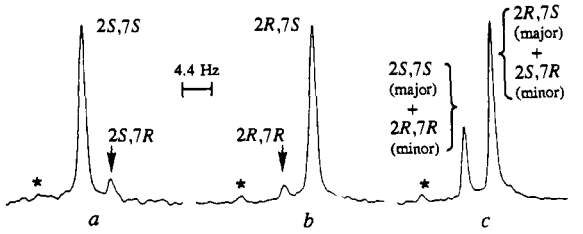
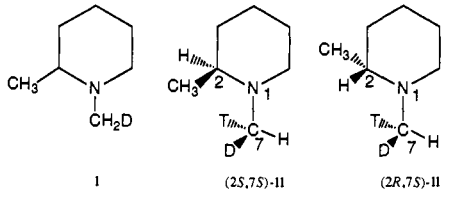


Figure 1. $^3H\{^1H,^2H\}$ NMR spectra at 320 MHz of the reaction products of (*R*)-CHDTN(Tos)$_2$ (86% enantiomeric excess, 3.4 and 6.6 mg, 0.32 mCi/mg) with (a) (*S*)-(+)- and (b) (*R*)-(−)-2-methylpiperidine having 96 and 100% enantiomeric excesses, respectively, and (c) spectrum of the mixture of the a and b samples in the ratio of 0.7:1 (note that the configuration of the methyl group inverts during the S_N2 reaction). The reactions were carried out in sealed tubes at 130 °C for 7 h, and the products were diluted with CD_2Cl_2 to give solution heights of ~2.0 cm in standard 5-mm NMR tubes. The FIDs were accumulated in an unlocked block mode, with a pulse angle of about 40°, an acquisition time of 2.8 s/FID, and total times of 112, 102, and 84 min for a, b, and c, respectively. The data were processed to give the optimum signal-to-noise (S/N) ratio with a broadening of 0.3 Hz; the smaller S/N ratio in a compared to b arises from a lower tritium concentration. The small peaks labeled with an asterisk (*) are assigned to CH_2T groups in isotopic impurities.

tively, by tritium NMR.[9,13,14] The work reported here, however, demonstrates for the first time that the configuration of stereogenic methyl groups can be assigned by direct tritium NMR analysis of a molecule containing an intact CHDT group. Although this new method is not as sensitive as the enzymatic procedure,[1-3] it allows a more accurate determination of the enantiomeric purity of the CHDT group.

A method for determining the enantiomeric excess in a CHDT group by a direct tritium NMR technique should be feasible because of the recent demonstration of diastereotopic protons with observably different ^1H chemical shifts in the CH_2D group of a chiral molecule (I) (as its racemate).[15] All that is required is



I (2S,7S)-II (2R,7S)-II

the N-methylation by a S_N2 mechanism of (*R*)- or (*S*)-2-methylpiperidine by a molecule of the type CHDTX, where X is a leaving group. The prediction is that the diastereomers (2*S*,7*S*)-II and (2*R*,7*S*)-II (and likewise their respective mirror-image forms) should have tritium chemical shifts differing by 0.015 ± 0.001 ppm, with the 2*R*,7*S* isomer containing the more shielded tritium.[15] Any compound that can be converted into CHDTX by a sequence of reactions of known stereochemistry is also a candidate for this method, and this includes chiral acetic acid, $CHDTCO_2H$. The Schmidt reaction (sodium azide and concentrated sulfuric acid, a reaction that takes place with configurational retention) on this acid gives chiral methylamine, which is converted by a two-step tosylation procedure into chiral *N*,*N*-ditosylmethylamine, CHDTN(Tos)$_2$,[7,16] where Tos_2N^- is a known leaving group in S_N2 reactions.

For demonstration purposes, we have treated (*R*)-CHDTN(Tos)$_2$ (expected to be >80% enantiomerically pure),[16] prepared from (*R*)-$CHDTCO_2H$,[17] separately with a 20-fold molar excess of (*S*)-(+)- and (*R*)-(−)-2-methylpiperidine.[18-20] The $^3H\{^1H,^2H\}$ NMR spectra of the crude reaction mixtures in CD_2Cl_2 solution were then measured, first separately and then after the samples were mixed together (Figure 1). The results clearly demonstrate that the diastereomeric [7-$^2H_1$,^3H]-1,2-dimethylpiperidines have tritium NMR chemical shifts differing by 4.4 Hz (0.014 ppm), with the tritium more shielded in the 2*R*,7*S* than in the 2*S*,7*S* diastereomer (and in their respective mirror images), in excellent agreement with predictions.[21]

The tritium NMR spectrum in Figure 1b gives an accurate estimate (86 ± 1%) of the enantiomeric excess in the (*R*)-CHDTN(Tos)$_2$ starting material. Presumably, the *S* enantiomeric impurity is the result of exchange between the protonated acetic acid or a related species with the strongly acidic solvent during the Schmidt reaction because the labeled acetic acid does not contain observable amounts of (*S*)-$CHDTCO_2H$ or of CH_2TCO_2H; this is consistent with the presence in the spectrum of a peak assignable to ~3% of the CH_2T analogue of II and an H/D kinetic isotope effect of ~5 in the exchange.

Acknowledgment. We thank Drs. T. Zydowsky, J. P. Lee, and C. Fox for the preparation of chiral *N*,*N*-ditosylmethylamine, Dr. U. Mocek for help in handling the tritiated compounds, and the National Institutes of Health and Research Corporation for financial support.

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(21) Actually, the ^1H chemical shift difference at 500 MHz in the CH_2D group of I prepared and examined as above (i.e., containing a small amount of the protonated amine) is 6.4 Hz rather than the 6.9 Hz reported[15] for the pure amine in CD_2Cl_2, and this leads to a predicted tritium chemical shift difference of 4.4 Hz at 320 MHz.

Coenzyme B_{12} Chemistry: The Crystal and Molecular Structure of Cob(II)alamin[‡,1]

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According to the available information, the homolysis of the organometallic bond of the protein-bound coenzyme B_{12} (**1**, 5′-adenosylcobalamin) induces the coenzyme B_{12} catalyzed enzymatic reactions.[2] However, the rates of these latter reactions typically exceed that of the homolysis of **1** in homogeneous solution by more than 10^{10} times at room temperature, so that the Co–C bond cleavage appears remarkably activated in the enzyme.[3,4] This is believed to result from specific interactions of the apoenzyme[4-9]

‡Dedicated to Prof. Dr. J. Schurz, on the occasion of his 65th birthday.
(1) Presented in preliminary form at the autumn meeting of the Swiss Chemical Society, Bern, Oct 1987, and at the 5th International Symposium on Organic Free Radicals, Zürich, Sept 1988.
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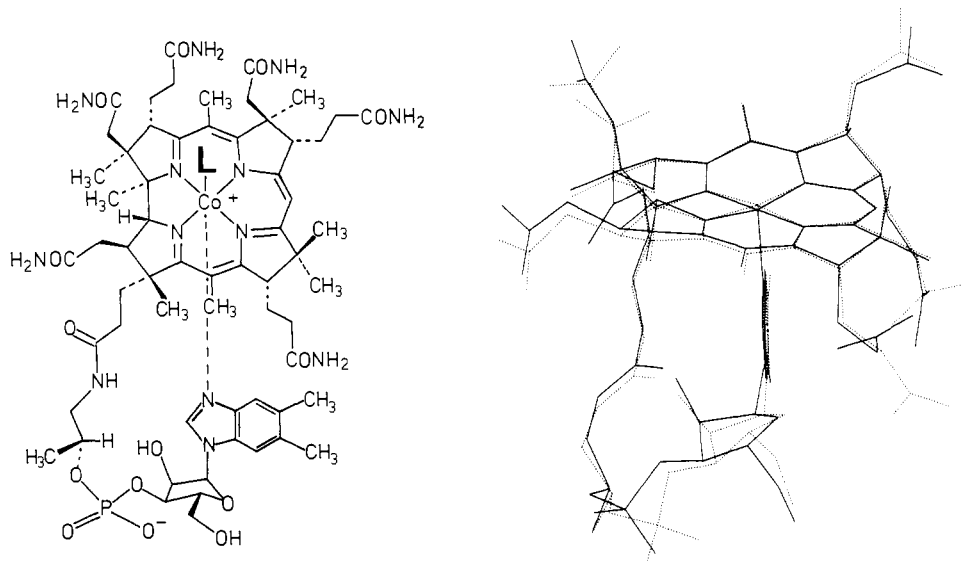

Figure 1. Left: structural formulas of coenzyme B_{12} (**1**, Co-L = Co^{III}-5′-adenosyl); cob(II)alamin (**2**, Co-L = Co^{II}), aquocobalamin (**3**, Co-L = Co^{III}-$H_2O^+(Cl^-)$), methylcobalamin (**4**, Co-L = Co^{III}-CH_3). Right: superposition of structures of the cobalt corrin part of coenzyme B_{12} (**1**, ···)[17c] and of cob(II)alamin (**2**, —); superposition at the four corrin nitrogens; orientation as in Figure 2, left picture.

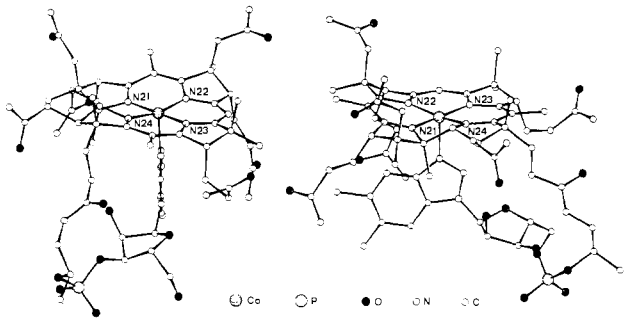

Figure 2. Crystal structure of cob(II)alamin (**2**).

with both the cobalamin and the adenosyl moieties of **1**.[5,6]

In spite of extensive experimental[4-8] and theoretical[9] work, the factors that promote the homolysis reaction are still a matter of dispute: the rate enhancement is believed to result from a "steric distortion" of the protein-bound coenzyme[2,4-10] (e.g., an "upward conformational distortion" of the corrin ring,[4a] movement of the benzimidazole base toward the corrin ring[10e]), while "electronic effects" of the trans ligand[10a,11] are considered less important.[9,12] *The structural changes that occur during the Co–C bond homolysis of the coenzyme* **1**, *which is a transition from a cobalt(III) corrin to a cobalt(II) corrin, are still unknown, however.* We are interested in the structures of cobalt(II) corrins[13] and report here on the preparation[14] and crystal structure[15,16] of cob(II)alamin (**2**, "B_{12r}"; see Figure 1), the corrinoid homolysis fragment of coenzyme B_{12} (**1**)[17a-c] (and of methylcobalamin (**4**)[17d]).

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(14) (a) The cobalt(II) corrin **2**, from catalytic hydrogenation of aquocobalamin (**3**, Fluka, AG, Buchs, Switzerland) according to a modification of an earlier procedure[14b,c] (370 μmol **3** in 20 mL of H_2O; 66 μmol platinum oxide (Baker); 1 atm of H_2; 3 h, room temperature; filtration and drying), was crystallized from deoxygenated aqueous acetone under N_2 (<10 ppm O_2, glove box (Mecaplex Switzerland)), 78% yield; spectral data: UV/vis (H_2O) λ (nm) (log ε): 260 (4.25), 289 (4.29), 312 (4.46), 404 (3.88), 474 (3.99); CD (H_2O) λ_{max} (Δε): 245 (−10.0), 279 (−4.8), 311 (24.1), 360 (−0.5), 378 (6.0), 442 (−4.6), 474 (−3.1), 506 (2.0), 663 (−1.6); magnetic susceptibility in the crystalline state: μ_{eff} = 1.89 μ_B (300 K, linear Curie plot down to 77 K; Faraday method). (b) Blaser, H. U.; Halpern, J. *J. Am. Chem. Soc.* **1980**, *102*, 1684–1689. (c) Dolphin, D. In *Methods Enzymol.*; McCormick, D. B., Wright, L. D., Eds.; 1971; Vol. XXVIII(C), pp 34–52.

(15) Crystals for X-ray analysis grew under N_2 in Mark capillaries (1 mm) at room temperature during several weeks from aqueous solutions of **2** by vapor diffusion of acetone. They were anaerobically sealed in the presence of the mother liquor.

(16) Crystal data for **2**: $C_{62}H_{88}N_{13}O_{14}PCo\cdot 19H_2O\cdot 3C_3H_6O$ (without consideration of SOFs), $M_{B_{12r}}$ = 1329.5; M_{LM} = 516; space group $P2_12_12_1$; a = 15.952 (4) Å, b = 21.732 (9) Å, c = 26.688 (10) Å, V = 9252 Å^3; Z = 4; D_{calcd}^{191} = 1.33, D_{obsd}^{293} = 1.34 g/cm^3 (flotation method, $CHCl_3$/acetone); data were collected on a STOE four-circle diffractometer with Mo Kα radiation (λ = 0.710 69 Å, graphite monochromator) at 191 (1) K for two octants, with 4° < 2θ < 45°; 12413 unique reflections (8782 with $F > 4\sigma(F)$), R = 0.102, R_w = 0.085 ($1/\sigma_1$ weights), 679 parameters and 8782 observations. Atomic coordinates, thermal coefficients, and geometric parameters are available as supplementary material. (Data collected at room temperature are less precise, but produce essentially the same structure).

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In the crystal, the cobalt(II) corrin **2** is found to contain a pentacoordinate[18] low-spin[14] Co(II) center, bound equatorially by the four corrin nitrogens and (α-)axially by a benzimidazole nitrogen (Figure 2). The Co(II) center is displaced by 0.12 Å from the plane of the corrin N's toward the axial base (in **1** and **4**, the deviations of Co(III) from the plane of the four corrin N's are within experimental error[17]). The equatorial Co–N bond lengths in **2** (average: 1.89 Å) are within experimental error of the corresponding bond lengths of **1** (1.90 Å)[17c] and of **4** (1.92 Å).[17d] The axial Co–N bond in the cobalt(II) corrin **2** (2.13 Å) is shorter than in the two cobalt(III) corrins **1** (2.24 Å) and **4** (2.19 Å).[17]

*The structure of the cobalt(II) corrin **2** is strikingly similar to that of the corrin moiety of coenzyme B_{12} (**1**, Figure 1) and of methylcobalamin (**4**).* In particular, the "upward folding"[8] of the corrin ligand in **2** (16.3°) compares to that in **1** (13.3°) and in **4** (15.8°).[8,17] Position and orientation of the nucleotide base with respect to the corrin ring are virtually unchanged in **1** and **2** as well, as the shorter axial bond is compensated for by the downward movement of the cobalt center in **2**. Significant differences in ligand structure between **1** and **2** are only evident for the D ring and its substituents, the nucleotide bearing f side chain and the g acetamide group: The peripheral ring-D substituents are displaced upward, as a consequence of a change in the tilt of ring D with respect to the corrin ring plane, which accompanies the downward axial shift of the Co(II) center in **2**. In the nucleotide loop, this results in considerable conformational changes in the ribophosphate segment.[19]

If the concept of activation of the bound coenzyme B_{12} by protein-induced deformation[4-12] is meaningful, we expect a deformation of **1** toward the structure of the homolysis products to be relevant, i.e., to **2** and to a 5′-deoxyadenosyl radical. As concerns the corrin fragment, our data give little support to the major modes of deformation considered, an increase of the "upward folding" of the corrin ligand[4a,8,10] and a movement of the benzimidazole base.[10e,f] On the other hand, the cobalt corrin core of **2** differs from that of **1** mainly by an upward deflection of the ring-D periphery and downward movement of the Co center. A corresponding deformation of **1** would result in increased strain between the adenosyl group and the corrin ligand.[8b,c] This could lead to "sterically induced labilization" of the Co–C bond of the type predicted[9b-d] for activation of the coenzyme.

However, protein-induced activation of coenzyme B_{12} does not necessarily require steric deformation of the bound starting material, but will result from differential binding of the coenzyme **1** and of the homolysis products (**2** and the 5′-deoxyadenosyl radical) to the apoenzyme.[20] Cobalt corrin based potential contributors to the protein-induced activation of the homolysis are provided by the structural differences between the unstrained **1** and **2**. In the crystal, these are noted at the nucleotide loop in particular, which has a high density of groups capable of strongly interacting with a protein.

In conclusion, the crystallographic data point at a remarkable similarity of the structures—*including the "upward folding"*[8]—of the corrin part of the coenzyme B_{12} (**1**) and the homolysis fragment cob(II)alamin (**2**). In view of the structural information presented, *the interactions (apoenzyme/coenzyme) at the corrin moiety of the coenzyme **1** appear insufficient to provide by themselves the major means*[21] *for a protein-induced activation of the bound **1** toward homolysis of its Co–C bond.* Instead, the organometallic bond of the bound **1** may be labilized largely by way of apoenzyme (and substrate) induced separation of the homolysis fragments, made possible by strong binding[5,6] of both separated fragments to the protein. On the other hand, the structural similarity between the corrin part of **1** and **2** presumably is an important factor for lowering the activation barrier in the Co–C bond homolysis of **1** and in the radical trapping reactions of **2**.

Acknowledgment. We thank Prof. Dr. A. Eschenmoser for stimulating discussions and Dr. C. Gatterer (Technical University of Graz) for carrying out the magnetic susceptibility measurements. This work was supported by the National Science Foundations of Switzerland (B.K.) and Austria (C.K.).

Supplementary Material Available: X-ray structural data for cob(II)alamin (**2**), including crystal data, atomic positional and thermal parameters, anisotropic temperature coefficients, observed and calculated hydrogen atom coordinates, bond lengths and bond angles, least squares plane analyses, and stereoscopic crystal packing diagrams, and a comparison of distances of equivalent atoms in the crystal structures of **1** and of **2** (15 pages). Ordering information is given on any current masthead page.

(18) An ill-defined solvent molecule produces electron density near the second axial coordination site, but ca. 3.42 Å from the metal center.

(19) The ribose ring assumes a C2′-exo conformation in **2** and a C3′-endo conformation in **1**. Dihedral angles along the nucleotide side chain differ by up to 20°, leading to positional discrepancies of up to 2.1 Å for the phosphate oxygens. Apart from that, the b and e carboxamide termini differ in position (by up to 4.8 Å), due to adaption (trans vs gauche) of the side-chain conformations to the particular H-bonding pattern in the crystals (see, e.g., ref 8b).

(20) See, e.g.: Fersht, A. *Enzyme Structure and Mechanism*, 2nd ed.; W. H. Freeman & Co.: New York, 1985; pp 311–346.

(21) Potential minor contributors are discussed in the previous two paragraphs.

Tetrakis(trifluoromethyl)cyclopentadienyl Ligands for Transition Metals†

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The introduction of substituents into the cyclopentadienyl (Cp) backbone is well-known to dramatically affect the reactivity of transition metals containing these ligands.[1] While complexes of electron-rich derivatives such as pentamethylcyclopentadienide have been extensively studied, metal centers that contain cyclopentadienyl groups bearing electron-withdrawing substituents[2] remain relatively unexplored. Recently, Gassman and Winter demonstrated the effects of placing one trifluoromethyl group on a Cp ring.[3] Our objective has been to develop general routes to highly electron withdrawing cyclopentadienyl ligands containing multiple trifluoromethyl substituents. Such ligands should lead to electrophilic, yet nonoxophilic, late-transition-metal complexes with potentially useful properties such as oxidation resistance and fluorocarbon solubility. We report the preparation and properties of several transition-metal complexes containing the tetrakis(trifluoromethyl)cyclopentadienyl ligands, η^5-$C_5(CF_3)_4X$(X = H, $OSiEt_3$).

Our initial efforts focused on establishing the coordinating ability of the known[4] tetrakis(trifluoromethyl)cyclopentadienide salt, $[C_5(CF_3)_4H]^-Me_4N^+$ (**1**). No reaction occurred between **1** and metal halides, probably owing to the low nucleophilicity of this anion. Reaction with the solvated metal cation $[(\eta^5$-$C_5Me_5)Ru(MeCN)_3]^+O_3SCF_3^-$,[5] however, resulted in essentially

† We dedicate this paper to Dr. George W. Parshall on the occasion of his 60th birthday.

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