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*Asia-Pacific Journal of Molecular Medicine 2017, 7 (SUPP 1)*

**Abstracts for 7th Regional Conference on Molecular Medicine (RCMM)**

 **in Conjunction with 3rd National Conference for Cancer Research 2017**

**10-12th November 2017, Auditorium UMBI, Kuala Lumpur**

**Genetic and Non-genetic Studies of Type 2 Diabetes In Three Susceptible Asian Populations: Malays, Chinese and Indian**

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**ABSTRACT**

Although numerous epidemiological studies have been conducted in various populations, the Malaysian society remains relatively understudied to date, despite having a relatively high prevalence of type 2 diabetes among Asian countries. Utilising the same samples from 1,604 Malays, 1,654 Chinese and 1,728 Indians, a total of 62 individual candidate single nucleotide polymorphisms (SNPs) previously associated with type 2 diabetes and the effects of environmental (non-genetic, or lifestyle) risk factors were assessed. After Bonferroni correction, seven (7) individual SNPs showed association with type 2 diabetes in Malaysian sample. The genetic risk score showed strong association with type 2 diabetes but the genetic risk score explained only 1.0 to 1.7% of total risk variance. In contrast, four non-genetic risk factors, age, gender, waist-to-hip ratio and physical inactivity, accounted for about 20% of total type 2 diabetes risk variation in the Malaysian samples. The effect of increasing waist-to-hip ratio was higher in Chinese than Indian or Malay participants, suggesting anthropometric risk differences between groups. We found some evidence for gene by environment effect modification, with the genetic risk score showing a gradient of decreasing effect sizes across increasing strata of body mass index. Taken together, these studies suggest that environmental, rather than genetic risk factors are the major contributors to the epidemic of type 2 diabetes in Malaysia. Our findings have some public health significance in relation to mitigating type 2 diabetes risk in Malaysia. First, these findings may inform targeted interventions focussing on abdominal obesity, especially in Chinese Malaysians. Second, these results suggest a need for the development of ethnicity-specific anthropometric cut-point. Third, these findings suggest a relatively greater contribution of genetic factors to disease among lean individuals. Future studies in larger samples could further clarify the respective roles of genetic and environmental risk factors to disease, and inform personalised interventions.