Microbiome Diversity

The pathogenesis of allergic diseases, including asthma, is in part attributable to the composition of microbial colonies in the gastrointestinal tract. A collaborative relationship has been identified between the development of allergic diseases in childhood and factors such as early-life antimicrobial exposure, Caesarian delivery, and formula feeding. The capacity for microbial composition to shape the immune system has been demonstrated in studies of antimicrobial administration.[9,10]

THE DEVELOPMENT OF CHILDHOOD ALLERGIC ASTHMA MAY BE IN PART DUE TO MICROBIOME PERTURBATIONS IN EARLY LIFE.

Given that the identified early risks occur immediately before and after birth, the development of childhood allergic asthma may be in part due to microbiome perturbations in early life. In support of this hypothesis, studies have shown that neonates have a higher likelihood of allergic disease development, or atopy, with a higher fecal burden of *Clostridium difficile* and a higher *C. difficile* to *Bifiobacteria* ratio.[10] Another study demonstrated these findings with a diversity of bacteria. In both experimental cases, early-life bacterial colonization produced significantly decreased circulating IgE concentrations in adulthood. However, the administration of these bacterial strains in adult animal models did not lead to protection, suggesting the importance of early life environmental exposure to diverse microbes.[9,11] From an immunological standpoint, the altered inflammatory response from the lack of microbial diversity in the gut microbiota may be due to compromised functional capacity of regulatory cells. Studies have already shown a relationship between the gut microbiome, regulatory T cells (Tregs), and allergic diseases. However, given that regulatory B cells (Bregs) have been shown to act earlier and facilitate the

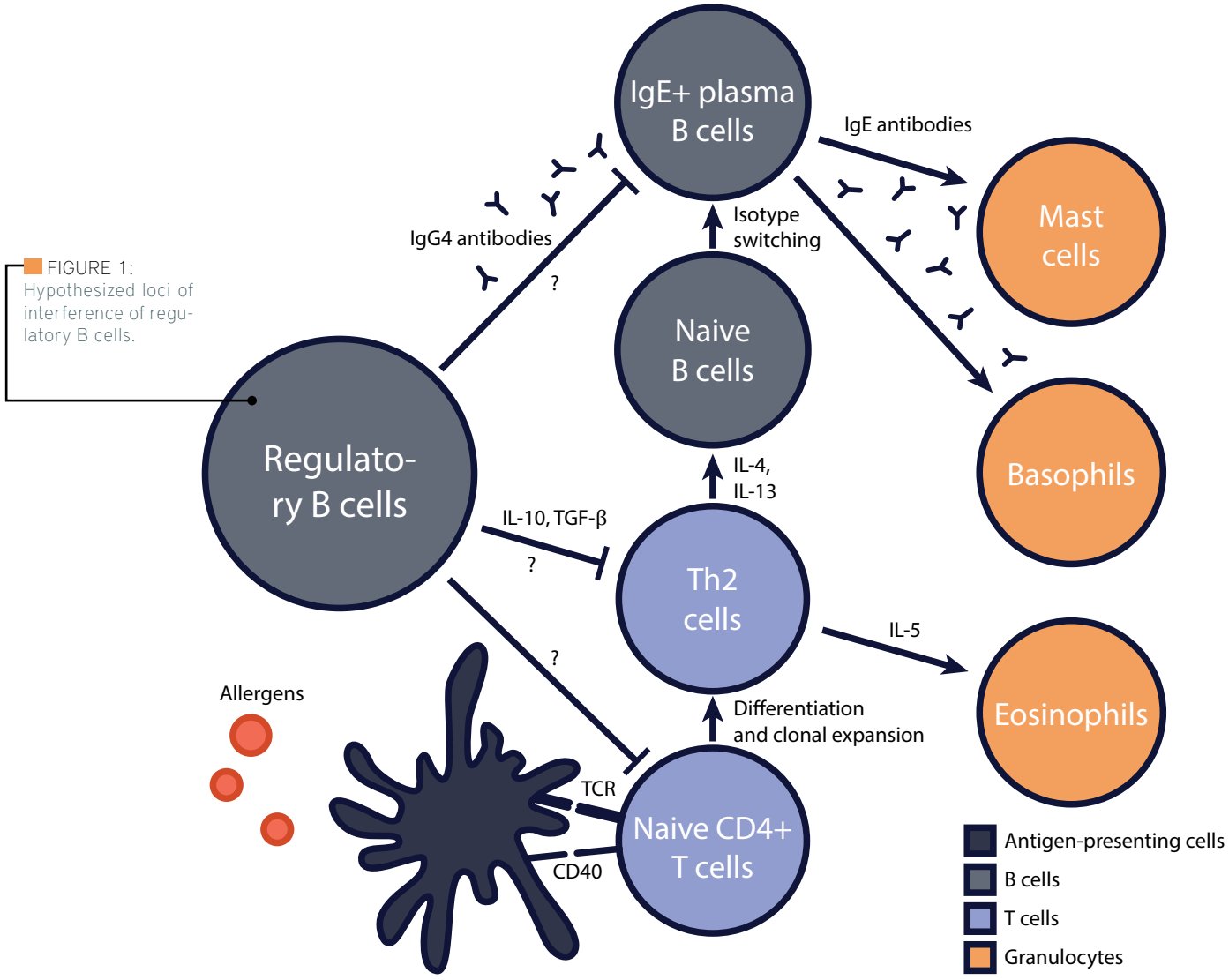

FIGURE 1: Hypothesized loci of interference of regulatory B cells.