Division of Bio-Medical Informatics  
Department of Epidemiology

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In this department, Hirofumi Maruyama took up its new post on August 1, 2006.  
A main study has been aimed for epidemiological studies on the late effects of atomic bomb (A-bomb) survivors and the actual conditions from socio-medical viewpoints. A purpose as this master does not change, but it passes after being bombed in 60, and A-bomb survivors have gone old aged, and a lifestyle-related disease and a problem in geriatrics become important as a healthy problem of A-bomb survivors, too. Therefore, as well as a cohort study based on a database; we take in the new study method how we used molecular markers and genetic polymorphic markers for, and neurological diseases to increase with senior people.

1. A-bomb survivors cohort study.

Kawakami H, Maruyama H, Morino H, Sumida O*, Hiraoka M* (*International Radiation Information Center)

Purpose: From November, 1965, we followed it in “Hiroshima atom bomb Survivors cohort” resident in Hiroshima from biological influence and points of view such as the later living environment of survivors. There is it for the purpose of making basics document contributing to health care / the welfare of A-bomb survivors. As for the examination problem, next is put up.

1) Analysis by A-bomb dose based on ABS93D  
2) Analysis of exposure status  
3) Analysis by the distance from the hypocenter  
4) Analysis of the effects by environment factors  
5) Study of disease development by family analysis

2. Study of hereditary spinocerebellar degeneration (SCD)

Morino H, Maruyama H, Liu L, Tanaka E, Kawakami H, Ito H*, Kusaka H* (*Kansai Medical School, Department of Neurology)

Purpose & Results: One third of the patients with SCD are hereditary, and many types of Spinocerebellar degeneration are caused by extension of three base repeat to call “triplet repeat disease”, but there is still a group of unknown causative gene. We study it aiming at discovery of a new causative gene. We also reported the
first neuropathological study of SCA8.

3. Collaborative analysis of alpha-synuclein gene promoter variability and Parkinson’s disease

Kawakami H, Morino H, Maruyama H, Genetic Epidemiology of Parkinson’s Disease Consortium.

Purpose, Methods and Results: Parkinson’s disease shows the pathology of accumulation of Lewy-body in the brainstem. alpha-synuclein is a major constituent of Lewy-body, and the triplication and the mutations of the gene cause hereditary Parkinson’s disease. To determine whether allele-length variability in the dinucleotide repeat sequence (REPI) of the SNCA gene promoter is associated with Parkinson disease susceptibility, we performed a collaborative analysis of complete data for 2692 cases and 2652 controls. The SNCA REPI alleles differed in frequency for cases and controls (P<0.01). Genotypes defined by the 263 base-pair allele were associated with Parkinson disease (odds ratio, 1.43; 95% confidence interval, 1.22-1.69; P<.001 for trend).

4. Collaborative analysis of 13 single-nucleotide polymorphisms and Parkinson’s disease:

Kawakami H, Morino H, Maruyama H, Genetic Epidemiology of Parkinson’s Disease Consortium.

A genome-wide association study identified 13 single-nucleotide polymorphisms (SNPs) significantly associated with Parkinson’s disease. Investigators from three Michael J Fox Foundation for Parkinson’s Research-funded genetics consortia-comprising 14 teams-contributed DNA samples from 5526 patients with Parkinson’s disease and 6682 controls, which were genotyped for the 13 SNPs. Our results do not lend support to the finding that the 13 SNPs reported in the original genome-wide association study are genetic susceptibility factors for Parkinson’s disease.


CP Zabetian*,1, H Morino,1, H Maruyama, H Kawakami (*1University of Washington School of Medicine)

LRRK2 G2019S is the most common known cause of Parkinson disease (PD) in patients of European origin, but little is known about its distribution in other populations. The authors identified two of 586 Japanese patients with PD heterozygous for the mutation who shared a haplotype distinct from that observed in Europeans. This suggests that G2019S originated from separate founders in Europe and Japan and is more widely dispersed than previously recognized.

A. Original papers


Wirdefeldt*1, CP Zabetian*2, M Dehem*2,2, JS Montimurro*3, A Southwick*2,2, RM Myerz*2, TA Trikalinos*4


7. Ito, H*1, Kawakami, H, Wate, R*1, Matsumoto, S*2, Imai, T*1, Hirano, A*4, Kusaka, H*1: The Department of Neurology, Kansai Medical University, *2 Department of Neurology Kitano Hospital, *3 Department of Neurology Shiroyama Hospital, *4 Department of Pathology, Montefiore Medical Center) Clinicopathologic investigation of a family with expanded SCA8 CTA/CTG repeats. Neurology, 67, 1479-81, 2006. (I)


9. Tanimoto S*1, Tamura H*1, Ue T*1, Yamane K*1, Maruyama H, Kawakami H, Kiuchi Y*1: A polymorphism of LOC387715 gene is associated with age-related


B. Meeting Presentations


4. K Kondo*1, A Ishihara*1, H Maruyama, T Ohshita*1, T Takahashi*1, T Miyachi*1, T Kohriyama*1, M Matsumoto*1 (*1 Department of Clinical Neuroscience and Therapeutics): A case of Tolosa-Hunt syndrome: lesion detectable using 3T MRI. 95th Chugoku Meeting of the Japanese Society of Internal Medicine, Hiroshima, 2006

