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BOOK REVIEW

Directory of Therapeutic Enzymes

(McGrath, B. M., and Walsh, G. (eds.) CRC Press, Boca Raton-London-New York-Washington, D. C., 2006, 303 p., $159.95)

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The book consists of 13 chapters written by an international group of authors.

Chapter 1 (N. Shanley, G. Walsh) begins with a historical overview of the main stages of development of enzymology. It gives enzyme classification and various examples of enzyme applications in clinical biochemistry, food technologies, and industry. Special sections of this chapter characterize proteases, immobilized enzymes, enzyme-based biosensors, and enzymes employed in enzyme-linked immunoassay.

Chapter 2 (B. M. McGrath) considers approaches used in enzyme engineering. It discusses enzyme stability, substrate specificity of enzymes, features of enzyme catalysis in organic solvents, and chemical modification improving properties of enzymes employed clinically.

Chapters 3 (G. Walsh), 4 (B. W. Grinnell et al.), and 5 (N. N. Sanders et al.) sequentially describe such enzymes as tissue plasminogen, activated protein C, and deoxyribonuclease I, results of clinical trials of these enzymes, and the main steps in their industrial production. Special attention is paid to positive and negative factors affecting clinical application of these enzymes.

Chapters 6 (T. Edmunds) and 7 (D. Barngrover) characterize and describe medical application of β-glucocerebrosidase and α-galactosidase. Inherited deficiencies of these enzymes cause lysosomal storage diseases known as Gaucher and Fabry disease, respectively. Technologies developed by Genzyme (USA) for application of these enzymes for medical treatment of such patients are now widely used in clinical practice.

Chapters 8 (A. Bayol et al.), 9 (C. Mauz-Korholz et al.), and 10 (E. Erhardtsen et al.) provide analysis of such enzymes as urate oxidase, L-asparaginase, and recombinant factor VIIa.

Chapters 11 (B. M. McGrath) and 12 (E. Kakkis) describe factor IX and α-L-iduronidase, respectively.

Chapter 13 (S. O'Connell) highlights a wide spectrum of other enzymes (uro-, strepto-, and staphylokinases, ancrod, acid α-glucosidase, superoxide dismutase, hyaluronidase, lactase, pancreatin, and enzymes as debriding and anti-inflammatory agents) employed clinically.

Based on the title and description of this book, the authors intend it to cover "all approved therapeutic enzymes currently used in medicine". However, the directory is incomplete as there is no information regarding several of the therapeutic enzymes either widely used today or currently in clinical development.

First, a reasonable question arises as to why the authors and editors of this book have omitted any reference to α-galactosidase A (Replagal®; Shire Pharmaceuticals, Human Genetic Therapies, Cambridge, MA, USA), the only human cell line derived therapeutic enzyme for the treatment of Fabry disease (α-galactosidase deficiency). Replagal® was first introduced in August 2001 by Transkaryotic Therapies (Cambridge, MA, USA) and is approved in 35 countries to date. It was added to Shire's portfolio following the acquisition of Transkaryotic Therapies (Cambridge, MA, USA). It should be noted that Shire Human Genetic Therapies, together with Genzyme and BioMarin, is one of the major companies in the field of lysosomal storage diseases (LSDs) (Hopwood, J., et al. (2006) *Nature Rev.*, **5**, 101-102). For further information on the above-mentioned companies, please visit the following websites: www.shire.com; www.genzyme.com; www.biomarinpharm.com.

Second, we would like to attract attention to the enzyme preparations assigned for correction of mucopolysaccharidosis (MPS) including galsulfase (Nagalzyme™; BioMarin) for treatment of patients with MPS type VI (Maroteaux–Lamy syndrome) and idursulfase (Elaprase™; Shire Human Genetic Therapies) for treatment of MPS type II (Hunter syndrome). MPS II is a serious, life threatening, genetic disorder with no historically effective therapy. Individuals with MPS II lack iduronate-2-sulfatase (I-2-S), which is essential for the continuous process of breaking down and recycling glycosaminoglycans (GAG). As a result, GAG build up in the lysosomes of various types of cells in the human body causing progressive severe disease. The therapeutic

enzyme Elaprase™ is a human I-2-S derived from a human cell line and it replaces the missing enzyme in Hunter syndrome patients. Elaprase™ was recently approved by the U. S. Food and Drug Administration (FDA) and was filed with regulatory authorities in Europe in the fourth quarter of 2005. The product represents the first human enzyme replacement therapy for Hunter syndrome.

Third, we would like to note new developments for the treatment of Gaucher's disease including a human gene-activated glucocerebrosidase from Shire Human Genetic Therapies (see Zimran, A., et al., in *Program and Abstracts. The American College of Medical Genetics. Annual Clinical Genetics Meeting*, March 23-26, 2006, p. 124, Abstract # 185).

The book is illustrated with many tables and figures, contains main references for each chapter, and has a subject index at the end. All these features support the text and will be very helpful for readers.

This book can be recommended for students and teachers of biological and medical faculties, medical doctors involved into treatment of various enzymopathies, and also to a large audience of biochemists, cell and molecular biologists, and biotechnologists.

G. Ya. Wiederschain, M.D., Ph.D., Doc.Biol.Sci.,
Scientist, Shire Human Genetic Therapies, Cambridge, MA 02139,
gwiederschain@shire.com

M. Baldry, BSc., MBA,
Head of World Wide Marketing,
Shire Human Genetic Therapies, Cambridge, MA 02139,
mbaldry@shire.com