**Toxoplasma gondii**
is a widespread and obligate intracellular parasitic protist. It can infect virtually all types of nucleated mammalian and avian cells. As an opportunistic human pathogen, this parasite causes diseases in immunocompromised individuals and in neonates following congenital infection. *T. gondii* belongs to the phylum Apicomplexa, which also includes *Plasmodium*, the causative agent of malaria, and *Cryptosporidium*, responsible for intestinal cryptosporidiosis. Recently, there are some reports that apicomplexan parasites are producing some plant hormones and using them to regulate their proliferation. Here, I investigate whether one of the plant hormones, cytokinin, regulates the proliferation of the apicomplexan parasite, mainly used *T. gondii* as a model. Cytokinins are a particular type of plant hormone that are involved in regulation of cell proliferation, cell cycle progression, and cell and plastid development. At the first, to investigate whether apicomplexan parasite produces cytokinins, cell lysate of *T. gondii* and a rodent malaria, *Plasmodium berghei*, were prepared and analysed by mass spectrometry. Cytokinins, same with higher plant, were detected from the lysates of both parasites. Accordingly, we analysed the effect of cytokinins on *T. gondii* proliferation and found that natural cytokinin, trans-zeatin, accelerated the proliferation of parasites. On the other hand, a synthetic cytokinin, thidiazuron, inhibited the parasite proliferation in culture. Since cytokinins regulate the cell cycle progression by enhancing the expression of one of cyclins in plant system, we analysed the *T. gondii* cell cycle by flowcytometry. Indeed, trans-zeatin hastened the cell cycle progression from G1 to S phase while thidiazuron caused the halt at G1 to S phase progression. By qPCR analysis, we found that trans-zeatin upregulated the expression of one cyclin gene (*TgCYC4*), and that thidiazuron downregulated its expression. These suggest that *TgCYC4* acts as an on/off switch playing a crucial role in controlling the cell cycle progression in *T. gondii* and concomitantly its proliferation. Since cytokinins also regulate chloroplast development in plants, we observed an organella called apicoplast, which is a homologous organella to chloroplast in *T. gondii*. Immunofluorescence microscopy revealed that trans-zeatin increased the number of apicoplast but thidiazuron extinguished apicoplast. The change in the number of apicoplast in cytokinin-treated parasites was confirmed by qPCR analysis. These results strongly suggest that cytokinins also regulate the proliferation of apicoplast in *T. gondii*. On the other hand, because centrin, which is known to regulate the proliferation of apicoplast directly, did not change the localization nor expression level, it does not relate to the regulation of the proliferation of apicoplast by the stimulation with cytokinins. Together, the present results led me to conclude that a plant hormone, cytokinin, is important for the host cell infection of *T. gondii*.

The inhibitory effect of the synthetic cytokinin, thidiazuron, was also observed in laboratory animals. We thus believe that thidiazuron can be a potential drug to inhibit *T. gondii* proliferation, and that the biosynthesis pathway of this hormone might be a good candidate for the development of anti-toxoplasmosis drugs.