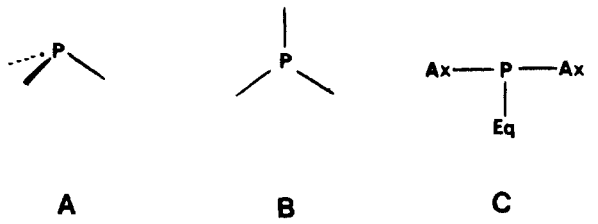


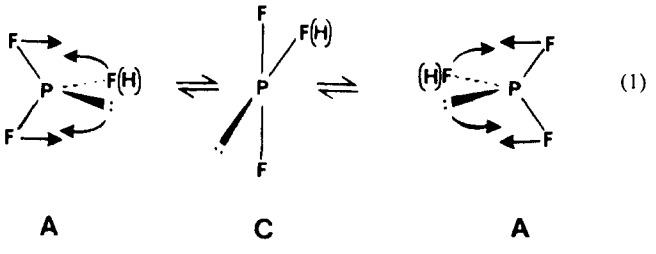
ramidal structures (A) is also found with the calculations of Boggs and co-workers.[4a] We also find good agreement for the structures of the classical inversion transition states B except for PF_3 where Boggs and co-workers[4a] placed the lone pair in an a_2'' orbital. Comparison of the pyramidal A and planar B structures (excluding PF_3) shows that the bond lengths decrease in going from the pyramidal to planar structures in agreement with previous observations.[11] For PF_3, the bond length in structure B increases which is consistent with the lone pair occupying an in-plane a_1' orbital.

The planar T-shaped structures C show novel features. For PH_2F and PHF_2 C_{2v} symmetry was maintained and thus the hydrogens are in the axial positions in the former and fluorines are in the axial positions in the latter. In all cases, the three ligands optimize to the same side of the phosphorus (the axial substituents are bent back toward the equatorial substituent). As expected the axial substituents have long bond lengths. For example, in PH_2F (C), the P–H bond is 13.1 pm longer than in structure A and 17.8 pm longer than in structure B. The P–F equatorial bond in PH_2F (C) is slightly longer, 0.5 pm, than in B but is shorter by 1.0 pm as compared to A. The axial P–H bonds in PH_3 (C) are even longer, 24.1 pm, as compared to PH_3 (A) while the equatorial P–H bond is shorter by 0.5 pm as compared to A. The axial P–F bonds in C for PHF_2 and PF_3 show a less dramatic lengthening of 7.0 pm. For PF_3 (C) the equatorial P–F bond is 8.0 pm shorter than the axial P–F bonds.

The barriers for inversion through B are slightly lower (excluding PF_3) than those reported by Boggs and co-workers[4a] (Table I). The classical inversion barriers through a B structure increase as F is substituted for H as previously observed.[4a] Our value for PH_3 is only 1 kcal/mol above that calculated with a DZ + P basis set of STO's.[11] Our value for PF_3 (B) is essentially the same (3.4 kcal/mol higher) as Marynick's value[5] (a_1' HOMO).

Phosphine will invert through structure B as structure C is 121.2 kcal/mol higher in energy than structure B. For PH_2F, structure C is still 67.2 kcal/mol above structure B and clearly PH_2F will invert via a classical planar structure. However, structure C for PHF_2 is 35.6 kcal/mol below structure B, and for PF_3, structure C is 56.2 kcal/mol below structure B. Thus PHF_2 and PF_3 will invert through a T-shaped structure C but not via a classical planar-trigonal structure B.

The inversion process may be visualized as the one involving simple angle deformation motions (eq 1). For PHF_2 and PF_3,



(1)

the T-shaped structure C can arise from a pyramidal configuration by simultaneous opening of the F–P–F and F(H)–P–lone pair angles. Microscopic reverse of the process can occur in two degenerate ways and provides A or its inverted structure.

Table I. Optimized Geometry Parameters[a] and Inversion Barriers[b] for Fluorinated Phosphines

property	PH_3	PH_2F	PHF_2	PF_3
r(PH) A	140.7	140.8	140.9	
r(PF) A		160.2	158.1	156.3
θ(APA) A	95.2	94.1	98.5	97.1
θ(HPF) A		98.1	96.3	
r(PH) B	137.1	136.1	135.2	
r(PF) B		158.7	157.1	166.4
θ(APA) B	120.0	131.2	109.6	120.0
θ(HPF) B		114.4	125.2	
r(PH) C	164.8 (ax) 140.2 (eq)	153.9 (ax)	139.7 (eq)	
r(PF) C		159.2 (eq)	165.5 (ax)	155.3 (eq) 163.3 (ax)
θ(axPax) C	166.0	158.0	168.0	172.8
θ(axPeq) C	83.0	79.0	84.0	86.4
barrier (B)	37.9	59.5	101.6	124.9
HOMO (B)	a_2''	a_2''	a_2''	a_1'
barrier (C)	159.1	126.7	66.0	68.7

[a] Bond distances in pm; Bond angles in degrees: ax = axial; eq = equatorial. [b] Inversion barriers (geometry of planar structure) in kcal/mol.

Throughout the process the lone pair remains on the same face of the P and electronic repulsions between the lone pair and the ligands' electrons are kept minimum. By contrast, such electron repulsions significantly increase in the classical process involving structure B that inverts the lone pair through the plane. Inversion via structure C amounts to edge inversion of a tetrahedral structure through a square-planar species, whereas the classical inversion through a planar-trigonal species B represents face or vertex inversion.

The accessibility of the planar T-shaped structure not only should allow for a new process for changing conformation at pnictogen centers but also suggests a plausible explanation for the stability of compounds such as **1**. Experimental demonstration of this novel inversion process and studies of factors that influence relative energies of structure A, B, and C are in progress.

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Structure of a Metalated Schiff Base[1]

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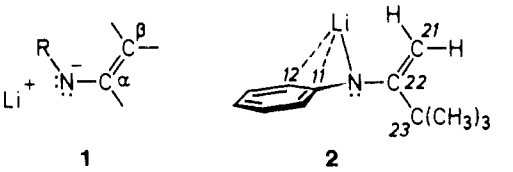
1-Azaallyllithium reagents **1** have aroused widespread interest because of their synthetic utility and fascinating aspects of (stereo)isomerism. Whereas the problem of *E*/*Z* isomerism about the CαCβ double bond appears to be provisionally settled,[3-6] exploration of the CαN configuration of **1** had to rely on more



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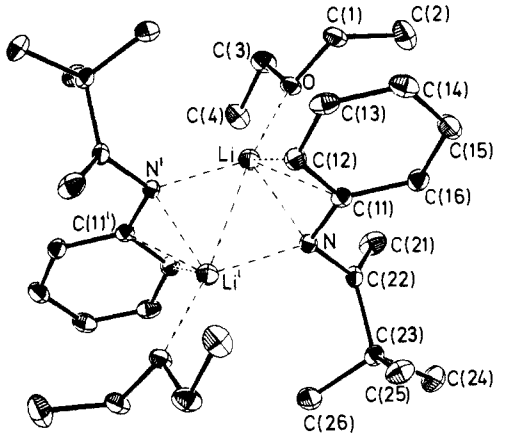

Figure 1. Molecular structure **3** of $[(H_2C{=}C(t\text{-Bu})NPh)Li(OEt_2)]_2$, the dimer of **2**. Ellipsoids correspond to 50% probability. Distances: Li–Li′, 2.544 (2); Li–N, 2.076 (2); Li–N′, 2.000 (2); Li–O, 1.937 (2); Li–C11, 2.437 (2); Li–C12, 2.662 (2); N–C11, 1.380 (1); N–C22, 1.436 (1); C22–C21, 1.326 (1); O–C_{av}, 1.443 Å. Angles: Li–N–C11, 87.28 (7)°; Li–N′–C11′, 119.36 (7)°; Li–N–Li′, 77.22 (7)°; N–Li–N′, 102.78 (7)°; N–Li–O, 121.93 (9)°; N′–Li–O, 134.7 (1)°; C22–N–C11, 116.61 (7)°.

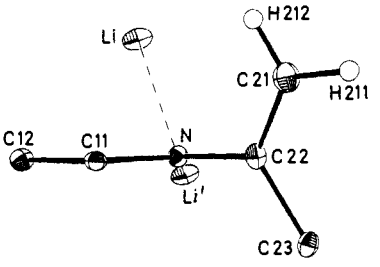

Figure 2. Partial structure of **2**, viewing the dimer **3** in Figure 1 from the right and along the bisector of the angle C22–N–C11.

indirect evidence,[7,8] and a syn-coplanar arrangement of the CβCαNR moiety has been taken for granted by almost all authors,[9] including ourselves.[3,4] Moreover, since the actual structure of **1** is not known, the binding of lithium by the π-azaallyl system or the nonconjugating lone pair at the nitrogen atom or by ionic interaction remained an open question.[10,11] We now report the structure[12] of *N*-(2,2-dimethyl-1-methylenepropyl)-*N*-lithiobenzenamine (**2**), showing that two lithium cations can bind each to one of the two nitrogen lone pairs simultaneously and that aggregation of **1** as well as unorthodox conformations about the CαN bond should be seriously considered.

The monoetherate of **2**, prepared[4] by deprotonation of pinacolone anil, separated from ethereal solution as colorless, transparent platelets which are sensitive toward atmospheric oxygen. The compound was found to be a centrosymmetric dimer (**3** in Figure 1) by structural analysis[12,13] from which the following salient features emerge (using the crystallographic numbering):

(i) Each nitrogen atom is bound to its three next neighbors (e.g., N to C11, C22, and Li′) in an approximately planar fashion, as shown in Figure 2 and expected for typical enamines[14] and furthermore to the second cation (Li) at an almost right angle (Li–N–C11 = 87.3°). In contrast to enamines, the C21–C22 double bond is rotated almost completely out of conjugation with the N–Li bond by a dihedral angle (C21–C22–N–C11) of 80.9° and appears to be weakly conjugated with the N–Li′ bond, with a C22–N bond length of 1.436 (1) Å which is ca. 0.05 Å longer than in enamines but shorter than in saturated amines.

(ii) The cation Li binds to N, C11, and C12 in much the same way as in benzyllithium.[15] Its ligand shell is satisfied by coordination to a pyramidal oxygen atom of diethyl ether and to the N′ atom, thus forming a planar NLiN′Li′ core.

(iii) The phenyl groups occupy transoid positions at the four-membered ring and are significantly distorted within their planes, presumably due to charge delocalization from the NLi and N′Li′ bonds, respectively. This characterizes compound **3** as a lithium anilide and, together with feature ii, as very similar in this part of its structure to the dimeric etherate of lithium 2,4,6-tri-*tert*-butylanilide,[16] in close accord with predictions.[17]

The present structure **3**, of the dimer of **2**, differs greatly from that of a η^4,η^1-lithiated *N*,*N*-dimethylhydrazone[18] and from all previous proposals for **1**, including computational results on the free 1-azaallyl anions,[8,19] π-complexes,[20] and models with internal chelation.[5,9] A formal relationship to the latter models might be recognized if the slightly nonplanar atomic chain Li–N–C11–C12 in Figure 2 is taken as the basic structure, as suggested by a referee, with auxiliary π-coordination to the other lithium cation. However, these models had been proposed[5,9] to explain the stereoselectivity of electrophilic attack at the olefinic double bond that does not belong to that chain.

Viewed from the nearly planar olefinic fragment C21–C22(–N)–C23, the lithium anilide moiety maintains an almost orthogonal conformation, thus explaining why the $C_6H_5NLi_2$ substituent appeared to be "smaller than benzyl"[21] and hence why a trans arrangement of substituents at Cα and Cβ in (*Z*)-**1** (R = C_6H_5) prevails in equilibrium.[4] Since the C22–N conformation is neither syn nor anti, the lithiated Schiff base **3** cannot be classified as a 1-azaallyl anion. However, the existence in solution of lithiated heterocyclic imines[19] attests to the possibility of the anti (Cα–N) conformation as another alternative to **1**; furthermore, lithiated, open-chain, *N*-alkyl-substituted imines **1** are invariably observed to accept alkylating reagents in the syn position of their Schiff base products under kinetic control, i.e., at the Cβ atom most remote from the nitrogen lone pair. Therefore, loss of 1-azaallyl conjugation may not be tolerable without the opportunity to delocalize negative charge from nitrogen atoms into the phenyl rings as in **2**/**3**, and the reversed preference for *E*-(CαCβ) conformations observed[5] for *N*-alkyl-substituted derivatives of **1** becomes plausible.

Spectroscopic and chemical observations may appear in a new light if the dimeric nature carries over to lithiated imines in solution. NMR decoalescence phenomena had previously[8] been considered to be the only direct evidence for a syn (CαN) conformation of anions **1**. If these effects are also observed for the *N*-*tert*-butyl derivatives,[6] they can no longer be interpreted by *N*-alkyl rotation;[8] two equilibrating diastereomeric dimers with differing maximal symmetries, $C_2^{\parallel}$ (LiLi′ axis) for the trans-substituted NLiN′Li′ core and C_s for the cis arrangement, should provide an alternative. Chiral N substituents destroy the C_i (in **3**) and C_s symmetries and permit easier access of Cβ-alkylating

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reagents (whether assisted by Li[5] or not[20]) from that hemisphere about the dimer which does not enclose the bulkier groups.[22] Even the poorer enantioselectivity of the Z-(CαCβ) imine anions[5] may be explained without[20] recourse to dechelation,[5] because Cβ substituents in cis relation to the nitrogen alkyl substituents would force the face-differentiating groups further away from the reacting Cβ atom.

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Supplementary Material Available: Tables of refined atomic coordinates and temperature factors for $[(CH_2{=}C(t\text{-Bu})NPh)\text{-}Li(OEt_2)]_2$ (2 pages). Ordering information is given on any current masthead page.

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Mechanism of Induction of Parkinson's Disease by 1-Methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). Chemical and Electrochemical Characterization of a Geminal-Dimethyl-Blocked Analogue of a Postulated Toxic Metabolite

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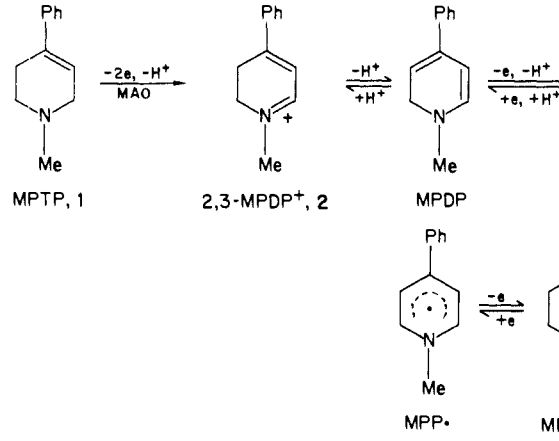
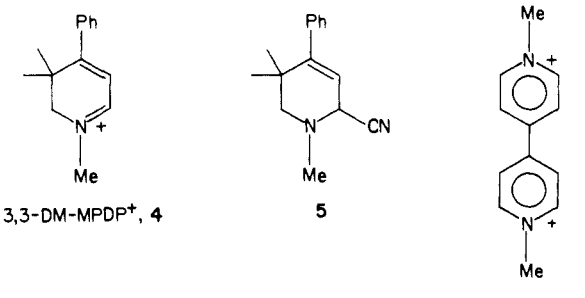
The discovery that 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP, **1**), present as an impurity in an illicit narcotic preparation, produced an irreversible Parkinson's Disease (PD) syndrome in man[1] has led to vigorous research aimed at elucidating the molecular mechanisms responsible for this effect.[2-6] As in PD, MPTP neurotoxicity is associated with the selective destruction of the substantia nigra, a pigmented (neuromelanin) dopaminergic cell group at the base of the brain. The finding that monoamine oxidase B (MAO-B) inhibitors protect against this effect[7] suggests that oxidized metabolites of MPTP are responsible for neurotoxicity. MPTP is oxidized by MAO-B in the brain to 1-methyl-4-phenyl-2,3-dihydropyridinium (2,3-MPDP$^+$, **2**),[8] which is ultimately converted to 1-methyl-4-phenylpyridinium (MPP$^+$, **3**), the major metabolite detected in brain tissue (eq 1).[9] Although



(1)

MPP$^+$ has recently been shown to be a potent cytotoxin in its own right,[10] it is not definitively known if it is entirely responsible for MPTP neurotoxicity. It is also conceivable that 2,3-MPDP$^+$ is neurotoxic, perhaps via a catalytic redox cycle in which it oxidizes dopamine to cytotoxic dopamine quinone and then is regenerated by MAO,[8] or through its action as an alkylating[11] and/or oxidizing agent toward biological nucleophiles (e.g., thiol enzymes). A direct assessment of the neurotoxicity of 2,3-MPDP$^+$ is complicated by its rapid oxidation in biological tissue to MPP$^+$,[12] and an evaluation of its potential physiologically relevant chemistry is hampered by its redox disproportionation at pH 7 to equal amounts of MPTP and MPP$^+$.[8,13] For these reasons we synthesized the geminal 3,3-dimethyl analogue of 2,3-MPDP$^+$ (3,3-DM-MPDP$^+$, **4**),[14]



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