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申請者（ふりがな）	岩﨑　なつみ　　（　　いわさき　なつみ　　）
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発表者（※学会発表の場合のみ記載、共同発表者の氏名も記載すること）	Natsumi Iwasaki, Naohiro Nomura, Ryoji Nakano, Jiro Seto, Shinji Matsuo, Saki Shiota, Hideo Satsu, Yuki Marugame, Taichi Hara
発表題目（※学会発表の場合のみ記載）	Identification of a Lactic Acid Bacterium that Enhances Intestinal Barrier Function via mTORC1-Independent Autophagy

発表の概要と成果（抄録を公開している URL がある場合、「概要・成果」を記載した上で、URL を末尾に記してください。また、抄録 PDF は別途ご提出ください。なお、抄録 PDF は Web 上には公開されません。）

Autophagy is a cellular recycling mechanism for intracellular components and is essential for maintaining cellular homeostasis. It is also posited to mitigate aging and disease. Consequently, effective activation of autophagy by food-derived components is anticipated to support preventive medicine and promote health. Autophagy has been implicated in maintaining barrier function in intestinal epithelial cells. Lactic acid bacteria are recognized for diverse physiological activities, including immune modulation and enhancement of intestinal health. Moreover, proper autophagy in intestinal cells is intricately linked to lifespan and crucial for sustaining the intestinal barrier. Based on these insights, this study aimed to identify lactic acid bacterial strains with autophagy-activating effects in intestinal cells, investigate their molecular mechanisms, and assess their impact on intestinal barrier function. Using Caco-2 cells stably expressing a GFP-LC3-RFP probe capable of quantitatively measuring autophagy degradation activity (autophagy flux), we identified a strain of lactic acid bacteria that enhanced autophagy flux. This strain activates autophagy independent of mTORC1. RNA-Seq and comprehensive gene expression analysis identified candidate intracellular signaling pathways in this novel mechanism. Furthermore, evaluation in differentiated Caco-2 cells demonstrated that this strain enhanced intestinal barrier function, confirming that this function is autophagy-dependent. In summary, this study identified a lactic acid bacterial strain that activates autophagy via a novel mTORC1-independent pathway and improves intestinal barrier function. Detailed validation of the novel signaling pathway identified by RNA-Seq is expected to provide new insights into autophagy regulation. These findings are anticipated to provide a valuable foundation for gut environment control, focusing on autophagy.