Additions and Corrections

Monophotonic Ionization of 7-Azaindole, Indole, and Their Derivatives and the Role of Overlapping Excited States [*J. Am. Chem. Soc.* **1994**, *116*, 735–746]. F. GAI, R. L. RICH, AND J. W. PETRICH*

Page 740: The variation of the fluorescence quantum yield as a function of excitation wavelength for indole and its derivatives depicted in panels a–c of Figure 5 is incorrect owing to an improper correction of the lamp intensity of our fluorimeter. There is no measurable variation of fluorescence quantum yield with excitation wavelength. Consequently these data cannot be used to argue that the ^1L_b state is the origin of the solvated electron whose appearance is complete within 1 ps.

C–H Bond Activation by Metal Oxo Species: Oxidation of Cyclohexane by Chromyl Chloride [*J. Am. Chem. Soc.* **1994**, *116*, 1855–1868]. GERALD K. COOK AND JAMES M. MAYER*

Page 1867: The calculation of $\Delta H°$ in Scheme 4 is in error. Since the normal hydrogen electrode (the second equation) relates $H^+(aq)$ with $H_2(g)$, the fourth equation should relate $H^•(aq)$ with $H_2(g)$ and the calculation of $\Delta G°$ for this line should not include the solvation of $H_2(g)$. The corrected values are shown in italics in the scheme below. The expression for $\Delta G°$ in footnote 62 should be as follows: $\Delta G°[H^•(aq) \rightarrow {}^1/_2H_2(g)] = -48.6 - 6.6 = -55.2$ kcal/mol. This correction slightly changes the shape of Figure 5 (p 1868) but does not alter the conclusions. We are very grateful to Ms. Kimberly Gardner for finding this error.

Scheme 4. Calculation of $\Delta H°$ for the Addition of $H^•$ to Permanganate

$MnO_4^- + e^- \rightarrow MnO_4^{2-}$	$E° = +0.564$ V[60]
${}^1/_2H_2(g) \rightarrow H^+ + e^-$	
$MnO_4^{2-} + H^+ \rightarrow HMnO_4^-$	$pK_a = 10.5$[61]
$H^• \rightarrow {}^1/_2H_2(g)$	*$\Delta G° = -55$ kcal/mol*[62]
$MnO_4^- + H^• \rightarrow HMnO_4^-$	*$\Delta G° = -82$ kcal/mol*
	$\Delta H° = -83$ kcal/mol

Book Reviews *

Protein Kinase C. Current Concepts and Future Perspectives. Edited by David S. Lester (CACSS, Yona Microscope and Instrument Co.) and Richard M. Epand (McMaster University Health Sciences Centre). Ellis Horwood: New York, London, Toronto, Sydney, Tokyo, and Singapore. 1992. xii + 365 pp. $75.00. ISBN 0-13-720186-9.

Protein kinases were the subject of 4914 articles during the past year (or more precisely, from the 13th week of 1993 through the 12th week of 1994) according to *Current Contents*. To place this number in perspective, fulleranes, which are the current rage in the chemical community, appeared in only 73 articles during the same period! Why the fascination with protein kinases? Much of their attraction is due to the general phenomenon known as signal transduction. The latter term is defined in Lester and Epand's monograph as "events occurring at the plasma membrane whereby extracellular signal molecules (e.g. hormones, neutrotransmitters, cytokines, extracellular matrix) can bring about specific intracellular events to alter cell behavior". For example, a particular signal molecule, upon binding to an appropriate receptor on the cell membrane, may trigger a series of enzyme-catalyzed events that ultimately induce the cell to divide. The vast majority of enzymes that comprise these signal transduction pathways are protein kinases. We now know that in many cancer cell lines certain signal transduction pathways are permanently turned on. The cell continues to divide even though no extracellular signal molecule is bound to the cell surface receptor. Given the relevance of protein kinases to carcinogenesis, as well as to other biological phenomena, it is not surprising that this enzyme family has received such intense scrutiny. However, of all the protein kinases that have been studied to date, none have received more attention than protein kinase C ("PKC"). Of the 4914 articles referred to above, more than 60% directly deal with PKC. Undoubtedly, much of the interest here is due to the observation by Nishizuka and his colleagues that PKC serves as the receptor for the tumor-promoting phorbol esters. Lester and Epand have compiled a series of 14 reviews that focus on the biochemical and biophysical properties, as well as the biological action, of protein kinase C. All of the articles cover the primary literature up to 1991. The book is divided into two main sections, the first dealing with *in vitro* properties and the second with the *in vivo* role of PKC. Rather than describe all 14 reviews in detail, I will focus on a few in order to give a general sense of the monograph.

The readers of the *Journal of the American Chemical Society* and, in particular, organic chemists will find Rando and Kishi's article The Structural Basis of Protein Kinase C Activation by Diacylglycerols and Tumor Promoters an absolute joy to read. Rando and Kishi nicely demonstrate that organic synthesis can serve as a powerful tool in addressing questions of biological significance. Indeed, this article could have easily been entitled "Advice to young natural products chemists" given the current funding situation. The authors propose a model for PKC activation by tumor promoters and diacylglycerol based upon the activity of a series of phorbol ester and glycerol derivatives. The narrative in this article is crisp and easy to follow. Unfortunately, not all of the reviews in this monograph live up to the standard set by Rando and Kishi. In particular, the article entitled Membrane-Associated Protein Kinase C is somewhat disappointing. In the resting cell, PKC is primarily located in the cytosol. Upon activation, PKC undergoes a net transfer to the plasma membrane. The expressed purpose of this review is to "correlate the factors controlling cellular localization with the cellular activity". Unfortunately, the extraordinarily stilted sentence structure contained

*Unsigned book reviews are by the Book Review Editor.