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Endogenous and

exogenous mineralocorticoids

affect the threshold of

vasopressin-induced mesenteric

vascular contraction. The aim

of the present study was to

determine the effect of

exogenous and endogenous

mineralocorticoids on the

threshold of vasopressin-

induced vasoconstriction in the

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perfused mesentery of Sprague-Dawley rats. The mesentery was perfused with Krebs-Henseleit buffer and the perfusion pressure was continuously measured. A submaximal dose of arginine vasopressin (AVP) (1 U) produced a transient and dose-dependent increase in perfusion pressure. The threshold concentration of AVP necessary for vasoconstriction was 1 U/ml (0.5 nmol/l), which is 4-fold higher than the concentration required for contraction in the portal vein. The threshold of AVP-induced

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vasoconstriction was not influenced by the coadministration of aldosterone (250 ng/ml), corticosterone (250 ng/ml) or dexamethasone (250 ng/ml) in the perfusion buffer. When given alone at 500 ng/ml, dexamethasone and aldosterone increased the threshold concentration of AVP necessary for vasoconstriction approximately 2-fold compared to AVP alone. The threshold of AVP-induced vasoconstriction was elevated approximately 2-fold in aldosterone-pretreated rats compared to control rats. Perfusion with an

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antiglucocorticoid  
(hydrocortisone) (100 ng/ml)  
increased the threshold of AVP-  
induced vasoconstriction  
approximately 4-fold compared  
to AVP alone. The results  
indicate that (i) the threshold of  
AVP-induced vasoconstriction  
is higher in the mesentery than  
in the portal vein and (ii) that  
mineralocorticoids increase the  
threshold of vasopressin-  
induced vasoconstriction,  
possibly by enhancing the  
sensitivity of the vascular  
smooth muscle cell to AVP.1.  
Field of the Invention The  
present invention relates to the

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field of signal input circuits.  
More particularly, the present invention relates to a high-speed signal input circuit that is used in an electronic device

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