fore to higher growth rates, facilitating successful invasions.

In the paper, "resource" is specified as N rather than "biomass" as generally referred to in previous studies, and where "resource" is allocated are also explicitly designated, i. e. the photosynthetic apparatus (growth), and cell walls and N-based defense chemicals (defenses). It also helps to understand the different effects of specialist and generalist enemies on evolution of introduced plant species. Furthermore, the paper elucidates for the first time how the reallocation of "resource" from defenses to growth contributes to invasiveness of alien plant species, i. e. via increasing resource-capture ability and-use efficiency. This research provides the first potential mechanism behind the commonly observed and genetically based increase in plant growth and vigor when they are introduced to new ranges, taking the investigation of mechanistic causes for invasiveness to a new level.

* * * *

New Findings in Anti-viral Infection and Control of Inflammation

Based on previous studies on phosphotase SHP-1-mediated promotion of interferon and pro-inflammatory cytokine production in immune cells such as dendritic cells and subsequent elimination of invading virus and other pathogens published in *Nature Immunology* in May, 2008, Academician Cao Xuetao's group from the National Key Laboratory of Medical Immunology and Institute of Immunology in Shanghai had another new story in *Nature Immunology* recently. After almost 10 years' investigation on the molecular mechanism underlying the initiation and regulation of anti-viral immunity and control of inflammation, the group independently identified a new E3 Ub ligase, Nrdp1, which can preferentially induce the production of type I interferon but inhibit the production of pro-inflammatory cytokines in macrophages and dendritic cells once stimulated by pathogen components or infected by virus, leading to the elimination of invading pathogens and attenuation of the inflammatory responses. This is the fourth time for Prof. Cao and his group to present their research findings in Nature Immunology in the past 4 years.

Supported by NSFC (projects No. 30572122, 30721091, 30771118), Prof. Cao and his group have cloned new molecules from human dendritic cell cDNA library since 1998. They firstly cloned a new molecule that can make the tumor cells resistant to death induction once the new gene was transfected into the tumor cells, so the new molecule was designated as death-resistant protein (DRP). The mouse homolog of this human molecule reported by foreign scientists 3 years later proved and identified the regulation of tumor cells apoptosis and carcinogenesis, which was named as Nrdp1 etc.

The finding indicates that Nrdp1 may prevent viral infection by promoting interferon production, which has provided new insight into the molecular mechanisms for the immune recognition and regulation, and also will contribute to the possible drug design to selectively activate Nrdp1 to induce antiviral immunity and control the pathogenesis of inflammatory diseases.