大学院特別講義のご案内

◆ **Date： 2025 December 9th（12月 9 日（火）） 17:00-18:30**

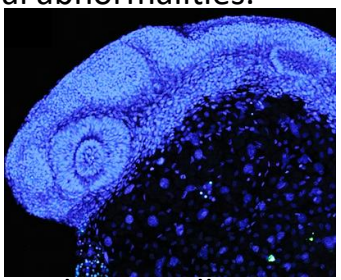
◆ Room： **大講義室**

Lecturer：Dr. Hiroshi Kurosaka（17:00-17:30）

Associate Professor. Department of Orthodontics and Dentofacial Orthopedics, Graduate School of Dentistry, The University of Osaka

Title：Cranial Neural Crest Cells: Significance for Understanding Craniofacial Diseases and Evolution

Neural crest cells are multipotent cells that arise from the neural ectoderm through epithelial–mesenchymal transition. Cranial neural crest cells contribute to the majority of craniofacial mesenchyme and differentiate into various facial structures in response to region-specific signaling pathways. In this lecture, we will demonstrate that retinoid signaling plays a critical role in specifying cranial neural crest cells toward a frontonasal-specific fate. We will also discuss how these insights enhance our understanding of the etiology of rare human diseases associated with craniofacial abnormalities.



Lecturer：Dr. Kristin Watt（17:30-18:30）

Assistant Professor. Department of Craniofacial Biology
University of Colorado Anschutz Medical Campus, Aurora, CO, USA.

Title：Ribosome Biogenesis in Craniofacial Development and Disease

Craniofacial development is a complex morphogenetic process that begins early in embryogenesis. Neural crest cells are a migratory progenitor cell population critical for forming much of the craniofacial bone, cartilage, and connective tissue. Disruptions in the development of neural crest cells or their differentiation underlie a variety of craniofacial syndromes. These include mandibulofacial dysostoses such as Treacher Collins syndrome (TCS), which is characterized by features including mandibular and malar hypoplasia, and microtia, although the severity is quite variable. Surprisingly, TCS is associated with disruptions in ribosome biogenesis, an essential process in all cells. To understand how disruptions in ribosome biogenesis specifically affect craniofacial development, we previously established zebrafish and mouse models with mutations in RNA Polymerases I and III. This work demonstrated that disruptions in ribosome biogenesis resulted in increased cell death of neural crest cell progenitors and raised the possibility of tissue-specific requirements for ribosome biogenesis and translation in neural crest cells. Our most recent studies have demonstrated that there are distinct responses to reduced ribosome biogenesis and translation within the developing craniofacial cartilage in zebrafish models. These data support the idea that there are unique requirements for ribosome biogenesis and translation during craniofacial development that contribute to disease presentation. Future studies using these models will further our understanding of disease etiology and treatments for craniofacial syndromes.

*These lectures will be conducted in English.

問い合わせ先：（顎顔面口腔矯正学教室 山城・黒坂 内線2958）