CAMD セミナー

(Center for Development of Advanced Medicine for Dementia)

The importance of old proteins for age-related diseases

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平成 26 年 9 月 8 日(月)午後4時～5時
第 1 研究棟 2 階 大会議室

加齢に伴う蛋白変性研究で著名なTruscott先生のご講演です。先生は老人性白内障における水晶体蛋白の加齢性変性メカニズムを解明され、最近では脳のミエリン塩基性蛋白（MBP）の変性がMBPを主標的とする自己免疫疾患である多発性硬化症の発症原因となっている可能性を示されるなどご活躍されています。

The human body appears to contain a number of long-lived proteins. A key question is, what happens to these proteins as we age and what are the consequences of this deterioration? Over time, the crystallin proteins of the human lens, which do not turn over, progressively degrade. Using proteomic techniques we have been able to elucidate the major modifications that take place with age. The main processes were found to be racemisation, deamidation, and truncation.

In many cases, the extent of modification at particular sites did not differ between the normal and the cataract lenses, however at certain sites there was a significantly greater degree of modification in the cataract lenses. Cataract appears to result from site-specific decomposition of particular long-lived macromolecules in the human lens.

We are now exploring the extent to which the lens data can be extrapolated to other long-lived proteins, particularly those found in the brain. I will present very recent data on myelin basic protein (MBP). MBP appears to be a life-long protein and it undergoes many modifications as we age. Specific MBP modifications may play a role in the onset of multiple sclerosis.

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