

Category (科目区分)	Cluster of Metabolism and Information Systems		
Course Title (授業科目名)	Metabolism		
Instructors (担当者名)	Yosuke Shimodaira	Academic Year (配当年次)	1,2
Required Course / Elective Course (必修/選択)	Elective Course	Credits (単位数)	1
Class Format (授業形態)	Lecture		
Schedule (開講期間)	4/1/2026-3/31/2027		
Class Date/Period (開講曜日・時間)	to be determined		
Course Outline/ Course Objectives (授業の概要・到達目標)			
<p>Aim: To learn the intestinal immune system and the pathogenesis of inflammatory bowel disease Goal: To understand and be able to explain the pathogenesis of IBD Summary of this class: Gastrointestinal tract is luminal organ from the mouth to the anus and plays a role in the digestion and absorption of water and nutrients. Intestinal homeostasis is tightly regulated by complex mechanisms of the intestinal mucosal immune network against to foreign substances such as intestinal microorganisms and dietary antigens. Inflammatory bowel disease include ulcerative colitis (UC) and Crohn's disease (CD) which are chronic intestinal inflammatory diseases increasing globally. Although the main cause is not clear yet, many genetic and environmental factors have been involved in the development of IBD. Inflammation mainly occurs in colonic mucoasa from the rectum to proximal continuously in UC, on the other hand, CD causes discontinuous inflammation of the entire gastrointestinal tract from the mouth to the anus, and is characterized by intestinal stenosis and fistula formation with granulomas. Lots of susceptibility genes related to intestinal immunity have been identified in IBD. Susceptible host affected by environmental factors could be the key for the initiation of IBD. In recent years, various molecular-targeted therapeutic agents such as biologics and small-molecules have been developed and clinically applied in the field of IBD. Part of the pathophysiology of IBD can be understood from the effectiveness of these drugs. In our laboratory, we are analyzing the relationship between endoplasmic reticulum stress, autophagy and enteritis. The endoplasmic reticulum stress is caused by the accumulation of excess proteins and denatured proteins in cells, and promotes suppression of transcriptional translation involved in protein synthesis and protein degradation. When the abnormal protein cannot be processed, apoptosis is finally initiated, leading to cell death. It has been suggested that endoplasmic reticulum stress, which is an important cell function maintenance mechanism that controls protein quality in cells, is associated with inflammatory bowel disease. In this lecture, we will introduce the pathophysiology of IBD, and based on that, we will read and discuss related basic papers. Both clinical and basic medicine are important for the development of medicine, and it should be useful for future clinical practice and setting of research subjects to spend time to study for basic medicine in a graduated school.</p>			
Course Planning (授業計画)			
	Course Outline/ Course Objectives (授業の概要及び到達目標) (Contents of Class) (授業内容)	Instructor (担当教員名)	Department (講座名) Class Room [実施場所]
	Pathogenesis of Inflammatory Bowel Disease	Yosuke Shimodaira	Department of Gastroenterology and Neurology
Grading Criteria (成績評価の基準と方法)			
30 hours of lectures + 15 hours of practical training, 45 hours in total, will be one credit, and the evaluation will be made in consideration of the attendance status, the results of oral examinations or written examinations, and the contents of the submitted report.			
Contact Information (問い合わせ先(氏名, メールアドレス等))			
Name: Yosuke Shimodaira / E-mail: yosuke.shimodaira@med.akita-u.ac.jp			
Comment (その他特記事項)			
<p>Information about courses: If you cannot attend the training due to work, such as a graduate student who is a member of society, we will adjust the schedule. Textbooks / References: Distribute materials as needed. Alternatively, specify the document. Self-study content during self-study time: It is desirable to carry out preparatory learning according to the goals to be achieved and the content of the lesson.</p>			