中文題目：幽門桿菌感染與胃癌-胃類器官的研究

英文題目：Helicobacter pylori infection and gastric cancer - research on stomach organoids

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Background: The human gastric cancer- and normal-organoids from cancer tissues and iPS cells were established. Owing to the cellular diversity observed in the human stomachs, we attracted significant interest as a novel model system for precision medicine. However, many questions remain about the extent to which these cultures recapitulate gastro development and mechanism of *Helicobacter pylori* infected cancer progression.

Method: To clarify the recapitulation of human organoid models, we found several key points for *in vitro* culturing and differences between normal and cancerous organoids. 1. Effect of ROCK inhibitor; 2. Effect of GSK inhibitor CHIR-99021; 3. Dose of niche factors.

Results: (1) Effect of ROCK inhibitor; Rho kinase inhibitor was added to the primary cultures to inhibit anoikis and apoptosis were previously observed in purified colonic epithelial cells. (2) Effect of GSK inhibitor CHIR-99021; Treatment with CHIR-99021 ubiquitously and strongly activated beta-catenin-mediated transcription, induced discordant phenotypic alterations in intestinal organoids. Of note, CHIR treatment, in combination with histone deacetylase inactivation via valproic acid, comprised an effective procedure for the enrichment of Lgr5+ cells in intestinal organoids. Thus, we used only for 3 days and removed. (3) Dose of niche factors; R-SPONDIN 1 concentration of normal and cancer organoids are the same as 10 %, but WNT3A in cancer case is 10% and normal organoids required 20%.

Conclusion: Here, we demonstrate not only the precise culture conditions of the human gastric organoids *in vitro* to improve the accuracy of generation of organoid models for apply the therapeutic and medical use, but also the effect of the growth factors such as HDGF/TNFalpha and the antioxidation reagents/ROS controls, and for cancer progression by *Helicobacter pylori* on stomach organoids.