$$\Delta F_c = -nRT \int_0^1 \ln Q_c \, dY \qquad \text{(Eq. 4)}$$

Therefore, as $\Delta F_a = \Delta F_b - \Delta F_c$:

$$\Delta F_a = 2.3nRT \int_0^1 (\log Q_c - \log Q_b) dY \qquad \text{(Eq. 5)}$$

In Fig. 1, curve b represents log Q_b, so the area under the curve multiplied by $-2.3nRT$ is equal to ΔF_b. However, Q_c cannot be obtained in a similar manner, because, in general, reaction c cannot be followed experimentally. A good approximation for log Q_c can be obtained, because all protein molecules are in the R state when Y approaches 1 so the limiting value of log Q_b and log Q_c must be the same. Therefore, an estimate of log Q_c can be obtained from Fig. 1, because line c should equal log Q_c when it is assumed that all sites are equal in the R state. This assumption immediately implies that, from Eq. 5, ΔF_a is equal to the shaded area of Fig. 1 multiplied by $2.3nRT$. In the figure, this area is equal to 5.0 kcal. From the figure, it can be seen that ΔF_a is always a positive quantity in a cooperative process, so the associated equilibrium constant is less than 1 or, in other words, the T conformation is highly favored in the absence of ligand.

This important result is obtained following the assumption that all sites in the R state are equal. A qualitative picture of the situation, when this assumption does not hold, can be obtained in the following way. All protein molecules are in the R state when Y approaches 1, so the limiting value of log Q_b when Y approaches 1 should form one point of the line representing log Q_c. However, when the binding sites are unequal, log Q_c is no longer equal to line c in Fig. 1. Log Q_c cannot lie below line c in Fig. 1, because this would indicate cooperativity in a molecule totally in the R state. Under these circumstances, log Q_c must lie somewhere above line c. This means that the true value of ΔF_a for nonidentical sites is higher than that obtained by applying the method for identical sites. Only when some specific model is assumed can the exact value of ΔF_a be calculated (8). It is obvious that dissimilarity of binding sites in the T state has no influence on the determination of ΔF_a.

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BOOKS

REVIEWS

Topics in Infectious Diseases. Vol. 1. Drug Receptor Interactions in Antimicrobial Chemotherapy. Edited by J. DREWS and F. E. HAHN. Springer-Verlag, 175 Fifth Ave., New York, NY 10010, 1975. 314 pp. 17 × 24.5 cm. Price $19.40.

This book contains the papers presented at the Sandoz Symposium held in Vienna on September 4–6, 1974. It is divided into five general areas, *i.e.*, receptor hypothesis, DNA as a drug receptor, ribosomes as drug receptors, mode of action of chloramphenicol, and microbial enzymes as drug receptors, and includes papers that were contributed by 20 participants. The text of the book has 300 pages, 130 figures and illustrations, and 60 tables.

Drug–receptor interaction is the key to the effect and fate of a drug in the biological system. The authors exemplified the underlying mechanism of antibiotic–receptor interactions by systematically quantifying the relationship between physicochemical parameters and biological responses elicited by interactions of the drug with bioreceptors. Recent advances in research on binding sites of drug molecules to DNA and ribosomal subunits are presented.

The book offers a further insight into the mechanism of development of resistance in microorganisms and the role of plasmids in transmitting resistant genetic elements into the new cell line. Evidence has been produced demonstrating that there can be a surge of R factors in nonpathogenic enterobacteria which may be transferred to the pathogens such as *Shigellae* and *Salmonellae* due to the worldwide indiscriminate use of antibiotics. This is part of the endless race between medical science and microorganisms. The authors discussed the elimination of the genetic determinant elements from plasmids by binding DNA with a number of antibiotic as well as nonantibiotic agents. Enzyme inhibitory actions demonstrated by antibiotics and nonantibiotics suggest potential development of a bacterial enzyme inhibitor as an antimicrobial agent.

The authors suggest that when theory and knowledge of drug–receptor interactions are put into practice, a more ideal drug molecule with precise effect and anticipated mode of action may be designed with less time and expense.

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Medication Law and Behavior. By J. TYRONE GIBSON. Wiley, 605 Third Ave., New York, NY 10016, 1976. 407 pp. 16 × 23.5 cm. Price $15.95.

"The book is designed to help the reader learn more about the influence of medication law on the behavior of health care personnel who assist in providing medication and medication services." The author establishes this objective in the preface and in a highly readable style achieves it in the text.

Nonlawyers and those not in the health professions, as well as health professionals, can learn from reading this book. The book