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$$\begin{aligned} \text{Let } R &= \mathbb{Z}[i] \text{ be the ring of integers of } \mathbb{Q}(i) \text{ and } T := R \cdot i \\ \text{Then } \forall z \in \mathbb{Q}(i) & \text{ we have } \frac{1}{1-i} = (1+i)^{-1} = 1 + (1-i) = 1 - i + (1-i)^2 - (1-i)^3 + \dots \\ \frac{1}{1+i} &= (1-i)^0 + (1-i)^1 + (1-i)^2 + (1-i)^3 + \dots \\ \text{Then } \forall z \in \mathbb{Z}[i] & \end{aligned}$$

The following seems plausible to me, but I don't know if it is correct and I can't prove it.

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hormones, and soluble molecules that function in a paracrine fashion to regulate cellular development, growth, differentiation, and function.

Some cytokines have important roles in host response to infectious agents, whereas others may contribute to the host response to noninfectious diseases. Chemokines are chemotactic molecules that direct the migration of leukocytes from the blood into tissues. Cytokines and chemokines are produced by activated monocytes, macrophages, lymphocytes, epithelial cells, and certain types of cells within the vascular wall. To date, proinflammatory cytokines have been identified, and their effects have been determined. These molecules are referred to as interleukins (IL-1, IL-2, IL-4, IL-5, IL-6, IL-8), interferons (IFN-alpha, IFN-gamma), tumor necrosis factor-alpha (TNF-alpha) and the lymphotoxin alpha-beta (LT-alpha-beta). Collectively, these factors are believed to be involved in development and progression of inflammatory and autoimmune diseases, infectious diseases, and neoplastic processes. IL-1 production and release are regulated by corticosteroids. Some cytokines act in an autocrine manner. They may be produced by cells of the immune system and therefore serve in an autocrine manner as a feedback signal. Thus, the cytokines are expressed by activated T cells, B cells, macrophages, neutrophils, mast cells, and monocytes. Cytokines are also expressed by other cells such as epithelial cells and fibroblasts.

Cytokines are pleiotropic, and they are highly regulated at the transcriptional, translational, posttranslational and posttranslational levels. Each cytokine is controlled by specific mechanisms and often has a limited half-life. Thus, human cytok