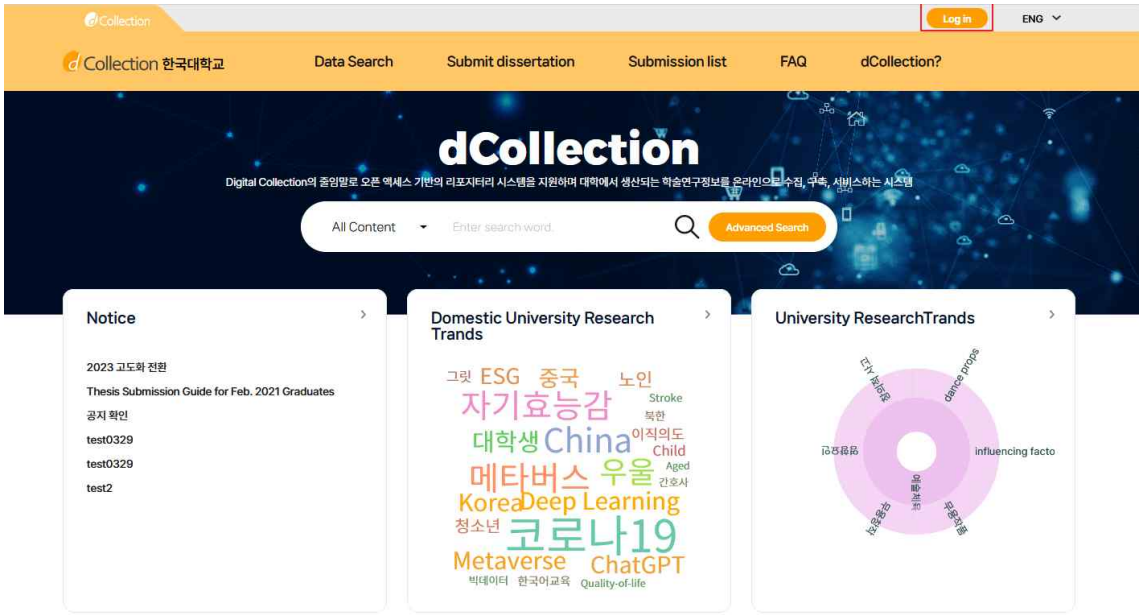
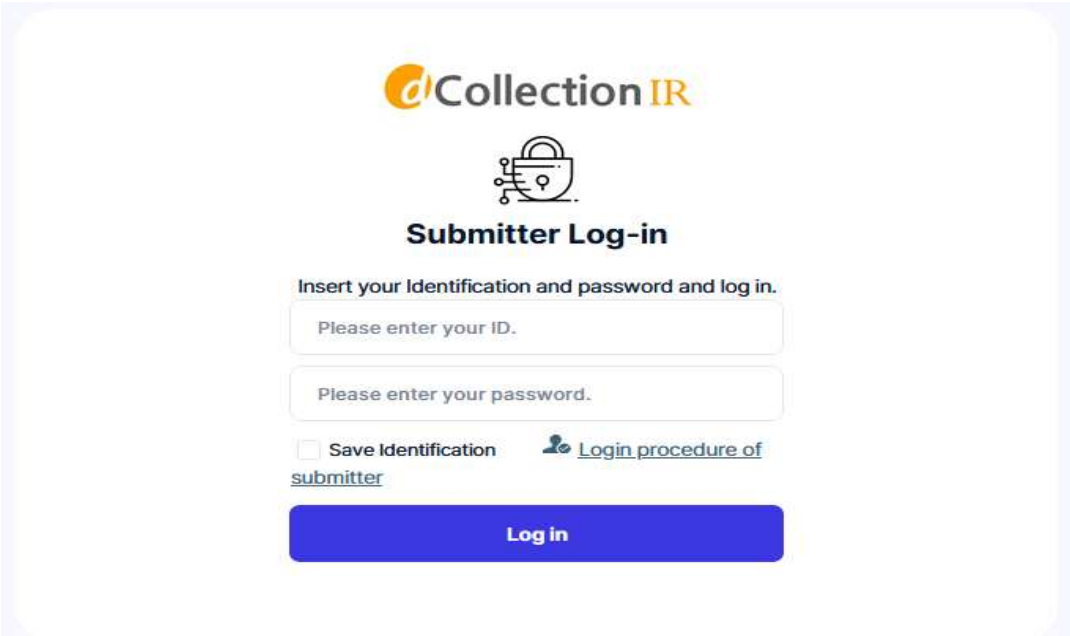
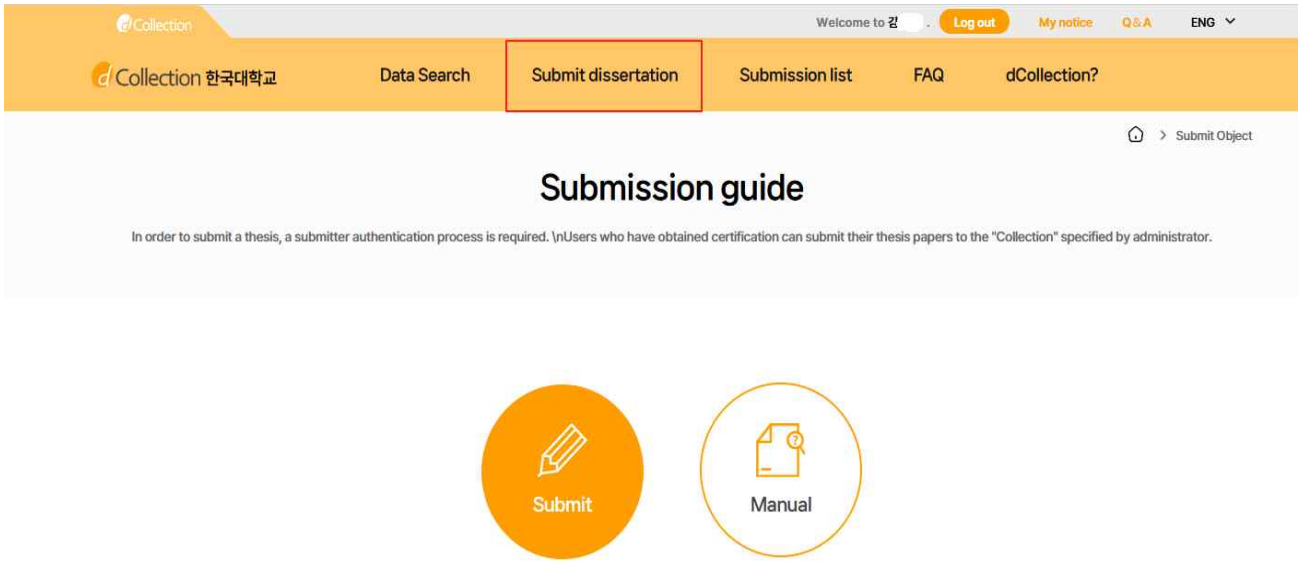
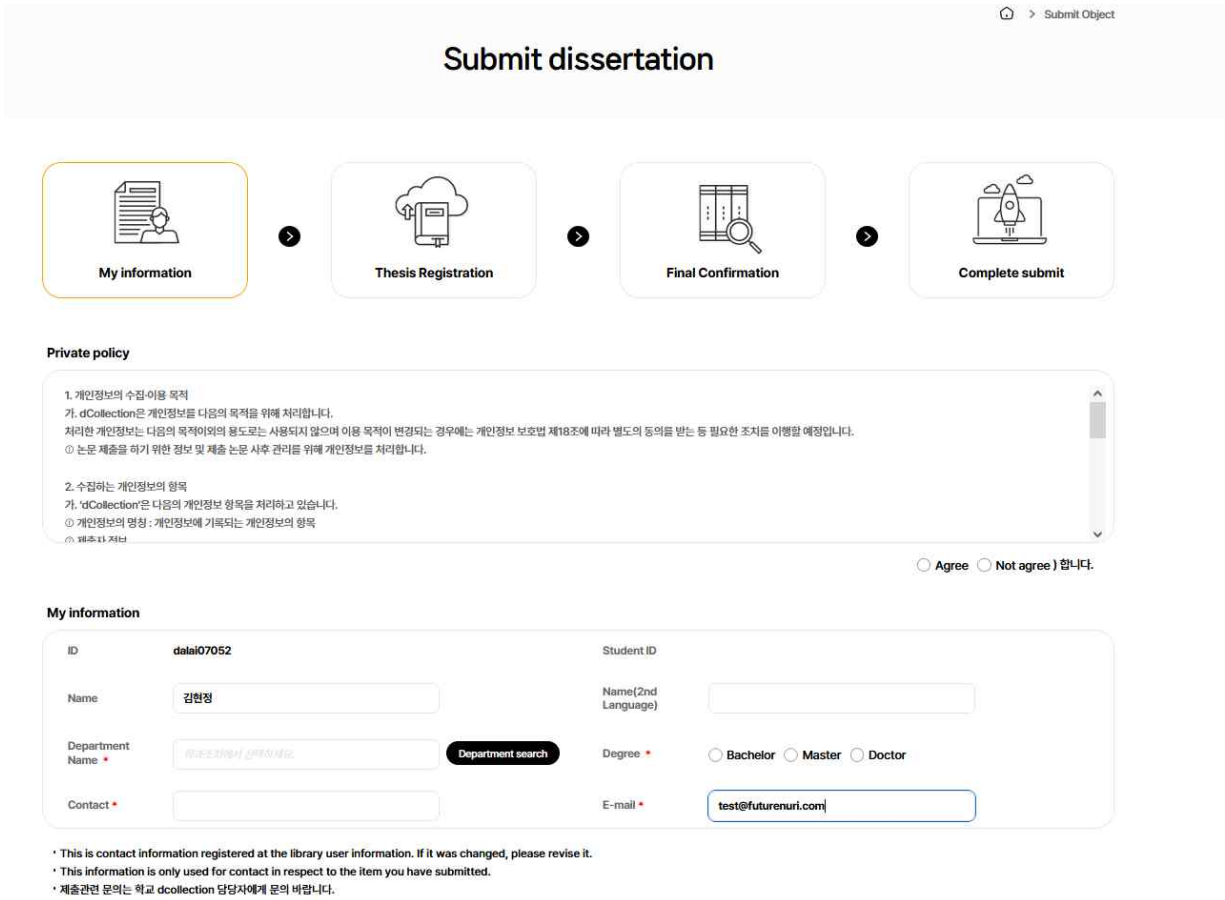




1. Select the login button.
2. To log in, enter the submitter's ID (library account ID–student number, faculty number, or portal ID) and password.
3. First-time users of the dCollection system should click the **"Submitter Login Authentication"** icon on the login **page** to authenticate themselves. After authentication, a URL to set a password will be sent via email. You can log in after setting the password.
4. After logging in, submitters can start the submission process by selecting the "Submit Material" menu.



1. Clicking the "Submit Material" button will take you to the screen for entering submitter information.
2. You will be directed to the collection selection screen if multiple collections are submitted.
3. Refer to the submission manual when submitting materials.



1. Choose whether to agree to the collection and use of personal information.

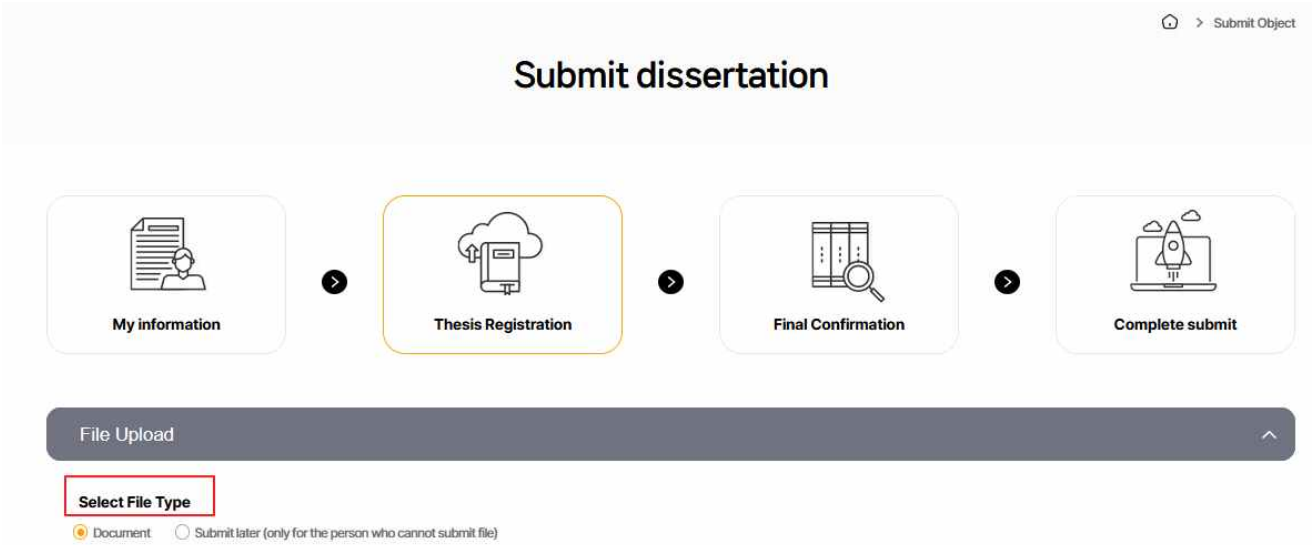
2. At the submitter information verification step, confirm the submitter's basic information (ID, student/faculty number, name, department name, degree) and contact details (phone, mobile number, email address).

※ The English name is only required for institutions where it is mandatory or if the administrator has registered it in English.

3. If there are changes to your contact information, please update your submitter's contact details.

※ Library administrators will only use this information to contact you regarding your submitted thesis.

4. Clicking the "Next Step" button will take you to the thesis registration screen.



1. The thesis registration step is divided into file registration, thesis information registration, and copyright settings. In the file registration step, you register the full text of your submitted thesis.

2. First, select the method for submitting the full text.

※ The types of full-text submission options may vary depending on the administrator's settings.

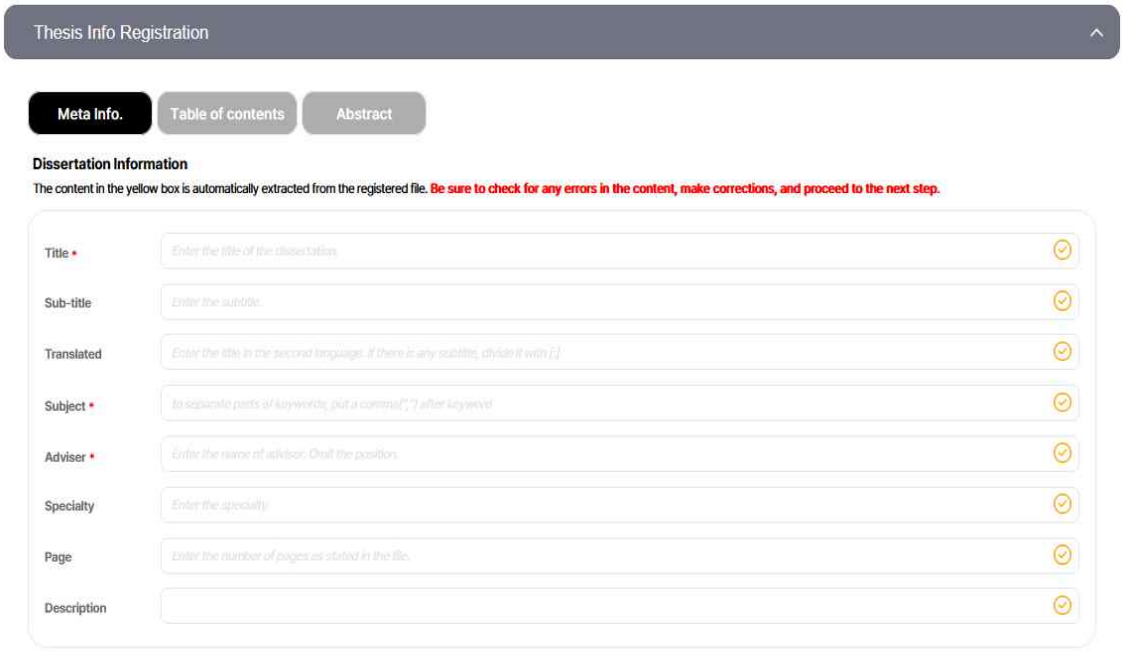
@ Document (PDF): Only PDF files can be submitted

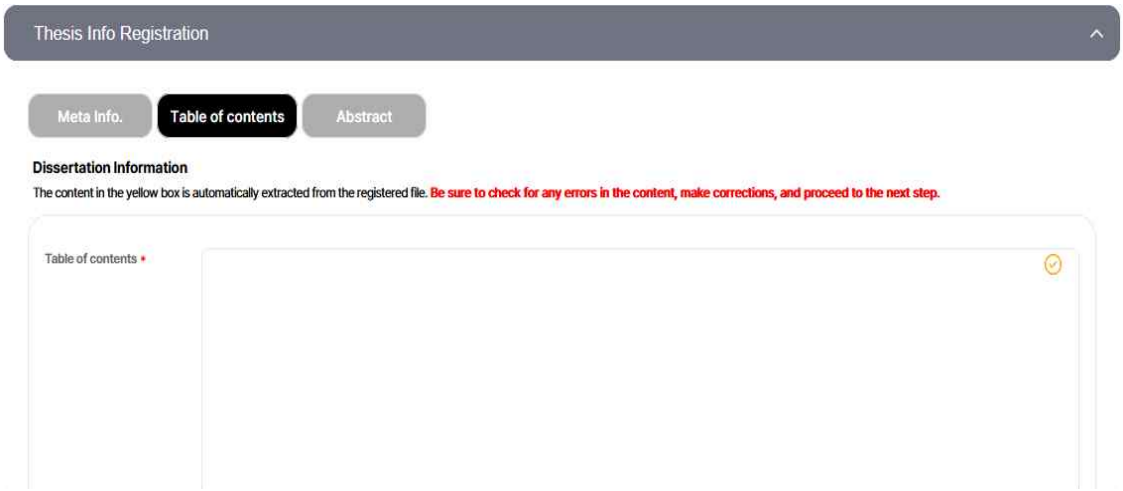
* The file registration document may differ depending on the school's settings

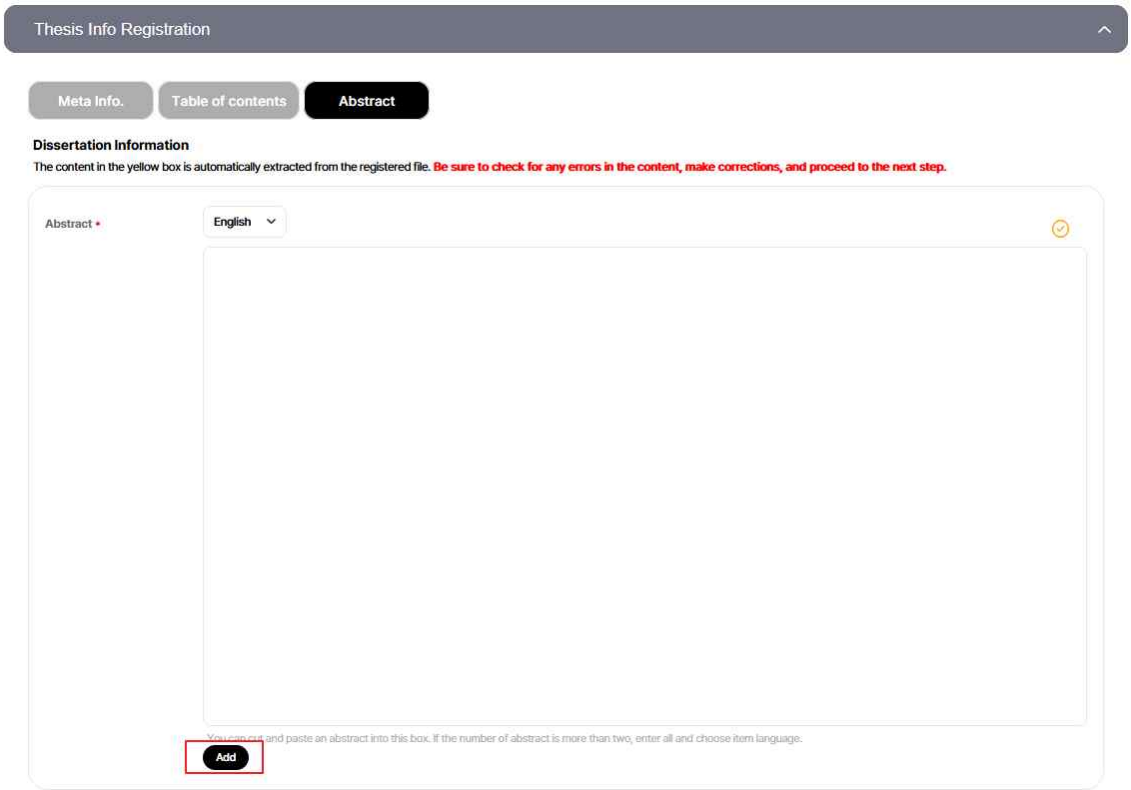
@ Separate submission: For cases where the full-text file is too large for online submission (over 100MB), when you want to submit the full-text file directly (CD or diskette), or when submitting in book form

3. File Registration

(1) **Input method when selecting full-text registration OFF**



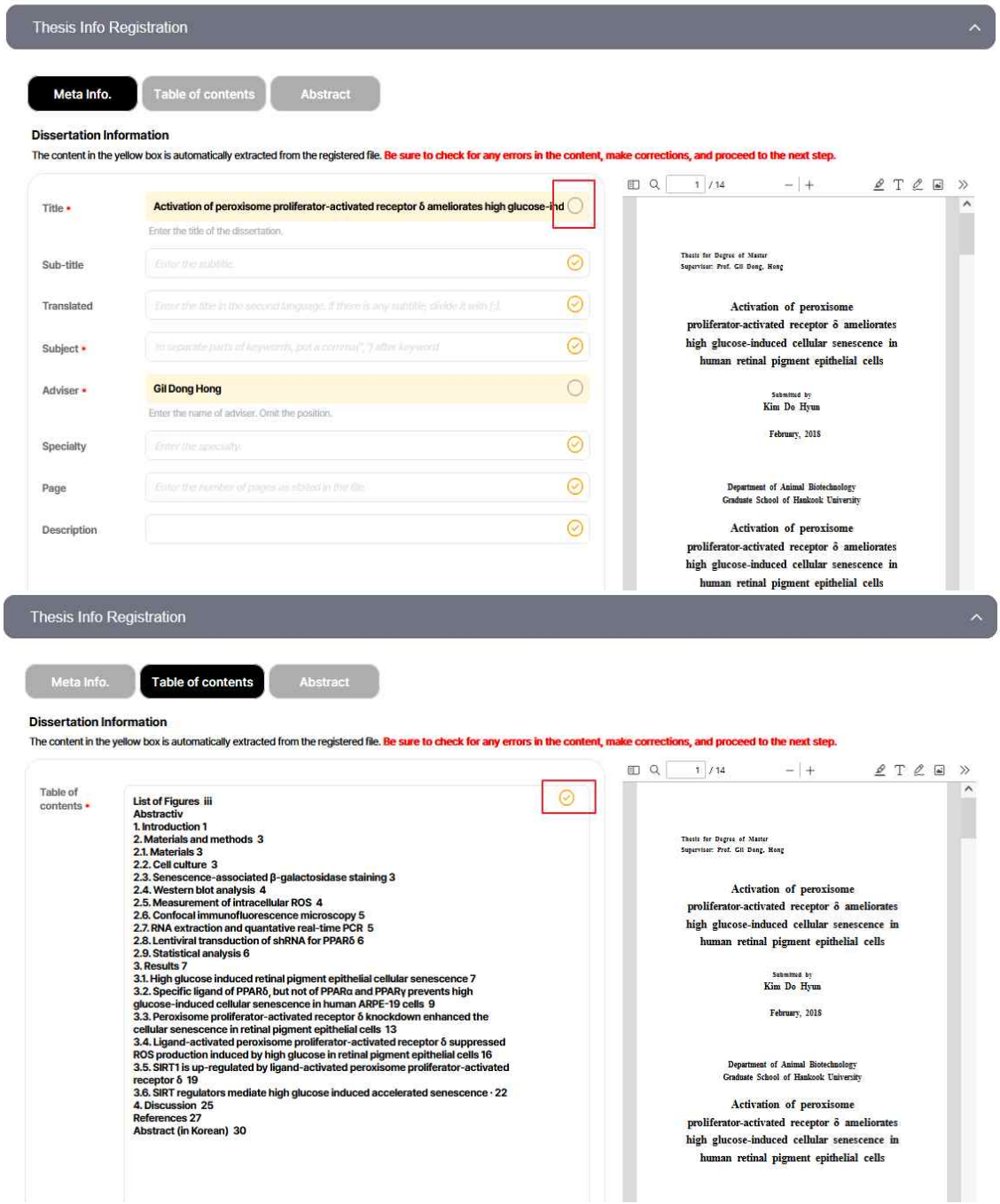




- When setting the button next to the full-text registration to OFF and attaching a PDF file, directly input basic information (title, subtitle, title (2nd language), subject (keywords), advisor, detailed major, etc.).

※ Items marked with * on the input screen are mandatory and must be entered. If multiple keywords exist, separate them with a [,] in the input field. After entering the basic information, input the table of contents and abstract. If the abstract is in two or more languages, click the add button below to enter abstracts in other languages.

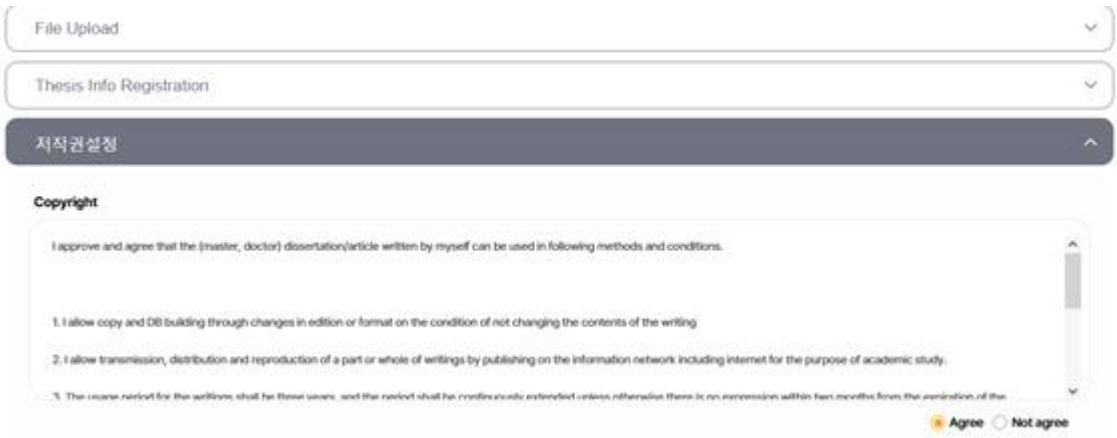
(2) Input method when selecting full-text registration ON



- When setting the button next to the full-text registration to ON and attaching a PDF file, the basic thesis information (title, subtitle, title (2nd language), advisor, table of contents, abstract) will be automatically extracted in the thesis information registration–basic information section.

If keywords and some required items are not extracted, enter them manually. Check the accuracy of the extracted content and mark the circle to confirm.

4. After confirming the thesis information registration (basic information, table of contents, abstract), proceed to copyright settings.



1) In the copyright agreement step, select whether to agree to the copyright for the submitted thesis.

(1) When agreeing to the copyright - **Select the CCL license.**

If you agree, the submitted thesis will be converted to PDF format and available to general users.

(a) In the license (CCL) settings, set options for non-commercial use, no derivatives, and share-alike.

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@ Non-Commercial

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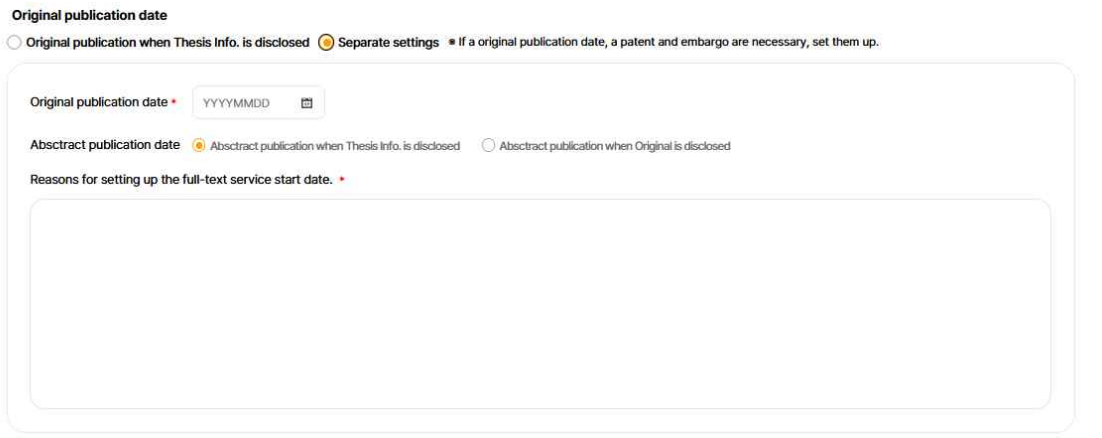

(2) When not agreeing to the copyright - Enter the reason for not agreeing.

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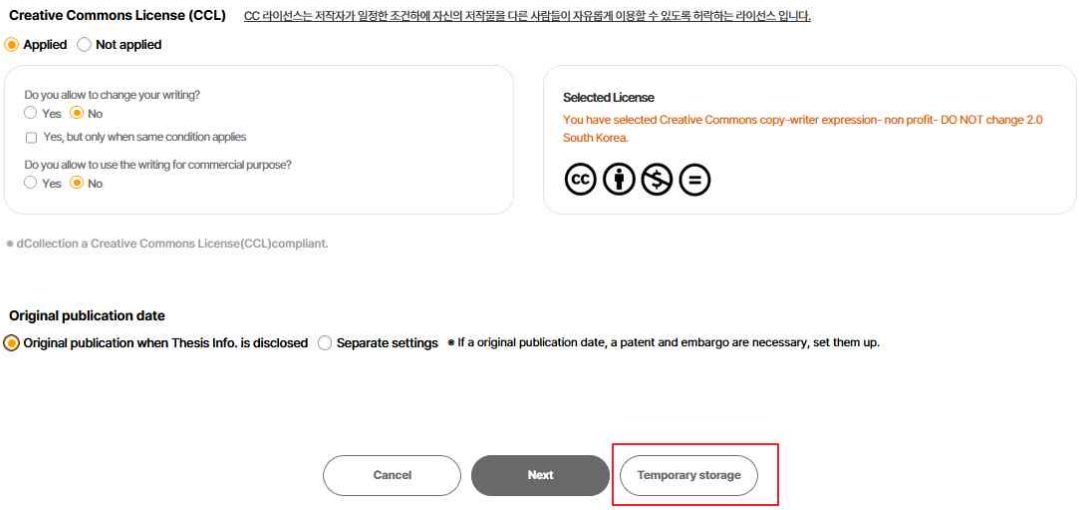
2. I allow transmission, distribution and reproduction of a part or whole of writings by publishing on the information network including internet for the purpose of academic study.

3. The usage period for the writings shall be three years, and the period shall be continuously extended unless otherwise there is no expression within two months from the expiration of the

○ Agree ◉ Not agree

저작권 비동의 사유 *



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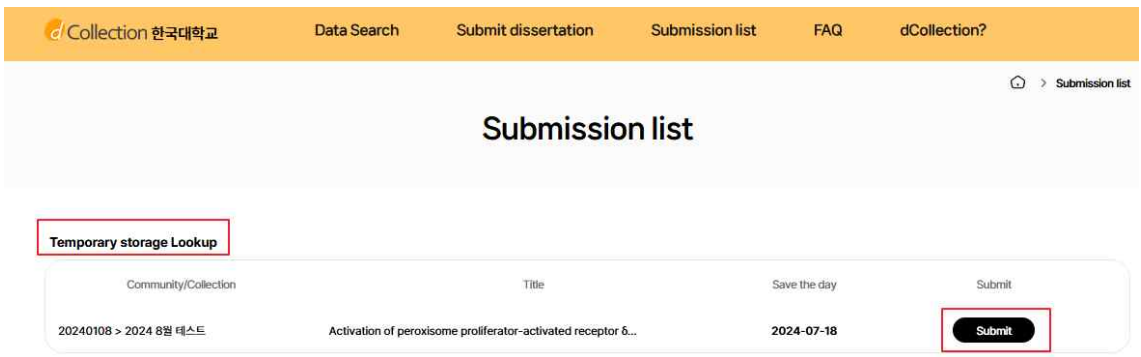

Dissertation Information

Title	proliferator-Activation of peroxisomeactivated receptor δ ameliorates high glucose-induced cellular senescence in human retinal pigment epithelial cells Kim Do Hyun
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 incr easing 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 e pithelial 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 hum an 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 sene scence 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 gene ration 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 a bolished on cellular senescence compared with shControl-ARPE-19 cells. Treatment of GW501516 inhibited the high glucose-induced generation of reactive oxygen species (ROS) in s hControl-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 SI RT1 in time- and concentration-dependent manners. In addition, GW501516-activated PPARδ recovered high glucose-inhibited expression of SIRT1. Finally, GW501516-induced inhibiti on of cellular senescence treated with D-glucose restored by pre-treatment of SIRT1 inhibitor. Thus, current study indicated that GW501516-induced PPARδ activation significantly sup pressed high glucose-induced cellular senescence via upregulating the expression of SIRT1 in human ARPE-19 cells. keyword : High glucose; oxidative stress; cellular senescence; hu man adult retinal pigment epithelial cell; PPARδ; SIRT1
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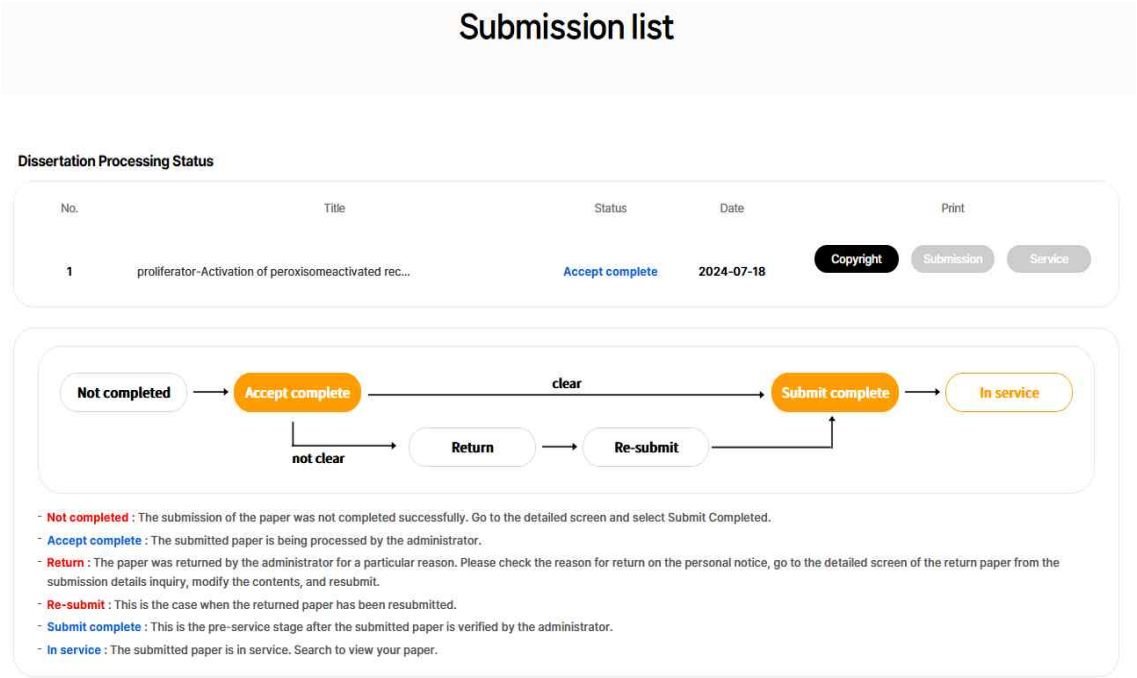

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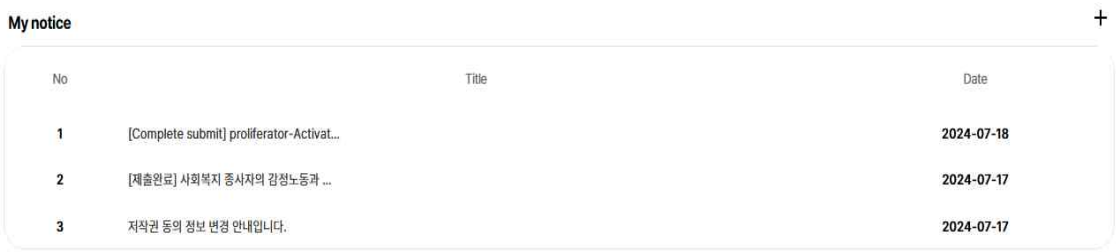

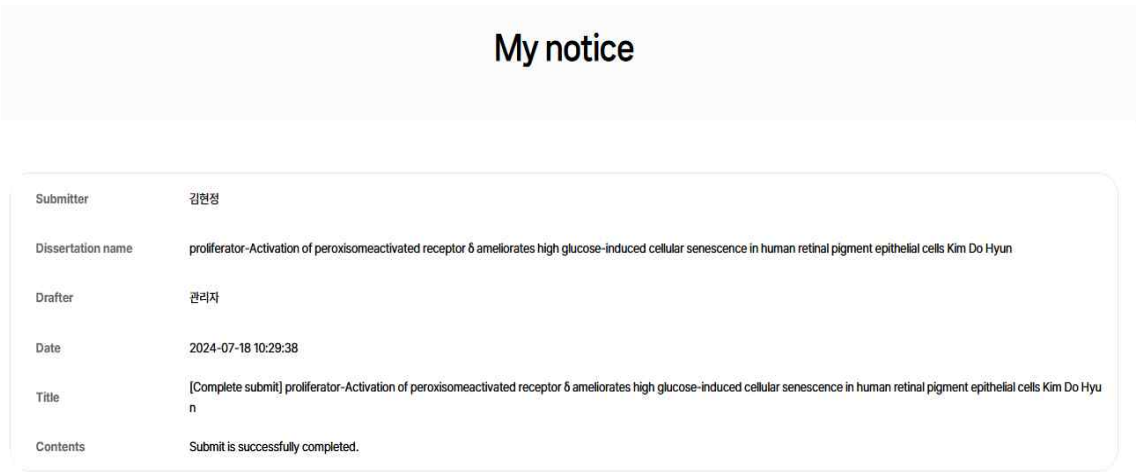

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