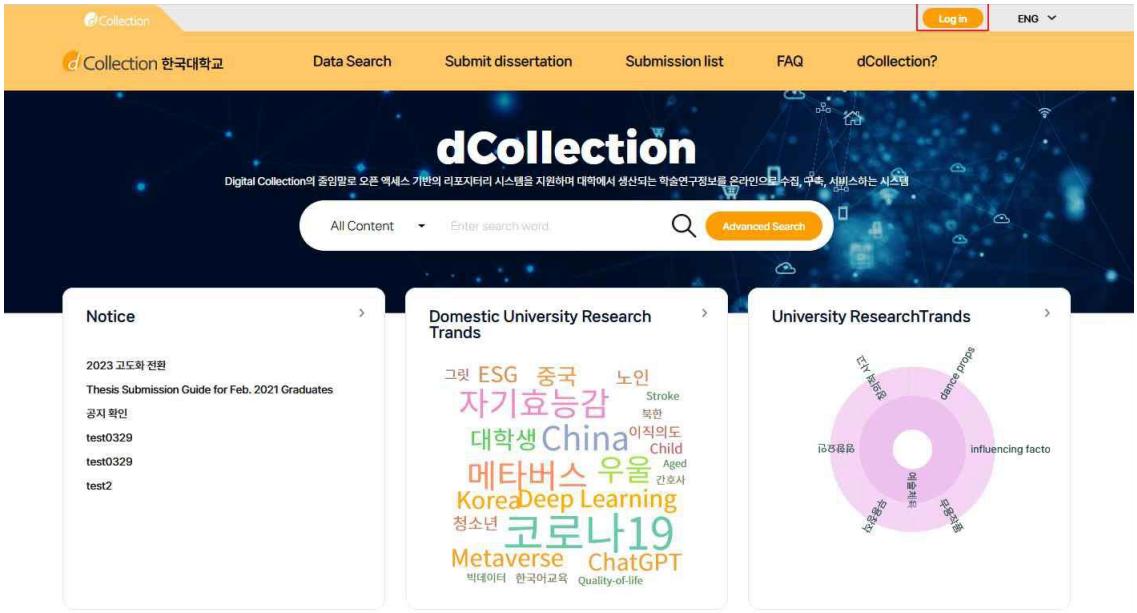
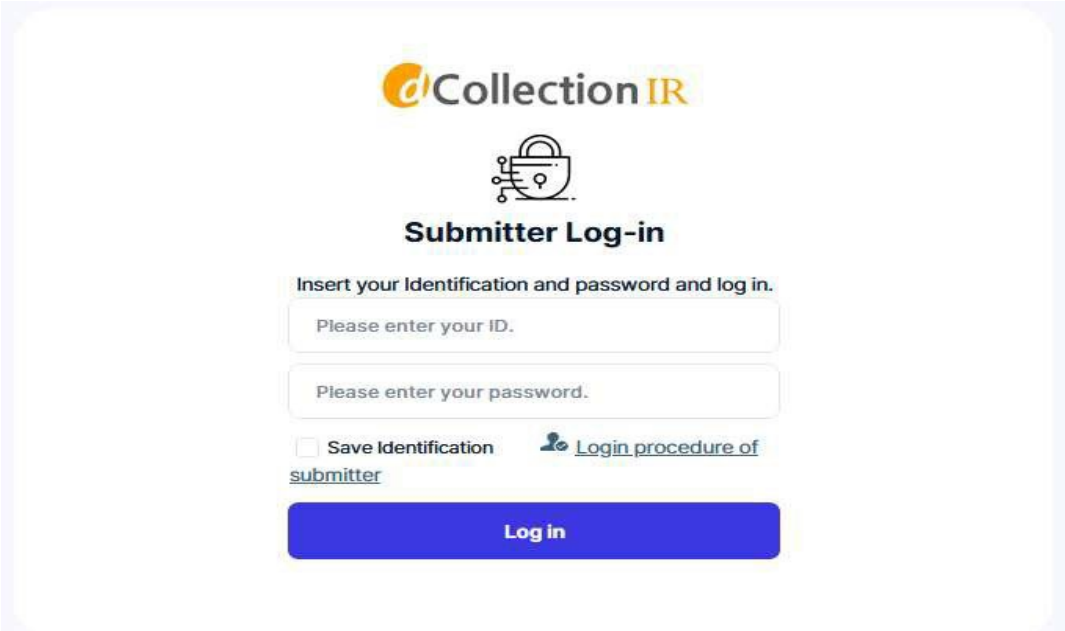
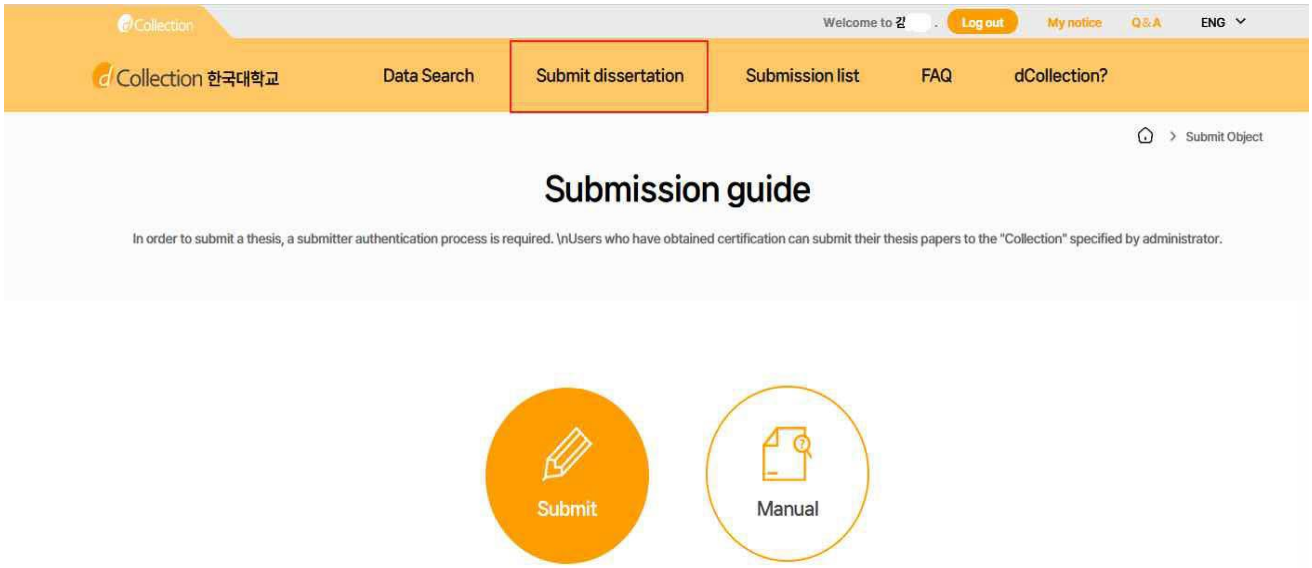


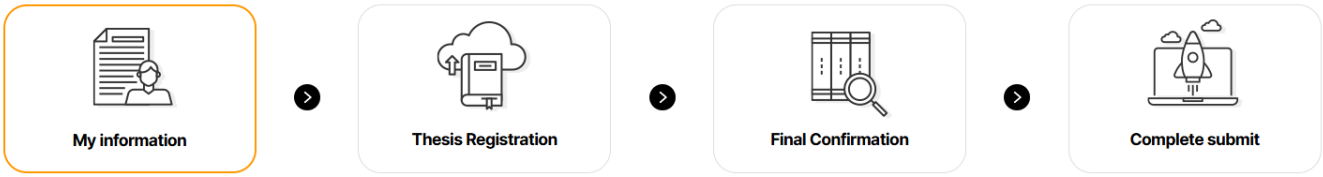


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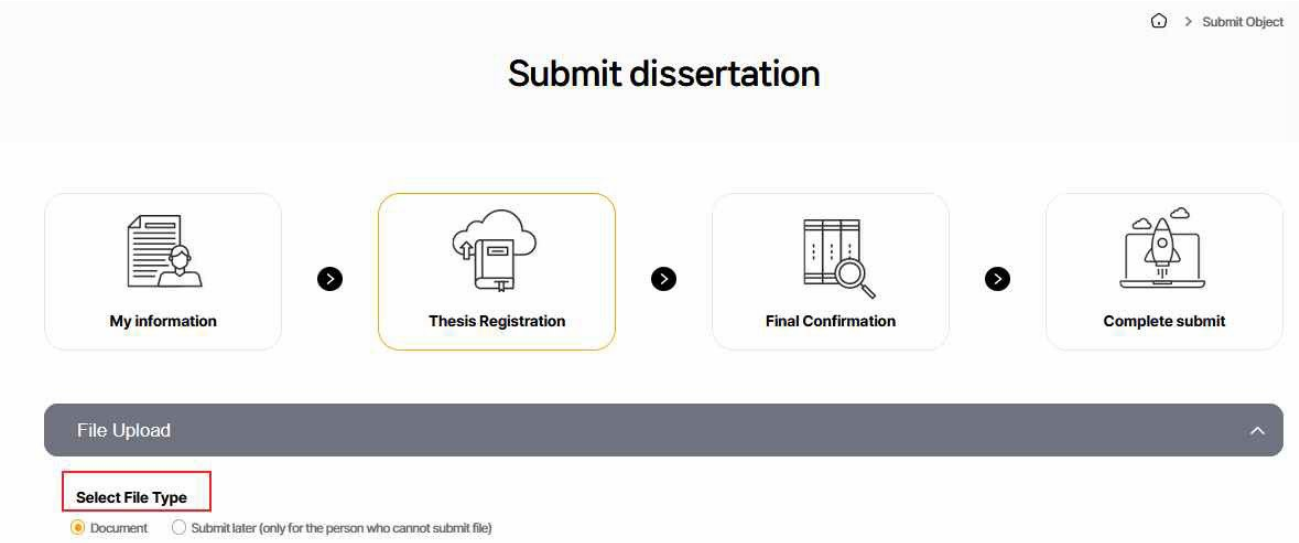
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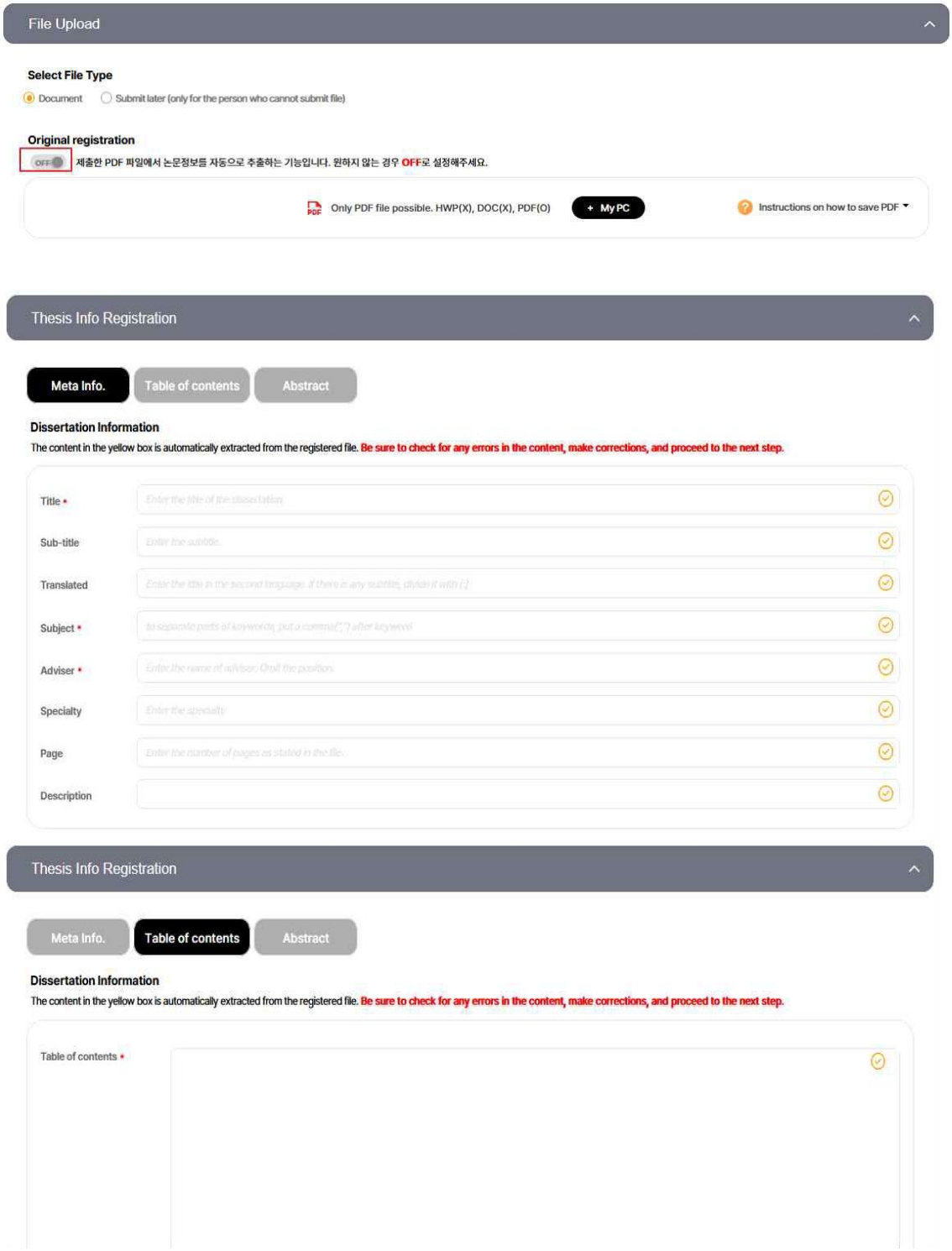
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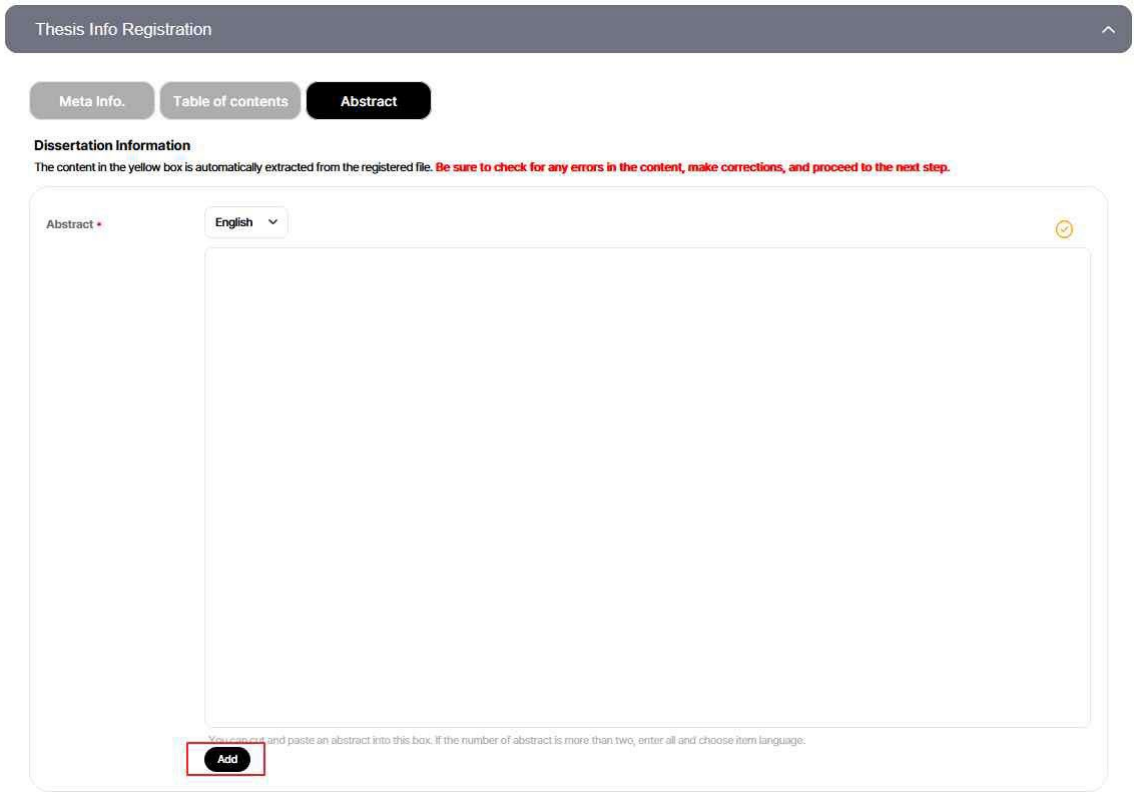


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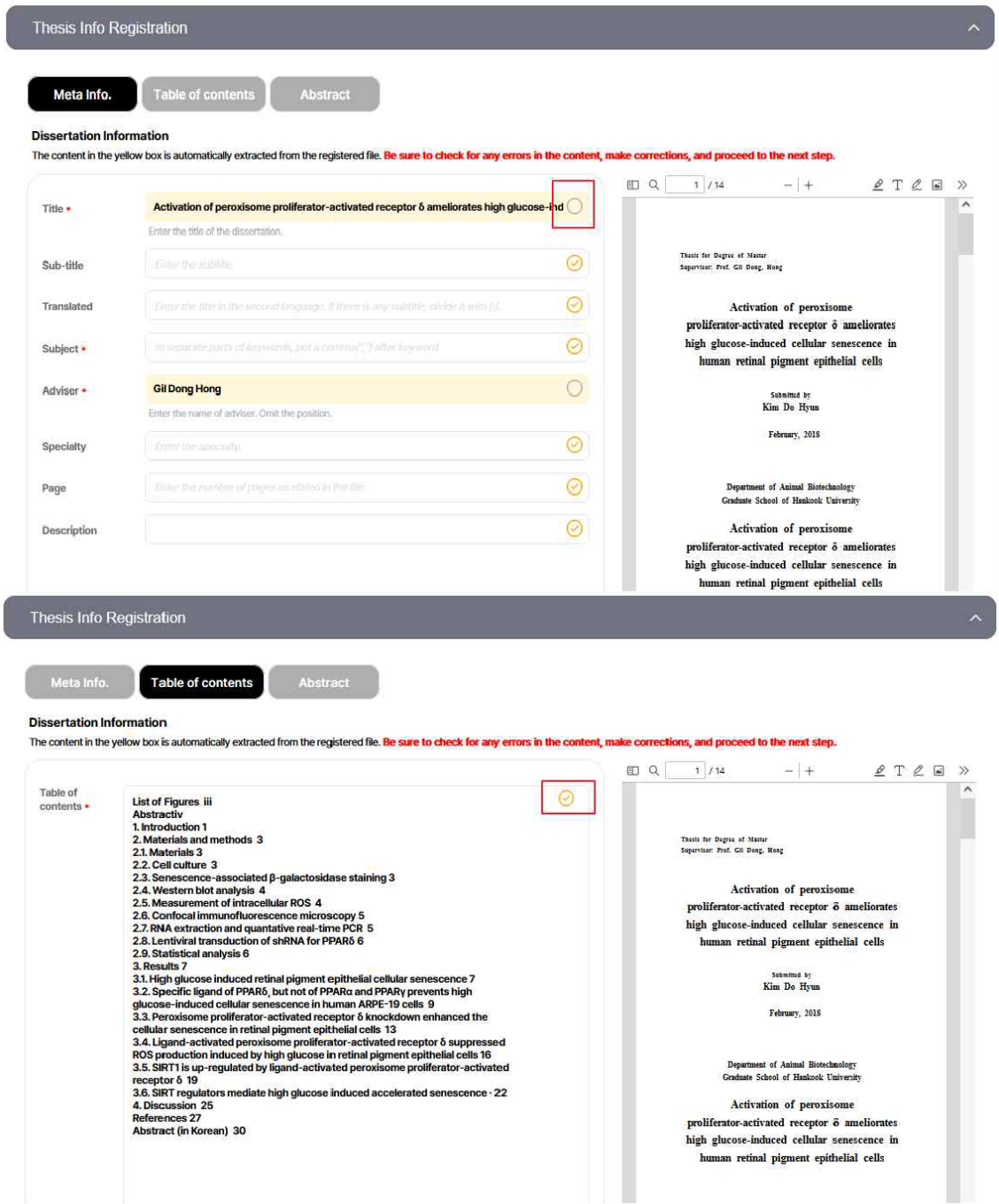


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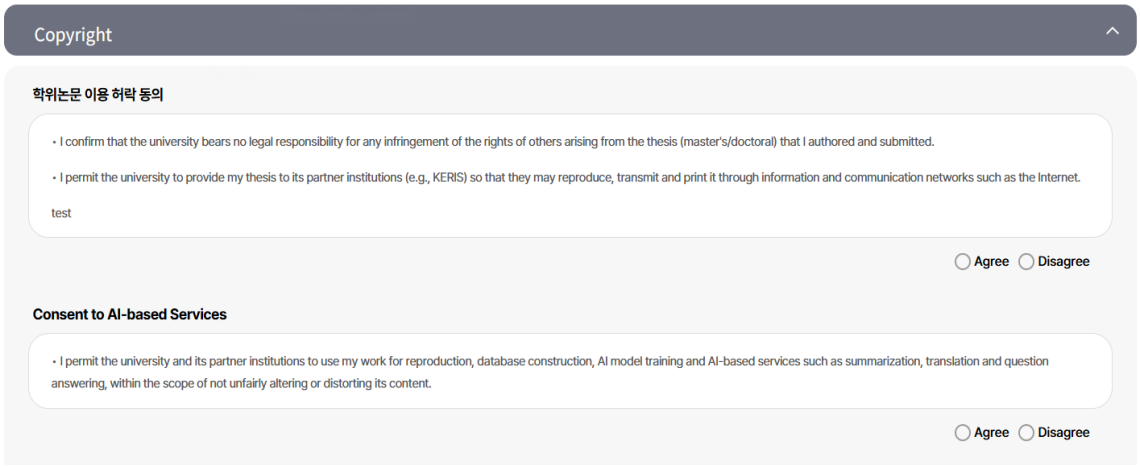


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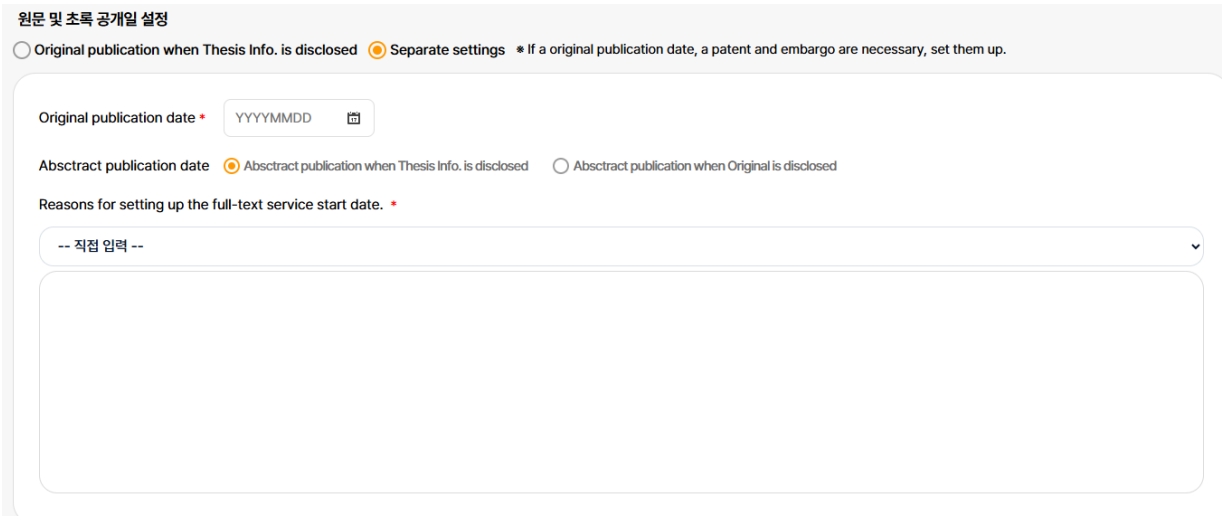
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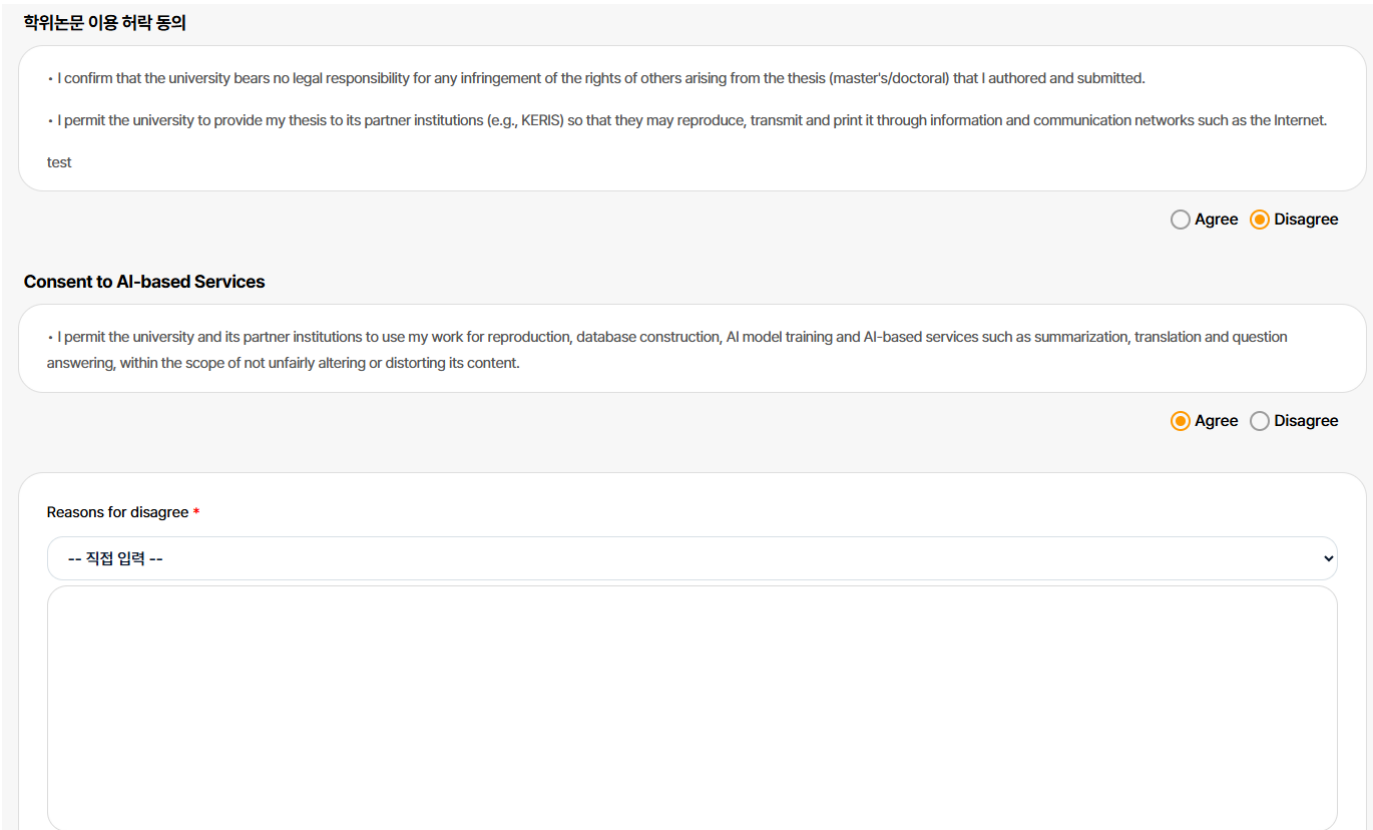
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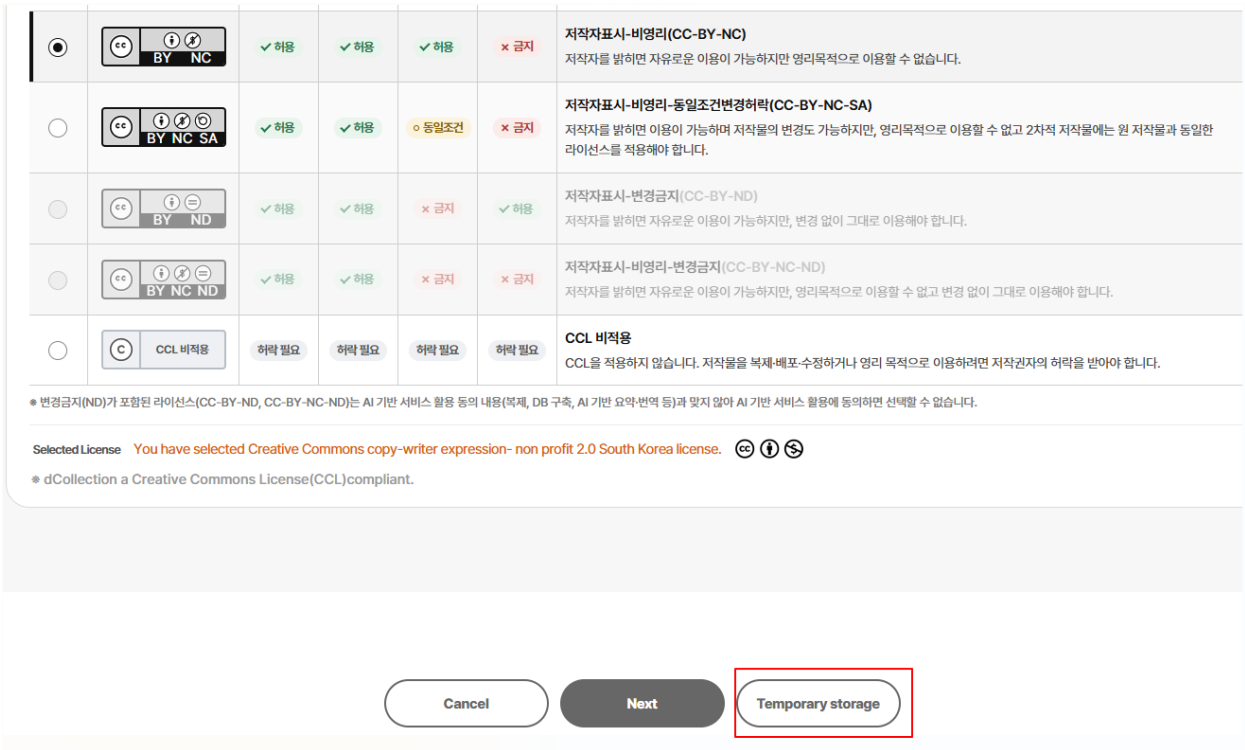
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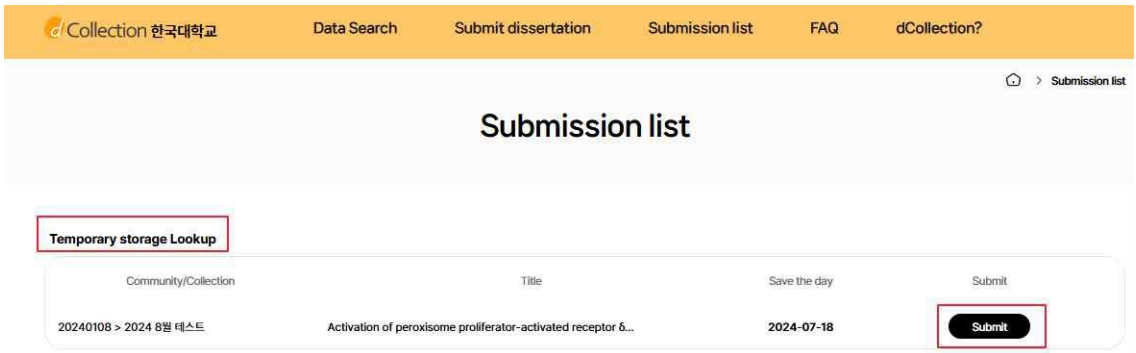




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Dissertation Information

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Author	Author	김현정	Affiliation	한국대학교 경영대학원2
	E-mail	test@futurenuri.com		
Subject	Activation of peroxisome, peroxisomea			
Abstract	Activation of peroxisome proliferator-activated receptor δ ameliorates high glucose-induced cellular senescence in human retinal pigment epithelial Kim Do Hyun Department of Animal Biotechnology Graduate School of Hankook University Diabetic retinopathy is one of the major cause on the visual impairment in adult patients with diabetes mellitus. Although the increasing evidence indicates that various cells enter the state of senescence earlier following exposure to high glucose, the high glucose-induced cellular senescence in retinal pigment epithelial cells is largely unknown. In this study, we investigated the role of peroxisome proliferator-activated receptor (PPAR) δ on the high glucose-induced cellular senescence in human adult retinal pigment epithelial cell line, ARPE-19 cells. Treatment of D-glucose significantly induced cellular senescence in human ARPE-19 cells. High glucose-induced cellular senescence was markedly suppressed by the activation of PPARδ by GW501516, a specific ligand of PPARδ, but not of PPARα or PPARγ ligands. Activation of PPARδ also inhibited the generation of reactive oxygen species (ROS) in ARPE-19 cells treated with D-glucose. High glucose-induced cellular senescence was markedly suppressed by pre-treatment of GW501516, a specific ligand of PPARδ, but not of WY14643, a specific ligand of PPARα or rosiglitazone, a specific ligand of PPARγ. In the shPPARδ-ARPE-19 cells, the effects of GW501516 were abolished on cellular senescence compared with shControl-ARPE-19 cells. Treatment of GW501516 inhibited the high glucose-induced generation of reactive oxygen species (ROS) in shControl-ARPE-19 cells. However, the effects of GW501516 on ROS generation were eliminated in shPPARδ-ARPE-19 cells. Activation of PPARδ significantly increased expression of SIRT1 in time- and concentration-dependent manners. In addition, GW501516-activated PPARδ recovered high glucose-inhibited expression of SIRT1. Finally, GW501516-induced inhibition of cellular senescence treated with D-glucose restored by pre-treatment of SIRT1 inhibitor. Thus, current study indicated that GW501516-induced PPARδ activation significantly suppressed high glucose-induced cellular senescence via upregulating the expression of SIRT1 in human ARPE-19 cells. keyword : High glucose; oxidative stress; cellular senescence; human adult retinal pigment epithelial cell; PPARδ; SIRT1			
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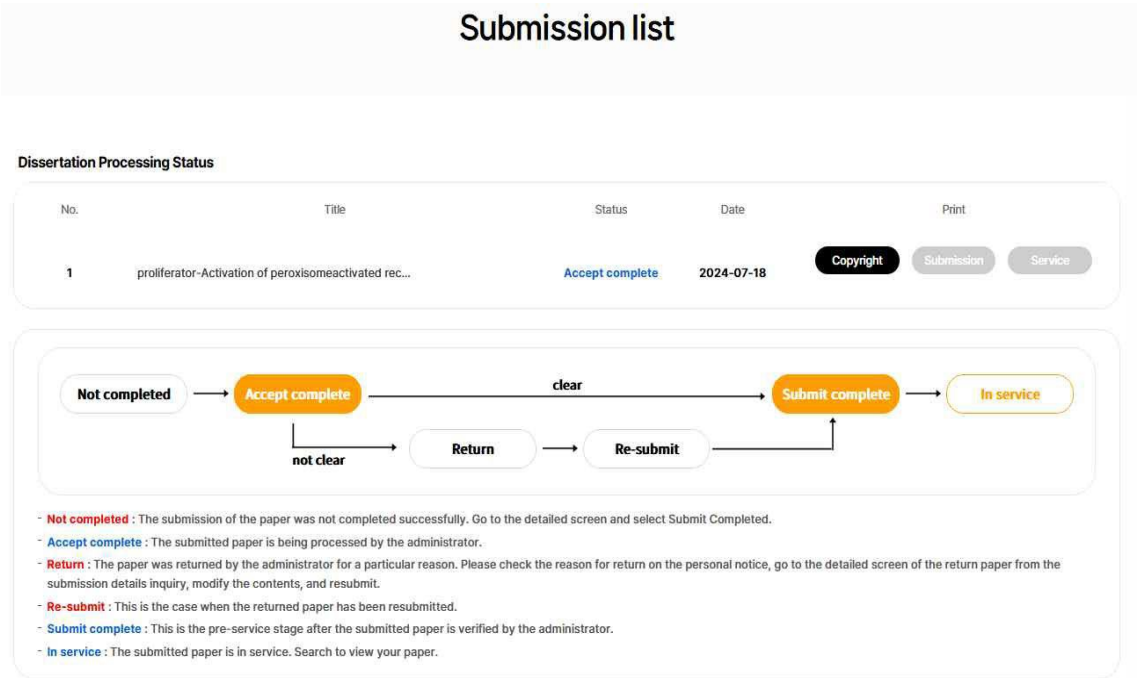
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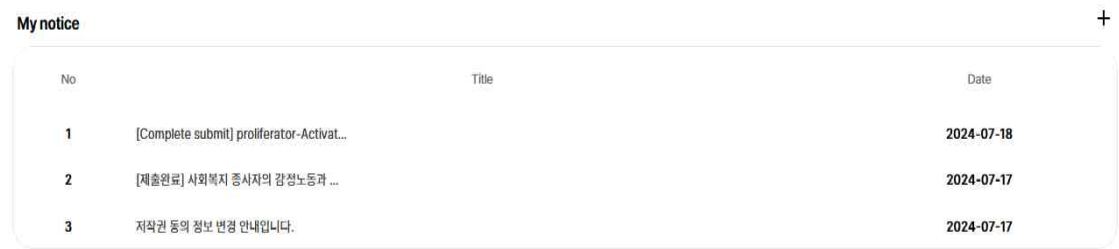
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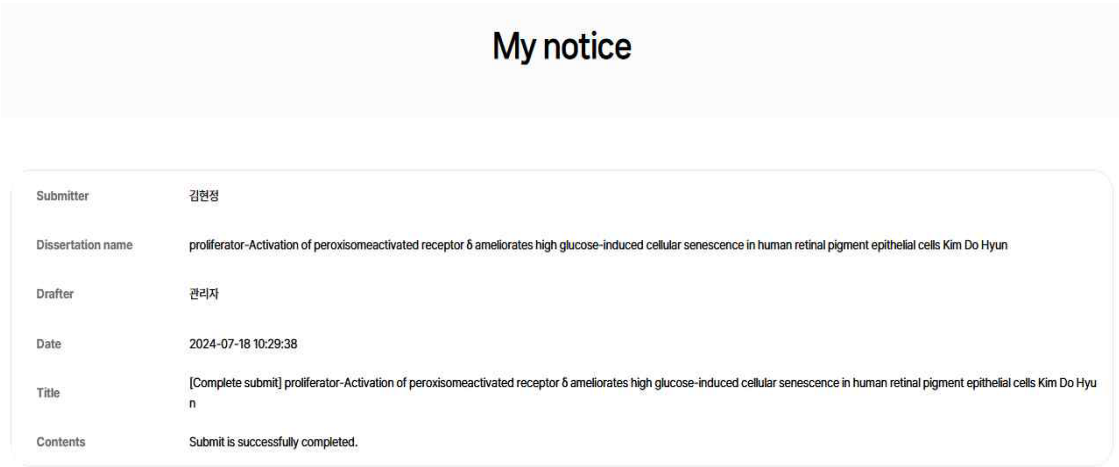
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