“Fighting Disease: Cancer Treatments and Their Role in Plant Defenses”

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In many developed countries, cancer is the leading cause of death among 15- to 64-year-olds and the main contributor to lost years of life (Pujol et al., 2007). Rituximab, a monoclonal antibody, approved as a cancer therapy in 1997, improved patient survival and quality of life (Dent et al., 2020; Biogen). Since the introduction of Rituximab, hundreds of biologic drugs have been clinically evaluated. However, biologic drugs are expensive. The demands to expand the therapeutic resources against cancer, boost the production of existing/underdeveloped cancer drugs, and decrease costs to patients are driving forces behind the continuous improvement in this field. One opportunity is plant made pharmaceuticals (PMPs).

PMPs offer advantages such as low cost of cultivation and high product recovery. Plants perform eukaryotic protein post-translational modifications, such as folding and multimeric assembly, and typically render active proteins. This is in contrast to prokaryotic systems, in which proteins may be non-functional or insoluble. Lectins are proteins found in plants used to store nitrogen and provide defense against animals that are eating the plant. Recently, lectins have obtained a lot of interest due to their cancer-targeting abilities (Dent et al., 2020).

In this review, plant-expressed pharmaceuticals and the potential of plant-derived lectins as anti-cancer therapies will be discussed. In addition, we will discuss the reverse: anti-cancer drugs playing a role in plant defenses (Hadwiger et al., 2018). Actinomycin D is a DNA specific anti-cancer therapy that treats a number of cancers, and it also been shown to elicit defense responses in pea plants in a concentration dependent manner. The data show that plant genes can be affected by anti-cancer drugs and subsequently, they influence plant immune responses.

Plant disease is defined as anything that interrupts its normal and vital functions (Raskin et al., 2002). Disease can be caused by biotic agents, such as bacteria or fungi, or by abiotic factors, such as drought or extreme temperatures. Unlike animals that have an adaptive immune system consisting of immune cells and antibodies, plant defense is solely reliant upon defenses described in a ‘zig-zag’ model (Jones et al., 2006). This model can be divided into four phases. Upon pathogen challenge, pathogen associated molecular patterns (PAMPs) are detected by host pattern recognition receptors (PRRs) to elicit PAMP-triggered immunity (PTI). This serves as the first line of defense and is not specific. For a pathogen to be successful, it produces effectors to suppress PTI and cause effector-triggered susceptibility (ETS). In turn these effectors can be recognized by cytosolic host receptors to mount effector-triggered immunity (ETI). ETI is stronger and may cause programmed cell death to trap the biotrophic pathogen in dead cells. Subsequently, selection pressure in pathogen populations to evade detection by the host will lead to an evolutionary arms race between pathogen and host. This battle has been investigated by researchers because humans depend on plants for food and nonfood products such as soaps and medicines.

Overall, basic cancer research will have the benefits of novel plant-based therapeutics and provide insights on the commonalities of animal and plant defenses.

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References