whole-mount cornea on day 14 post-inoculation. Anti-LYVE-1 antibody, anti-CD11b antibody and anti-F4/80 antibody were used for immunostaining. Corneal infection was graded by a previously reported method and central corneal thickness as a measure of corneal edema was evaluated by anterior segment optical coherence tomography（CASIA SS-1000 ; Tomey, Nagoya, Japan）.

【Results】 Lymphangiogenesis was significantly reduced by macrophage depletion on day 14 post-inoculation. There is no significant difference the control group and the macrophage depleted group on day 7 postinoculation. However, clinical infection score and corneal edema significantly increased in the macrophage depleted group on day 14 post-inoculation.

【Conclusions】 These results suggest that the process of lymphangiogenesis in bacterial infection of the cornea presumably suppresses keratitis in the late stage of infection.

P1-05.

Amyloid and tau positron emission tomography in diabetes-related dementia

（社会人大学院博士課程 3 年高齢総合医学）
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Our proposed clinical entity, referred to as diabetes-related dementia（DrD）, describes a dementia state predominantly associated with type 2 diabetes mellitus（DM）-related metabolic abnormalities. We studied 11C-PiB and 11C-PBB3 positron emission tomography（PET）in 29 subjects with DrD and 5 subjects with Alzheimer disease（AD）associated with DM to assess amyloid and tau deposits in the brain. Different from AD, only 11 out of 29 subjects（38%）with DrD showed positive PiB, whereas 17 out of 19（89%）showed positive PBB3. Depending on positivity of PiB and PBB3, we classified subjects with DrD into a negative PiB and positive PBB3 pattern（53%）, indicating tauopathy, a positive PiB and positive PBB3 pattern（32%）, indicating AD pathology, or a negative PiB and negative PBB3 pattern（16%）, indicating non-specific neuronal damage. DrD showed variable amyloid and tau accumulation patterns in the brain. DrD may be associated predominantly with tau pathology, in addition to AD pathology and non-specific neuronal damage due to DM-related metabolic abnormalities.

P1-06.

Analysis of aggregated proteins in HSPB8 myopathy using zebrafish models

（病態生理学）
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Heat shock protein B8（HSPB8）, a member of the small heat shock protein family, is known to have chaperone activity and be involved in protein quality control. Previous studies reported that mutations in *HSPB8* cause several neuromuscular diseases. Recently, two novel candidate mutations of *HSPB8* were identified in families with protein-aggregated myopathy. However, the pathogenic mechanisms of HSPB8 myopathy remains to be elucidated. In this study, we firstly establish zebrafish models of HSPB8 myopathy to confirm the pathogenicity of these novel *HSPB8* mutations. We also tried to identify abnormal aggregated proteins for the purpose of clarifying the pathological mechanisms of HSPB8 myopathy. We carried out microinjection of wild-type or mutant human *HSPB8* mRNA in zebrafish embryos at 1-2 cell stage. Then we analyzed phenotype of these fish at 5 days post-fertilization. Overexpression of mutant *HSPB8* mRNA resulted in morphological abnormalities at higher rate compared to expressing wild-type *HSPB8* mRNA-injected and uninjected fish. Furthermore, it revealed that these abnormal fish had severe muscle degeneration and protein aggregation. Our data suggest that the

novel mutations of *HSPB8* may cause myopathy with protein aggregation.

P1-07.

Do sedative antihisitamines prolong febrile seizure duration?

（社会人大学院博士課程 3 年小児科学）
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【Background】 Use of sedative antihistamines（sAH）is not recommended in the guideline released by the Japanese Society of Child Neurology in 2015 because sAH might prolong durations of febrile seizure. However, reports about effect on febrile seizure by sAH are limited. Moreover their results show diverse outcomes. In this study, we used a template for febrile seizure in daily medical examination and performed a cross-sectional study to evaluate the relation between the febrile seizure duration and antihistamines.

【Methods】 We collected the data of 475 patients who visited St. Luke's International Hospital due to febrile seizure from August 2013 to February 2016. Patient with epilepsy, perinatal disorders, developmental disorders, acute encephalopathy or encephalitis and aged less than 6 months old or over 6 years old were excluded. We defined the seizure duration as a primary outcome, and performed univariate and multivariate analysis.

【Results】 In 475 patients, sAH group who were given sAH were 24 cases aged 10 months to 5 years-old. The control group were 425 patients who where administered neither sedative nor nonsedative antihistamines. The median seizure duration of seizure was all 3 minutes, as well as there was no statistical difference within 3 groups（sAH group, nonsedative antihistamines group, and control group）by using the univariate and multivariate analysis（p>0.05）.

【Conclusion】 Since there was no statistical difference in the seizure duration between patients with and without sAH, the uniformly recommendation about the use of sAH should be reconsidered.

P1-08.

シナプトタグミンアイソフォームに依存したダイナミン駆動シナプス小胞リサイクリング

（細胞生理学）
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神経終末に到達した活動電位により放出された神経伝達物質が神経信号伝達を担っており、安定したシナプス伝達の維持には多様な活動電位発火に応じたシナプス小胞リサイクル経路の活性化が必須である。培養ラット上頸交感神経節細胞では、活動電位の生理的発火パターンの違いが、神経終末への小胞膜再取り込みに重要なダイナミン 1, 2, 3 のいずれかを活性化して速さの異なる小胞リサイクル経路を駆動することを既に報告している。しかし、活動電位発火に応じた小胞リサイクル経路選択に機能する分子機構の詳細は未だ解明されていない。これらの小胞リサイクル経路選択機構には活動電位に伴って上昇する神経終末内 Ca^{2+} 濃度が重要であると考え、本研究では Ca^{2+} センサー蛋白質であるシナプトタグミンに着目して、ダイナミン活性化による小胞リサイクル経路選択の分子機構を推測した。

siRNA を導入してシナプトタグミン 1, 2 の発現を抑制した培養ラット上頸交感神経節シナプス前細胞（Syt1-KD, Syt2-KD）に、5 Hz で 4 分間連続発火させて小胞放出部位のシナプス小胞を枯渇させた後の興奮性シナプス後電位を測定し、小胞補充速度を推測・比較した。その結果、Syt1-KD は速い小胞補充を遅延し、Syt2-KD は速い小胞補充と遅い小胞補充の両方を遅延した。

これらの結果から交感神経系では、シナプトタグミン 1 はダイナミン 1 依存的な速い小胞補充を担う経路を、シナプトタグミン 2 は速い小胞補充経路に加えてダイナミン 3 依存的な遅い小胞補充を担う経路も駆動することが示唆された。