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## Letter to Editor

# Can Strict Control of Renalase Present a New Treatment Alternative in Regulating Blood Pressure?

in addition to the aforementioned vasodilators, or may be even primarily, by the renalase enzyme (an adenine dinucleotide-FAD-dependent monoamine oxidase) [3]. Renalase metabolizes dopamine, epinephrine, and norepinephrine catecholamines, respectively. Remembering that catecholamines serve as the major actors in the regulation of blood pressure through modulating myocardial contractility, heart rate and the tonus of resistance vessels [2], it is certain that as a catecholamine-destroying enzyme, renalase has a decisive role in reducing blood pressure. The main findings supporting this claim are reports showing that renalase administration lowers circulating catecholamines and significantly reduces blood pressure [4,5].

Considering the physiological effect of renalase on biological systems, when its circulating concentrations are above the physiological threshold value [5], renalase can metabolize a large amount of catecholamines, that is, dopamine, epinephrine, and norepinephrine, over a unit of time, and thus hypotension may arise. In this case, administration of monoamine oxidase inhibitors (phenelzine, iproniazid, isocarboxazid, tranilcypromine, deprenyl, moclobemide, brofaromine) may prevent the destruction of catecholamines [6], and mediate the elimination of hypotension. However, it should be kept in mind that the amino acid similarity of renalase is lower than other known FAD-dependent monoamine oxidases A and B. It is a new class of FAD-dependent monoamine oxidase [4, 5]. Therefore, the development of new monoamine oxidase inhibitors appropriate to this new group remains to be accomplished as a new generation of medications to be used in the regulation of blood pressure. Epinephrine-evoked renalase expression was blocked by  $\alpha$ -adrenoceptor blocker as well [7]. It has been also reported that renalase attenuates hypertension and renal injury in animals subjected to subtotal nephrectomy [8,9]. All this researches indicated that this enzyme serving in the regulation of blood pressure (renalase) is released to the circulation mainly from the kidneys [4]. It is also produced, though to a lesser extent, by the heart, skeletal muscle and small intestines, oocytes, granulosa, luteal and interstitial cells of the ovaries, spermatogenic cells of the testes, and cortex cells of the adrenal gland [4,10]. Renalases secreted

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Blood pressure is the pressure the blood in arteries applies on vessels. Abnormal blood pressure is a major public health problem commonly encountered in the population with normal blood pressure and intensive care patients. The two types of action that the heart performs are pumping blood and relaxing. The pressure exerted when the heart is pumping blood is the “systolic” blood pressure, and the pressure applied during the relaxation of the heart is the “diastolic” pressure. Although blood pressure is not always at the same level (with slight fluctuations), normal blood pressure in adults is 120/80 mmHg. Lower blood pressure of 90/60 mmHg is hypotension, while higher blood pressure of 140/90 mmHg is hypertension [1]. Circulating high catecholamines (epinephrine and norepinephrine) can lead to a hypertensive crisis [2]. Renalase, a recently discovered flavoprotein, degrade catecholamines [3, 4, 5]. Here it has been therefore claimed that blocking or enhancing the action of renalase might help a strict control of regulating blood pressure.

It is also known that in healthy individuals, arterial blood pressure equals to the multiplication of the cardiac output (CO) which is a product of the heart rate (HR) and stroke volume (SV), with total peripheral resistance (TPR).  $BP = CO \times TPR = (HR \times SV) \times \text{vasoconstriction}$ . An increase in the elements of this equation, either individually or in combination, results in elevated blood pressure. In the early stages of elevated blood pressure, endothelial cells increase the release of nitric acid and prostacyclin (vasodilators) from local parenchymal cells to compensate for this elevation and thus to regulate blood pressure [2].

It is here hypothesized in this editorial that in cases where the blood pressure is elevated, blood regulation is undertaken,

to the circulation are normally inactive, and are activated by catecholamines. If renalase is launched as a medicine, it may prevent endothelial damage in cases with extremely high blood pressure, and if monoamine oxidase inhibitors compatible with renalase are developed, these may help eliminate hypotension, and thus, renalase seems to be a significant factor in regulating the erratic blood pressure, holding considerable promise for both intensive case and emergency polyclinic patients.

Use of renalase during ischemia preserves the myocardium and reduces the infarct area. Besides, catecholamine elevation is characteristic in heart failure. Therefore, renalase replacement is promising. Since chronic kidney diseases are marked by renalase deficiency and renalase reduces renal tubular necrosis, apoptosis and inflammation, it can be both a marker of acute kidney injury and a new agent in devising treatment alternatives.

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