$(Me_2CSO_2Ph)^-$ and [Li·[2.1.1]cryptand]$^+$ ions; the anionic centers are well separated from the Li^+ ion, and no unusual short intermolecular contacts are observed. Hence, **1** may be regarded as a model for a solvent-separated ion pair. The anion of **1** has, like that of **2**, a strongly pyramidalized anionic carbon atom (C_α atom) with the methyl groups bent away from the O atoms. It features the typical C_α–S conformation of α-sulfonyl carbanions[1-4,6,7] wherein the lone electron pair is almost exactly orientated gauche to both O atoms. This allows inter alia for a stabilization by negative hyperconjugation (n_C–σ_{SPh}^*).[7] A comparison of the bonding parameters of the unassociated anion of **1** and of the lithium-associated one of **2** reveals a close similarity except for one feature (Figure 1): the trigonal C_α atom in the cryptate **1** is significantly less pyramidalized than that in the contact ion pair **2** as shown by the pyramidalization angle χ[11] of 32.5° and 40.8°, respectively. There is much evidence from crystal structure analysis of acyclic O–M-associated α-sulfonyl carbanion alkali-metal salts that the pyramidalization of the alkyl-substituted C_α atom originates primarily from the minimization of torsional strain around the C_α–S bond whereas the planarization of the phenyl-substituted C_α atom stems from the maximization of stabilizing p_π–p_π overlap.[1-4] This, however, implies that electronically the energy difference between a pyrmidalized and planar (at the C_α atom) α-sulfonyl carbanion is only small, a conclusion that is supported by ab initio calculations.[7b] Thus, in dimeric solvated contact ion pairs like **2**, intramolecular packing forces could reinforce the pyramidalization of the C_α atom because of its shallow pyramidalization potential. A space-filling model of **2** (C_i) reveals indeed a packing that encompasses a close proximity of methylene and methyl groups of both diglyme molecules and the methyl groups at the C_α atom. The greater pyramidalization of the C_α atom in **2** therefore may be attributed to a steric intraaggregate chelate ligand/anion interaction. In dimeric solvated O–Li contact ion pairs of benzylic lithio sulfones,[1,2,3c,f,4a,c] manifestation of such an effect is not to be expected because of a steeper pyramidalization potential of the C_α atom bearing a phenyl group. Accordingly, the C_α atom of the analogous dimer {[Ph(Me)CSO_2Ph]Li·diglyme}$_2$ (C_i) is almost planar, and no close proximity exists between the diglyme molecules and the groups at the C_α atom.[3c]

The complex ion [Li·[2.1.1]cryptand]$^+$ has already been crystallographically characterized; its present structure shows no significant deviation.[12] Since with **1** the separation of an O–Li-associated lithium carbanion salt has been achieved in the solid state,[13] isolation of inclusive cryptates of other synthetically important lithium carbanion salts with O–Li and/or N–Li association[4c,14] may be feasible too.[15]

In summary, the Li^+ gegenion effect on an alkyl-substituted or benzylic α-sulfonyl carbanion in dimeric solvated lithium salts is apparently small. The extent of the pyramidalization of the C_α atom, however, can be determined in the former case to a certain degree by intraaggregate interactions.

A fair structural picture of the associated as well as the unassociated α-sulfonyl carbanion has now emerged. This should aid considerably an investigation of the asymmetric induction exerted by the sulfonyl group at the C_α atom which is now underway in our laboratories.

Acknowledgment. This work was supported by the Deutsche Forschungsgemeinschaft. We thank Dr. H. Paulus, TH Darmstadt, for the crystallographic data set, Dr. E. Keller for a version of SCHAKAL-88, and N. Reuter for the SCHAKAL drawings.

Supplementary Material Available: Details of the X-ray structural analysis of **1** including tables of refined atomic coordinates, bond lengths and angles, calculated hydrogen atom coordinates, and anisotropic thermal parameters and figures showing space-filling models of the molecular structures of **2** and {[Ph(Me)CSO_2Ph]Li·diglyme}$_2$ (46 pages); listing of observed and calculated structure factors (18 pages). Ordering information is given on any current masthead page.

(9) Crystal data for **1**: $C_{23}H_{39}LiN_2O_6S$, monoclinic, space group $C2/c$ with a = 30.113 (19) Å, b = 8.446 (3) Å, c = 25.248 (8) Å, β = 127.50 (1)°, V = 5094 Å^3, Z = 8, D_{calcd} = 1.247 g cm^{-3}. The final residuals for 306 variables refined against 3014 data with $|F| \geq 2\sigma(F)$ ($3° < 2\theta < 45°$) were R = 4.8% and R_w = 6.0%.

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Construction, DNA Binding, Two-Dimensional Nuclear Magnetic Resonance Spectrum, and Structure of a Mutant *lac* Repressor Headpiece

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Received October 8, 1990

The *lac* repressor–operator system has been the prototype for studying protein–DNA interactions.[1] While site-specific mutagenesis has provided information on the role of individual amino acids in recognition,[2,3] it is not understood at a detailed molecular level.[4,5] Many mutants have been generated by nonsense mutations[6,7] (although the substitutions are limited to amino acids whose codons have suppressible mutations) and cassette mutagenesis.[8] Here, we describe the secondary structure of a 56-residue *lac* repressor mutant headpiece studied by two-dimensional NMR. The tyrosine 7 to isoleucine (Y7I) mutant of the repressor has been designed to test the importance of proposed tyrosine 7–tyrosine 17 stacking in the stabilization of the protein and the role this might play in DNA recognition. This interaction reported by Jardetzky[9,10] remains one of the first NOEs observed for *lac* headpiece. While some NMR spectral differences have been reported for mutant repressors,[11,12] we believe this study to represent the first direct comparison of the structural and biological

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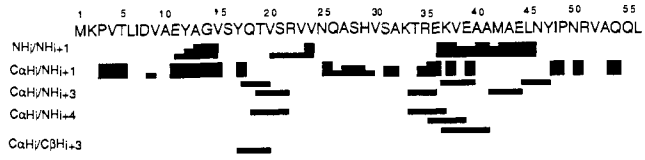

Figure 1. Summary of the short- and medium-range NOEs observed for the Y7I mutant headpiece. Relative sizes of the NOEs are defined by the sizes of the bars. Note that the NH–NH connectivity of the second helix is weaker in intensity than that reported for the second helix of the wild-type repressor, where all of the helices are of approximately equal intensity. As reported for the wild-type headpiece, not all of the sequential backbone connectivities are observed.

functional changes between a wild-type and mutant repressor which was accomplished by NMR spectroscopy.

The *lac* repressor is proposed to bind through a helix–turn–helix motif which shares homology with other bacterial DNA-binding proteins.[13-15] The smaller *lac* repressor headpiece appears to be able to duplicate the basic *lac* operator–*lac* repressor interaction.[16-21] While tyrosines 7 and 17 are at the beginning of the first and second (or recognition) α-helices, the two tyrosine side chains are proposed to stack so as to stabilize the tertiary structure and orientation of the recognition helix in the complex.[22]

The Y7I mutant was constructed by oligonucleotide site-directed mutagenesis of pHIQ6[23] which overproduces the *lac* repressor. The mutant was produced[24-26,28] by nicking pHIQ3 with Hae III under restrictive conditions, digesting with exonuclease III, and reannealing a synthetic oligonucleotide producing a new Cla I site and coding for the Y7I substitution. Screening was performed by hybridization of a ^{32}P-labeled oligonucleotide with washing at 55 °C.[26-28] The mutation was confirmed by the presence of a new Cla I site, dideoxy sequencing of the (−) strand, and Edman degradation of the purified protein.[28] The mutant repressor and headpiece were prepared as previously described.[21,29]

The Y7I headpiece (ca. 10 mg) was dissolved in 0.6 mL of 0.4 M KCl, 0.05 M potassium phosphate buffer, pH 6.0. Most of the ^{1}H NMR signals of the mutant repressor were assigned through the NOESY (mixing times of 40–350 ms) and TOCSY 2D NMR spectra in H_2O and D_2O by using sequential assignment methodology developed by Wüthrich[30] and performed as previously described[31] (spectra and assignments available upon request). The secondary structure of the Y7I mutant headpiece derived from

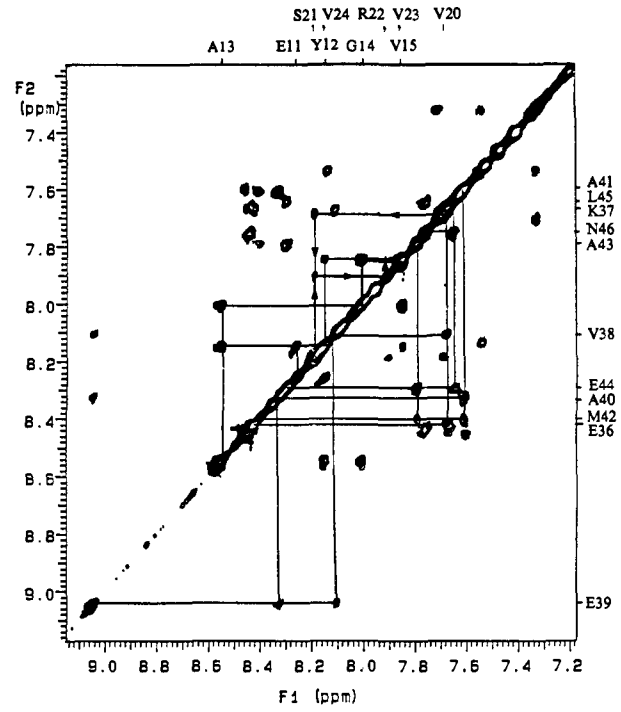

Figure 2. Expanded 2D NOESY spectrum at 600 MHz with a mixing time of 200 ms showing the NH–NH connectivities observed for the Y7I mutant headpiece at ambient temperature. The second helix, shown by a dashed line, has weaker NH–NH connectivities than those observed for the first and third helices as well as fewer NH–NH connectivities observed for this helix compared to the second helix of the wild-type repressor headpiece.

the relative intensities of the NOESY cross peaks (see Figure 1) is similar to that of the wild-type headpiece. Proton chemical shifts of the mutant are remarkably similar to those of the wild-type protein (with the exception of T5 and I7). However, the aromatic protons of tyrosine 17 are shifted ca. 0.2 ppm upfield of those in the wild-type headpiece. This is consistent with a ring-current shift (although not necessarily stacking) of the aromatic rings of the two tyrosines in the wild-type headpiece. Several of the NH–NH sequential NOESY cross peaks in the second α-helix are missing, and those present are much weaker than those found in the wild-type headpiece (see Figures 1 and 2). Also, many of the αH_i–NH_{i+3} and αH_i–NH_{i+4} are not found in the first (residues 6–13) and second (residues 17–25) helices of the Y7I headpiece. Note, however, that the NH–NH intensities in "helix" 3 (residues 34–47) are *not* altered relative to the wild-type repressor. The loss of eight αH_i–NH_{i+3} and αH_i–NH_{i+4} cross peaks in "helix 1" of mutant relative to the wild-type headpiece indicates that the conformations of these residues in the mutant are also quite different from their conformations in the wild-type protein. Presumably loss of the Y7–Y17 side-chain interaction in the mutant selectively destabilizes helices 1 and 2. The presence of nearly all of the tertiary structure cross peaks (Karslake et al., to be published) in the mutant indicates that the overall folding has not been dramatically altered.

Binding affinity of the wild-type and mutant proteins for the operator was determined by gel retardation assays[32] using a horizontal polyacrylamide gel of 4% acrylamide/0.7% agarose.[33] The polymerase chain reaction[35] (PCR) was used to prepare a ^{32}P-labeled 322-bp operator-containing fragment of *Pvu*II-digested pUC19[28,34] with high specific activity.

The DNA was run with 20000 cpm per sample. Wild-type and mutant repressor concentration varied from 2.6 to 31.1 and

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16 to 162 pM, respectively. The gel was loaded with a final volume of 40 μL in 10 mM Tris, 0.2 M KCl, 2.5% glycerol, 10 mM magnesium acetate, 0.3 mM dithiothreitol, 100 μg/mL salmon sperm DNA, and 50 μg/mL bovine serum albumin, pH 7.75. After electrophoresis, the gels were exposed to X-ray film overnight at −80 °C and scanned. The $K_{D(app)}$ for the *lac* wild-type and mutant repressors are 9.5×10^{-12} M and 30.6×10^{-12} M, respectively. (We believe this to be the first time PCR and a gel retardation assay have been used to measure protein–DNA dissociation constants in the picomolar concentration range.[28]) This is consistent with previous reports[6,7] that the tyrosine 7 to leucine mutant is a "weak binding" repressor.

While the mutation has significantly disrupted the overall structure and stability of the recognition helix, it appears to have had a lesser effect on DNA recognition and binding, suggesting that the operator may induce proper folding and stabilization of the recognition helix. Alternatively, the reduction in operator affinity may simply reflect the degree of helix disruption that we observe by NMR. Interestingly, the DNA recognition portion of the "bZIP" motif of GCN4 has been shown to increase in α-helical content in the presence of its target DNA.[36] If we are to understand the origin of protein–DNA recognition, it will be essential that we correlate biological functional changes with structural changes. Perhaps a certain degree of flexibility in the recognition helix will prove to be an important factor in the binding of proteins to DNA.

Acknowledgment. Supported by the NIH (AI27744), the Purdue University Biochemical Magnetic Resonance Laboratory, which is supported by the National AIDS Research Center at Purdue (AI727713), and the NSF National Biological Facilities Center on Biomolecular NMR, Structure and Design at Purdue (Grants BBS 8614177 and 8714258 from the Division of Biological Instrumentation). We appreciate the gift of the plasmid and *Escherichia coli* cell line from Prof. Joan Betz and contributions of Martial Piotto.

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Sulfur versus 2,3-η^2 Coordination of Benzo[*b*]thiophene (BT) in Cp′(CO)$_2$Re(BT)

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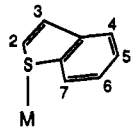
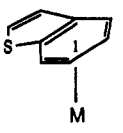
Received December 3, 1990

Recent studies of thiophene coordination and reaction in transition-metal complexes have suggested new modes of thiophene adsorption and activation on hydrodesulfurization (HDS) catalysts.[2,3] Much less is known about benzo[*b*]thiophene (BT) coordination in transition-metal complexes. Although there is one example of an S-bound BT complex, Cp(CO)$_2$Fe(*S*-BT)$^+$,[4] all other characterized complexes contain an η^6-BT ligand which is coordinated via the π-system of the benzene ring: CpRu(η^6-BT)$^+$,[5] Cp*Rh(η^6-BT)$^{+2}$,[5] Cp*Ir(η^6-BT)$^{+2}$,[5] and Cr(CO)$_3$(η^6-BT).[6]



S-BT



η^6-BT

However, η^6-binding to a metal site does not account for deuterium exchange or C–S bond cleavage of BT on HDS catalysts since the sites of deuterium exchange in BT are different over the catalyst and in CpRu(η^6-BT)$^+$.[7,8] In the present communication, we describe the complexes Cp′(CO)$_2$Re(BT), where Cp′ = η^5-C_5H_5 (Cp) or η^5-C_5Me_5 (Cp*), which exist as S- and 2,3-η^2-bound BT isomers in equilibrium with each other. The novel 2,3-η^2-BT bonding mode provides a basis for understanding initial steps in the hydrodesulfurization of BT.

A solution of Cp′(CO)$_2$Re(THF), generated by UV irradiation of a THF (30 mL) solution of Cp′Re(CO)$_3$ (0.20 g) at −20 °C,[9] was stirred with BT (0.30–0.40 g) at room temperature for 10 h. After removal of the solvent under vacuum, the residue was chromatographed on neutral alumina by using CH_2Cl_2/hexanes (1:4) as eluent. The yellow band was concentrated under vacuum and slowly cooled to −20 °C to give pale yellow, moderately air stable crystals of **1** (0.053 g, 21%) and **2** (0.071 g, 27%) respectively. Although elemental analyses and the mass spectrum establish the composition of **1** as Cp*(CO)$_2$Re(BT),[10] it is evident from the number of bands in the solution IR and ^{1}H and ^{13}C NMR spectra that it consists of two isomers, the η^2-bound (**1a**) and S-bound (**1b**) isomers, which are present at equilibrium in a 1.6:1 ratio in $CDCl_3$ solution at room temperature (Scheme I). The H2 and H3 ^{1}H NMR signals (δ 4.25, d; 3.96, d) of the BT in the major isomer (**1a**) are substantially upfield of those in free BT (δ 7.33 (H2) and 7.22 (H3)).[11] Also, two of the ^{13}C NMR resonances (δ 47.9 and 46.6),[10] presumably those of C2 and C3, are substantially upfield of those (δ 126.2 and 123.8)[11b] in BT. Such upfield ^{1}H and ^{13}C NMR shifts were observed previously in Cp*(CO)$_2$Re(η^2-selenophene)[12] and are characteristic of η^2-olefin[13] and η^2-arene[14] bonding. Thus, the major isomer (**1a**, Scheme I) contains 2,3-η^2-BT. This was further supported by an X-ray diffraction study[15] of a crystal of **1a** selected from product **1**.

The ^{1}H and ^{13}C NMR chemical shifts of the BT ligand in the minor isomer (**1b**)[10] are similar to those in free BT[11] (^{1}H NMR (CCl_4) δ 7.79, 7.72, 7.33, 7.26, 7.24, 7.22; ^{13}C NMR ($CDCl_3$) δ 139.7, 139.6, 126.2, 124.2, 124.1, 123.8, 123.6, 122.4), which are also similar to those[4] of the S-coordinated BT in Cp(CO)$_2$Fe(*S*-BT)$^+$. These comparisons together with the similarity of the ν(CO) bands (1932 and 1871 cm^{-1}) of **1b** and those (1934

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(15) Details of the structure of **1a** will be published elsewhere.

0002-7863/91/1513-4005$02.50/0