Dexamethasone induces sodium and water loss in skin

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We previously reported that endogenous glucocorticoid elevation induces skin-specific sodium and water loss. In the present study, we examined the effect of exogenous glucocorticoid administration on body sodium and water balance in mice by using dexamethasone, a potent corticosteroid with predominantly glucocorticoid actions. At 24 hours after dexamethasone injection (1 mg/kg/day, s.c.), dexamethasone-treated mice exhibited the decrease in skin sodium and water content with the increase in urinary sodium excretion and urine volume, suggesting that acute dexamethasone injection induces skin sodium and water loss accompanied by renal sodium and water excretion. On the other hand, continuous administration of dexamethasone for 3 weeks also reduced skin sodium and water content although urinary sodium excretion and urine volume were not significantly altered. This skin-specific sodium loss independently of renal function was associated with increased lymphatic vessel endothelial hyaluronan receptor-1 mRNA levels in the skin, suggesting that expanded skin lymph vessels enhanced lymphatic sodium and water clearance in continuous dexamethasone-treated mice. Our findings confirm that fluid homeostasis is maintained by kidney and skin barriers. Glucocorticoid induces skin-specific sodium osmolyte loss via the lymphatic clearance system, which causes skin dehydration.