Analysis of compensatory hypertrophy-associated enhancement of salivary secretionusing the intravital Ca^{2+}imaging and comprehensive gene analysis.

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Dysfunction of unilateral salivary glands causes compensatoryhypertrophy of the contralateral salivary gland. To examine a functional change of the hypertrophiedsubmandibular gland (SMG) after the ligation of main excretory duct (MED) of unilateral SMGs, we monitored Ca^{2+}responses and salivary secretion in rat SMG using the intravital Ca^{2+}imaging system and the fibre-optic pressure sensor, simultaneously. Submaximal dose of ACh (<120 nmol/min) induced 2 times larger increase in intracellular Ca^{2+}concentration and salivary secretion in hypertrophied SMGs than in controlSMGs, whereas the maximal dose of ACh (360 nmol/min) induced comparable responses in these SMGs. These results indicate that the ligation of MED of unilateral SMGs increased the sensitivity of contralateral SMGs to ACh. To clarify the molecular mechanism for inducing "the compensatory hyperfunction", we examined gene expression of the hypertrophiedSMG by a comprehensive analysis using the next-generation sequencing and a quantitative RT-PCR analysis. Currently, we identified 57 candidate genes of which 6 genes showed significant up- or down-regulation after MED ligation in the contralateralSMGs. Our data suggest the increase in acinar cell by the proliferation and transition from ductal cells, and the involvement of some growth factors and clock genes.