of $J-V$ curves of Figure 2 at 0 V or from the ohmic $J-V$ curves of thicker LB membranes was found to be less than 10^{-14} S/cm. The breakdown voltage of the MIM cell of monolayer PDPF was ca. 8 V, i.e., 10^{10} V/m. Compared with the similar MIM device of [Al/fatty acid monolayer/Hg] by Kuhn et al.,[7] our PDF systems gave much better results. Note that, in the fatty acid system, (1) the oxide insulator layer on the substrate surface played an important role[8] in preventing short circuit (the fatty acid monolayer membrane was an imperfect tunneling barrier unless the metallic substrate plates covered with insulating oxides, such as Al-Al_2O_3, Si-SiO_2, and so on, were used), and (2) liquid mercury was used as the counter electrode in order to minimize the damage to the LB membrane.[7] It was found that our polymer LB membrane could be prepared even on Nesa glass, semiconductors, derivative semiconductors, organic semiconductors, or fresh metal surfaces including Pt, Au, etc. Furthermore, due to the thermally stable properties of the PDF LB membranes, it was possible to use vacuum-deposited counter electrodes. In the example shown in Figure 2, the aluminum layer was vacuum-deposited within 5 s, during which the temperature of the LB membrane surface was elevated to ca. 100 °C. Such high stability of PDF LB membranes may have wide applications to MIM or MIS (metal/insulator/semiconductor) devices.

Registry No. PDPF, 39050-69-6; PDBF, 41700-07-6; PDHF, 101490-50-0.

(7) Mann, G.; Kuhn, H. *J. Appl. Phys.* **1971**, *42*, 4398.
(8) Mann, G.; Kuhn, H.; Szentpaly, L. V. *Chem. Phys. Lett.* **1971**, *8*, 82.

Why Does Nature Not Use the Porphyrin Ligand in Vitamin B_{12}?

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It is now widely recognized that coenzyme B_{12}-dependent rearrangements are initiated by homolytic dissociation of the Co-C bond to generate a 5′-deoxyadenosyl radical.[1,2] It has been suggested that this dissociation is triggered by a steric perturbation involving an enzyme-induced conformational distortion of the corrin ring toward the 5′-deoxyadenosyl group, thereby weakening the cobalt-carbon bond.[1,3-6] Indeed, the X-ray analysis of coenzyme B_{12} reveals a crowded structure with several close contacts between atoms of the 5′-deoxyadenosyl group and atoms in the corrin ring and its substituents.[3] Furthermore, structural studies of different corrinoid complexes reveal highly puckered and variable conformations of the corrin ring attesting to its flexibility.[3]

Previously, we have cited parallels between the roles of hemes as reversible O_2 carriers and the role of coenzyme B_{12} as a reversible "free radical carrier".[1] Indeed, many aspects of the chemistry of organocobalt porphyrin complexes, including the range of Co-C bond dissociation energies (BDE's) reported below, closely parallel those of coenzyme B_{12} and related cobalamins. Why, then, does nature use the corrin ligand specifically in vitamin B_{12} and its coenzymes, rather than the porphyrin ligand which is used in so many other biological contexts? In this paper we report results of studies that bear on this theme.

Table I. Summary of Kinetic Data

chelate	PR_3	T, °C	10^4k_1, s^{-1}	$\Delta H_1^{\ddagger}$, kcal/mol	$\Delta S_1^{\ddagger}$, cal/(mol K)	D_{Co-R}, kcal/mol
$(DH)_2$	PMe_2Ph	66.0	0.20	32.4	15	30.4
		71.8	0.43			
		82.0	2.2			
		91.0	6.2			
		100.0	14			
	P-*n*-Bu_3	62.0	0.58	30.9	13	28.9
		73.6	2.8			
		87.0	20			
		92.0	22			
		100	69			
	$PEtPh_2$	49.6	0.26	28.8	10	26.8
		66.2	2.4			
		71.8	5.1			
		82.3	18			
	PPh_3	47.0	0.16	27.8	6	25.8
		57.0	0.69			
		67.0	2.5			
		75.0	5.8			
	P(*c*-C_6H_{11})$_3$	10.0	0.07	24.8	6	22.8
		15.0	0.12			
		23.2	0.55			
		28.4	1.0			
		35.0	3.7			
		45.6	10			
OEP	PMe_2Ph	55.0	0.096	29.1	7	27.1
		65.0	0.31			
		71.5	0.60			
		89.0	6.6			
		90.4	7.7			
	P-*n*-Bu_3	65.0	0.031	31.3	7	29.3
		75.0	0.088			
		85.0	0.40			
		90.4	0.80			
		100	2.4			
	$PEtPh_2$	55.0	0.20	28.1	6	26.1
		72.8	2.0			
		85.0	8.2			
		90.4	15			
	PPh_3	50.4	0.46	25.8	2	23.8
		55.4	0.92			
		60.4	1.6			
		70.4	5.2			
	P(*c*-C_6H_{11})$_3$	72.8	0.018	31.6	6	29.6
		82.8	0.069			
		85.0	0.088			
		91.2	0.194			

One possible rationale that occurred to us is that porphyrins might be unsuitable ligands for coenzyme B_{12} because they are insufficiently flexible, compared with corrins, to sterically modulate the Co-C BDE. Unfortunately, it is not possible to make direct comparisons of the steric and electronic influences of axial ligand variation for organocobalt porphyrin complexes with the corresponding corrin complexes because of the limited tendency of the latter to bind axial ligands. Accordingly, we have compared the influence on the Co-C BDE of varying the electronic and steric properties of the axial ligand (L) in a series of benzylcobalt octaethylporphyrin complexes [$PhCH_2$-Co(OEP)L] and in a corresponding series of benzylcobalt complexes of another flexible equatorial ligand, dimethylglyoxime (DH_2). For the latter family of complexes, [R-Co$(DH)_2$L], it has been shown that increasing the size of the axial ligand L does induce, in a manner analogous to that proposed for coenzyme B_{12}, Co-C bond lengthening and weakening due to conformational distortion of the equatorial $(DH)_2$ ligand away from L and toward the R group.[7]

(1) Halpern, J. *Science (Washington, D.C.)* **1985**, *227*, 869 and references therein.
(2) (a) Babior, B. M. *Acc. Chem. Res.* **1975**, *8*, 376. (b) Abeles, R. H. In *Vitamin* B_{12}; Zagalak, B., Friedrich, W., Eds.; de Gruyter: Berlin, 1979; p 373. (c) Golding, B. T. In B_{12}; Dolphin, D., Ed.; Wiley: New York, 1982; Vol. 2, p 543.
(3) Glusker, J. In B_{12}; Dolphin, D., Ed.; Wiley: New York, 1982; Vol. 1, p 23 and references therein.
(4) Chemaly, S. M.; Pratt, J. J. *J. Chem. Soc., Dalton Trans.* **1980**, 2259, 2267, 2274.
(5) Marzilli, L. G.; Toscano, P. J.; Randaccio, L.; Bresciani-Pahor, N.; Calligaris, M. *J. Am. Chem. Soc.* **1979**, *101*, 6754.
(6) (a) Babior, B. M. In B_{12}; Dolphin, D., Ed.; Wiley: New York, 1982; Vol. 2, p 263. (b) Babior, B M. In *Vitamin* B_{12}; Zagalak, B., Friedrich, W., Eds.; de Gruyter: Berlin, 1979; p 461.
(7) (a) Randaccio, L.; Bresciani-Pahor, N.; Toscano, P. J.; Marzilli, L. G. *J. Am. Chem. Soc.* **1981**, *103*, 6347. (b) Bresciani-Pahor, N.; Calligaris, M.; Nardin, G.; Randaccio, L. *J. Chem. Soc., Dalton Trans.* **1982**, 2549. (c) Summers, M. F.; Toscano, P. J.; Bresciani-Pahor, N.; Nardin, G.; Randaccio, L.; Marzilli, L. G. *J. Am. Chem. Soc.* **1983**, *105*, 6250 and references therein.

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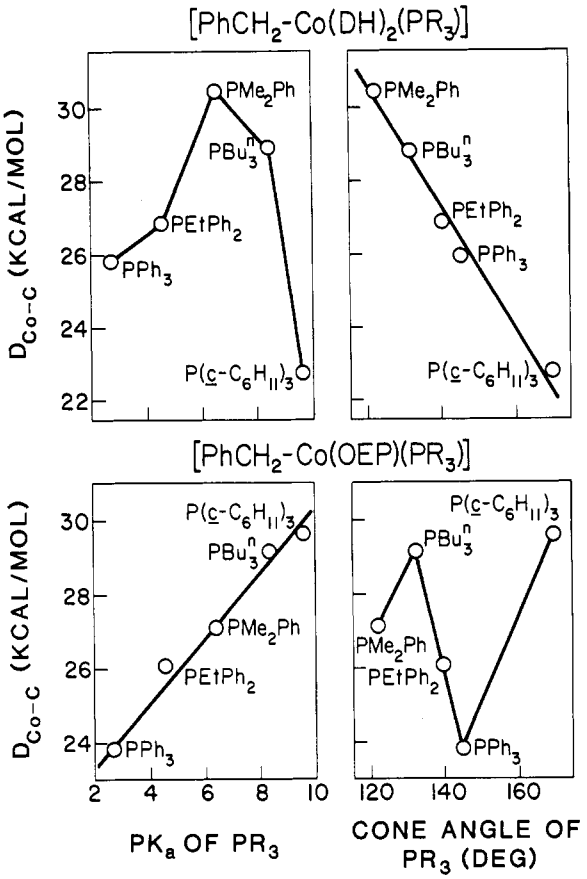

Figure 1. Dependence of D_{Co-C} on the pK_a and cone angle of PR_3.

Our studies encompass determination of the Co–CH_2Ph BDE's (D_{Co-C}) of a series of [$PhCH_2$–Co(OEP)(PR_3)][8] and [$PhCH_2$–$Co(DH)_2(PR_3)$][9] complexes in which the pK_a and size (as expressed by the cone angle)[11] of PR_3 were varied over a considerable range. The BDE's were determined by the kinetic method that we described earlier,[12] using 2,2,6,6-tetramethylpiperidineoxy (TEMPO)[13] as the free radical trap. In the presence of TEMPO, the cobalt complexes reacted cleanly according to the stoichiometry of eq 3 and the rate law (eq 4), derived for the mechanism depicted by eq 1 and 2 (where "chelate^{2-}" = OEP^{2-} or $(DH^-)_2$). The results of our kinetic measurements are summarized in Table I.[14]

$$[PhCH_2\text{–}Co^{III}(chelate)(PR_3)] \underset{k_{-1}}{\overset{k_1}{\rightleftharpoons}} PhCH_2^{\bullet} + [Co^{II}(chelate)(PR_3)] \quad (1)$$

$$PhCH_2^{\bullet} + TEMPO \xrightarrow{k_2} PhCH_2\text{–}TEMPO \quad (2)$$

$$[PhCH_2\text{–}Co^{III}(chelate)(PR_3)] + TEMPO \rightarrow PhCH_2\text{–}TEMPO + [Co^{II}(chelate)(PR_3)] \quad (3)$$

$$-d \ln [PhCH_2\text{–}Co^{III}(chelate)(PR_3)]/dt = k_{obsd} = \frac{k_1k_2[TEMPO]}{k_{-1}[Co^{II}(chelate)(PR_3)] + k_2[TEMPO]} \quad (4)$$

Earlier studies have demonstrated that recombination of organic radicals with cobalt(II) complexes typically is diffusion controlled.[12,16,17] Accordingly, D_{Co-R} can be deduced from the kinetic measurements by subtracting ~2 kcal (the estimated diffusion-controlled activation enthalpy, $\Delta H_{-1}^{\ddagger}$) from $\Delta H_1^{\ddagger}$ (Table I).

Our earlier studies have shown that for a series of [Ph-(CH_3)CH–$Co(DH)_2$L] complexes, where L is pyridine or a para-substituted pyridine (hence of constant steric influence), the Co–C BDE increases systematically with the pK_a of L, consistent with the formal reduction of Co^{III} to Co^{II} during homolysis of the Co–C bond (eq 1).[16] On the other hand, when L is a tertiary phosphine, the Co–C BDE exhibits a marked inverse dependence on the size (cone angle) of L, which masks the influence of varying basicity.[18] The latter trend is consistent with results of structural studies on [R–$Co(DH)_2$L] complexes which reveal, with increasing size of L, a bending away of the flexible $(DH)_2$ ligand from L and toward R, resulting in lengthening of the Co–C bond.[7]

Our results on the [$PhCH_2$–$Co(DH)_2(PR_3)$] complexes, plotted in Figure 1, reveal a similar trend, i.e., a marked inverse dependence of D_{Co-R} on the cone angle of PR_3. On the other hand, the data for [$PhCH_2$–Co(OEP)(PR_3)] reveal a systematic dependence of D_{Co-R} on the pK_a of PR_3 (similar to that previously found for the sterically constant para-substituted pyridines),[16] unperturbed by the substantial variations in the size of PR_3 (Figure 1). We conclude that the porphyrin ligand is not sufficiently flexible to respond to the steric pressures of bulky axial ligands and bend toward the $PhCH_2$ group to weaken the Co–C bond. Thus, the porphyrin appears to act effectively as a rigid "barrier", shielding the $PhCH_2$–Co bond from steric perturbations.

If, indeed, conformational deflection of the corrin toward the 5′-deoxyadenosyl group contributes significantly to the mechanism of enzyme-induced Co–C bond weakening and dissociation in coenzyme B_{12}, our results suggest that the porphyrin ligand is insufficiently flexible to play this role.

We call attention to other possible interpretations of the reasons for the choice of the corrin ligand in vitamin B_{12}, for example, that corrins may have preceded porphyrins in the evolutionary time scale.[19]

Acknowledgment. A generous gift of octaethylporphyrin from Professor David Dolphin and a grant (AM 13339) from the National Institutes of Health are gratefully acknowledged. The NMR facilities used in this research were supported in part through the University of Chicago Cancer Center Grant NIH-CA-14599.

Registry No. [$PhCH_2$-$Co(DH)_2(PMe_2Ph)$], 106095-14-1; [$PhCH_2$-$Co(DH)_2$(P-*n*-Bu_3)], 55886-61-8; [$PhCH_2$-$Co(DH)_2(PEtPh_2)$], 106114-

(8) The porphyrin adducts were synthesized by dissolving solid [$C_6H_5CH_2$Co(OEP)] in deoxygenated benzene and adding an excess of the phosphine ligand followed by precipitation with ethanol/water. The solid products were collected, filtered under nitrogen in the dark, recrystallized from CH_2Cl_2/CH_3OH (yields 81–94%), and characterized by ^1H NMR and UV–visible spectrophotometry and (satisfactory) C, H, and N analysis.

(9) [$C_6H_5CH_2Co(DH)_2(PR_3)$] complexes were synthesized according to Schrauzer and Windgassen[10] and characterized by ^1H NMR and UV–visible spectrophotometry.

(10) Schrauzer, G. N.; Windgassen, R. J. *J. Am. Chem. Soc.* **1967**, *89*, 1999.
(11) Tolman, C. A. *Chem. Rev.* **1977**, *77*, 3131.
(12) Tsou, T. T.; Loots, M.; Halpern, J. *J. Am. Chem. Soc.* **1982**, *104*, 623.
(13) (a) Finke, R. G.; Smith, B. L.; Mayer, B. J.; Molinero, A. A. *Inorg. Chem.* **1983**, *22*, 3677. (b) Collman, J. P.; McElwee-White, L.; Brothers, P. J.; Rose, E. *J. Am. Chem. Soc.* **1986**, *108*, 1332.

(14) The kinetic measurements were made in toluene solution. Initial [$PhCH_2$Co(chelate)L] concentrations were in the range 1.0×10^{-5} to 2.5×10^{-5} M with sufficient added L (typically ca. 0.1 M) so that dissociation of the latter was negligible. The reactions were monitored spectrophotometrically (at 455 and 350 nm for the OEP complexes and at ca. 350 nm for the $(DH)_2$ complexes). Schlenk cuvettes (filled with reaction solution under nitrogen) were immersed in a thermostated oil bath, withdrawn periodically, quenched by cooling to 25 °C, and subjected to spectral measurement. For two representative complexes, [$PhCH_2$Co(OEP)($PMePh_2$)] and [$PhCH_2Co(DH)_2$-(PPh_3)], the full dependence of the rate on the Co^{II} and TEMPO concentrations was determined and found to be in excellent agreement with eq 4. For the other complexes, only the limiting rates with excess TEMPO (typically 6×10^{-3} to 1.5×10^{-2}, with no added Co^{II}), i.e., in the region where $k_{obsd} = k_1$ (independent of the TEMPO concentration), were determined. The reactions were followed and exhibited good pseudo-first-order kinetics for at least two half-lives. The formation of the $C_6H_5CH_2$–TEMPO product was confirmed gas chromatography by (silica gel capillary column) separation (typically 86–93% yield) and comparison by 500-MHz ^1H NMR with an independently synthesized authentic product.[15]

(15) Hill, C. L.; Whitesides, G. M. *J. Am. Chem. Soc.* **1974**, *96*, 870.
(16) Ng, F. T. T.; Rempel, G. L.; Halpern, J. *J. Am. Chem. Soc.* **1984**, *104*, 621.
(17) Halpern, J. *Acc. Chem. Res.* **1982**, *15*, 238 and references therein.
(18) Ng, F. T. T.; Rempel, G. L.; Halpern, J. *Inorg. Chim. Acta* **1983**, *77*, L65.
(19) (a) Decker, K.; Jungermann, K.; Thauer, R. K. *Angew. Chem., Int. Ed. Engl.* **1970**, *9*, 138. (b) Eschenmoser, A. *Chem. Soc. Rev.* **1976**, *5*, 377.

23-2; [$PhCH_2$-Co$(DH)_2(PPh_3)$], 15977-36-3; [$PhCH_2$-Eco$(DH)_2$(P(c-$C_6H_{11})_3$)], 106095-15-2; [$PhCH_2$-Co(OEP)(PMe_2Ph)], 106095-16-3; [$PhCH_2$-Co(OEP)(P-n-Bu_3)], 106095-17-4; [$PhCH_2$-Co(OEP)-($PEtPh_2$)], 106095-18-5; [$PhCH_2$-Co(OEP)(PPh_3)], 106095-19-6; [$PhCH_2$-Co(OEP)(P(c-$C_6H_{11})_3$)], 106095-20-9; [$C_6H_5CH_2$Co(OEP)], 106095-21-0; PMe_2Ph, 672-66-2; P-n-Bu_3, 998-40-3; $PEtPh_2$, 607-01-2; PPh_3, 603-35-0; P(c-$C_6H_{11})_3$, 2622-14-2; vitamin B_{12}, 68-19-9; benzene, 71-43-2.

Evidence for Diazenyl Diradicals in the Photoisomerization of 4-Methylene-3,3,5,5-tetramethylpyrazoline

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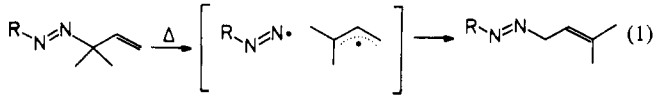
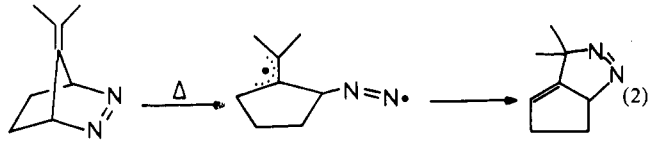
The mechanistic question whether extrusion of nitrogen from azo compounds proceeds concertedly or stepwise was recently[1] convincingly answered in the thermolysis of the unsymmetrically substituted acyclic azo compounds (eq 1). The fact that an



(1)

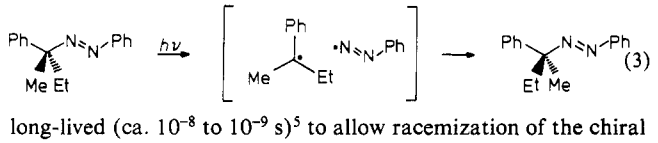
isomerized azo compound is formed, besides the usual denitrogenated products, implies rupture of the weaker C-N bond, leading to the caged diazenyl radical and allyl radical pair followed by recombination at the other allyl radical terminal prior to nitrogen loss from the diazenyl radical. Moreover, also for symmetrical bicyclic azo compounds, such thermal isomerizations have been documented[2] to proceed via diazenyl diradicals (eq 2). Theoretical



(2)

work substantiates[3] this mechanistic course, suggesting an appreciable activation energy (ca. 6-10 kcal/mol) for the loss of nitrogen from the diazenyl radical.

Experimental evidence indicates also for the photochemical process that on n,π^*-excitation photoracemization takes place (eq 3).[4] Again, the intermediary diazenyl radical is sufficiently

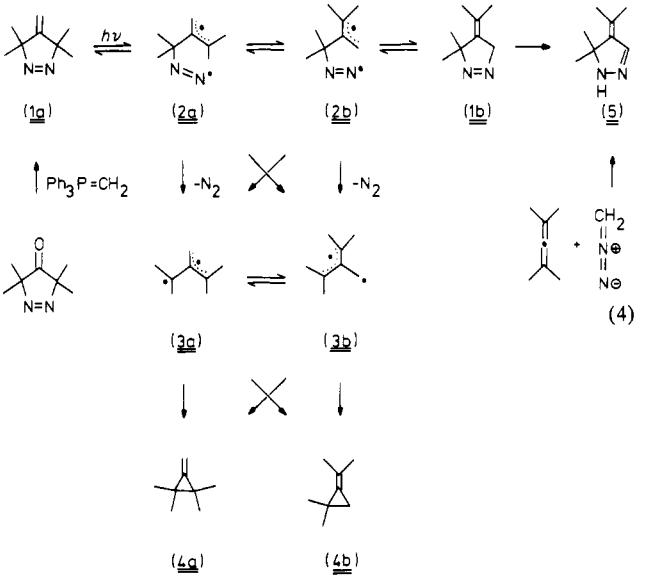


(3)

long-lived (ca. 10^{-8} to 10^{-9} s)[5] to allow racemization of the chiral alkyl radical, followed by cage recombination prior to nitrogen elimination. This is not surprising, since theoretical work shows,[6] with the parent diazene as model, that on n,π^*-excitation a π-type diazenyl radical is formed which should be reluctant toward denitrogenation. In this context, it is significant to mention that the bicyclic azo compounds in eq 2 did not isomerize during photolysis.[2]

In our recent publications,[7] we have proposed that diazenyl diradicals intervene quite generally in the photochemical denitrogenation of cyclic azo compounds. However, rigorous experimental proof is still lacking. Consequently, we decided to probe this mechanistic query by choosing an azo substrate capable of photoisomerization. Fortunately, the previously studied[8] 4-methylene-3,3,5,5-tetramethylpyrazoline (**1a**) proved useful for our purposes, and presently we communicate our results.

Azo compound **1a** was prepared in 75% yield from its pyrazolone by Wittig reaction with methylenetriphenylphosphorane.[8] Direct photolysis (ca. 0.7 M solution in *n*-pentane under a nitrogen atmosphere) at 350 nm and 20 °C afforded the methylenecyclopropanes **4a** and **4b** (eq 4) in the previously observed[8] relative



(4)

proportions of 75:25 (capillary GC on a 50-m OV-101 column, operated at column, injector, and detector temperatures of 80, 150, and 200 °C, respectively, and a carrier gas pressure of 1.0 kg/cm^2). However, careful examination of the photolyzate by means of capillary GC revealed a trace component (ca. 0.5%) of lower volatility, presumably still containing nitrogen.

This fact was indeed confirmed by means of capillary GC/MS, affording the expected *m/e* value of 138. Moreover, its fragmentation pattern was significantly different from the starting pyrazoline **1a**. That this product was not **1a** could readily be confirmed by means of capillary GC comparison with the authentic material.

Suspecting that the isomerized azo compound **1b** had been formed in the photolysis of **1a** (eq 4), we attempted to prepare an authentic sample. Cycloaddition of diazomethane and tetramethylallene in ether at 0 °C in an autoclave for 4 weeks gave

† Fonds der Chemischen Industrie doctoral fellow 1984-1986.

(1) Engel, P. S.; Gerth, D. B. *J. Am. Chem. Soc.* **1983**, *105*, 6849.
(2) (a) Cichra, D. A.; Duncan, C. D.; Berson, J. A. *J. Am. Chem. Soc.* **1980**, *102*, 6527. (b) Berson, J. A. *Acc. Chem. Res.* **1978**, *11*, 446. (c) Cichra, D. A.; Platz, M. S.; Berson, J. A. *J. Am. Chem. Soc.* **1977**, *99*, 8507. (d) Berson, J. A. In *Diradicals*; Borden, W. T., Ed.; Wiley: New York, 1982.
(3) (a) Dannenberg, J. J.; Rocklin, D. *J. Org. Chem.* **1982**, *47*, 4529. (b) Hiberty, P. C.; Jean, Y. *J. Am. Chem. Soc.* **1979**, *101*, 2538.
(4) Porter, N. A.; Landis, M. E.; Marnett, L. J. *J. Am. Chem. Soc.* **1971**, *93*, 795.
(5) Bartlett, P. D.; Pincock, R. E.; Rolston, J. H.; Schindel, W. G.; Singer, L. A. *J. Am. Chem. Soc.* **1965**, *87*, 2590.
(6) Bigot, B.; Sevin, A.; Devaquet, A. *J. Am. Chem. Soc.* **1978**, *100*, 2639.
(7) (a) Adam, W.; Gillaspey, W. D.; Peters, E.-M.; Peters, K.; Rosenthal, R. J.; von Schnering, H. G. *J. Org. Chem.* **1985**, *50*, 580. (b) Adam, W.; Dörr, M.; Hill, K.; Peters, E.-M.; Peters, K.; von Schnering, H. G. *J. Org. Chem.* **1985**, *50*, 587. (c) Adam, W.; Oppenländer, T.; Zang, G. *J. Org. Chem.* **1985**, *50*, 3303. (d) Adam, W.; Hill, K. *J. Am. Chem. Soc.* **1985**, *107*, 3686.
(8) (a) Crawford, R. J.; Tokunaga, H. *Can. J. Chem.* **1974**, *52*, 4033. (b) Crawford, R. J.; Tokunaga, H.; Schrijver, L. M. H. C.; Godard, J. C. *Can. J. Chem.* **1978**, *56*, 998.
(9) Pale yellow wax, sublimated: mp 35-40 °C; IR (CCl_4) 3420, 3020, 3000, 2970, 1610, 1550, 1230, 1000, 980, 970 cm^{-1}; ^{1}H NMR ($CDCl_3$, 90 MHz) δ 1.11 (s, 6 H, N-C(CH_3)$_2$), 1.26 (s, 1 H, NH), 2.12 (s, 3 H, =C-(CH_3)$_2$), 2.21 (s, 3 H, =C(CH_3)$_2$), 7.80 (s, 1 H, N=CH); ^{13}C NMR ($CDCl_3$, 100 MHz) δ 16.46 (q, =C(CH_3)$_2$), 20.07, (q, =C(CH_3)$_2$), 27.58 (q, N-C-(CH_3)$_2$), 51.55 (s, C-5), 123.10 (s, C=C), 131.75 (s, C=C), 167.49 (d, C-3); MS (70 eV), *m/e* 138 (18%, M$^+$), 95 (7%), 82 (100%), 67 (6%), 55 (6%), 54 (16%), 53 (7%), 41 (9%), 39 (17%). Anal. Calcd for $C_8H_{14}N_2$ (138.2): C, 69.52; H, 10.21; N, 20.28. Found: C, 69.51; H, 10.02; N, 20.46.

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