第２２回　名市大生物多様性研究センターセミナー

○ 日時：平成２５年１月１６日（水）　午後７～８時
○ 場所：名古屋市立大学　山の畑キャンパス
　　４号館（南棟）３階大講義室
○ 講師：鈴木　善幸　氏（名古屋市立大学　システム自然科学研究科、教授）
○ 題 目 ：『Roles of N-glycosylation and net-charge of hemagglutinin in influenza virus evolution』（講演は日本語）

The number of N-glycosylation sites (NGS) as well as the positive charge (+charge) of hemagglutinin (HA) are known to have increased after influenza A virus subtype H3N2 (H3N2 virus) entered the human population in 1968. Experimentally, it has been shown that N-glycans attached to NGS around the antigenic sites (AS) block the binding of antibodies (Ab) to AS and increases in the +charge enhance the receptor-binding avidity of HA. Therefore, these were considered to have contributed to the immune escape of the virus. However, since N-glycans may impair the receptor-binding avidity by covering the receptor-binding pocket and increases in the +charge may inhibit the release of progeny virus from infected cells, evolutionary mechanisms for these phenomena remain an enigma. To clarify the role of NGS in HA, we examined natural selection operating at AS before and after gains of NGS. Positive selection was detected before but not after gains of NGS, supporting the hypothesis that NGS generated in HA contributed to blocking the binding of Ab to AS. In addition, by designing a single-substitution analysis method for detecting episodic natural selection, we demonstrate that gains of N-linked glycosylation sites in HA during evolution of H3N2 virus were subject to positive selection. Although gains of NGS possibly reduced the receptor-binding avidity of HA, we observed that gains of NSG occurred almost coincidentally with increases in the +charge. These results suggest that increases in the +charge of HA, which enhance the receptor-binding avidity, may have occurred to compensate for the reduced receptor-binding avidity caused by gains of NGS during evolution of H3N2 virus.

山の畑キャンパスへの道順：http://www.nsc.nagoya-cu.ac.jp/location.html

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