



# Smoking Exposure Leads Physiological and Pathological Transfer, Especially in the Elderly



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## Abstract

Smoking exposure is always associated with aging, especially in old age. Aged-related changes in an old-aged smoker, but otherwise normal heart mimic those changes associated with cardiac diseases, including myocardial infarction and alterations to cardiac valves and coronary arteries. Human cardiac aging generates a complex phenotype. Experimental evidence in animal models has indicated smoking exposure attenuation cardioprotective with age such as left ventricular hypertrophy and heart failure, yet information regarding myocardial dysfunction in old age is limited. Cardiac aging is a human physiologic change which has the slowly progressive functional declines and structural changes with age from physiologic to pathologic transfer. Normal aging in old man must be in the absence of major cardiovascular risks such as high blood pressure. Smoking may stimuli first induce a phase of cardiac hypertrophy, especially in left ventricular individual. Aging affects cardiovascular function in the same manner as smoking exposure. However, aging also shows relative adaptive responsiveness to eliminate damaged and exhausted cells from birth to senescence. Inflammation response is the major role in the adaptive pathological aging from physiological responsiveness. Therefore, remodeling of the aging cardiac typically involves a large net loss of active cardiac myocytes, reactive cardiac of the remaining cells, and increased accumulation of connective tissue..

**Keywords:** Smoking exposure; Cardiac diseases; Cardiac aging; Physiologic to pathologic transfer; Cardiac hypertrophy; Inflammation response

## Introduction

Aging is a physiological process involving progressive impairment of normal functions, due to an increasing vulnerability to injuries, which reduces the ability of the organism to survive. The physiologic changes of the aging cardiac include left ventricular hypertrophy, increased cardiac fibrosis and valve degeneration [1]. However, cardiovascular disease is a major risk factor for aging cause of death. Aging-related cardiac disease changes in cardiac morphology and functions include decreased myocyte numbers, increased myocyte size, increased left ventricular wall thickness, and decreased conduction fiber density, while functional alterations include a decrease in intrinsic contractility, increased myocardial contraction time, decreased myocardial contraction velocity, and increased myocardial stiffness in left ventricular function [2]. Smoking increases arterial stiffness and coronary cardiac disease. Smoking exposure involves the combination of the smoke emitted by the burning end of a tobacco cigarette and the smoke exhaled by the smoker into the environment [3]. Cardiac pathological hypertrophy due to smoking exposure was observed in old-age patients, which leads to left ventricular remodeling and loss of function. Left ventricular hypertrophy is an initial adaptive response [4]. During LVH development, there is an imbalance of progressive remodeling at the cellular level, involving aging cardiomyocytes physiological and pathological disease transfer.

Aging changes of the elderly heart is associated with physiological cardiac hypertrophy, which is expected or normal aging changes, smoking exposure is associated with pathological hypertrophy. Smoking exposure induced age-related cardiac hypertrophy is associated with numerous molecular and biochemical changes [5]. Calcineurin/NFAT is an originally implicated as pathological hypertrophy signaling pathway. Calcineurin/NFAT is regulated by MAPKs cascades mediated directly and indirectly [6]. However, calcineurin/NFAT regulate cardiac hypertrophy is associated with MEK1-ERK1/2 (physiologic) and MEK5/ERK5 (pathologic) signaling pathways [7]. The subclassified branches MEK5/ERK5 pathways have been implicated in pathological hypertrophy regulation in the heart [8]. In addition, MEK1-ERK1/2 regulate myocytes growth and physiological cardiac hypertrophy function [9]. Indeed, age-associated disease underlies much of the physiological deterioration of old age. Distinctions may be made between “physiological aging” and “pathological aging”. The most well recognized risk factor for many chronic diseases in pathological aging. Interactions between the aging process and the aged-related disease has not been seriously addressed or systematically explained. Aging is an inevitable process of life [10]. Become progressively disorganized and degraded with age occurring as consequence of physiological aging. Aging process can be described

as a progressive function (left ventricular hypertrophy to heart failure) decline that lead to the accumulation of errors that damage repair systems and compromise stem cell function [11]. Aging is a human inevitable adaptive response to exhausted cells, while others regard it as a process that starts at conception and continues until death [12]. Consider aging to be a human physiologic change which has the slowly progressive structural changes and loss in body function with age.

## Conclusion

Smoking exposure always make human pathological cardiac hypertrophy in this environment in old age. Smoking exposure may stimuli a phase of cardiac hypertrophy, especially in left ventricular individual. Smoking exposure induced cardiac inflammation led to left ventricular pathological hypertrophy remodeling transfer from aging and increases the risk of a cardiovascular event and mortality.

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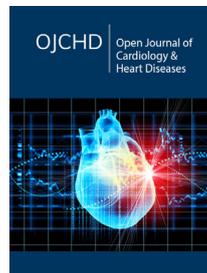
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