

## **Le Système Vasopressine/Hydratation dans la physiopathologie du diabète de type 2 et des complications vasculaires du diabète**

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Diabetic kidney disease (DKD) is now the most common cause of chronic kidney disease (CKD), is a major cause of end-stage renal disease (ESRD), and is associated with increased cardiovascular and all-cause mortality. Circulating levels of vasopressin (or anti-diuretic hormone) are increased in people with type 1 or type 2 diabetes, and in animal models with spontaneous or streptozotocin-induced diabetes. From an adaptive perspective, high levels of vasopressin may be beneficial in the short term by limiting the water loss in urine induced by glycosuria. However, in the long term, persistently high levels of vasopressin might be deleterious to renal function. A large body of experimental data support a direct causal role for vasopressin via its renal V2 receptor in the pathogenesis of CKD and DKD. Epidemiological studies using surrogate markers of vasopressin (hydration markers or circulating levels of copeptin, the stable C-terminal portion of the precursor of vasopressin) provide evidence in humans of the deleterious effects of vasopressin in the kidney. Vasopressin has many physiological actions in addition to its well-defined role in the control of fluid homeostasis and urine concentration. It stimulates hepatic gluconeogenesis and glycogenolysis, and the release either of glucagon or insulin, depending on the extracellular glucose level. An increasing body of data suggests that the vasopressin–hydration axis plays a role in glucose homeostasis, and that high vasopressin levels might be a risk factor for hyperglycemia and diabetes. In this presentation, we will review the evidence regarding the association between vasopressin, diabetes, CKD and DKD. This evidence provides a rationale for investigations aiming to reduce vasopressin secretion or action as potential preventive and therapeutic strategies in CKD and DKD.