



ANTIDIURETIC HORMONE¹

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This short review summarizes selected recent developments in our understanding of the mechanism of action of antidiuretic hormone (ADH). The subject has been reviewed in greater detail elsewhere (4, 22, 24-26, 30, 56).² After briefly considering the effects of ADH along the nephron, this review focuses on the control and the cellular mechanism of those effects that result in antidiuresis.

SITES OF THE EFFECTS ON TRANSPORT AND PERMEABILITY

In the limited range of mammalian species studied, ADH directly affects the glomerulus, medullary and cortical thick ascending limb, the connecting tubule, cortical and medullary collecting ducts, and papillary lining cells.

In the physiological concentration range ADH does not alter glomerular filtration rate (GFR) in mammals. Ichikawa & Brenner (27) found, however, that the glomerular ultrafiltration coefficient (K_f) fell as rats went from a state of water diuresis to a state of hydropenia. When a variety of manipulations were used to alter plasma ADH over a broad range, the inverse relationship between presumed plasma ADH concentration and K_f still held. Though one would expect the fall in K_f to result in a fall in GFR, virtually no change in either whole-kidney or single-nephron GFR was observed. The decrease in GFR was prevented by a concomitant rise in

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²The terminology is that used by Andreoli & Schafer (4). ADH connotes all species of antidiuretic hormone unless a specific analog is mentioned. P_f is the coefficient of hydraulic conductivity ($\text{cm} \cdot \text{sec}^{-2}$) evaluated by measuring water flow along an osmotic gradient. PD_w is the coefficient of diffusional permeability to water ($\text{cm} \cdot \text{sec}^{-1}$) evaluated by measuring the rate of exchange of isotopic water across a membrane. PD_i is the apparent diffusional permeability coefficient for the i th solute.

Antidiuretic Hormone Volume 3 Annual Research Reviews

Jessica J Manson



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