2021년도 제1차 한국유전학회 미니심포지엄

일시: 2021년 6월 17일(목요일), 15:00-17:00

방법: 온라인 심포지엄 (Zoom 이용), 연사발표 20분, 토론 10분

- Zoom 회의 참가:
https://zoom.us/j/98709344390?pwd=VlRQQVhNcC9BL1Z3UUFpQ1doQTI1Zz09

- 회의 ID: 987 0934 4390

- 암호: 509699

♦ 일정

15:00-15:05 [미니심포지엄 안내] 이경호 박사 (학술위원 한국생명공학연구원)

15:05-15:10 [학회장님 인사말씀] 정기화 교수 (한국유전학회 학회장 공주대학교)

[미니심포지엄 강연 1~3] 좌장 김미경 교수 (한국유전학회 학술부위원장 삼육대학교)

15:10-15:40 [강연 1] 김석형 박사 (제주대학교 해양과학연구소)

15:40-16:10 [강연 2] 황하영 박사 (대구경북첨단의료산업진흥재단 신약개발지원센터)

16:10-16:40 [강연 3] 이경호 박사 (한국생명공학연구원 항암물질연구센터)

16:40-16:50 [총평] 김미경 교수 (한국유전학회 학술부위원장 삼육대학교)

16:50-17:00 [종료 및 안내] 이경호 박사 (한국유전학회 학술위원 한국생명공학연구원)

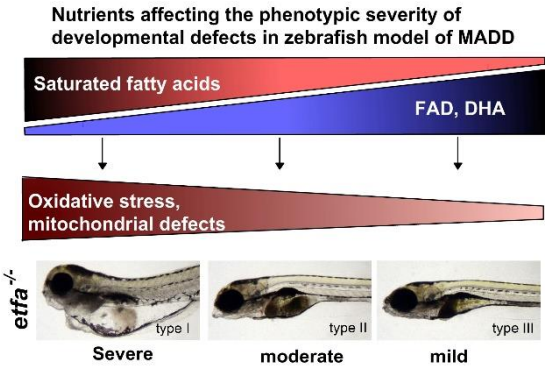
[강연 1] 김석형

(제주대 해양과학연구소, Zebrafish team director)

♦ 발표초록

Nutritional factors affecting the phenotypic severity of Multiple Acyl-CoA Dehydrogenase Deficiency (MADD)

Multiple acyl-CoA dehydrogenase deficiency (MADD) is a severe mitochondrial genetic disorder caused by mutations in the electron transfer flavoprotein complex or riboflavin metabolism associated genes. Although the severity of developmental defects in MADD is dependent to the type of mutation (mild to moderate defects by hypomorphic mutations and severe defects by null mutations), it is also obvious that the severity of developmental defects differs in siblings with identical mutations – pointing to an environmental component of the disease. Such data raise the possibility that maternal nutrients may affect the severity of developmental defects in MADD. Currently, there is a knowledge gap regarding the impacts of nutritional factors on developmental defects in MADD. Furthermore, although riboflavin supplementation appears to be effective for MADD patients with mild symptoms, there is no effective treatment for MADD patients with severe developmental defects. Here, we report current results about impacts of nutritional factors on the phenotypic severity of developmental defects in MADD and new therapeutic targets that might attenuate developmental defects in MADD.



[강연 2] 황하영

(대구경북첨단의료산업진흥재단, 신약개발지원센터, 책임연구원)

♦ 발표초록

Drug metabolism and pharmacokinetics in drug discovery

Drug discovery requires diverse scientific research areas including chemistry, biology, veterinary medicine, structural biology and pharmacology. In pharmacology research, pharmacokinetic study is very important to discover and develop drug-like compounds because blood concentrations of drugs are closely associated with efficacy and safety. Pharmacokinetic properties including maximum and total concentrations, half-life, and clearance in blood can be determined by ADME properties (absorption, distribution, metabolism, and excretion). Therefore, drug discovery researchers assess ADME properties of developing compounds as well as efficacy and toxicity. Many kinds of in vitro assays to figure out ADME properties of compounds have been developed, and US/Europe medicine agencies have been providing guidance in respect of DMPK (drug metabolism and pharmacokinetics) study. Today's presentation overviews the importance of pharmacokinetics in drug discovery and diverse assays used for determination of ADME properties followed by international guidance.

[강연 3] 이경호

(한국생명공학연구원, 항암물질연구센터, 선임연구원)

♦ 발표초록

Role of Wnt signaling in primary cilia assembly and disassembly

The primary cilium is a non-motile microtubule-based structure, which functions as an antenna-like cellular sensing organelle. The primary cilium is assembled from the basal body, a mother centriole-based structure, during interphase or a quiescent cell stage, and rapidly disassembles before entering mitosis in a dynamic cycle. Defects in this ciliogenesis dynamics are associated with human diseases such as ciliopathy and cancer, but the molecular mechanisms of the ciliogenesis dynamics are still largely unknown. To date, various cellular signaling pathways associated with primary cilia have been proposed, but the main signaling pathways regulating primary cilia assembly/disassembly remain enigmatic. In this seminar, I will focus on the Wnt signaling pathway in the regulation of primary cilia assembly and disassembly.