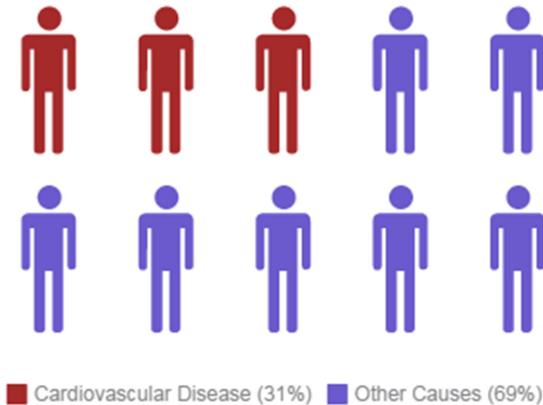


## Global Mortality from Cardiovascular Disease



Cardiovascular disease (CVD) is the leading cause of death in the United States and an increasingly significant issue in countries worldwide. In 2014, CVD was responsible for almost 17 million deaths globally and over 800,000 in the US. Although it is traditionally understood as a disease arising from diet, obesity, poor physical fitness and family history, research is increasingly being conducted on the effects of the environment--from air pollution to lead poisoning--on CVD risk.

### What Is Cardiovascular Disease?

The term CVD includes a collection of diseases involving the heart and circulatory system. Some of the most common, and serious, forms of CVD:

- **Coronary Artery Disease**

[Coronary artery disease](#) (CAD. Also known as atherosclerotic heart disease) is the most common type of heart disease in the United States and often leads to heart attacks. The condition is caused by plaque buildup (atherosclerosis) in the lining of arteries that supply blood to the heart. This plaque, often made up of cholesterol, causes the arteries to narrow over time, blocking blood flow. These plaques can erode or rupture, leading to blood clots and blockages in the artery.

- **Heart attack (myocardial infarction/cardiac arrest)**

A [heart attack](#) occurs when blood flow to part of the heart muscle is insufficient, usually due to a blood clot. CAD is the main cause of heart attacks; however, a severe spasm or sudden contraction of a coronary artery can also stop blood flow to the heart muscle.

- **High blood pressure (hypertension)**

[Hypertension](#) is a disease characterized by blood flowing through blood vessels at higher than normal pressures. Blood pressure when the heart beats and pumps blood (systolic pressure) should be below 120 mmHg, while blood pressure when the heart is at rest between beats (diastolic pressure) should be below 80 mmHg. Pressures above 120/80 mmHg weaken and damage blood vessels, leading to conditions such as **heart attack**, **heart failure**, **peripheral artery disease** and **stroke**. About one in three US adults have high blood blood pressure<sup>1</sup>.

Blood pressure that is too low (called [hypotension](#)) can also be a symptom of cardiovascular diseases such as heart failure (see below), heart attack and valve problems. Systolic pressures below 90 mmHg and diastolic pressures below 60 mmHg are considered lower than normal.

- **Stroke**

A [stroke](#) occurs when blood does not flow properly to the brain, leading to injury to brain tissue. This can result from a clot that blocks the flow of blood (called an **ischemic stroke**) or a rupture of a blood vessel in the brain, leading to bleeding (called a **hemorrhagic stroke**). Each year, it is estimated that 795,000 people in the U.S. suffer strokes, and roughly 130,000 will die of the event<sup>2</sup>.

- **Heart Failure**

[Heart failure](#) occurs when the heart is unable to pump a sufficient supply of oxygen-rich blood to the body due to weakening or stiffening of the heart muscle. Heart failure can result from coronary artery disease or high blood pressure. About 5.8 million people in the United States have heart failure, and about half of those who develop heart failure will die within five years of diagnosis<sup>3</sup>.

## Prevalence of Cardiovascular Disease

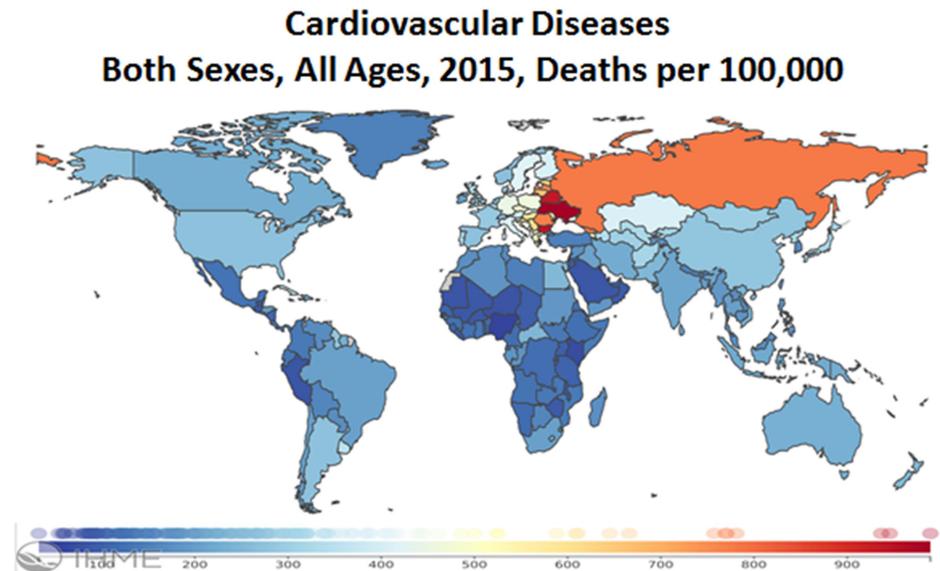
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<sup>1</sup> Writing Group Members, Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, Das SR, de Ferranti S, Després JP, Fullerton HJ, Howard VJ, Huffman MD, Isasi CR, Jiménez MC, Judd SE, Kissela BM, Lichtman JH, Lisabeth LD, Liu S, Mackey RH, Magid DJ, McGuire DK, Mohler ER 3rd, Moy CS, Muntner P, Mussolino ME, Nasir K, Neumar RW, Nichol G, Palaniappan L, Pandey DK, Reeves MJ, Rodriguez CJ, Rosamond W, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Woo D, Yeh RW, Turner MB; American Heart Association Statistics Committee; Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2016 Update: A Report From the American Heart Association. *Circulation*. 2016 Jan 26;133(4):e38-360.

<sup>2</sup> Writing Group Members, Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, Das SR, de Ferranti S, Després JP, Fullerton HJ, Howard VJ, Huffman MD, Isasi CR, Jiménez MC, Judd SE, Kissela BM, Lichtman JH, Lisabeth LD, Liu S, Mackey RH, Magid DJ, McGuire DK, Mohler ER 3rd, Moy CS, Muntner P, Mussolino ME, Nasir K, Neumar RW, Nichol G, Palaniappan L, Pandey DK, Reeves MJ, Rodriguez CJ, Rosamond W, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Woo D, Yeh RW, Turner MB; American Heart Association Statistics Committee; Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2016 Update: A Report From the American Heart Association. *Circulation*. 2016 Jan 26;133(4):e38-360.

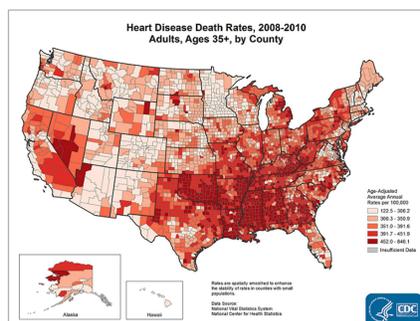
<sup>3</sup> Rodger VL. Epidemiology of Heart Failure. *Circ Res*. 2013 Aug 30; 113(6): 646–659.

Nearly one out of every three deaths worldwide is related to cardiovascular disease (CVD), making it the leading cause of global mortality<sup>4</sup>. The burden is not limited to high-income countries: 80 percent of CVD deaths occur in low- and middle-income countries<sup>5</sup>. A 2010 report by the Harvard School of Public Health estimated that CVD was responsible for \$863 billion in costs per year from treatment and loss of life-years and productivity<sup>6</sup>.



(Source: IHME: <http://vizhub.healthdata.org/gbd-compare/>)

In the US, over 85 million people live with at least one type of cardiovascular disease, with roughly half of those affected aged 60 or older. CVD remains the leading cause of death in the US, with roughly 800,000 deaths (31 percent of US total) in 2014 resulting from these diseases<sup>7</sup>.



(Source: CDC. <https://www.cdc.gov/heartdisease/facts.htm>)

<sup>4</sup> Bloom DE, Cafiero ET, Jané-Llopis E, Abrahams-Gessel S, Bloom LR, Fathima S, Feigl AB, Gaziano T, Mowafi M, Pandya A, Prettner K, Rosenberg L, Seligman B, Stein AZ, & Weinstein C. The Global Economic Burden of Noncommunicable Diseases. 2011. Geneva: World Economic Forum.

<sup>5</sup> Laslett LJ, Alagona P Jr, Clark BA 3rd, Drozda JP Jr, Saldivar F, Wilson SR, Poe C, Hart M. The worldwide environment of cardiovascular disease: prevalence, diagnosis, therapy, and policy issues: a report from the American College of Cardiology. J Am Coll Cardiol. 2012 Dec 25;60(25 Suppl):S1-49.

<sup>6</sup> Bloom DE, Cafiero ET, Jané-Llopis E, Abrahams-Gessel S, Bloom LR, Fathima S, Feigl AB, Gaziano T, Mowafi M, Pandya A, Prettner K, Rosenberg L, Seligman B, Stein AZ, & Weinstein C. The Global Economic Burden of Noncommunicable Diseases. 2011. Geneva: World Economic Forum.

<sup>7</sup> Kochanek KD, Murphy SL, Xu JQ, Tejada-Vera B. Deaths: Final data for 2014. National vital statistics reports; vol 65 no 4. Hyattsville, MD: National Center for Health Statistics. 2016.

## Trends in Cardiovascular Disease

As countries get better at preventing and treating communicable diseases like malaria and influenza, their residents are living longer lives. Because our risk for CVD increases as we age, rates of CVD are increasing worldwide. Additionally, increasing rates of known CVD risk factors, including smoking, physical inactivity, poor diet, and hypertension in low- and middle-income countries contributes to the rise in CVD. Globally, 17 million people die each year from CVD, and that is expected to increase to more than 27 million in the next 13 years<sup>8</sup>.

In the US, the trends show the opposite: deaths from cardiovascular disease have been falling for decades. After peaking in the 1950s and 1960s, CVD deaths fell steadily by roughly three percent per year until 2010<sup>9,10</sup>. Several factors have contributed to this decrease: improvements to medical treatment of CVD and its precursors has allowed patients to live longer, fuller lives with the disease. At the same time, efforts to improve population health by local and national organizations have led to better management of blood pressure and cholesterol, lower rates of smoking, and improved physical activity<sup>11,12</sup>.

## Cardiovascular Disease Risk Factors

### Lifestyle Factors

#### Nutrition

Nutrition is the most important factor influencing heart health, and any efforts at CVD prevention must address dietary choices. Diet influences not only weight and obesity, but can have strong impacts on insulin sensitivity and diabetes, inflammation throughout the body, low-density lipoprotein (LDL) levels, and oxidative stress, among others<sup>13</sup>. With constantly changing dietary fads and recommendations, it can be hard to understand what foods are helpful or harmful to

<sup>8</sup> Bloom DE, Cafiero ET, Jané-Llopis E, Abrahams-Gessel S, Bloom LR, Fathima S, Feigl AB, Gaziano T, Mowafi M, Pandya A, Prettner K, Rosenberg L, Seligman B, Stein AZ, & Weinstein C. The Global Economic Burden of Noncommunicable Diseases. 2011. Geneva: World Economic Forum.

<sup>9</sup> Cooper R, Cutler J, Desvigne-Nickens P, Fortmann SP, Friedman L, Havlik R, Hogelin G, Marler J, McGovern P, Morosco G, Mosca L, Pearson T, Stamler J, Stryer D, Thom T. Trends and disparities in coronary heart disease, stroke, and other cardiovascular diseases in the United States: findings of the national conference on cardiovascular disease prevention. *Circulation*. 2000 Dec 19;102(25):3137-47.

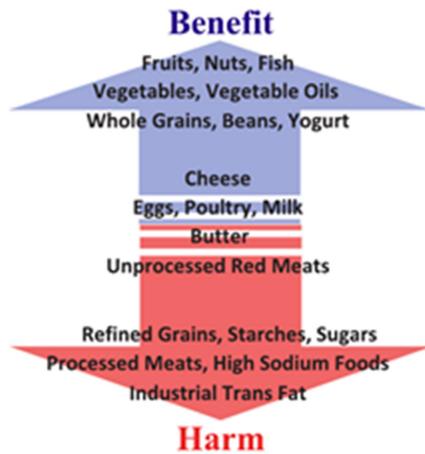
<sup>10</sup> Sidney S, Quesenberry CP Jr, Jaffe MG, Sorel M, Nguyen-Huynh MN, Kushi LH, Go AS, Rana JS. Recent Trends in Cardiovascular Mortality in the United States and Public Health Goals. *JAMA Cardiol*. 2016 Aug 1;1(5):594-9.

<sup>11</sup> Ford ES, Ajani UA, Croft JB, Critchley JA, Labarthe DR, Kottke TE, Giles WH, Capewell S. Explaining the decrease in U.S. deaths from coronary disease, 1980-2000. *N Engl J Med*. 2007 Jun 7;356(23):2388-98.

<sup>12</sup> Writing Group Members, Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, Das SR, de Ferranti S, Després JP, Fullerton HJ, Howard VJ, Huffman MD, Isasi CR, Jiménez MC, Judd SE, Kissela BM, Lichtman JH, Lisabeth LD, Liu S, Mackey RH, Magid DJ, McGuire DK, Mohler ER 3rd, Moy CS, Muntner P, Mussolino ME, Nasir K, Neumar RW, Nichol G, Palaniappan L, Pandey DK, Reeves MJ, Rodriguez CJ, Rosamond W, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Woo D, Yeh RW, Turner MB; American Heart Association Statistics Committee; Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2016 Update: A Report From the American Heart Association. *Circulation*. 2016 Jan 26;133(4):e38-360.

<sup>13</sup> Mozaffarian D. Dietary and Policy Priorities for Cardiovascular Disease, Diabetes, and Obesity: A Comprehensive Review. *Circulation*. 2016 Jan 12;133(2):187-225.

cardiovascular health. While new research on dietary health is ongoing, researchers have a good understanding of how most foods can improve, or impair, cardiovascular health.



1. Caption: From Mozaffarian D. Dietary and Policy Priorities for Cardiovascular Disease, Diabetes, and Obesity: a Comprehensive Review. *Circulation*. 2016;133:187-225.

In thinking about healthy diet, it is tempting, but usually unhelpful, to focus on a single aspect—like “low-fat” or “low-carb”. Not all fats have the same effect on cardiovascular disease risk, and not all carbs influence health equally. More important is focusing on a balanced diet. Eating more fruits, vegetables, fish, whole grains, nuts, and beans is important for cardiovascular health, and is correlated with lower rates of coronary heart disease and stroke<sup>14,15,16</sup>. Vegetable oils like extra virgin olive oil, though they are high in fat, have shown protective effects against cardiovascular disease. A 2013 study called the PREDIMED trial found that participants given extra virgin olive oil and nuts and recommended to follow a Mediterranean diet high in fish, vegetables, fruit and white meats saw a nearly 30 percent decrease in major cardiovascular events when compared to participants following a low-fat diet<sup>17</sup>. On the other hand, foods that are high in sugar, sodium, and industrial trans-fats show a harmful effect on cardiovascular health, with more heart disease and stroke<sup>18,19,20</sup>.

<sup>14</sup> Tang G, Wang D, Long J, Yang F, Si L. Meta-analysis of the association between whole grain intake and coronary heart disease risk. *Am J Cardiol*. 2015 Mar 1;115(5):625-9.

<sup>15</sup> Afshin A, Micha R, Khatibzadeh S, Mozaffarian D. Consumption of nuts and legumes and risk of incident ischemic heart disease, stroke, and diabetes: a systematic review and meta-analysis. *Am J Clin Nutr*. 2014 Jul;100(1):278-88.

<sup>16</sup> Zheng J, Huang T, Yu Y, Hu X, Yang B, Li D. Fish consumption and CHD mortality: an updated meta-analysis of seventeen cohort studies. *Public Health Nutr*. 2012 Apr;15(4):725-37.

<sup>17</sup> Estruch R, Ros E, Salas-Salvadó J, Covas MI, Corella D, Arós F, Gómez-Gracia E, Ruiz-Gutiérrez V, Fiol M, Lapetra J, Lamuela-Raventos RM, Serra-Majem L, Pintó X, Basora J, Muñoz MA, Sorlí JV, Martínez JA, Martínez-González MA; PREDIMED Study Investigators. Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med*. 2013 Apr 4;368(14):1279-90.

<sup>18</sup> Li XY, Cai XL, Bian PD, Hu LR. High salt intake and stroke: meta-analysis of the epidemiologic evidence. *CNS Neurosci Ther*. 2012 Aug;18(8):691-701.

<sup>19</sup> Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC. Trans fatty acids and cardiovascular disease. *N Engl J Med*. 2006 Apr 13;354(15):1601-13.

<sup>20</sup> Xi B, Huang Y, Reilly KH, Li S, Zheng R, Barrio-Lopez MT, Martinez-Gonzalez MA, Zhou D. Sugar-sweetened beverages and risk of hypertension and CVD: a dose-response meta-analysis. *Br J Nutr*. 2015 Mar 14;113(5):709-17.

Dietary choices are not only a matter of individual choice, but are influenced by an array of environmental and societal factors. Foods high in fat and sugar are often seen as being cheaper or more readily prepared than vegetables and whole grains. The availability and affordability of fresh fruits and vegetables can vary widely by neighborhood, influenced by socioeconomic status and ethnicity<sup>21</sup>. Advertisements and cultural influences can also shift people towards or away from particular foods. Like other environmental factors, diet is an important determinant of cardiovascular disease risk that is influenced at many levels.

## Exercise

Exercise has been shown to decrease blood pressure and reduce insulin resistance, both of which are important cardiovascular disease risk factors<sup>22, 23</sup>.

## Comorbidities

Several comorbidities are important for understanding cardiovascular disease risk and the environment.

- [Diabetes](#) patients develop cardiovascular disease at greater rates than those without diabetes, and those who develop cardiovascular disease tend to have poorer prognoses than those without diabetes<sup>24</sup>.
- Similarly, individuals with [metabolic syndrome](#) have a 53 percent increase in risk of developing CVD and 75 percent increase in risk of death from CVD<sup>25</sup>.
- High blood pressure can injure blood vessels, increasing the risk of stroke, atherosclerosis and other cardiovascular diseases<sup>26</sup>.
- Chronic kidney disease is a serious condition resulting from damage to the kidneys and leading to their failure to properly filter the blood. Risk of death from cardiovascular disease can be 10-30 times greater in people with chronic kidney disease compared to the regular population.

## Highlight Box: Metabolic Disease

<sup>21</sup> Hilmers A, Hilmers DC, Dave J. Neighborhood disparities in access to healthy foods and their effects on environmental justice. *Am J Public Health*. 2012 Sep;102(9):1644-54.

<sup>22</sup> Fagard RH. Exercise characteristics and the blood pressure response to dynamic physical training. *Med Sci Sports Exerc*. 2001 Jun;33(6 Suppl):S484-92; discussion S493-4.

<sup>23</sup> Thompson PD, Crouse SF, Goodpaster B, Kelley D, Moyna N, Pescatello L. The acute versus the chronic response to exercise. *Med Sci Sports Exerc*. 2001 Jun;33(6 Suppl):S438-45; discussion S452-3.

<sup>24</sup> Grundy SM, Benjamin IJ, Burke GL, Chait A, Eckel RH, Howard BV, Mitch W, Smith SC Jr, Sowers JR. Diabetes and cardiovascular disease: a statement for healthcare professionals from the American Heart Association. *Circulation*. 1999 Sep 7;100(10):1134-46.

<sup>25</sup> Galassi A, Reynolds K, He J. Metabolic syndrome and risk of cardiovascular disease: a meta-analysis. *Am J Med*. 2006 Oct;119(10):812-9.

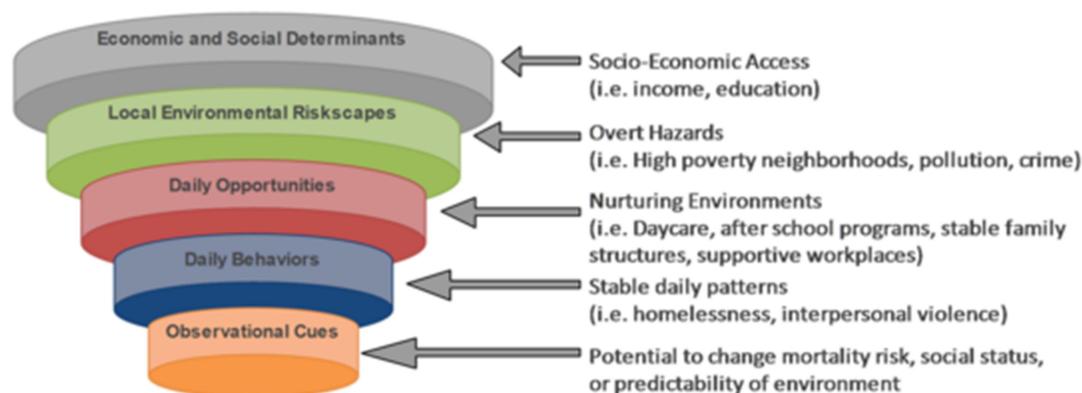
<sup>26</sup> MacMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J, Abbott R, Godwin J, Dyer A, Stamler J. Blood pressure, stroke, and coronary heart disease. Part 1, Prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. *Lancet*. 1990 Mar 31;335(8692):765-74.

Metabolic syndrome is a term for a collection of conditions that sharply increase a person's risk for cardiovascular disease, diabetes, and other outcomes. If a person has three or more of the following conditions described below, they are considered to have metabolic syndrome.

1. Abdominal obesity—or a waistline exceeding 41 inches in men
2. Blood glucose levels above 100mg/dL
3. Elevated blood pressure, with either systolic blood pressure above 130 or diastolic blood pressure above 85.
4. High levels of triglycerides in the blood—over 150mg/dL.
5. Low HDL cholesterol levels—below 40g/dL in women or below 50g/dL in men

### Cardiovascular Disease and the Social Ecological Model of Health

Our health and well-being are profoundly shaped by many aspects of the social and environmental setting in which we live. The laws and policies of our countries, states or provinces and cities can influence the conditions in which we live, work and play. They can also determine our access to health care, our education and the food we eat. These conditions can themselves influence our social interactions and community networks. Finally, our social and community environment can influence the personal life and health choices we make, and the effects those choices can have on our bodies. The social ecological model of health (SEM, covered in greater detail at the CHE [Psychosocial Environment page](#)) helps us to understand how factors influencing changes at one “level” of influence (see figure below for these levels) is situated within the larger model, and how such changes can influence other “levels.” The figure below illustrates some of these levels of influence.



**Figure by Lorelei Walker.**

Another feature of the ecological model of health is its treatment of risks and exposures for a disease like CVD as largely interrelated and unable to be easily separated from one another. For instance, a person's race can influence exposure to microaggressions and discrimination that causes stress. These experiences influence opportunities in the workplace and personal relationships at home, and can contribute to personal health choices. Because cardiovascular disease is influenced by many of these factors, as we will later learn, at many levels of the environment, they cannot be entirely separated when discussing cardiovascular disease prevention and treatment.

## Developmental Origins of Health and Disease (DOHaD) and Life Course Model

Environmental exposures in utero, or early in a child's life, can have wide-ranging and long-term effects on cardiovascular health in adulthood. Conducting research on adults in England and Wales from the 1980s into the 1990s, Dr. David Barker found that infants of lower birth weights were at an increased risk of dying from ischemic heart disease in adulthood. The trio of papers he subsequently published, outlining the important role played by environmental exposures and nutrition during pregnancy and early childhood, paved the way for the theory of Developmental Origins of Health and Disease (DOHaD)<sup>27,28,29</sup>. Underlying DOHaD theory is the idea that early life is a time of tremendous plasticity in development during which children are particularly susceptible to environmental factors. The health effects of these environmental factors may then persist long into adulthood. While this idea makes intuitive sense--that effects of childhood exposures do not simply disappear in adulthood--it shows the inadequacy of treating CVD exclusively in adults, once they are at high risk for the disease or have been diagnosed. These reports suggest that resources could be effectively aimed at decreasing childhood exposure to conditions known to increase risk of CVD later in life, such as poor nutrition and exposures to stressful situations, rather than simply treating the disease where it arises.

**Text box:** Developmental Origins of Health and Disease was described at length in a 2015 CHE presentation. Details, including slides and a recording of the talk, can be found here

([https://www.healthandenvironment.org/partnership\\_calls/17996](https://www.healthandenvironment.org/partnership_calls/17996))

## Stress and Cardiovascular Disease

When a person is exposed to a stressful environment, the cortical centers of the brain naturally respond by releasing hormones such as cortisol and adrenaline to better react to the stressor. For the cardiovascular system, this can include an increase in blood pressure, heart rate, and blood glucose in order to keep the person alert and ready for exertion. In healthy environments, when the stressor passes or is resolved, the body is able to return to its previous state and downregulate the stress-induced hormones. This process of self-regulation and homeostasis is called **allostasis**, and is explained in more detail on our [Psychosocial Environment](#) webpage.

However, in instances of repeated or chronic stress, the body's ability to regulate the stress response is impaired. This impairment due to chronic stress is called **allostatic load**, defined as "the wear and tear on the body and brain resulting from chronic overactivity or inactivity of physiological systems that are normally involved in adaptation to environmental challenge."<sup>30</sup>. Chronic stress can create a chronic state of heightened cardiovascular activity and decrease a

<sup>27</sup> Barker DJ, Gluckman PD, Godfrey KM, Harding JE, Owens JA, Robinson JS. Fetal nutrition and cardiovascular disease in adult life. *Lancet*. 1993 Apr 10;341(8850):938-41.

<sup>28</sup> Barker DJ, Winter PD, Osmond C, Margetts B, Simmonds SJ. Weight in infancy and death from ischaemic heart disease. *Lancet*. 1989 Sep 9;2(8663):577-80.

<sup>29</sup> Barker DJ, Osmond C. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet*. 1986 May 10;1(8489):1077-81.

<sup>30</sup> McEwen BS. Stress, adaptation, and disease. Allostasis and allostatic load. *Ann N Y Acad Sci*. 1998 May 1;840:33-44.

person's ability to regulate inflammation<sup>31, 32</sup>. In these instances of chronic stress, a person is at greater risk of developing high blood pressure, atherosclerosis, myocardial infarction and coronary heart disease<sup>33, 34, 35</sup>.

Populations subject to chronic stressors, particularly those of low socioeconomic status or subject to routine discrimination are at risk for high allostatic load. Levels of stress hormones, for instance, are significantly higher among those of lower socioeconomic status, independent of race and age<sup>36</sup>. Perception of discrimination can also lead to chronic stress, associated with higher blood pressure among both adults and children<sup>37, 38</sup>.

Over the course of a person's life, these chronic stressors can have a cumulative effect, increasing the risk of cardiovascular disease at each stage. When children are exposed to regular stressors, such as housing instability, childhood maltreatment and other adverse experiences, regular discrimination or those included in the box to the right, not only is their immediate risk of cardiovascular events increasing, so too is their lifetime risk. One model for understanding this steady accrual of risk is called the **life course model**, which suggests that a person's risk of disease is influenced by exposures at every age. Similar to a person's allostatic load, lifetime risk of a disease is an accumulation of risks earlier in life, from developmental exposures, to those in childhood, adulthood and old age.

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<sup>31</sup> Black PH, Garbutt LD. Stress, inflammation and cardiovascular disease. *J Psychosom Res.* 2002 Jan;52(1):1-23.

<sup>32</sup> Cohen S, Janicki-Deverts D, Doyle WJ, Miller GE, Frank E, Rabin BS, and Turner RB. Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk. *Proc Natl Acad Sci U S A.* 2012 Apr 17; 109(16): 5995–5999.

<sup>33</sup> Black PH, Garbutt LD. Stress, inflammation and cardiovascular disease. *J Psychosom Res.* 2002 Jan;52(1):1-23.

<sup>34</sup> Dimsdale JE. Psychological Stress and Cardiovascular Disease. *J Am Coll Cardiol.* 2008 Apr 1; 51(13): 1237–1246.

<sup>35</sup> Bosma H, Peter R, Siegrist J, and Marmot M. Two alternative job stress models and the risk of coronary heart disease. *Am J Public Health.* 1998 January; 88(1): 68–74.

<sup>36</sup> Cohen S, Doyle WJ, Baum A. Socioeconomic status is associated with stress hormones. *Psychosom Med.* 2006 May-Jun;68(3):414-20.

<sup>37</sup> Thomas KS, Nelesen RA, Ziegler MG, Bardwell WA, Dimsdale JE. Job strain, ethnicity, and sympathetic nervous system activity. *Hypertension.* 2004 Dec;44(6):891-6.

<sup>38</sup> Gosby BJ, Malone S, Richardson E, Cheadle JE, Williams D. Perceived Discrimination and Markers of Cardiovascular Risk among Low-Income African American Youth. *Am J Hum Biol.* 2015 Jul 8; 27(4): 546–552.

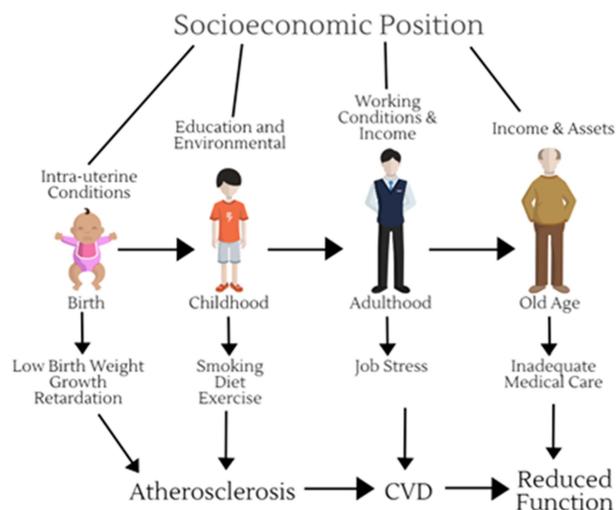


Figure Text: **Lifecourse Model**. Figure created using PiktoChart. Content developed from Berkman LF, Kawachi I, editors. *Social Epidemiology*. New York: Oxford University Press; 2000.

#### List of Common Stressors:

- Poverty and Socioeconomic Stress
- Community Violence, Crime
- Household Violence
- Housing Instability
- Discrimination/Perceived Racism
- Work Related Stresses
- Adverse Childhood Experience

### Infectious Disease and Cardiovascular Disease Risk

While cardiovascular disease is not infectious, there are well established links between CVD and some infectious diseases. The reason for this may be that systemic inflammation seen in infections can have damaging effects on blood vessels and the heart, increasing the risk for heart disease down later in life. Periodontal disease—an infection of the gum often precipitated by poor oral hygiene—has been shown to be associated with cardiovascular disease, though it is not clear whether the infection *causes* CVD itself<sup>39, 40</sup>. Inflammation throughout the body are seen in Herpes Simplex 1 and Chlamydia pneumoniae and may increase risk for cardiovascular disease. Finally, ongoing research is investigating the relationship between HIV and cardiovascular disease, people living with HIV are shown to have a greater risk of CVD.

### Environmental Exposures and Disease Associations

<sup>39</sup> DeStefano F, Anda RF, Kahn HS, Williamson DF, Russell CM. Dental disease and risk of coronary heart disease and mortality. *BMJ*. 1993 Mar 13;306(6879):688-91.

<sup>40</sup> Beck J, Garcia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease and cardiovascular disease. *J Periodontol*. 1996 Oct;67(10 Suppl):1123-37.

Below is a table outlining environmental exposures, risk factors, and associations with cardiovascular diseases.

Disease	Social, Behavioral, or Medical Risk Factors	Toxicants	
		Strong Evidence	Good Evidence
Coronary Artery Disease	Diets high in sodium Physical Inactivity Smoking Tobacco	Air Pollution Tobacco Smoke Carbon disulfide Dioxins	Lead Arsenic Mercury Cadmium Bisphenol A
Heart Attack	Physical Inactivity Unhealthy diet Smoking tobacco High stress High levels of noise	Tobacco smoke Nitrates/nitrites Hydrogen sulfide Cyanide Solvent Air pollution	Arsenic Lead Mercury Bisphenol A
Chest Pain	Diets high in sodium and fat Physical inactivity Tobacco use		Bisphenol A (BPA) Tobacco smoke Air pollution
Heart Failure	Coronary heart disease Heart attacks High blood pressure Diabetes Smoking tobacco Diets high in fat, cholesterol, and sodium Physical inactivity Obesity	Air pollution	Cadmium
Cardiomyopathy	Family History of cardiomyopathy, heart failure, or sudden cardiac arrest Connective tissue disease Coronary heart disease Heart attack Alcoholism or cocaine abuse		

	Muscle conditions		
Stroke	High blood pressure High cholesterol Heart disease Diabetes Overweight or obesity History of stroke Smoking tobacco Excess alcohol consumption Poor diet Physical inactivity	Air pollution Tobacco smoke	Cadmium Lead
Arrhythmias	Increasing age High blood pressure Obesity European ancestry Diabetes Heart failure Ischemic heart disease Hyperthyroidism Chronic kidney disease Excess alcohol consumption	Cyanide Air pollution	Antimony Arsenic Pesticides Solvents Lead Tobacco smoke Chloroform
Congenital heart defects			Anesthetic gases Solvents Tobacco smoke Pesticides Air pollution
Peripheral Arterial Disease		Arsenic	Lead Cadmium Bisphenol A (BPA)
High Blood Pressure	Obese or overweight Diet high in sodium Physical inactivity High alcohol consumption Smoking Tobacco Family history of high blood pressure	Air pollution Solvents Mercury	Arsenic Cadmium Pesticides PCB Bisphenol A (BPA) Lead
Vasculitis	Chronic Hepatitis B or C Autoimmune diseases such as lupus, rheumatoid		

	arthritis, and scleroderma Smoking Tobacco		
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For clearer explanation of the criteria for each designation of association, see the [CHE Toxicants and Disease Database](#)

## Secondhand Smoke

Dozens of studies have looked at the effects of secondhand smoke (SHS) on cardiovascular disease. When their results are studied collectively, they show an increase in stroke of roughly 25 percent and an increase in heart disease between 20 and 40 percent. Secondhand smoke can influence the cardiovascular system in many ways, from increasing inflammation, insulin resistance and arterial resistance, to decreasing metabolism and HDL cholesterol levels<sup>41</sup>. SHS is known to increase platelet formation among those exposed, which may damage the lining of the arteries and increase the risk of blood clots. Although secondhand smoke may seem less serious compared to smoking, as exposure time increases, risks quickly approach those of smokers<sup>42</sup>.

As the long-term health effects of smoking have become more readily understood, policymakers have looked to decrease exposure to harmful smoke through laws prohibiting smoking in workplaces, some housing and public spaces. The aim of these policies is both to decrease exposure to secondhand smoke and to dissuade smokers from continuing their habit<sup>43</sup>. Seattle, for instance, following the national trend, adopted a “Smoking in Public Places” policy in 2005 that prohibited smoking in workplaces and indoor public places, which included restaurants and bars. The Washington State Department of Health touts the law as having led to a decrease in secondhand smoke, indoor air pollution, and smoking overall<sup>44</sup>. When the effects of many policies throughout the US are studied together, they show a 39 percent decrease in chest pain, coronary heart disease and sudden cardiac death. Additionally, a roughly 15 percent decrease in myocardial infarction, coronary syndrome, coronary events ischemic heart disease and stroke have been shown<sup>45</sup>.

## Air Pollution

<sup>41</sup> Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking. *Circulation*. 2005 May 24;111(20):2684-98.

<sup>42</sup> Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking. *Circulation*. 2005 May 24;111(20):2684-98.

<sup>43</sup> Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking. *Circulation*. 2005 May 24;111(20):2684-98.

<sup>44</sup> Washington State Department of Health. Smoking in Public Places Law. <http://www.doh.wa.gov/YouandYourFamily/Tobacco/LawsRegulations/SmokinginPublicPlacesLaw>. Accessed 2 Mar 2017.

<sup>45</sup> Tan CE, Glantz SA. Association between Smokefree Legislation and Hospitalizations for Cardiac, Cerebrovascular and Respiratory Diseases: A Meta-Analysis. *Circulation*. 2012 Oct 30; 126(18): 2177–2183.

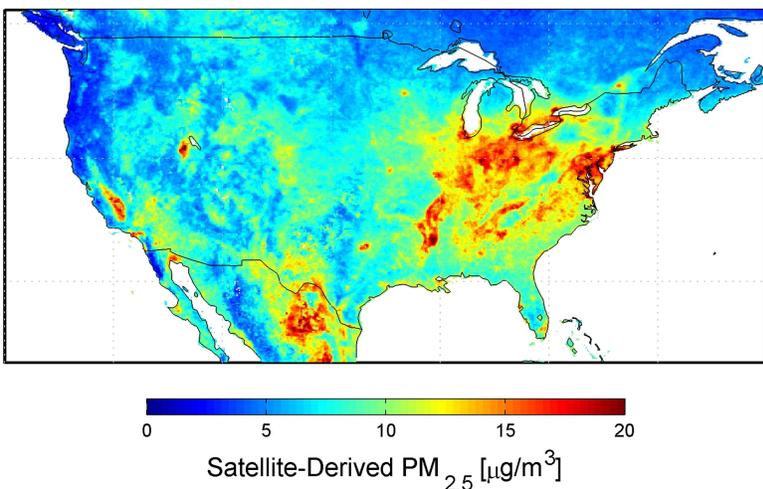


Image Text: Pollution levels in the United States, averaged between 2001-2006. Source: NASA

Air pollution resulting from factories, auto exhaust, smoking, and other sources of particulate matter are a primary concern when discussing environmental exposure and cardiovascular disease risk. When we talk about air pollution and its health risks, we may refer to fine particles (PM<sub>2.5</sub>: 2.5 micrometers in diameter), which arise largely from coal, oil, or wood combustion, or coarse particles (PM<sub>10</sub>: 10 micrometers or smaller in diameter), those arising from mining, construction and road traffic<sup>46</sup>. Pollution tends to be concentrated in urban areas or cities with large amounts of manufacturing. In low-income countries, additionally, the burning of coal, wood, or other materials in the home can lead to higher levels of household air pollution<sup>47</sup>. A recent report by the World Health Organization (WHO, an arm of the United Nations focused on international public health) on household air pollution estimates that globally 3.7 million deaths were associated with ambient air pollution in 2012, with the majority of those coming from cardiovascular disease<sup>48</sup>.

Air pollution from fine particles has been associated with risk of ischemic heart disease, arrhythmias, heart failures and cardiac arrest, in addition to overall cardiovascular mortality. Indeed, the majority of deaths arising from air pollution are caused by cardiovascular disease, primarily ischemic heart disease and stroke<sup>49, 50</sup>. Exposure to pollution appears to increase not only someone's immediate risk of cardiovascular events, but can have longer-term effects as well. Though research is continually being done on the mechanisms through which air pollution

<sup>46</sup> Laden F, Neas LM, Dockery DW, Schwartz J. Association of fine particulate matter from different sources with daily mortality in six U.S. cities. *Environ Health Perspect*. 2000 Oct; 108(10): 941–947.

<sup>47</sup> Bruce N, Perez-Padilla R, Albalak R. Indoor air pollution in developing countries: a major environmental and public health challenge. *Bull World Health Organ*. 2000;78(9):1078-92.

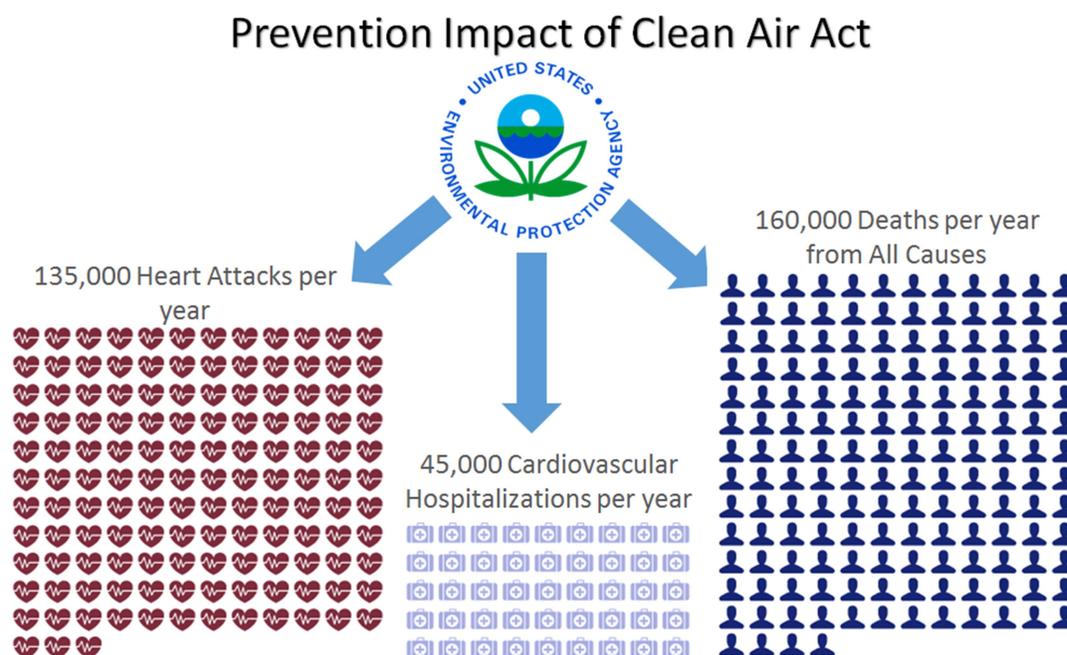
<sup>48</sup> Gall ET, Carter EM, Earnest CM, Stephens B. Indoor Air Pollution in Developing Countries: Research and Implementation Needs for Improvements in Global Public Health. *Am J Public Health*. 2013 April; 103(4):

<sup>49</sup> Pope CA 3rd, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. *Circulation*. 2004 Jan 6;109(1):71-7.

<sup>50</sup> Bhatnagar A. Environmental cardiology: studying mechanistic links between pollution and heart disease. *Circ Res*. 2006 Sep 29;99(7):692-705.

acts on the circulatory system, recent studies suggest that it can activate inflammatory pathways, leading to atherosclerosis, and interfere with heart rhythm<sup>51</sup>.

Air pollution is recognized by many governments for its danger to public health, particularly cardiovascular disease. In the U.S., National Ambient Air Quality Standard (NAAQS) were established with the Clean Air Act in 1990, with the primary goal of protecting public health, particularly for groups at greater risk, including children and the elderly. These standards are updated every five years to ensure they keep pace with our scientific understanding of the risks of air pollution. Though it is difficult to precisely estimate the effects of such large-scale policies, the Environmental Protection Agency (EPA) in 2010 estimated that 130,000 heart attacks and 45,000 cardiovascular hospital admissions, in addition to 160,000 deaths from all causes, were prevented yearly because of Clean Air Act regulations<sup>52</sup>.



**Figure Text:** Impact of the Clean Air Act on prevention of disease. Each icon represents 1,000 cases prevented due to clean air act regulations (heart attack, cardiovascular hospitalization, all-cause death). Data from the U.S. Environmental Protection Agency Office of Air and Radiation.

Arsenic

<sup>51</sup> Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F, Hong Y, Luepker RV, Mittleman MA, Peters A, Siscovick D, Smith SC Jr, Whitsel L, Kaufman JD; American Heart Association Council on Epidemiology and Prevention, Council on the Kidney in Cardiovascular Disease, and Council on Nutrition, Physical Activity and Metabolism. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. *Circulation*. 2010 Jun 1;121(21):2331-78.

<sup>52</sup> U.S. Environmental Protection Agency Office of Air and Radiation. The Benefits and Costs of the Clean Air Act from 1990 to 2020. April 2011. Final Report

Exposure to arsenic, a particularly toxic heavy metal at high levels, can arise from a number of environmental sources. Found naturally in the earth's crust, arsenic can leach into groundwater. Human activity can also release arsenic and introduce it to the water supply through mining waste and after use as a treatment for wood and agricultural fertilizer. It is introduced to the air through tobacco smoke and industrial activities<sup>53, 54</sup>. In several studies in Taiwan, long-term exposure to high levels of arsenic in drinking water has been associated with a thickening of small and medium-sized arteries in children and young adults, putting them at greater risk for CVD-related death<sup>55</sup>. In the United States, where arsenic in drinking water is more strictly regulated and levels of the toxicant are low, research on arsenic has not shown a similarly strong effect on CVD risk<sup>56</sup>.

## Lead

Lead is a common toxic heavy metal. Previously widely used as an additive in gasoline, house paint, water pipes and batteries, lead has since been shown to have toxic properties, particularly among children. Lead is absorbed into the blood upon exposure and accumulates in bones, where it can remain for decades, steadily releasing lead into the bloodstream<sup>57</sup>. Its use in paint was prohibited in 1978, and it was removed from gasoline through the 1970s and 1980s. Lead is most widely known for its neurological effects, particularly on cognitive development, making it particularly dangerous if ingested or inhaled by children<sup>58</sup>. Increased cardiovascular disease risk is another consequence of lead poisoning, with several studies showing a small but significant risk of hypertension after lead exposure<sup>59</sup>. Additionally, lead exposure has been associated with a significant increase in CVD mortality, myocardial infarction, stroke, ischemic heart disease, and atherosclerosis<sup>60</sup>.

## Cadmium

Cadmium is a toxic element and known carcinogen used in batteries and electroplating to prevent corrosion. Environmental exposure to cadmium can additionally arise from cigarette smoke and fuel consumption. There is some evidence that much of the increased risk of peripheral arterial disease that is known to arise from smoking is the result of exposure to

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<sup>53</sup> Solenkova NV, Newman JD, Berger JS, Thurston G, Hochman JS, Lamas GA. Metal pollutants and cardiovascular disease: mechanisms and consequences of exposure. *Am Heart J*. 2014 Dec;168(6):812-22.

<sup>54</sup> Arsenic in Drinking-water: Background document for development of WHO Guidelines for Drinking-water Quality. World Health Organization, 2011

<sup>55</sup> Navas-Acien A, Sharrett AR, Silbergeld EK, Schwartz BS, Nachman KE, Burke TA, Guallar E. Arsenic exposure and cardiovascular disease: a systematic review of the epidemiologic evidence. *Am J Epidemiol*. 2005 Dec 1;162(11):1037-49.

<sup>56</sup> Navas-Acien A, Sharrett AR, Silbergeld EK, Schwartz BS, Nachman KE, Burke TA, Guallar E. Arsenic exposure and cardiovascular disease: a systematic review of the epidemiologic evidence. *Am J Epidemiol*. 2005 Dec 1;162(11):1037-49.

<sup>57</sup> Solenkova NV, Newman JD, Berger JS, Thurston G, Hochman JS, Lamas GA. Metal pollutants and cardiovascular disease: mechanisms and consequences of exposure. *Am Heart J*. 2014 Dec;168(6):812-22.

<sup>58</sup> Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. *N Engl J Med*. 1990 Jan 11;322(2):83-8.

<sup>59</sup> Pirkle JL, Schwartz J, Landis JR, Harlan WR. The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. *Am J Epidemiol*. 1985 Feb;121(2):246-58.

<sup>60</sup> Solenkova NV, Newman JD, Berger JS, Thurston G, Hochman JS, Lamas GA. Metal pollutants and cardiovascular disease: mechanisms and consequences of exposure. *Am Heart J*. 2014 Dec;168(6):812-22.

cadmium in cigarettes<sup>61</sup>. A recent review of the many studies investigating the health effects of cadmium exposure showed elevated risks of cardiovascular disease, coronary heart disease, stroke and peripheral artery disease<sup>62</sup>. Experimental studies have suggested that cadmium may act to increase CVD risk through atherosclerosis, initiating the buildup of plaques in the arteries of those exposed<sup>63</sup>.

## Epigenetic Factors

Though our understanding genetic, behavioral and environmental foundations of cardiovascular disease have improved significantly in recent decades, we still do not understand all of the variability in CVD risk among the population. One relatively new area of study called *epigenetics* may help us understand how environmental exposures can interact with genetics to influence CVD risk. Though genes are passed down from parents to children, and typically do not change throughout a person's life, environmental exposures and stressors can influence how the body *expresses* genes. They can do this by marking DNA, often by a process called methylation, which can modify the gene's expression. This process does not alter the underlying DNA, but these marks and their effects can remain with a person throughout their life.

Measurements of epigenetic changes or differences has allowed researchers to get a more detailed look at the mechanisms through which environmental exposure may affect cardiovascular health. For instance, studies have found different levels of DNA methylation when comparing people exposed to different levels of air pollution<sup>64</sup>. Additionally, different levels of methylation are seen comparing patients with hypertension, ischemic heart disease, and stroke to those without these conditions<sup>65</sup>. It is thought that environmental exposures may affect epigenetic regulation through several of the inflammatory responses they induce<sup>66</sup>.

## Policy and Cardiovascular Disease

Governmental policy, at both the local and national scale, has been tremendously important in translating scientific research on environmental exposures into meaningful changes in health. For cardiovascular disease, the key policies to decreasing environmental exposures have been those focused on smoking prevention and air pollution.

<sup>61</sup> Navas-Acien A, Selvin E, Sharrett AR, Calderon-Aranda E, Silbergeld E, Guallar E. Lead, cadmium, smoking, and increased risk of peripheral arterial disease. *Circulation*. 2004 Jun 29;109(25):3196-201.

<sup>62</sup> Tellez-Plaza M, Jones MR, Dominguez-Lucas A, Guallar E, Navas-Acien A. Cadmium exposure and clinical cardiovascular disease: a systematic review. *Curr Atheroscler Rep*. 2013 Oct;15(10):356.

<sup>63</sup> Messner B, Knoflach M, Seubert A, Ritsch A, Pfaller K, Henderson B, Shen YH, Zeller I, Willeit J, Lauffer G, Wick G, Kiechl S, Bernhard D. Cadmium is a novel and independent risk factor for early atherosclerosis mechanisms and in vivo relevance. *Arterioscler Thromb Vasc Biol*. 2009 Sep;29(9):1392-8.

<sup>64</sup> Baccarelli A, Wright RO, Bollati V, Tarantini L, Litonjua AA, Suh HH, Zanobetti A, Sparrow D, Vokonas PS, Schwartz J. Rapid DNA methylation changes after exposure to traffic particles. *Am J Respir Crit Care Med*. 2009 Apr 1;179(7):572-8.

<sup>65</sup> Baccarelli A, Tarantini L, Wright RO, Bollati V, Litonjua AA, Zanobetti A, Sparrow D, Vokonas PS, Schwartz J. Repetitive element DNA methylation and circulating endothelial and inflammation markers in the VA normative aging study. *Epigenetics*. 2010 Apr;5(3):222-8.

<sup>66</sup> Baccarelli A, Ghosh S. Environmental exposures, epigenetics and cardiovascular disease. *Curr Opin Clin Nutr Metab Care*. 2012 Jul;15(4):323-9.

In the US and worldwide, policies are being developed that acknowledge and intervene on risks of cardiovascular disease. These policies rely on reports generated by both national and international organizations. The World Health Organization releases a biennial “Global Status Report on Noncommunicable Diseases” whose stated target audience is ministers of health<sup>67</sup>. Similarly, the Institute for Health Metrics and Evaluation at the University of Washington generates its own reports on the global burden of disease, including cardiovascular disease, with the aim to inform and guide policymakers<sup>68</sup>. In a more disease-focused manner, national health associations and nonprofits often release statements, reviews, and recommendations with an eye towards policy makers. The American Heart Association, for instance, released a 2004 statement for healthcare professionals and regulatory agencies regarding Air Pollution and Cardiovascular Disease<sup>69</sup>. Because research findings can often be inaccessible to anyone outside the field of public health, such statements and reports are essential for translating the most up-to-date understanding of exposures and cardiovascular disease.



In the US, the EPA is a key agency with regards to cardiovascular disease prevention. Interventions such as the National Ambient Air Quality Standards (NAAQS) are important large-scale efforts to decrease cardiovascular disease risk factors. Often, cardiovascular disease prevention efforts focus on individuals at high risk, who are then offered advice on how to lower their own risk factors. This is often ineffective and does not fully address the inequalities that exist in CVD<sup>70</sup>. Population-level measures, however, including the NAAQS and others, work better to address cardiovascular risks in the whole population, not simply those in a position to make lifestyle or environmental changes themselves. This is particularly important because we know race to be a strong indicator of environmental inequality, and thus places non-whites at higher risk of CVD by dint of where they live<sup>71</sup>.

<sup>67</sup> World Health Organization. Global status report on noncommunicable diseases 2014. World Health Organization, 2014.

<sup>68</sup> Institute for Health Metrics and Evaluation. The Global Burden of Disease: Generating Evidence, Guiding Policy. Seattle, WA: IHME, 2013.

<sup>69</sup> Brook RD, Franklin B, Cascio W, Hong Y, Howard G, Lipsett M, Luepker R, Mittleman M, Samet J, Smith SC Jr, Tager I; Expert Panel on Population and Prevention Science of the American Heart Association. Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation*. 2004 Jun 1;109(21):2655-71.

<sup>70</sup> Capewell S, Graham H. Will cardiovascular disease prevention widen health inequalities? *PLoS Med*. 2010 Aug 24;7(8):e1000320.

<sup>71</sup> Downey L, Hawkins B. Race, Income, and Environmental Inequality in the United States. *Sociol Perspect*. 2008 Dec 1;51(4):759-781.

## Prevention

Cardiovascular disease is perhaps the greatest worldwide public health threat, but much can be done on the personal, community, national and global level to improve heart health and effectively prevent the disease. The most effective solutions are also the simplest. Maintaining a healthy diet low in sugars, processed meats and refined grains is particularly effective, along with including fruits, vegetables, fish, vegetable oils, nuts and beans in your daily diet (see the section above on diet). The American Heart Association includes [recommendations on their website](#) that are helpful for planning dietary choices.

Regular exercise is important for maintaining cardiovascular health, and even 30 minutes of walking per day is associated with a lower risk of coronary heart disease, stroke, and overall cardiovascular disease<sup>72, 73</sup>.

Smoking, as described above, is known to contribute strongly to cardiovascular disease. Refraining from smoking or being around secondhand smoke is an important to protect against CVD.

In your household, neighborhood, and community, be aware of factors that are known to contribute to cardiovascular disease. Lead paint in houses, air pollution, and other environmental exposures described above may influence your risk of developing cardiovascular disease.

At the policy level, efforts to monitor and regulate air pollution have been cost-effective in decreasing cardiovascular disease risk nationwide.

*This document is student work. CHE makes no claim that all the information has been verified.*

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<sup>72</sup> Carnethon MR. Physical Activity and Cardiovascular Disease: How Much is Enough? Am J Lifestyle Med. 2009 Jul;3(1 Suppl):44S-49S.

<sup>73</sup> Oguma Y, Shinoda-Tagawa T. Physical activity decreases cardiovascular disease risk in women: review and meta-analysis. Am J Prev Med. 2004 Jun;26(5):407-18.