



Calcium-sensing receptor SE haplotype in patients with primary hyperparathyroidism; expected magnitude of lowered serum calcium level

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In primary hyperparathyroidism (PHT), the problem of calcium metabolism is an important problem (1). The underlying molecular mechanism is very interesting. The calcium-sensing receptor (CSR) is widely mentioned for its effect on blood calcium control (2). The genetic polymorphism of CSR is related to the different phenotypic manifestation of blood calcium level in patients with PHT (3). The CSR AQ haplotype in patients with PHT is found to relate to the lower blood calcium level (4,5). In this short report, the authors assess the molecular change due to the mutated type SE haplotype and discussed on the magnitude of lowered serum calcium level.

This work is a quantum chemistry analysis using mathematical modeling study using the same technique as previously published by Joob and Wiwanitkit (6). The basic calculation for the molecular mass in the wild and mutated types of CSR polymorphisms was done. The basic calculation for the molecular mass in the wild and mutated types of CSR polymorphisms was done. The focused studied polymorphisms are A986S is 16 and Q1011E. First, the molecular masses of the wild types (986A and 1011Q) were performed. After that, the mutation S and E are assigned to 986A and 1011Q to form 986S and 1011E mutated types. Then the similar calculation for molecular masses of the mutated types (986S and 1011E) was performed. The change of molecular mass after mutation assignment is calculated. Comparison of magnitude of change was then done. According to the analysis, the molecular change due to the mutated type of AQ haplotype is shown in Table 1. The estimated the magnitude of change in mutated types of A986S is higher than Q1011E.

Calcium is an important bio-mineral. The alteration of blood calcium is a common problem seen in PHT. Risk

■ Implication for health policy/practice/research/medical education

It was suggested that some genetic variants may predispose to calcium stone formation or hypocalcemia.

■ **Keywords:** Primary hyperparathyroidism, Parathyroid gland, Calcium, Calcium-sensing receptor, Calcium stone

of CSR genotypes are widely mentioned in the patients with PHT. It was suggested that some genetic variants may predispose to calcium stone formation or hypocalcemia. Among patients with PHT, the cases with AQ haplotypes (986A and 1011Q) are reported for clinical association with lower serum calcium levels and hypercalciuria comparing to the SQ haplotypes (3,4). Based on this work, the lowering blood calcium due to the effect of genetic polymorphism in CSR should be higher in A986S polymorphism comparing to Q1011E. In the patient with 986S, higher blood calcium level could be expected.

Authors' contribution

SY and VW wrote the manuscript equally.

Conflicts of interest

The authors declare no conflicting interest.

Ethical considerations

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Table 1. The molecular change due to the mutated type of AQ

Corresponding polymorphisms	Change of molecular mass at mutation site per mol	magnitude of molecular mass change
A986S	89 to 105	+16
Q1011E	146 to 147	+1

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