

# Preface

In the beginning of 2005, after finishing the galley proofs for “The Molecular Basis of Fragile X Syndrome, Research Signpost” earlier that fall, I was invited to participate in a conference on fragile X syndrome. This was one of the famed Banbury conferences which were held on the picturesque campus of Cold Spring Harbor Laboratory. I had attended the inaugural one in 2000, where I met a childhood idol, Dr. James Watson. As with all conferences there are highlights, the things that leave an indelible impression on your memory, and there is the rest, which you, in short order, forget. For this particular Banbury conference, there was one talk which bears on the creation of this book that I will never forget.

The talk was given by Dr. Richard Paylor of Baylor University and it concerned the recent new behavioral tests that were being used in his laboratory to assess the several different fragile X mouse model strains that currently existed. His group’s work definitively showed that specific behaviors and particular phenotypes produced by the loss of the fragile X mental retardation protein were significantly affected by the mouse strain under investigation. He summarized his findings by constructing the behavior equivalent of a gene expression heat map and put forth the provocative thesis that in order to understand fragile X syndrome one must assess phenotypes in a variety of model strains. I remember afterwards thinking, in true Darwinian fashion, that if strains could produce such profound effects, how much more so the species. So to tease out the true fragile X phenotype, we may need to examine behaviors in several species and would not that make an interesting book project to edit.

Except perhaps for the closing fragment in that last sentence such an idea was not novel because the *Drosophila* dFmr1<sup>-/-</sup> model of fragile X syndrome was already well established in the literature and work characterizing the fragile X gene family member expression in frogs and zebra fish had just been published. Nevertheless, it took a few more years before an opportunity arose to gestate this project. That opportunity came by way of an inquiry from Dr. Henri Tiedge, co-editor of “*Results and Problems in Cell Differentiation*”, as to whether I would be interested in editing a volume on fragile X syndrome for the series. I jumped at the chance and

could not have been more pleased with the outcome. I hope that you, the reader and especially those who are my colleagues in the fragile X field, agree with this assessment.

It should be self-evident that like a symphony conductor an editor's role in the book-making process is mainly one of preparation and coordination; although often the focus of the audience's attention, a conductor should merely serve as a bridge, accepting the audience's applause on behalf of the orchestra. The real kudos belong to the individual members for their performances. This differentiates the roles of editors and conductors, as editors are often unheralded, anonymous fellows and that is how it should be. In contrast, authors are utterly like their orchestral counterparts in deserving praise. Therefore, I humbly and gratefully acknowledge my immense debt to each of the chapter authors: first for doing the majority of the primary research that enabled this project to be initiated and second for their willingness to cogently distill and disseminate their results here in these next pages. They have truly turned my dream into reality and collaborating with them has been one of the highlights of my short editing career.

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