scan). The cyclic voltammogram corresponded to a one-electron, quasi-reversible, Fe(III)/Fe(II) redox couple;[12] $E_{1/2} = -0.08$ V vs Ag/AgCl($i_{p,c}/i_{p,a} \sim 1$, $\Delta E_p = 0.10$ V). Multiple (>15) scans were carried out without significant loss of current amplitude, indicating that redox cycling proceeded without destruction of Fe·BLM or the electrode surface. Although facile metal-centered reduction of Fe·BLM was not possible in the absence of an electron mediator using other electrode materials,[13] this $E_{1/2}$ value was consistent with the redox potential of Fe·BLM measured by microcoulometric and optical absorption techniques.[13a]

Aerobically (Figure 1, upper scan), the cyclic voltammogram exhibited increased current for the reduction wave at −0.13 V relative to the anaerobic system and a multielectron reduction wave

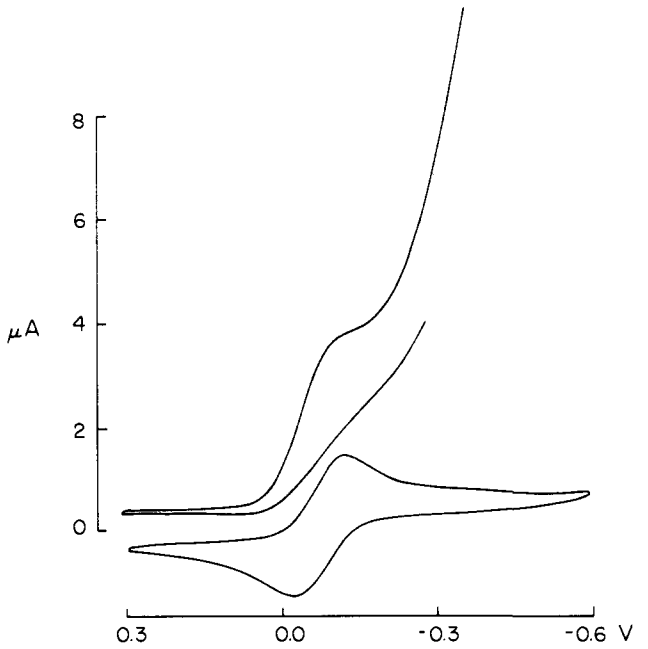


Figure 1. Cyclic voltammograms of Fe·bleomycin in the absence (lower scan) and presence (upper scan) of O_2. The experiments employed 0.5 mM Fe·BLM in 50 mM Na cacodylate, pH 7.2, at a scan rate of 20 mV/s; $E_{1/2} = -0.08$ V vs Ag/AgCl reference (Pt auxiliary electrode).

at more negative potentials. The increased current of the first reduction wave in the presence of O_2 suggests that Fe^{II}·BLM, once formed, can combine with dioxygen and undergo further reduction at the electrode surface.[14,15] The putative resulting activated Fe·BLM should undergo facile electrochemical reduction, consistent both with its known properties[7b,13c,16] and the absence of a reoxidation wave in the reverse sweep of the cyclic voltammogram.

The electrochemically reduced, oxygenated Fe·BLM was studied with DNA oligonucleotides. Accordingly, Fe^{III}·BLM solutions were electrolyzed at −0.22 V in the presence of O_2 and excess substrate oligonucleotides. Electrochemically activated Fe·BLM effected oligonucleotide degradation, producing multiple DNA lesions for each Fe·BLM present (Table I). Although the electrochemical reactions were slower than those carried out with Na ascorbate + O_2, undoubtedly reflecting limitations in maxi-

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(10) While such reagents have provided useful mechanistic data, they can also complicate interpretation of that data. E.g., dithiothreitol produced S-containing products when included in reaction mixtures containing Fe·BLM + olefinic substrate, and iodosobenzene destroyed BLM in a process unrelated to metallobleomycin activation (Murugesan, N.; Hecht, S. M., unpublished).
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(15) Alternatively, initial oxygenation of Fe^{II}·BLM could be followed by an electrode-independent disproportionation with another Fe^{II}·BLM.[7a] The observed lack of effect of catalase at high concentrations argues against the intermediacy of H_2O_2, as does the finding that intentional admixture of H_2O_2 to Fe·BLM in the presence of $CGCT_3A_3GCG$ gave products significantly less efficiently than electrochemically activated Fe·BLM.
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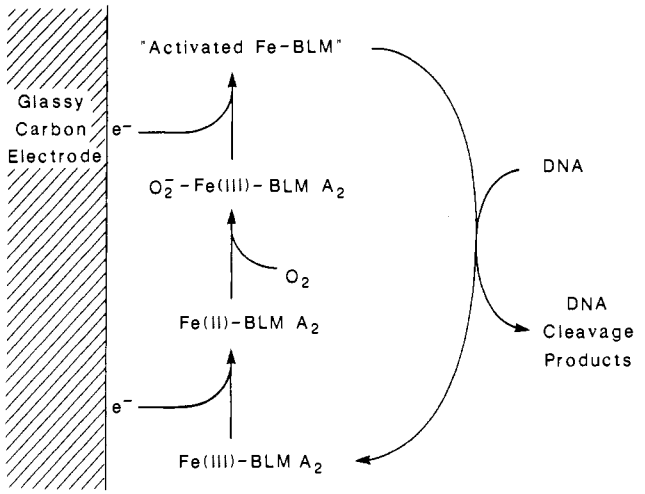
Scheme I. Proposed Catalytic Cycle for Fe·BLM Activation



mum current accessible in the present electrochemical cell, the products obtained via electrochemical and chemical activation were the same.[8,17]

Also studied was the effect of DNA on the electrochemical activation of Fe·BLM and on the putative self-inactivation of activated Fe·BLM. Admixture of excess CGCTAGCG or calf thymus DNA to Fe^{III}·BLM diminished the conversion to Fe(II)·BLM.[19] This paralleled a recent report[20] of the interaction of tris(1,10-phenanthroline)cobalt(III) with DNA and suggested that Fe^{III}·BLM was substantially bound to DNA under our experimental conditions. Quantitation of the current at high DNA concentrations indicated that Fe^{III}·BLM is reduced poorly, if at all, when bound to DNA.[21] Further, reductive activation of Fe^{II}·BLM under aerobic conditions at controlled potential was also inhibited at high DNA concentrations (not shown).

The presumed self-inactivation of Fe·BLM was studied by carrying out electrochemical activation in the presence and absence of DNA.[22] Aerobic electrolysis of Fe^{III}·BLM[23] resulted in a decrease in current to <20% (9.5 μA) of the initial value after passage of 10.2 electron equiv. Recovery of the electrolyzed Fe·BLM permitted verification that it had been substantially inactivated; by using fresh Fe(II), this sample degraded d($CGCT_3A_3GCG$) only 6% as well as a control sample of Fe·BLM. When the electrolysis was carried out in the presence of 0.63 mM d($CGCT_3A_3GCG$), the current was still 23.5 μA after passage of 14.4 electron equiv, indicating protection by DNA against Fe·BLM self-inactivation.

The foregoing data are consistent with a BLM activation mechanism involving initial reduction of Fe^{III}·BLM, followed by binding of O_2 and further reduction of the derived ternary complex (Scheme I). The data suggest strongly that both initial electrochemical reduction of Fe^{III}·BLM to Fe^{II}·BLM and reductive activation of Fe^{II}·BLM precede DNA binding. These observations place important constraints on the possible mechanistic schemes for Fe·BLM-mediated DNA degradation and should facilitate better definition of the individual steps involved.

Acknowledgment. This work was supported by NIH Grant CA-38544 and by an American Cancer Society postdoctoral fellowship to R.V.A. (PF-3125).

(17) Moreover, the specificity for modification at C_3 and $C_7(C_{11})$ as well as the strand selectivity (C_3 vs $C_7(C_{11})$), two characteristic features of individual activated BLMs,[8] were identical for electrochemically and chemically activated Fe·BLMs, suggesting that these species were the same. The maximum extent of DNA modification was also similar, presumably reflecting competitive self-inactivation of activated Fe·BLMs.[18] In fact, current flow diminished during the course of electrolysis.

(18) Although quantitation of the faradaic efficiency of Fe·BLM-mediated DNA degradation was hindered by the small volumes and limited amounts of substrates employed, the efficiency was clearly high in the context of the presumed stoichiometry of Fe·BLM activation.[7,8]

(19) In the presence of 1 and 2 mM calf thymus DNA, the current obtained with 500 μM Fe·BLM diminished from 1.19 to 0.94 and 0.63 μA, respectively.

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(21) That this was not due to physical exclusion of the DNA-bound Fe·BLM from the electrode surface was suggested by a parallel observation in other BLM activating systems. See: (a) Burger, R. M.; Peisach, J.; Blumberg, W. E.; Horwitz, S. B. *J. Biol. Chem.* **1979**, *254*, 10906. (b) Albertini, J.-P.; Garnier-Suillerot, A.; Tosi, L. *Biochem. Biophys. Res. Commun.* **1982**, *104*, 557. (c) Ciriolo, M. R.; Magliozzo, R. S.; Peisach, J. *J. Biol. Chem.* **1987**, *262*, 6290. A slight shift in the redox potential of Fe·BLM to a more negative value was observed by differential pulse polargraphy upon admixture of moderate concentrations of DNA. The significance of this observation is unclear, however, since the current obtained from 0.16 mM Fe·BLM and high concentrations (14 mM) of sonicated calf thymus DNA was <1.5% of that obtained in its absence.

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(23) Electrolysis was carried out using 0.20 mM Fe^{III}·BLM A_2 in 1 mL of 50 mM Na cacodylate, pH 7.2, in a cell containing a 3.14 cm^2 glassy carbon plate electrode. The background current was negligible (<2% of the initial current).

Rate-Determining Complexation in Catalytic Hydrolysis of Unactivated Esters in Neutral Water

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Nature had a considerable head start[1] in developing catalysts that hydrolyze simple esters such as acetylcholine. In the past 40 years many research teams worldwide have been trying hard to catch up to nature. Much has been learned by mimicking various catalytic features of esterases with elegant enzyme models.[2-7] However, one critical difference between the esterases and their models is their reactivity.[7,8] Nature has a monopoly on true catalysts that hydrolyze unactivated esters under mild conditions. In model studies, the esters are either highly activated (e.g., *p*-nitrophenyl acetate[2] or methyl trifluoroacetate[7]) or they are permanently anchored to various catalytic groups preventing any catalytic turnover.[3-6] Currently there is considerable interest in developing artificial esterases and peptidases that hydrolyze unactivated esters and amides.[9] Here we report on the first nonenzymic, catalytic hydrolysis of methyl acetate and acetylcholine in neutral water at 25 °C.

$[(trpn)Co(OH)(OH_2)]^{2+}$ (**1**)-catalyzed hydrolysis of methyl acetate was monitored by the pH stat method (trpn: tris(aminopropyl)amine). In a typical kinetic experiment, methyl acetate (1 M) and $[(trpn)Co(OH_2)_2]^{3+}$ (1 mM) in 5 mL of water were stirred at 25 °C. The pH of the reaction solution was maintained at 7.6 with a Radiometer PHM63 pH meter equipped with a Radiometer RTS822 automatic titrator.[10] Figure 1 shows the acetic acid production vs time plot. Three turnovers of the catalyst

(1) By about 4 000 000 000 years.

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0002-7863/89/1511-2724$01.50/0