For example, viruses frequently expose unique chemical structures only during their replication inside host cells. Many of these can be detected via intracellular receptors that bind exposed chemical moieties while still inside the host cell. This can trigger an immediate antiviral response in the infected cell that blocks further virus replication. At the same time, this initiates the secretion of chemical warning signals sent to nearby cells to help them guard against infection, after that categorizing happens via a subtle tracking system and allows the immune response to make note of which recognition molecules were involved in the initial detection event and relay that information to subsequent responding immune cells, it allowing the follow-up response to begin to focus attention on the likely type of assault underway.

Host-pathogen interactions are an ongoing arms race; pathogens evolve to express unique structures that avoid host detection, and the host recognition system co-evolves to match these new challenges. Because pathogens generally have much shorter life cycles than their vertebrate hosts, and some use error-prone DNA polymerases to replicate their genomes, pathogens can evolve rapidly to evade host-encoded recognition systems. The host immune response would quickly become obsolete thanks to these real-time pathogen avoidance strategies. How can the immune system prepare for this? How can our DNA encode a recognition system for things that change in random ways.

To favor randomness in the design of some recognition molecules strategy, called **generation of diversity**, is employed only by developing B and T lymphocytes. The result is a group of B and T cells in which each cell expresses many copies of one unique recognition molecule—collectively, a cell population with the theoretical potential to respond to any antigen. This feat is accomplished by rearranging and editing the genomic DNA that encodes the antigen receptors expressed by each B or T lymphocyte.

This system allows chance to play a role in generating a menu of responding recognition molecules. Thus, B and T cells make surface receptors unique to each individual, which are then not passed on to offspring. This is in direct contrast to the DNA that encodes PRRs, which are inherited and passed on to the Next generation.

(Generation of diversity and clonal selection in T and B lymphocytes. Maturation of T and B cells, which occurs in primary lymphoid organs (bone marrow for B cells and thymus for T cells) in the absence of antigen, produces cells with a committed and Antigenic specificity, each of which expresses many copies of surface receptor that binds to one particular antigen. Different clones Of B cells (numbered 1, 2, 3, and 4). Cells that do not die or become deleted during this maturation and Weeding-out process move into the circulation of the body and are available to interact with antigen.

These clonal selection occurs when one of these cells encounters its cognate or specific antigen. Clonal proliferation of an antigen-activated cell leads to many cells that can engage with and destroy the antigen, plus memory cells that can be called **a Subsequent exposure**. The B cells secrete antibody, a soluble form of the receptor, reactive with the activating antigen. Similar Processes take place in the T-lymphocyte population, resulting in clones of memory T cells and effector T cells. the latter activated T cells, which secrete cytokines that aid in the further development of adaptive immunity, and cytotoxic T lymphocytes (CTLs), which can kill infected host cells.)

This cutting and splicing of chromosomes is not without risk. This theory, proposed by Polly Matzinger at the National Institutes of Health.