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My gf india the best nickname you would use for a girlfriend you like? | Yahoo AnswersI feel like this would be slightly inappropriate. | Yahoo AnswersWhy is your daughter so obsessed with pink? | Yahoo AnswersMine is my G F. | Yahoo AnswersMine is my G F. | Yahoo AnswersWhy is your gf wearing a pair of sunglasses? | Yahoo AnswersYou were right when you said about a gay guy. The human body produces a number of compounds that can promote a host of biological processes, ranging from cell-to-cell signaling to proper metabolism. Yet, it can also produce compounds that can cause health problems, including detrimental effects on the cardiovascular system. Until a few decades ago, it was assumed that small changes in the levels of certain compounds, called natriuretic hormones, were the primary driving force behind the development of hypertension. The discovery of the essential hypertension-susceptibility gene (and its product, the CNP/BNP-A) disproved this theory. Since then, we have learned that this gene plays a role in the regulation of vascular smooth muscle growth. Recently, we made some important advances in understanding the role of natriuretic hormones in the cardiovascular system. We discovered that, in the absence of the CNP/BNP-A gene, vascular smooth muscle cells (VSMC) will stop growing during development. Thus, the CNP/BNP-A gene is essential for growth of vascular smooth muscle, and is required for the maintenance of vascular tone and blood pressure. These studies showed that the CNP/BNP-A gene product plays a critical role in regulating growth and death of vascular smooth muscle cells. Thus, CNP/BNP-A is required for physiological development of the vessel wall, not only for maintaining arterial blood pressure. The aim of this renewal project is to further investigate the role of CNP/BNP-A in the regulation of vascular growth and to understand how this gene product controls vascular remodeling. Our studies involve three main areas. First, we will investigate the pathway used by the CNP/BNP-A gene product to control VSMC growth. We will test the hypothesis that the CNP/BNP-A gene product promotes growth of VSMCs by phosphorylation of a MAP kinase. Second, we will investigate the mechanism for CNP/BNP-A-dependent growth arrest in VSMCs. We will test the hypothesis that this arrest requires the phosphorylation of 6d1f23a050

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