Preface

Créer, c'est recombiner.—François Jacob

As changes over time—evolution—may be called adaptation at the species level, we refer to somatic adaptations as those genetic changes occurring in an animal's cells during its lifetime. The latter phenomenon, with which this volume is concerned, deals with the genetic diversity generated in vegetative cells of individuals, whether parasite or host, enabling individualized responses for survival. This strategy allows members of the population to survive, procreate, or infect. And because the survival phenotypic constellation was somatically based, subsequent generations continue to take individual risks. Thus, it is not so much the resultant genetic diversity in itself but rather the mechanism generating this diversity that has been evolutionarily selected for.

The model systems here exemplify diversification pathways involving DNA, RNA, or posttranslational modifications and range from protozoan to mollusk to fishes to humans. In most cases, the diversifying mechanism, be it in host or in parasite, enables competing responses that escalate in scissors-rock-cloth fashion. The advantage of genetic diversification is rapid adjustment to novel (read: potentially adverse) circumstances, as in the switching of the variant surface glycoprotein coating a trypanosome during the host's mounting immune response, or as in the proliferation and expansion of vertebrate lymphocytes bearing immunoglobulin, T-cell receptor, or variable lymphocyte receptor (VLR) when activated in the course of an infection.

As respiration and nourishment have to be acquired from the external environment, complex interactions have originated at these interfaces; the epithelium of respiratory or filtering-feeding organs and the gut must be also be immunocompetent. The immune aspect of the vertebrate gut, with its associated lymphoid tissues and molecules, is represented here in bony fishes and in mammals, which have independently evolved specialized secretory immunoglobulins. Whereas V(D)J recombination provides fish lymphocytes with a variable region that directly splices to the IgT C region exons, in mammals it is additional somatic signaling that

induces class switch recombination to IgA, diversifying the immunoglobulin effector function. Junctions of contact with the environment may have been the primary sites where decisive selective advantages were exerted.

Just as the host systems evolved to recognize invaders, the invaders evolved to become evaders. However, there is yet another important facet: the host needs to distinguish beneficent microorganisms. Negotiation with gut microbiota is thus not unilaterally defensive but complex in ways we are only beginning to explore. The protochordate VCBP molecules constitute the only germline-based system presented in this volume. These highly diverse molecules have undergone selection through evolution, and their role in the gut involves not only immune function but also regulating microbiota homeostasis. And, intriguingly, stimulation of lymphocytes by the gut flora is not always a host protective response. As demonstrated in rabbit appendix, emerging B lymphocytes interact with gut commensals, and antibody repertoire expansion through gene conversion and somatic hypermutation is driven by the local superantigen-like molecules. The developing immune system within an individual is subjected to selection for tolerance versus immunity, self versus nonself, but the interaction between its immune system and microbiome is emerging as multilayered and, to a great but unknown extent, symbiotic.

If the CRISPR system in bacteria and archaea is included, then in most branches of the tree of life organisms have evolved somatic mechanisms in this "arms race" between host and parasite. As shown in models reviewed in this volume, when the diversification pathway involves combinatorial associations of elements, the gene organization tends toward minimalism. That is, in stark contrast to their diversified output, there are far fewer germline elements. Fewer components facilitate regulation of the system. Fewer building blocks also permit more stringent selection for efficacy; mutations in a germline component will have amplified effects. What we discover in gene systems to be simplicity and perceive as elegance, it is in Nature mere functionality.

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