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Exploring How Salt Loading Affects Acute Kidney Injury through the Spleen

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Summary

We previously reported that stimulation of vagal afferent fibers or restraint stress can activate the C1 neurons -> sympathetic nervous system -> splenic nerve -> spleen axis, resulting in the protection against acute kidney injury. On the other hand, it was demonstrated that elevated blood Na^+ concentrations induced by the administration of hypertonic saline activated OVLT -> PVN -> C1 neurons -> sympathetic nervous system, resulting in an elevation of blood pressure. Based on these findings, we hypothesized that elevated blood Na^+ concentrations induced by the administration of hypertonic saline activates the OVLT -> PVN -> C1 neurons -> sympathetic nervous system -> splenic nerve -> spleen -> kidney pathway, resulting in the altered phenotype of the splenocytes and kidney protection against acute injury. This year, we focused on investigating the missing link between the spleen and kidney. We used photoconversion to trace splenocytes *in vivo*. We optimized the photoconversion protocol and obtained 83% efficiency with 365 nm light without increasing the number of dead cells. However, the efficiency was high only in the superficial region of the spleen. Thus, we need to further optimize the protocol (e.g., using a longer wavelength).