# Preface

The initial studies on autophagy began in the late 1950s and focused on the response of organisms to glucagon. The methods for analyzing autophagy at that time were limited primarily to electron microscopy and subcellular fractionation. In subsequent decades the analyses were concerned with the regulation of autophagy, and additional biochemical methods including sequestration assays became part of the arsenal for following autophagy activity. Most recently, molecular genetic studies have focused on the function of the autophagy-related proteins, and many of the assays have been designed around these newly identified components.

Now, in part, we are seeing a return to whole organism analyses, bringing to bear some of the increased knowledge we have gained from the recent wide range of molecular studies. These analyses include an examination of autophagy in new model systems, such as planaria, hydra and ticks as described in volume A of this series. Similarly, extensive studies have been carried out in standard organisms, most notably mice, and some of the corresponding techniques are included in volume B. These animal studies have certainly paved the way for the examination of autophagy in humans. In addition, however, the renewed interest in whole organisms has resulted from the many health implications of autophagy. Recent studies have indicated a role for autophagy in tumor suppression and immunity, in preventing certain types of neurodegeneration, in liver and lung disease, in myopathies and heart disease, and in some lysosomal storage disorders.

I think it is fair to say that one goal, even of the most basic research on autophagy, is to find information that may ultimately be of therapeutic use. For example, rapamycin or its derivatives are being used in clinical trials for treating certain types of cancer, and there is a high level of interest in the use of autophagy (either through stimulation or inhibition) for potentiating anti-cancer therapies. Similarly, a number of labs have carried out screens for drugs that can modulate autophagy for the potential mitigation of neurodegenerative diseases or myopathies. Accordingly, this final volume of the series on autophagy focuses on disease connections with autophagy and on methods for monitoring autophagy in clinical settings. Once again, certain techniques are presented in more than one chapter, with the emphasis on details that are essential for successful sample preparation and

assay using the particular tissue sample being described. There is no question that these methods should facilitate the application of similar techniques to study new systems and additional diseases that become linked with autophagy in the future.

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