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Editorial

# Vaccine against Cardiovascular Disease: Possible and Indispensable

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## Editorial

Since the 1940s Cardiovascular Disease (CVD) was the leading cause of death in the United States [1] and worldwide. Despite significant progress in therapy and prevention resulting in decline of CVD mortality rates, this trend continues. Several epidemiological studies have suggested that vaccination for seasonal influenza virus infection significantly reduces the risk of major cardiovascular events in patients with acute coronary syndrome or coronary artery disease [2]. A key question is whether preventing influenza virus infection, particularly by means of influenza virus vaccination, can prevent influenza virus triggered major cardiovascular events, or if the vaccine through an independent mechanism to protect against CVD. Despite plausible evidence for beneficial effects of the vaccination against influenza in CVD patients very limited studies have been carried.

Available data suggest the lack of correlation between an effectiveness of influenza vaccine and its protective effect against CVD. So, during the influenza season 2004/2005, the vaccine effectiveness was only 10% [3] and the virulence of circulating viruses was very high [4]. Despite these unfavorable conditions, the risk for the Major Adverse Cardiovascular Events (MACE) was significantly decreased in persons who received influenza virus vaccination [5]. Contrary, in the influenza season 1991/1992, which was characterized with the low virulence [6] and the vaccine effectiveness of 58% [7], incidence of MACE was very high [8]. Negative correlation between the effectiveness of influenza vaccine and cardiovascular risk suggests that influenza vaccine may act against CVD through independent pathways.

Knowing of the molecular mechanism underlying the protective effect of influenza vaccine against CVD is the key precondition for its further use as preventive intervention and development of specific CVD vaccine. Prior studies have proposed that some antibodies elicited by influenza vaccines act as agonists, which activate a bradykinin-2 receptors-associated signaling pathway contributing to the protection against CVD [9]. Activation of this pathway would be expected to induce nitric oxide production, vasodilatation, and natriuresis. Vaccine virus strains which could be suited for future investigations to further elucidate the role of influenza vaccines as primary and secondary prevention against CVD, have been also suggested [9].

Despite the fact that CVD still is the leading cause of death worldwide and a strong evidence that that vaccination for seasonal influenza virus infection significantly reduces the risk of major cardiovascular events acting as an independent protective factor against CVD, the interest of biotech industry for development of the CVD vaccine still is disappointedly low. It is hoped that this situation will be changed in the future and that significant research and financial resources will be deployed in development of vaccine against CVD. In the meantime, since there is no safe and efficient vaccine, we encourage patients with or at high risk for CVD receive their annual influenza virus vaccination.

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