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Theoretical Identification of Behavioral Risk Factors Among Multiple Risk Factors Causing Morning Onset of Cardiac Events Due to Circadian Variations

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1. Introduction

Circadian variations of cardiac events exist, although times of onset differ slightly between studies (Atkinson et al., 2010; Muller et al., 1989; Quyyumi, 1990; Scheer et al., 2010; Willich et al., 1987). However, several studies have confirmed the occurrence of more events in the morning than in the evening (Atkinson et al., 2010; Mulcahy et al., 1988; Scheer et al., 2010). Two peaks of onset of cardiac events were found: a high frequency of cardiac events in the hours after rising in the morning and in the evening hours (Atkinson et al., 2010; Hjalmarson et al., 1989; Mulcahy et al., 1988; Muller, 1987).

Because cardiac events occurred more frequently in the morning than at any other times of the day, the occurrences of these events are discussed relative to the time after awakening (Kiowski & Osswald, 1993). A specific single factor causing circadian variation in the morning has not been identified. It has been suggested that interactions between multiple factors during transition from the sleep state to the waking stage may occur simultaneously or as a self-sustaining reaction (Atkinson et al., 2010; Scheer et al., 2010).

Evidence regarding physiological risk factors for morning onset of cardiac events has been used when selecting the most appropriate time for taking antihypertensive or anti-arrhythmic medications (Athyros et al., 1998; Mulcahy, 1999; Redon, 2004). For example, Redon (2004) indicated that the angiotensin II blocker telmisartan with a long half-life given once daily is likely to confer benefit in terms of 24-hour blood pressure control and reduce early morning surge in blood pressure. Despite applications of this evidence to medical treatment, few studies examine use of behavioral risk factors related to morning onset of cardiac events. For example, final decisions based on evidence regarding the safest time to exercise during the day are still pending (Atkinson et al., 2006; Atkinson & Davenne, 2007; Murray et al., 1993; Shiotani et al., 2009; White, 2003).

Even though Ebrahim et al. (2006, 2011) indicated that intervention using educational method may be effective in reducing mortality in cardiac high risk population, few discussions have taken place regarding how to apply the current evidence regarding

morning onset of cardiac events to patient education. In particular, knowledge about preventive strategies for self-care that minimize the chance of recurrent morning onset of cardiac events is lacking. Amazingly, most patient educational materials about cardiac disease do not contain information about risks of morning behaviors.

Although behavioral risk factors for cardiac events will not significantly influence daily life if they occur once in a while at an early age, human behaviors tend to be unconscious and habitual throughout life. By identifying behavioral risk factors among multiple risk factors that can trigger morning onset of cardiac events, patients with cardiac disease may be able to avoid life-threatening conditions and reconsider their daily lives, including the cycle of activity, rest, and sleep that is associated with quality of life (Baas, 2004; Condon & McCarthy, 2006; Kristofferzon et al., 2007; Noriss et al., 2004). By controlling behavioral risks among the multiple risk factors for cardiac events in the morning, it might be possible to reduce the frequency of early morning cardiac events.

Reviewing evidence regarding behavioral risk factors of morning onset of cardiac events may be helpful for health professionals including doctors and nurses in the cardiovascular community to educate the patients. Furthermore, interventions supported by research that ameliorate life-threatening conditions may improve the quality of patient education. Thus, the primary aim of this chapter is to identify behavioral risk factors among possible causes of morning onset of cardiac events. To develop interventions leading to evidence-based practice, the secondary aim is to suggest strategies for controlling behavioral risk factors that trigger morning onset of cardiac events.

2. Methods

Material reviewed in this chapter was based on journals reporting risk factors related to cardiac events. First, focus of the review was put on the physiological perspective in nature, and then behavioral risk factors were reviewed to suggest strategies for controlling such risk factors. Because physiological conditions are usually deteriorated by behavioral risk factors under the occurrence of cardiac events, it is importance to recognize how each physiological risk factor is progressed and interacted with each other (Deedwania, 1996). Recognition regarding physiological risk factors is also important to develop effective strategies for controlling behavioral risk factors (Baxendale, 1992).

The electronic databases of PubMed (1950 to 2011), CINAHL (1998 to 2011), and the COCHRANE Library (1998 to 2011) were searched for topics related to morning cardiac events and their mechanism. Key words were as follows: circadian variation, sleep, ischemic episodes, cardiac events, cardiac disease, autonomic nervous system activation, physical stress (abrupt upright posture), mental stress, endothelial response of the coronary blood vessels, morning surge of blood pressure, and blood components (Carnethon et al., 2002; Cooke-Ariel, 1998; Furukawa et al., 1998; Khoury et al., 1992; Scheer et al., 2010; Thrall et al., 2007; White, 2003). By searching the database for these key words, evidence regarding behavioral risks factors triggering morning cardiac events was identified, and preventive patient education strategies to decrease morning onset of cardiac events are proposed.

All journals used for this review were limited in only English language, and findings from the animal study were included to understand mechanism of the morning onset of cardiac events in patients with cardiac disease. Also the findings reported in the 1998 review

(Furukawa et al., 1998) were used in part in this review. Permission for utilization of the findings was obtained from the editor.

3. Results

Possible risk factors for cardiac events were as follows: 1) coronary artery blood flow dynamics during sleep; 2) physiology of sympathetic nervous system activation in the morning; 3) pathophysiology of sympathetic nervous system activation in the morning, including coronary blood flow, α - and β -adrenergic receptor responses of coronary artery endothelial cells to sympathetic nerves, platelet aggregation and atherosclerosis with sympathetic nervous activation, neurotransmitters and hormonal elements, and other risk factors for cardiac events, such as morning surge in blood pressure and seasonal variations; and 4) behavioral risk factors immediately after awakening, including abrupt changes of body position.

3.1 Coronary artery blood flow dynamics during sleep

Because of the sleeping state in relation to patterns of REM and non-REM sleep (Aserinsky & Kleitman, 1953; Jouvet, 1967; Steriade, 1992), the morning onset of cardiac events should be discussed from this point. One sleeping cycle is made up of one REM and one non-REM sleep period, and four to six cycles are repeated during the night in adults. Non-REM sleep has four sub-stages, from I to IV, during one cycle, and the depth of sleep increases progressively toward stage IV and then decreases in REM sleep. People wake up mostly at the end of the last rapid eye movement sleep (Willis, 1981).

During the night, arterial blood pressure falls progressively by 5% to 23% from the wakening state to stage IV of non-REM sleep (Martin, 1984). Giles (2005) indicated blood pressure in most people is from 10% to 20% lower than the mean daytime value (Giles, 2005). Cardiac output is minimized during the last rapid eye movement sleep period before arousal and is 25% lower than that during wakening (Martin, 1984). Changes in blood flow of the coronary artery vessels from the beginning of sleep to the time of awakening make insufficient blood flow in cardiac muscles at the time of restart of physical activity immediately upon awakening.

In healthy subjects, increased blood flow by sustained physical activity might lead to vasodilatation, which is induced by endothelial derived relaxing factor released from endothelial cells (Furchgott, 1983). This is a compensation mechanism to increase the blood supply to cardiac muscle. However, such mechanism may not work in patients with cardiac disease. The sudden rises in heart rate at each transition from non-REM sleep (deeper sleep) to REM sleep (lighter sleep), or increase in blood pressure at awakening may precipitate cardiac events (Viola et al., 2002).

3.2 Physiology of sympathetic nervous system activation in the morning

Coronary blood flow is determined by five factors (West, 1990): metabolic control (oxygen demand), coronary perfusion pressure, systolic compression, autonomic nervous system, and circulating catecholamine and other vasoactive substances. Determinants for coronary blood flow are conveyed by auto-regulatory mechanisms. Sympathetic nerve fibers

stimulate α -adrenergic receptors of the endothelial cells to respond to vasoconstriction so as to increase coronary vascular resistance (Laxson et al., 1992; Baran et al., 1992; Quyyumi et al., 1992). Stimulation of β -adrenergic receptors of the endothelial cells is a response to coronary vasodilatation (Baran et al., 1992; Quyyumi et al., 1992).

Epinephrine, norepinephrine, and acetylcholine also act through adrenergic receptors of smooth muscle in the coronary arteries (Furchgott, 1983; Kuo et al., 1993). In the transition period from resting to activity in healthy person in the morning, coronary arteries of the human heart can receive signals normally from the sympathetic nervous system due to the increased physical and mental stress associated with awakening, and the compensatory mechanisms of the coronary artery function are well activated to meet oxygen demands.

3.3 Pathophysiology of sympathetic nervous system activation in the morning

In this section, 1) coronary blood flow, 2) α - and β -adrenergic receptor responses of coronary artery endothelial cells to the sympathetic nervous system, 3) platelet aggregation and atherosclerosis with sympathetic nervous activation, 4) neurotransmitters and hormonal elements under sympathetic nervous activation, and 5) other risk factors are reviewed.

3.3.1 Coronary blood flow

Ischemic episodes occur if the increase in coronary blood flow is inadequate for the myocardial oxygen demand. The frequency of ischemic episodes is maximized in the first 2 hours after awakening (Quyyumi, 1990; Rocco, 1990). Coronary spasms due to vasoconstriction are a trigger for ischemic episodes (Sugiishi & Takatsu, 1993). Coronary artery constriction is more severe in the early morning than in the late afternoon, and coronary blood flow at rest in the morning is lower than in the afternoon under comparable hemodynamic conditions (Fujita & Franklin, 1987).

Vascular resistance increases due to the decreased blood flow in patients with hypertrophy or elevated left ventricular diastolic pressure (Duncker et al., 1993). When coronary vascular resistance is increased in the morning, the ischemic threshold is lower than in the afternoon (Benhorin et al., 1993; Quyyumi et al., 1992). Also, the cardiac output during the last REM sleep period before arousal is lowest, and it is one fourth of that while wakening (Martin, 1984). These studies indicate that abrupt and vigorous physical stress becomes a risk factor for cardiac events.

3.3.2 α - and β -adrenergic receptor response of coronary artery endothelial cells to the sympathetic nervous system

The vascular endothelium modifies the contractile characteristics of vascular smooth muscle (Greenberg et al., 1990). When the vasodilator response to acetylcholine in endothelial dysfunction is eliminated, the coronary perfusion rate increases (Furchgott et al., 1980). Endothelial injury initiated by atherosclerotic change leads to plaque formation in a variety of conditions (Zemel & Sowers, 1990; Baxendale, 1992).

Similarly, injury to the endothelial wall triggers monocyte and platelet aggregation, which leads to release of growth factors into the smooth muscle of coronary arteries (Lucchesi,

1990). In particular, the injured endothelial vessel wall produces a paradoxical response that alters the normal response against α - and β -receptors on the vessel walls (Ludmer et al., 1986). The neural sympathetic tone, which increases in the morning, may cause an increased stimulation of α -adrenergic receptors, leading to coronary and peripheral vasoconstriction.

The presence of enough β -adrenergic receptors is necessary to adapt to the increased oxygen demand (Hammond et al., 1992; Figueras & Lindon, 1995). However, the number of β -adrenergic receptors decreases if there is volume overload-induced myocardial hypertrophy, or if the endothelial cells of the coronary artery are impaired (Figueras & Lindon, 1995; Hammond et al., 1992). Patients with unstable angina during bed rest and with significant coronary disease demonstrate a lower ischemic threshold early in the morning (Figueras & Lindon, 1995). Usually, plasma levels of norepinephrine are increased and myocardial levels of norepinephrine are decreased in patients with fewer β -adrenergic receptors (West, 1990; Hammond et al., 1992). The increased plasma norepinephrine overstimulates sympathetic nervous activity and acts mostly on α -adrenergic receptors, causing vessel constriction (Hammond et al., 1992; Kuo et al., 1993; West, 1990).

3.3.3 Platelet aggregation and atherosclerosis with sympathetic nervous activation

Accelerated platelets aggregation is one of significant risk factors for the morning onset of ischemic episodes (Andrews et al., 1996; Quyyumi, 1990; Braunwald, 1995). The plasma levels of fibrin peptide (thrombin generator) in patients with variant angina are significantly higher from midnight to early morning than other times (Masuda et al., 1994). The plasma norepinephrine concentration (Brezinski et al., 1988), renin activity, and angiotensin II concentration (Oparil et al., 1970; Schachter, 2004) are all increased with change of platelet levels.

Activated platelets release potent vasoactive factors due to adherence of atherosclerotic plaque and cause vasoconstriction or vasospasms (Kaul et al., 1993; Vanhoutte & Huston, 1985). A plaque rupture associated with well-developed atherosclerotic conditions in the coronary artery is often occurred in the morning as sympathetic activity is upregulated, and this induced increase in blood pressure (Shimada et al., 2001; Stone, 1990). Fibrinogen is also involved in this process (Palmieri et al., 2003), and leads to increased blood viscosity (Jay et al., 1990; Lowe et al., 1980).

Plasma fibrinogen has further been found to stimulate red cell aggregation in familial hypercholesterolemia (Jay et al., 1990). The increased fibrinogen and red blood cells lead to increased blood viscosity (Yarnell et al., 1991). In addition, body fluid during sleep usually shifts from the vascular space to the interstitial space (Jacob et al., 1998). These conditions may lead to vasoconstriction and cause vessel occlusion in the presence of atherosclerosis and physical stress in the morning.

3.3.4 Neurotransmitters and hormonal elements

Neurotransmitters and hormonal elements such as epinephrine, norepinephrine, neuropeptide Y, and acetylcholine also participate in cardiac events through aggregation (Eisenhofer et al., 1992; Pernow et al., 1988; Svendsen et al., 1990; Yang & Levy, 1993) and interrelations with endothelial dysfunction (Ludmer et al., 1986), sympathetic nervous

system activation (Drager et al., 2009), degrees of coronary blood flow (Fujita & Franklin, 1987; Kuo et al., 1993; Young & Vatner, 1986), level of co-existing atherosclerosis (Ludmer et al., 1986), sleep cycle during the morning transitional phase (Culebras, 1992; Steriade, 1992; Viola et al., 2002), and sleep duration (Cappuccio et al., 2011).

In particular, plasma epinephrine and norepinephrine levels were shown to increase rapidly while plasma volume induced by an abrupt upright posture fell by 13% over 14 minutes (Jacob et al., 1998). Epinephrine approached baseline values by 20 minutes of standing, but norepinephrine spillover increased by 80% and clearance decreased by 30% with 30 minutes of standing (Jacob et al., 1998). Inhibition of the morning surge in catecholamine levels may reduce the risk of thrombotic vascular events in atherosclerosis (Andwers et al., 1999).

3.3.5 Other risks factors

The morning increase in blood pressure, called the morning surge, is one of the risk factors for cardiac events (Giles, 2005; Ohkubo et al., 2008; White, 2001). In addition, morning onset of cardiac events might occur more easily in winter if the body is suddenly exposed to a cold environment (Peters et al., 1996). Mental stress also may cause stress-induced cardiac arrhythmic vulnerability associated with sudden cardiac death (Critchley et al., 2005).

3.4 Behavioral risk factors immediately after awakening

Physical activity, such as standing up immediately after awakening, affects the blood components. In healthy men, a significant change in platelet aggregation occurs as a result of a compensatory homeostatic mechanism against the immediate change to the upright position (Muller et al., 1989; Krantz et al., 1996). Adaption to the change of position after awakening causes persistent platelet aggregation for the next 90 minutes; this platelet aggregation is greater than walking up and down stairs. (Muller et al., 1989). If activities such as walking up and down stairs are performed immediately after waking, platelet aggregation becomes greater than just staying in the upright position (Muller et al., 1989). Because the abrupt upright position after awakening brings a significant change in platelet aggregation with persistent manner, patients with cardiac disease may experience an adverse effect of exercise in the morning (Hilberg et al., 2000).

Also, the upright posture from spine position made plasma volume fell by 13% over approximately 14 minutes in healthy subjects, after which time it remained relatively stable (Jacob et al., 1998). This occurs as a result of compensatory homeostatic mechanisms responding to the initial decrease in central blood and stroke volume from the heart induced by the physical stress (Ahmadizad et al., 2006; Thrall et al., 2007). The sudden rises in heart rate in this case may occur to compensate cardiac output based on the intense of activity. These changes in platelet aggregation and plasma volume occurred in healthy subjects may become severe triggers of cardiac events for patients with cardiac disease.

4. Discussion and conclusion

Cardiac events often occur in the morning after awakening. Many studies report similar findings in terms of the circadian variation of the cardiac events. The significant behavioral risk factor was an abrupt upright position at awakening and seriously acted on

physiological risk factors at the occurrence of the cardiac events. The most plausible underline cause of cardiac events in the morning in patients with progressed atherosclerosis was activation of the sympathetic nervous system due to activity immediately upon rising (Kiowski & Osswald, 1993; Stone, 1990; Umemura et al., 1987). Studies on physical stress for cardiac disease under variety of settings were available (Camici et al., 1992; Herd, 1991), but only a few studies of the relation between physical stress and morning onset of the cardiac events were found. The activation of the sympathetic nervous system by physical activity after rising (Umemura et al., 1987) should be considered in relation to levels of atherosclerosis and sleeping stages. The nature of sleep (Culebras, 1992; Steriade, 1992) plays an important role in the morning onset of cardiac events.

A shift change of domination in the autonomic nervous system occurs upon awakening, so that the sympathetic nervous system is activated to prepare for physical and mental activities in the morning. The morning surge of blood pressure can be explained by this shift change, because the sympathetic nervous system is activated by exogenous factors (Leary et al., 2002; van Eekelen et al., 2004a). It should be noted that domination of the sympathetic nervous system in the morning may play a major role on the occurrence of cardiac events (Atkinson et al., 2010; van Eekelen et al., 2004a, 2004b).

In particular, patients who have atherosclerosis may experience a paradoxical response in their vessels under sympathetic nervous system activation with abrupt physical activities undertaken after awakening in the morning (Ludmer et al., 1986). Atherosclerosis triggers cardiac events by creating a paradoxical response in the injured endothelial cells of coronary vessels, providing the greatest vulnerability to sympathetic nervous activity (Ludmer et al., 1986). Thus, normal daytime activity may become a powerful stimulus if it is undertaken in the morning by patients with coronary artery disease (Freed et al., 1989; Herd, 1991; Meller et al., 1979).

It can be assumed that we might experience cardiac events at the time of awakening in the morning because our lives repeat to follow a daily active-sleep cycle, and nobody can develop atherosclerosis with aging. The last REM sleep at the time of awakening is needed to create a smooth transition from parasympathetic to sympathetic nervous activation. As one study indicated, REM sleep works to increase pulse rate (Martin, 1984). The last REM sleep may bring a wake-up ready condition by increasing pulse rate through gradually stimulating the sympathetic nervous system (Martin, 1984) and decreasing parasympathetic nervous system activation (van Eekelen et al., 2004a). Using evidence on the nature of sleep is beneficial if strategies for controlling the morning onset of cardiac events are developed.

Waking-up at REM sleep, not at non-REM sleep may be important for decreasing cardiac events in the morning. REM sleep provide a waking-up ready condition to cardiovascular system. To facilitate waking-up at rapid eye movement sleep, it is better to avoid utilizing an alarm clock, and use non-artificial stimuli such as sunlight providing a natural wake-up call. People should sleep with the shades and curtains open so that they are woken up with natural sunlight that will lead us to wake up at REM sleep.

Similarly, it is important for patients with cardiac disease to not wake up in the morning during non-REM sleep. Because coronary blood flow is the lowest at the last non-REM sleep (Martin, 1984), cardiac output in patients with cardiac disease might not respond adequately to a sudden increase of coronary blood flow at the time of wakening (Kuo et al., 1993), so

that supply and demand become unbalanced (Li, 2003). If patients with cardiac disease show non-movement of eyelids, smooth movement of the chest, and quiet breathing indicating non-REM sleep (Aserinski & Kleitman, 1953; Aserinski & Kleitman, 2003), it is better for them to be left until waking-up by themselves.

In addition to sensitivity to REM sleep and non-REM sleep, levels of catecholamine and coronary tone should be considered because they are lowest just before awakening (Linsell et al., 1985). During the transitional phase from sleep state to physical activity state in patients with atherosclerosis, insufficient oxygen supply to the heart occurs. The high levels of catecholamine at awakening may produce a severe paradoxical response to the coronary vessel wall in patients with progressing atherosclerosis, leading to vasospasm (Ludmer et al., 1986). Such vasospasms might trigger a cardiac event. Atherosclerosis of artery walls develops over a long period and progresses with a high-fat diet and a sedentary lifestyle. The cardiovascular system should be maintained in a healthy condition by an adequate caloric intake and an active lifestyle (Ebrahim et al., 2011). From the viewpoint of human evolution, modern life with excess caloric intake and sedentary lifestyles may contribute to the increased number of cardiac events that occur in the morning.

Although atherosclerotic changes are significantly associated with aging, platelet aggregation may not be. Platelet aggregation has shown to be triggered in healthy young people who assume an abrupt upright position in the morning (Muller et al., 1989; Tofler et al., 1987) and works with fibrinogen levels to significantly contribute to morning onset of cardiac events (Braunwald, 1995). Abruptly assuming an upright position immediately after awakening is now a known risk factor for cardiac events, and may create a life-threatening condition in patients with the major cardiac risk factors. However, such behavior seems to be recognized as a small factor that can be modified, and the effects of this change have not been studied.

Abrupt changes in blood pressure and heart rate due to adoption of an upright position after awaking in the morning may trigger platelet aggregation, enhancing the degree of occlusion of the coronary artery with aging (Storm et al., 1989). Once platelet aggregation occurs, it lasts for hours (Muller et al., 1989). Activated platelets release potent vasoactive factors due to adherence of atherosclerotic plaque, and cause vasoconstriction or vasospasms (Kaul et al., 1993; Vanhoutte & Huston, 1985). This phenomenon may create a morning peak in cardiac events. Patients with cardiac disease should take their time getting up in the morning, engaging only in slow activities until platelet aggregation subsides. Furthermore, they should maintain ideal fibrinogen levels by taking part in regular exercise (Furukawa et al., 2008).

Cold temperatures with seasonal variations at the transitional phase in the morning are also a risk factor triggering cardiac events (Peters et al., 1996). These temperatures may increase blood pressure by constricting peripheral vessels. Increased blood pressure might create rupture of the vessel's wall and release clots, leading to ischemic events (Shimada et al., 2001). Here again, rupture of the vessel wall leads to an increase in platelets (Shimada et al., 2001). Abruptly assuming an upright posture, which causes platelet aggregation, will precipitate ischemic events during a few hours after awakening in the morning. Therefore, before patients rise from bed, they should lay quietly in bed for a few minutes.

In general, four strategies are recommended to reduced cardiac events in the early morning, especially for people with any cardiac disease: waking up at the end of rapid eye movement sleep, laying in bed for a few minutes after awakening, slowly raising the body from bed, and staying relaxed at least 30 minutes after awakening. Staying relaxed in 30 minutes is suggested because the once increased norepinephrine spillover by standing mostly decreased after 30 minutes (Jacob et al., 1998). Relaxation therapy is also recommended in the morning to control sympathetic nervous activation in patients with cardiac disease (Benson et al., 1974; Guzzetta, 1989; Hoffman et al., 1982; Melville, 1987).

From a preventive perspective for self-care, health professionals might intervene in awakening behavior to control one of the risk factors that triggers cardiac events in the morning. Based on findings of this review, an experimental study should be conducted to identify the effects of interventions associated with the four wakening behaviors identified.

In conclusion, evidence indicates that activation of the sympathetic nervous system due to physical stress in the morning is a significant factor causing morning cardiac events in relation to the pattern of REM and non-REM sleep stages in patients with atherosclerosis. In addition to controlling the major cardiac risk factors causing cardiac events, four strategies for controlling the abrupt upright position at wakening should be included in patient education to reduce cardiac events for patients with cardiac disease. These strategies include waking up at the end of REM sleep, laying in bed for a few minutes after awakening, slowly raising the body from bed, and staying relaxed at least 30 minutes after awakening. Health professionals play vital roles on identifying novel behavioral risk factors that trigger morning cardiac events. Because it is important to maintain continuity of the activity, rest, sleep cycle of daily life in patients with cardiac disease, strategies for patient education should focus on preventive behaviors that control triggers of cardiac events.

5. Clinical implications for managing behavioral risk factors for morning cardiac events

Health professionals should develop educational strategies to reduce the frequency of cardiac events by modifying the patient behaviors such as abruptly assuming an upright position upon awakening in the morning. When patients wake up, they should stay in bed for few minutes and slowly sit up, then stand and walk in relaxed manner. In addition, running up and down stairs, smoking, mental stress, and other behaviors immediately after awakening should be avoided.

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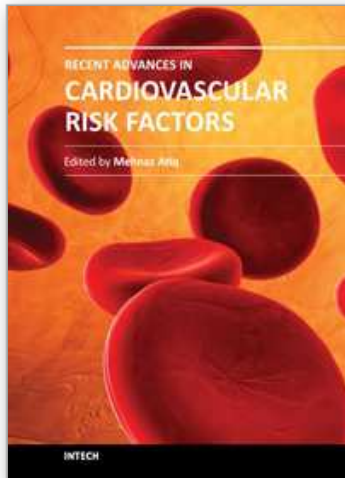
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Edited by Prof. Mehnaz Atiq

ISBN 978-953-51-0321-9

Hard cover, 522 pages

Publisher InTech

Published online 21, March, 2012

Published in print edition March, 2012

Among the non-communicable diseases, cardiovascular disorders are the leading cause of morbidity and mortality in both the developed and the developing countries. The spectrum of risk factors is wide and their understanding is imperative to prevent the first and recurrent episodes of myocardial infarction, stroke or peripheral vascular disease which may prove fatal or disabling. This book has tried to present an update on risk factors incorporating new research which has thrown more light on the existing knowledge. It has also tried to highlight regional diversity addressing such issues. It will hopefully be resourceful to the cardiologists, general practitioners, family physicians, researchers, graduate students committed to cardiovascular risk prevention.

How to reference

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Fumiko Furukawa and Tatsuya Morimoto (2012). Theoretical Identification of Behavioral Risk Factors Among Multiple Risk Factors Causing Morning Onset of Cardiac Events due to Circadian Variations, *Recent Advances in Cardiovascular Risk Factors*, Prof. Mehnaz Atiq (Ed.), ISBN: 978-953-51-0321-9, InTech, Available from: <http://www.intechopen.com/books/recent-advances-in-cardiovascular-risk-factors/theoretical-identification-of-a-behavioral-risk-factor-causing-morning-onset-of-cardiac-events-appea>

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