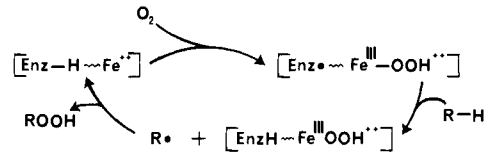

Figure 1.

in the eluent and 65 dpm remained on the C_{18} column (total recovery of radioactivity 99.5%). The radioactivity remaining on the column corresponds to 0.122 nmol of tritiated 14,15-DHA, that is, 0.87 molecule of 14,15-DHA per molecule of enzyme. It was demonstrated in a separate experiment that the lipoxygenase enzyme itself is not eluted from a C_{18} column under these conditions whereas labeled 14,15-DHA is. In a parallel experiment with 15-LO(SB) and tritiated 11,12-DHA, conducted exactly as described above for the enzyme and labeled 14,15-DHA, it was shown that by starting with 13 980 dpm of 11,12-DHA, 12 850 dpm were recovered by extraction and 718 dpm by elution of the C_{18} column, with essentially no radioactivity (0–10 dpm) remaining on the column. Taken together these results show that ca. 1 molecule of 14,15-DHA becomes tightly bound to each molecule of deactivated 15-LO(SB) enzyme, whereas the isomeric 11,12-DHA is neither a strong irreversible inhibitor nor tightly bound. Since covalent binding of 14,15-DHA to deactivated 15-LO(SB) seems a logical possibility, degradative studies on the inactivated enzyme are in order.

Paralleling the findings reported above on the deactivation of soybean lipoxygenase by 14,15-DHA are the results presented in the following communication, which demonstrate a similar irreversible inactivation of arachidonate cyclooxygenase by 11,12-DHA. Further, preliminary data indicate irreversible blockage of the 5-lipoxygenase (leukotriene) pathway by 5,6-DHA. Thus it appears that the presence of a single acetylenic unit in place of a *Z*-ethylenic unit at a site of lipoxygenation of arachidonic acid leads to potent and irreversible inhibition of the corresponding lipoxygenase pathway.

Despite the heuristic value of the hypothesis leading to the discovery of DHA's as lipoxygenase inhibitors, the mechanism by which enzyme deactivation occurs still remains to be established, as in fact is true also for the mechanism of the lipoxygenase reaction itself. Present thinking favors the intermediacy of pentadienyl free radicals[15] and a catalytic scheme of the general type shown in Figure 1,[16,17] in which RH is the polyunsaturated fatty acid and [Enz–H⋯Fe^{2+}] is the lipoxygenase enzyme. Autodestruction of the lipoxygenase enzyme during catalysis could be the result of any of the following: (1) reaction of Enz· with oxygen, ROOH, R·, or radicals produced from ROOH; (2) reaction of Enz–H at one or more vulnerable sites other than the group holding the explicit donor H with oxygen, ROOH, R·, or radicals produced from ROOH. Radical formation from ROOH might be promoted by the enzyme itself (e.g., at the Fe^{2+} site) or might be spontaneous. Clearly 14,15-DHA may function by masquerading as arachidonic acid and by enhancing one or more of these catalytically destructive processes.[18]

Registry No. 1, 80738-21-2; 5,6-DHA, 58688-54-3; 8,9-DHA, 80738-22-3; 11,12-DHA, 80738-23-4; 15-HPETE, 69371-38-6; arachidonic acid, 506-32-1; 14,15-oxidoarachidonic acid, 74868-37-4; methyl 14-bromo-15-hydroxy-5,8,11-eicosatrienoate, 80738-24-5; methyl 14-hydroxy-15-bromo-5,8,11-eicosatrienato, 80738-25-6; methyl (*Z*,*Z*,*Z*)-14-bromo-15-keto-5,8,11-eicosatrienoato, 80738-26-7; methyl (*Z*,*Z*,*Z*)-15-bromo-14-keto-5,8,11-eicosatrienoate, 80738-27-8; methyl 14,15-dehydroarachidonate, 80738-28-9.

(15) DeGroot, J. J. M. C.; Garssen, G. J.; Vliegenthart, J. F. G.; Boldingh, J. *Biochim. Biophys. Acta* **1973**, *326*, 279.
(16) DeGroot, J. J. M. C.; Veldink, G. A.; Vliegenthart, J. F. G.; Boldingh, J.; Wever, R.; van Gelder, B. F. *Biochim. Biophys. Acta* **1975**, *377*, 71.
(17) "Plant Lipid Biochemistry"; Hitchcock, C., Nichols, B. W., Eds.; Academic Press: New York, 1971; p 226.
(18) This research was assisted financially by grants from the National Science Foundation and the National Institutes of Health. We are indebted to Dr. Jahyo Kang and John Munroe for providing tritiated 11,12- and 14,15-DHA.

Irreversible Inhibition of Prostaglandin and Leukotriene Biosynthesis from Arachidonic Acid by 11,12-Dehydro- and 5,6-Dehydroarachidonic Acids, Respectively

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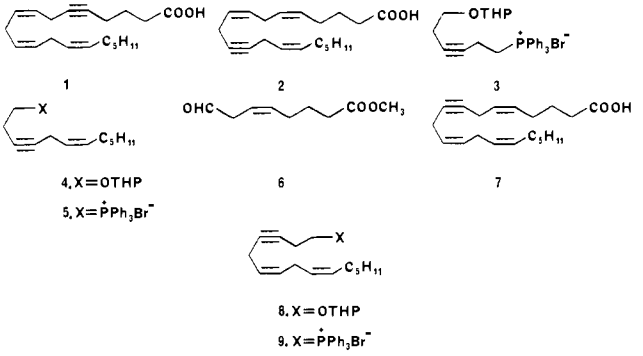
Four oxidative pathways are now known for the biosynthesis of physiologically active eicosanoids from arachidonic acid, each commencing with an enzymic lipoxygenase (LO) type reaction.[1] The 5-LO pathway leading to leukotrienes and the 11-LO pathway leading to prostaglandins (PG's) and thromboxanes are of special interest currently because of their relevance to a number of disease states.[1] Effective and selective inhibitors of specific LO pathways could be of service in unraveling the biological effects of the numerous naturally occurring eicosanoids. Nonsteroidal antiinflammatory agents such as aspirin inhibit the 11-LO (cyclooxygenase, CO) pathway only. In the preceding communication[2] a rationale was outlined for the use of monoacetylenic, dehydroarachidonic acids as position-selective lipoxygenase inhibitors and evidence was presented that the arachidonic 15-LO from soybean is subject to potent and irreversible inhibition by 14,15-dehydroarachidonic acid (14,15-DHA) but not by 5,6-, 8,9-, or 11,12-dehydroarachidonic acids. Described herein are the effects of these DHA's on prostaglandin biosynthesis and also initial studies on the inhibition of leukotriene biosynthesis by 5,6-DHA.

Synthesis of 5,6-, 8,9-, 11,12-, and 14,15-DHA's. 5,6-DHA (**1**)



was available from arachidonic acid as recently described[3] and also by an independent total synthesis.[4] 14,15-DHA was prepared as described in the foregoing paper.[2] 8,9- and 11,12-DHA's were made by short and efficient total syntheses utilizing as a common intermediate the readily available acetylenic phosphonium bromide **3**.[5] For the synthesis of 11,12-DHA (**2**) the ylide derived from **3** (1 equiv of *n*-BuLi in THF[6] at −78 to −30 °C, for 30 min) in 9:1 THF–HMPT at −78 °C was treated with hexanal (0.9 equiv) to afford after reaction at −78 °C for 30 min and at −78 to 0 °C

(1) For reviews see: (a) Bailey, D. M.; Chakrin, L. W. *Annu. Rep. Med. Chem.* **1981**, *16*, 213. (b) Schaaf, T. K. *Ibid.* **1977**, *12*, 182.
(2) Corey, E. J.; Park, H. *J. Am. Chem. Soc.* **1982**, *104*, 0000.
(3) Corey, E. J.; Park, H.; Barton, A.; Nii, Y. *Tetrahedron Lett.* **1980**, *21*, 4243.
(4) Corey, E. J.; Kang, J. *Tetrahedron Lett.*, in press. Credit is due to a referee for suggesting that this paper be published separately.
(5) The phosphonium bromide **3** was prepared from the known corresponding alcohol, the monotetrahydropyranyl ether of 3-hexyne-1,6-diol [Raphael, R. A.; Roxburgh, C. M. *J. Chem. Soc.* **1952**, 3875] by the following sequence: (1) reaction with 1.5 equiv of carbon tetrabromide, 1.1 equiv of pyridine, and 1.0 equiv of triphenylphosphine in methylene chloride (ca. 1 M in alcohol) at 23 °C for 2.5 h to form the bromide tetrahydropyranyl ether (90% isolated yield after nonaqueous isolation and passage through a column of silica gel); (2) reaction of the bromide with 1.5 equiv of triphenylphosphine in toluene (or acetonitrile where indicated) at reflux for 26 h and treatment of the resulting crude product with 0.003 equiv of *p*-toluenesulfonic acid and 1.1 equiv of dihydropyran in methylene chloride at 0 °C for 30 min (to replace any of the THP group lost in the previous step) to afford after nonaqueous workup the crystalline phosphonium salt **3** (93% overall yield), mp 111–115 °C.
(6) Chemical abbreviations: THF, tetrahydrofuran; HMPT, hexamethylphosphoric triamide.

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for 90 min, extractive isolation, and filtration through silica gel the eneyne **4** in 99% yield.[7] Conversion of **4** to the phosphonium salt **5** was effected in acetonitrile in 80% overall yield by deprotection and use of the method outlined below.[5] Condensation of the ylide from **5** (prepared in THF at −78 °C) with the *Z* unsaturated aldehyde **6**[8] under the above described Wittig cis-olefination conditions gave the methyl ester of **2** in 65% yield, and this was converted (100% yield) to **2** by treatment with 15 equiv of lithium hydroxide (0.5 M) in 1:1 THF–H_2O at 23 °C for 4 h.

For the synthesis of 8,9-DHA (**7**), the ylide from **3** was coupled under the Wittig cis-olefination conditions described above to 3(*Z*)-nonenal[9] to afford **8** in 57% yield. Conversion to the phosphonium salt **9** (in acetonitrile, method of ref 5, 85% overall yield) and coupling of the corresponding ylide in 9:1 THF–HMPT at −78 °C with methyl 4-formylbutyrate[8] (92% yield) produced the methyl ester of **7** which was saponified to **7** with 15 equiv of 0.5 M lithium hydroxide in 1:1 THF–H_2O at 23 °C for 4 h (96% yield).

11,12-DHA (**2**) and 8,9-DHA (**7**), synthesized as described immediately above, were identical with samples prepared by alternative synthetic routes described in ref 4. These acids were stored under argon in frozen benzene at −20 °C.

Inhibition of Prostaglandin Biosynthesis. PG biosynthesis was studied at 23 °C by using a magnetically stirred suspension of ram seminal vesicle microsomes (0.5 mg/mL) in 0.1 M Tris buffer (pH 8.3),[10] 1.0 mM in glutathione and 0.05 mM in hydroquinone, with arachidonic acid in the presence of air. The standard conditions for PG formation involved the use of 1 mL of microsomal suspension and 10 nmol of arachidonic acid (10 μM) and led to >90% conversion to PGE_2 as measured by treatment of an aliquot of product with 0.5 M potassium hydroxide in 3:1 methanol–water at 50 °C for 15 min, cooling, adjustment of pH to 7.0 with acetic acid, and analysis by reversed-phase high-performance liquid chromatography (RP HPLC) (Waters C_{18} μ-Bondapak column using 65:35:0.1 CH_3OH–H_2O–HOAc, buffered to pH 5.6 with ammonium hydroxide, for elution) for PGB_2 (λ_{max} 278 nm, e 27 000).[11] The rate of conversion of arachidonic acid to PGE_2 is nearly linear over the first 10 min, the interval of time over which the zero-order rate constants were calculated. That the enzyme is under saturating conditions was demonstrated by the finding that for a given reaction time the amount of product did not change with increasing arachidonate concentration.

The effects of the various DHA's were ascertained by incubation of the microsomal enzyme suspension at 23 °C with DHA for 30 min, then addition of arachidonate, and measurement of the rate of conversion to PGE_2. Deactivation was studied as a function of the concentration of DHA in the range of 0.1 to 10 μM with arachidonate at 10 μM, with comparison of the rate of PGE_2 formation to a concurrent control experiment without DHA. The most effective inhibitor of the PG-forming CO enzyme was found to be 11,12-DHA. By use of 11,12-DHA and a preincubation time of 30 min, the concentration of DHA required for 50% inhibition was found to be 0.25 μM. Under these same conditions 25% inhibition was observed at 0.10 μM and 87% inhibition was ascertained at 1.0 μM 11,12-DHA. At 0.25 μM 11,12-DHA but with only 3-min preincubation time 32% inhibition of PG synthesis was found.

In comparable experiments using 5,6-DHA at concentrations up to 1 μM and 30-min preincubation time, no inhibition of PG synthesis was observed. Some inhibition was observed at higher concentrations, but it appeared to be largely competitive in character. In fact, 5,6-DHA can serve as a substrate for the PG-synthetase system as is shown by the following results. Aerobic incubation of 110 nmol of 5,6-DHA with a suspension of 3 mg of ram seminal vesicle microsomes in 2.5 mL of a solution of 1.0 mM glutathione, 0.1 mM hydroquinone, and 0.1 M Tris buffer (pH 8.3) for 90 min at 23 °C, base treatment as above to effect PGE → PGB conversion, acidification to pH 6 with acetic acid, and separation of the mixture by RP HPLC (as above) afforded a UV-active product eluting at 3.3 column volumes as compared to 4.7 for PGB_2 under the same conditions. Concentration of the solution containing this UV-active product, acidification to pH 2.5, extraction with ether and esterification with diazomethane afforded in about 2% yield the 5,6-dehydro-PGB_2 methyl ester, λ_{max} 276 nm, purified by HPLC on a Waters μ-Porasil column (2.5% isopropyl alcohol in hexane; retention volume 14.7 vs. 11.3 for the PGB_2 methyl ester), which was further identified by the mass spectrum of the 15-trimethylsilyl derivative, which showed the expected M^+ (418) and fragmentation characteristic of the methyl ester of 15-(trimethylsilyl)-PGB_2. In summary, the evidence at hand suggests 5,6-DHA is a substrate (although a poor one) for the PG synthetase and that it is not a potent irreversible inhibitor as is 11,12-DHA.

8,9-DHA was also found not to be a potent irreversible inhibitor of PG synthesis; at ca. 50 times the concentration at which irreversible inhibition was clearly evident with 11,12-DHA, 8,9-DHA showed none. At higher concentrations appreciable inhibition could be detected. The nature of this inhibition (i.e., competitive substrate analogue type or some other sort) has not yet been determined.

14,15-DHA on the other hand was found to inhibit PG biosynthesis at concentrations of 0.25 μM (13% inhibition) and above and to show behavior paralleling that of 11,12-DHA. Since the 14,15 double bond becomes hydroperoxidized in PG biosynthesis, such behavior of the 14,15-acetylenic analogue of arachidonic acid is understandable on the basis of the hypothesis outlined in the preceding paper.[2] 11,12-DHA appears to be approximately 4 times more potent in the inhibition of PG biosynthesis than 14,15-DHA.

Inhibition of Leukotriene Biosynthesis. A study of inhibition of leukotriene biosynthesis by 5,6-DHA was also carried out. A homogenate of rat basophilic leukemia (RBL-1) cells was prepared at 0 °C in 50 mM phosphate buffer (pH 7.0) containing 1 mM ethylenediaminetetraacetic acid, 14 μM indomethacin, and 0.1% gelatin, and a crude supernatent fraction of enzyme was obtained by centrifugation at 10000*g* for 20 min.[12] With this preparation 5-LO activity was ascertained by aerobic incubation with 5 μM tritiated arachidonic acid at 30 °C, thin-layer chromatographic separation of 5-HETE, and measurement of radioactivity of the 5-HETE fraction.[13]

(7) Satisfactory infrared, proton magnetic resonance, and mass spectral data were obtained for each new compound described herein from a chromatographically purified and homogeneous sample. Unless otherwise noted products were colorless oils.

(8) The *Z* unsaturated aldehyde **6** was prepared by ketal cleavage (0.03 M HCl in CH_3OH, 4 °C, 16 h) of the acetonide of methyl *Z*-8,9-dihydroxynon-5-enoate followed by cleavage with $Pb(OAc)_4$ in CH_2Cl_2 at −20 °C for 15 min and −20 to 0 °C for 15 min and purified by rapid filtration through silica gel–Celite followed by azeotropic removal of traces of acetic acid with heptane at 20 mmHg (77% overall yield). The acetonide of methyl *Z*-8,9-dihydroxynon-5-enoate was generated from the ylide corresponding to 1-(triphenylphosphonio)butane-3,4-diol acetonide and methyl 4-formylbutyrate [Corey, E. J.; Niwa, H.; Knolle, J. *J. Am. Chem. Soc.* **1978**, *100*, 1942] in 9:1 THF–HMPT at −78 °C (70% yield).

(9) (*Z*)-3-Nonenal was conveniently prepared in 60% overall yield from dec-1-en-4-yne [Sucrow, W.; Klein, U. *Chem. Ber.* **1975**, *108*, 3518] by the following sequence: (1) catalytic olefinic hydroxylation [Van Rheenan, V.; Kelly, R. C.; Cha, D. Y. *Tetrahedron Lett.* **1976**, 1973]; (2) selective semihydrogenation of the triple bond using a Lindlar catalyst and 1 atm of hydrogen in THF containing 1% triethylamine, at 23 °C for 4.5 h; (3) lead tetraacetate cleavage of the resulting (*Z*)-dec-4-en-1,2-diol with 1.35 equiv of lead tetraacetate in CH_2Cl_2 as described above.[8]

(10) For microsomal preparation see: (a) Hamberg, M.; Samuelsson, B. *Proc. Natl. Acad. Sci. U.S.A.* **1973**, *70*, 899. (b) Hamberg, M.; Samuelsson, B. *J. Biol. Chem.* **1967**, *242*, 5344.

(11) It was demonstrated in control experiments that under these assay conditions PGE_2 is converted quantitatively to PGB_2. An internal standard of 4-phenylbenzoic acid was eluted at retention volume 2.7 under the above RP HPLC conditions, and PGB_2 was eluted at 4.7 retention volume. Biosynthetic PGB_2 from the enzymic studies was characterized by isolation, conversion to the methyl ester trimethylsilyl ether, and mass spectral analysis.

(12) The enzymic work on leukotriene biosynthesis was carried out in collaboration with Dr. T. J. Carty, Chas. Pfizer Co., Groton, Conn., and represents a preliminary examination. Definitive studies require further purification of the 5-LO enzyme. Further studies are underway which will be described in a more comprehensive joint paper.

(13) Jakschik, B. A.; Sun, F. F.; Lee, L. H.; Steinhoff, M. M. *Biochem. Biophys. Res. Commun.* **1980**, *95*, 103.

The ability of the crude 5-LO-containing homogenate to convert arachidonic acid to 5-HETE decreased in a time-dependent manner on aerobic preincubation of the homogenate with 5,6-DHA. For an experiment using preincubation with 0.5 μM 5,6-DHA at 10 °C, a plot of preincubation time vs. the log of the fraction of original activity remaining was linear, with the half-life being ca. 12 min. Similar measurements of 5,12-DIHETE formation showed falloff parallel to that for 5-HETE as a function of preincubation with 5,6-DHA. These results, even though obtained with a crude enzyme preparation, clearly indicate time-dependent, irreversible inhibition of the leukotriene pathway by 5,6-DHA. This initial study is being extended in several pertinent directions. It is interesting that 5,8,11,14-eicosatetraynoic acid has been found not to inhibit the conversion of arachidonate to 5-HETE in human leukocytes.[14,15]

Registry No. 1, 58688-54-3; **2**, 80738-23-4; **2** methyl ester, 80764-50-7; **3**, 80764-51-8; **4**, 80764-52-9; **5**, 80764-53-0; **6**, 80764-54-1; **7**, 80738-22-3; **7** methyl ester, 80764-55-2; **8**, 80764-56-3; **9**, 80764-57-4; 3 (*Z*)-nonenal, 31823-43-5; methyl 4-formylbutyrate, 6026-86-4; arachidonic acid, 506-32-1.

(14) Dr. Pierre Borgeat, unpublished results.

(15) This research was assisted by grants from the National Science Foundation and the National Institutes of Health. We thank Dr. Thomas J. Carty for enzymic data on the 5-LO pathway and for providing ram seminal microsomes.

Temperature Dependence of the Cycloaddition of Phenylchlorocarbene to Alkenes. Observation of "Negative Activation Energies"

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Chemists employ an extremely useful and effective model of activation energy as a barrier that must be overcome during the course of a reaction.[1] The barrier is usually positive in the energy sense, i.e., the energy of activation is higher than the energy of the reactants or the products. However, the activation energy, E_a, is merely a quantity associated with the slope of an experimental plot of log k_{obsd} vs. $1/T$ (Arrhenius plot). The magnitude of E_{obsd} may be positive, negative, or zero, depending on the complexity of the reaction being investigated.[2] When E_{obsd} is negative, a standard interpretation of the observation is available: the reaction under investigation is multistep and involves at least one intermediate that possesses at least two channels for reaction.[3-7]

Recently, small negative enthalpies of activation have been reported for the quenching of singlet fluorenylidene by several alkenes; the values were considered "statistically indistinguishable from zero".[8a] This report is one of the few dealing with measurements of absolute rate constants for carbene–alkene addition *in solution*. The others involve the additions to alkenes of diarylcarbenes,[8b] fluorenylidene,[8c] and phenylchlorocarbene (PhCCl).[9a] We have now determined activation parameters for the additions of PhCCl to several alkenes and report here results consistent with the occurrence of a dissociable intermediate in the carbene cycloaddition.

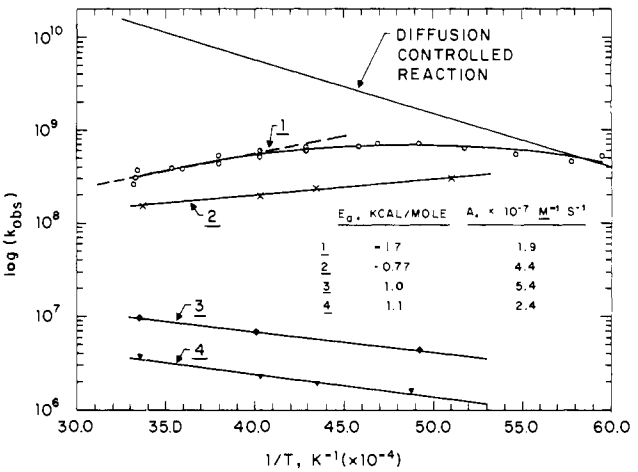

Figure 1. Arrhenius plot of the temperature dependence of the reaction of phenylchlorocarbene with olefins. Diffusion-controlled rate constants were calculated from the temperature dependence of the solvent viscosity.[11] The dashed line indicates results calculated between 301 and 248 K. Linear-regression coefficients for a second-order polynomial fit of all the data for **1** are $a_0 = 5.37$, $a_1 = 1436$, and $a_2 = -1.48 \times 10^5$.

The methods and system for the generation and monitoring of singlet PhCCl at various temperatures were similar to that described earlier.[9a] However, significant improvements were made to the laser flash photolysis apparatus previously employed to obtain the absolute rate constants for the addition of singlet PhCCl to olefins **1-4**.[9a] Specifically, we added a high-intensity pulsed

$Me_2C{=}CMe_2$ (**1**) $Me_2C{=}CHMe$ (**2**) *t*-MeCH=CHEt (**3**) $CH_2{=}CH$-*n*-Bu (**4**)

Xe lamp as the detection source, a newer, more sensitive monochromator, and a multiple-pulse delay unit to control the timing of the Xe lamp and the laser. Our new results, obtained with a different transient absorption system, differ significantly from the earlier results.[9a] The new rate constants are all higher by factors of 2–3. We attribute this to the systematic changes in the flash photolytic apparatus described above. As an independent check, Dr. J. C. Scaiano[9c] graciously determined k_{abs} for the addition of PhCCl to tetramethylethylene (**1**) on his laser flash system. The result, $k = 2.0 \times 10^8$ M^{-1} s^{-1}, is close to the average (2.3×10^8 M^{-1} s^{-1}) of our original[9a] and new (below) values. Clearly, the absolute rate constants are system dependent, and the accuracy of any given k_{abs} is low. However, it is the *precision* of the rate constant measurements that is important in examining temperature effects. In our case, the precision of the data points is ≤5%, and the reproducibility of the derived rate constants is ≤10%, so we can have reasonable confidence in the Arrhenius studies presented below.

(1) (a) Dewar, M. J. S.; Dougherty, R. C. "The PMO Theory of Organic Chemistry"; Prentice–Hall: Englewood Cliffs, NJ, 1975. (b) Michl, J. *Mol. Photochem.* **1972**, *4*, 253. (c) Michl, J. *Pure Appl. Chem.* **1975**, *41*, 507. (c) Salem, L.; Leforestier, C.; Segal, G.; Wetmore, R. *J. Am. Chem. Soc.* **1975**, *97*, 479. (d) Salem, L. *Ibid.* **1974**, *96*, 3486.

(2) Johnson, H.; Birks, J. *Acc. Chem. Res.* **1972**, *5*, 327.

(3) Benson, S. W. "The Foundations of Chemical Kinetics"; McGraw-Hill: New York, 1960; pp 308, 405.

(4) Kiselev, V. D.; Miller, T. G. *J. Am. Chem. Soc.* **1975**, *97*, 4036.

(5) Hui, M.-H.; Ware, W. R. *J. Am. Chem. Soc.* **1976**, *98*, 4718.

(6) (a) Gorman, A. A.; Lovering, G.; Rogers, M. A. J. *J. Am. Chem. Soc.* **1979**, *101*, 3050. (b) Gould, I. Ph.D. Dissertation, Manchester, England, 1981.

(7) Maharaj, U.; Winnik, M. A. *J. Am. Chem. Soc.* **1981**, *103*, 2328.

(8) (a) Zupanic, J. J.; Schuster, G. B. *J. Am. Chem. Soc.* **1981**, *103*, 944. See also: Wong, P. C.; Griller, D.; Scaiano, J. C. *Ibid.* **1981**, *103*, 5934. We thank Dr. Scaiano for communicating his results to us prior to publication. (b) Closs, G. L.; Rabinow, B. E. *Ibid.* **1976**, *98*, 8190. (c) Zupanic, J. J.; Schuster, G. B. *Ibid.* **1980**, *102*, 5958.

(9) (a) Turro, N. J.; Butcher, J. A.; Moss, R. A.; Guo, W.; Munjal, R. C.; Fedorynski, M. *J. Am. Chem. Soc.* **1980**, *102*, 7576. (b) The previous results[9a] were obtained with $\lambda_{ex} = 249$ nm, $\lambda_{obsd} = 320$ nm with a band-pass of 15 nm. Reexamination of the absorption spectrum of PhCCl in isooctane at room temperature showed a maximum at 308 nm (5-nm intervals, 4-nm band-pass), in excellent agreement with the spectrum recorded at 77 K in 3-methylpentane (See ref 9a). (c) National Research Council, Ottawa, Canada.

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