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The Importance of a Healthy Lifestyle in the Era of COVID-19

Kristina S. Petersen, PhD, APD, Andrew M. Freeman, MD,
Penny M. Kris-Etherton, PhD, RDN, Kim Allan Williams Sr, MD,
MACC, Koushik R. Reddy, MD, FACLM, FACC,
Monica Aggarwal, MD, FACC, Neal D. Barnard, MD,
Dean Ornish, MD, Caldwell B. Esselstyn Jr, MD,
Kathleen Allen, MS RD, Karen Aspry, MD, MS

Abstract

Existing cardiovascular disease (CVD) and its modifiable risk factors are associated with increased mortality from coronavirus disease 2019 (COVID-19). Clinical attention has focused on acute interventions for COVID-19, but reducing upstream risks associated with poor outcomes must occur in parallel. This is particularly urgent because risk factors for COVID-19 death are prevalent, and the pandemic has negatively impacted lifestyle and socioeconomic factors that augment these risks. Evidence-based lifestyle interventions have a generally short time-to-benefit, lower the risk of CVD, and improve markers of immune function. Wider promotion of healthy lifestyle practices by clinicians and other members of the healthcare team, including during telehealth visits, will improve population-wide cardiovascular health and could favorably impact COVID-19 outcomes. Research examining how lifestyle modification affects COVID-19 susceptibility and severity is urgently needed.

Keywords: Coronavirus 2019; Cardiovascular disease; Lifestyle; Immune function

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Introduction

Underlying cardiovascular disease (CVD) and the presence of CVD risk factors are associated with greater likelihood of adverse outcomes from coronavirus disease 2019 (COVID-19), including mortality.[1] Worse COVID-19 outcomes have been observed

Petersen [a,b*], Freeman [c*], Kris-Etherton [b], Williams Sr [d], Reddy [e], Aggarwal [f], Barnard [g], Ornish [h], Esselstyn Jr [j], Allen [j], Aspry [k]

*Co-First Authors

[a]Department of Nutritional Sciences, Texas Tech University, Lubbock, TX

[b]Department of Nutritional Sciences, Pennsylvania State University, University Park, PA

[c]Division of Cardiology, Department of Medicine, National Jewish Health, Denver, CO

[d]Division of Cardiology, Rush University Medical Center, Chicago, IL

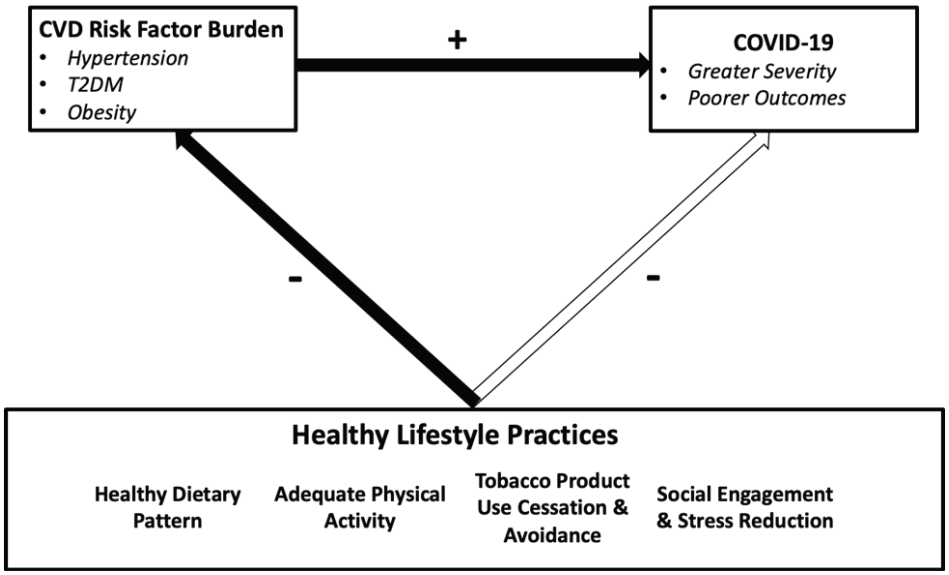
[e]College of Medicine, University of South Florida, Tampa, FL

[f]Division of Cardiology, University of Florida/Shands Hospital, Gainesville, FL

[g]Adjunct Faculty, George Washington University School of Medicine, Physicians Committee for Responsible Medicine, Washington, DC

[h]Preventive Medicine Research Institute, Sausalito, California, Department of Medicine, University of California, San Francisco, San

in individuals greater than 60 years of age, and those of any age with known CVD, obesity, type 2 diabetes mellitus (T2DM), hypertension, or current smokers.[2,3] Increased risk of severe COVID-19 is also observed with lung, kidney, or liver disease, and in those who are immunocompromised. However, the principal risks for poor outcomes from COVID-19 are modifiable and strongly impacted by lifestyle behaviors. In this narrative review, we summarize the effect of modifiable lifestyle factors on CVD risk and discuss evidence-based approaches that clinicians can implement in their practices to promote healthy lifestyle behaviors (central figure). We also highlight the impact of lifestyle behaviors on markers of immune function, which has clinical relevance in the era of COVID-19. In addition, we emphasize the need for research examining how lifestyle modification affects COVID-19 susceptibility and outcomes, which is currently lacking because of the novelty of COVID-19.



Central Figure: Known and hypothesized inter-relationships between healthy lifestyle practices, CVD risk factor burden, and COVID-19 severity and outcomes.

Notes: CVD risk factor burden is positively associated (+) with COVID-19 severity and poorer outcomes. Healthy lifestyle practices are inversely related (–) to CVD risk factor burden. We hypothesize that healthy lifestyle practices are inversely associated (–) with COVID-19 severity and poorer outcomes, although supportive evidence is currently lacking. Healthy lifestyle practices may also impact COVID-19 severity and outcomes by reducing CVD risk factor burden.

CVD risk factors and COVID-19

Differences in outcomes from COVID-19 are well documented. Among 5279 patients with confirmed COVID-19 diagnosed at an academic health system in New York, an outcomes analysis that used rigorous case ascertainment showed that the risk of hospitalization was greater than 2-fold higher for those with a body mass index (BMI) > 40 kg/m^2, heart failure, or diabetes mellitus (type not specified).[2] BMI > 40 kg/m^2 and heart failure increased the odds of critical illness and mortality by 1.5- and 1.9-fold, respectively. Similarly, in an analysis of 17 million electronic health records from the National Health Service in England, the risk of death from COVID-19 was ~2-fold higher in those with a BMI ≥ 40 kg/m^2, or poorly controlled diabetes mellitus (type not specified) defined as an HbA1c ≥ 7.5%.[4] A multi-center analysis, including 7337 hospitalized patients with COVID-19 in Hubei Province, China, that used propensity-score matching, showed that patients with T2DM who had poor versus good

glycemic control (HbA1c of 7.2–10.1% vs. 6.6–8.2%, respectively) had a 10-fold higher risk of death (11.0% vs. 1.1%, respectively), a finding hypothesized to be related to dysregulated immune function.[5] Finally, in a meta-analysis of 11 case series, the risk for severe COVID-19 outcomes was increased approximately 2-fold among current smokers; pre-existing chronic obstructive pulmonary disease also significantly increased the risk of death.[6]

In addition to clinical factors, data show that racial and socioeconomic factors are linked to higher rates of COVID-19 and mortality. Yancy found that among 131 predominantly black US counties, COVID-19 prevalence was 3-fold higher than in white communities, and the death rate was 6-fold higher.[7] In the National Health Service analysis, hospitalized black patients had a higher risk of death from COVID-19 compared to whites (HR 2.17, 95% CI 1.84, 2.57) that was partially reduced after adjusting for clinical factors and social disadvantage (HR 1.71, 95% CI 1.44, 2.02).[3] In this analysis, greater social disadvantage was associated with a near doubling of the adjusted death rate (HR 1.75, 95% CI 1.60, 1.91). In a recent analysis of 3481 COVID-19 positive patients from a Louisiana Health System, black race was not associated with increased risk of in-hospital mortality after adjustment for sociodemographic and clinical characteristics (OR 0.89, 95% CI 0.68, 1.17), suggesting risk factors drive mortality more than race.[8]

It is unclear whether improving CVD risk factors linked to more severe COVID-19 manifestations improves outcomes from the disease. However, evidence that a gradient of COVID-related risk exists for obesity and T2DM is an impetus to improve these risk factors through lifestyle and other medical interventions. The large burden of modifiable CVD risk factors in the United States and globally increases this urgency.[9] Data from the Behavioral Risk Factor Surveillance System suggest that 45% of US adults are at increased risk of complications from COVID-19 because of existing chronic conditions including obesity, T2DM, and hypertension.[10] Moreover, the negative impacts of COVID-19 on access to healthcare, healthy food, and physical exercise, and on traumatic stress and social isolation, may increase the burden of lifestyle-related preventable cardiometabolic diseases.[11] Finally, the established links between poor nutrition, obesity, inactivity, smoking, and psychological factors on immune function are another impetus to intensify efforts to improve lifestyle-related risk factors in the era of COVID-19.

Effects of lifestyle behaviors on CVD risk factors and outcomes

A healthy dietary pattern, maintenance of a healthy body weight, regular physical activity, avoidance of cigarette smoking, and stress reduction are cornerstones of CVD prevention and management.[12,13] In an analysis of 3 prospective and one cross-sectional study, a healthy lifestyle (3 of 4 criteria: no smoking, no obesity, regular physical activity, and a healthy diet) compared to an unhealthy lifestyle (≤1 of the 4 criteria) was associated with a 46% reduction in the relative risk of coronary heart disease (CHD) in individuals with high genetic predisposition.[14] This risk reduction was comparable to that observed in individuals at low genetic risk (45%). In a large cohort of individuals from 41 countries, adherence to diet and exercise recommendations, and smoking cessation following acute coronary syndrome, reduced the risk of myocardial infarction by 42-48% at 6 months[15]; indicating a short time-to-benefit, as previously found for lifestyle interventions.[16]

Nutrition

In the United States, only 1.4% of adults have an ideal diet, defined by the American

Francisco, CA
[i]The Cleveland Clinic Wellness Institute, Cleveland, OH
[j]Geisel School of Medicine at Dartmouth, Hanover, NH
[k]Lifespan Cardiovascular Institute, and Division of Cardiovascular Medicine, Brown University, Alpert Medical School, Providence, RI

Corresponding author
Karen Aspry, MD, MS
Lifespan Cardiovascular Institute,
1454 South County Trail,
East Greenwich, RI 02818
Email: KAspry@lifespan.org

Heart Association (AHA) Healthy Diet score; 50% of the population have a poor diet.[9] Of particular concern, <8% of US adults meet recommendations for sodium (< 2300 mg/day) and whole grain intake (3 ounce-equivalents/day), < 10% meet recommendations for intake of non-starchy vegetables (≥ 2 cups/day), < 20% meet recommendations for fruits (≥ 2 cups/day), and < 40% meet recommendations for saturated fat (< 10% calories) and added sugar intake (< 6.5% of calories); marked disparities exist in dietary intake across racial and ethnic groups.[9] Non-Hispanic black adults and those who have lower education or income levels or use food assistance programs are disproportionately affected by poor diet quality.[17-20] Based on these data, modeling analyses show that 48% of CVD deaths in the United States are caused by poor diet,[21] an attributable risk greater than for any other modifiable risk factor.[22] These trends are likely to worsen as a result of the pandemic, which has increased food insecurity, and impacted normal routines and feelings of stress and boredom, all of which are associated with overconsumption of calories and nutrient-poor, energy-dense "comfort" foods.[23,24] Early surveys have documented increased caloric intake and weight gain during COVID-19.[25-27]

Robust evidence shows improving diet quality lowers CVD risk. In randomized controlled trials, healthy dietary patterns significantly improve blood pressure, lipids/lipoproteins, glycemic control, markers of inflammation, and endothelial function.[28-33] In addition, randomized controlled trials show the Mediterranean diet reduces the risk of CVD events.[34,35] In alignment, an analysis of the Nurses' Health Study and Health Professionals Study showed that each 20-percentile increase in diet quality was associated with a 7–15% reduction in the relative risk of CVD death over 12 years,[36] and diet quality in the highest quintile was associated with a 33% reduction in the relative risk of CVD death.[37] In survivors of myocardial infarction in these cohorts, those in the highest quintile of diet quality, measured by the Alternative Healthy Eating Index, had a pooled adjusted hazard ratio for total mortality of 0.76 (95% CI 0.60, 0.96, $p = 0.02$).[38]

Recommended dietary patterns are appropriate for energy needs and abundant in fruits, vegetables, especially dark greens, legumes, whole grains, nuts and seeds, low-fat/fat-free dairy, and vegetable protein or lean animal protein, especially fish, in addition to being low in saturated fat, sodium, processed meats, and added sugars.[39-41] Several dietary patterns meet these criteria, including the 2015-2020 Dietary Guidelines for Americans eating patterns (i.e., Healthy US-Style, Healthy Mediterranean-Style, and Healthy Vegetarian diets), as well as the Dietary Approaches to Stop Hypertension diet.[39,41,42]

Diet should be assessed and discussed at the point of care with clinicians and other members of the healthcare team to reduce the incidence and improve the management of diet-related chronic disease.[43] The 5A model (Assess, Advise, Agree, Assist, Arrange) is identified by the US Preventive Services Task Force as a unifying framework for counseling in primary care[44] and can be used for brief dietary counseling. A systematic review conducted by the US Preventive Services Task Force concluded that primary care counseling to promote a healthy diet results in small but positive benefits to dietary intake that may assist in the prevention of CVD.[45,46] However, effective individualized dietary counseling is time-consuming and therefore referral to a registered dietitian nutritionist (RDN) for medical nutrition therapy may be necessary. Improvements in lipids/lipoproteins are observed with 2 to 4 medical nutrition therapy sessions with an RDN.[47] A team-based healthcare approach, with involvement of an RDN, is recommended in the current guidelines for prevention of CVD and management of

dyslipidemia and hypertension.[12,13,40,48] The United States Department of Agriculture[49] and the Academy of Nutrition and Dietetics[50] provide online resources about planning a healthy diet during COVID-19, which may assist patients with healthful eating during this period.

Physical activity

Only 24% of US adults meet physical activity guidelines and 26% do not engage in any leisure-time physical activity.[9] Estimates suggest that 7% of CVD deaths are caused by low physical activity levels.[22] Even as stay-at-home orders are lifted, the ongoing COVID-19 pandemic is likely to negatively impact physical activity levels, particularly in older individuals, because of reduced capacity of (or fears of attending) structured exercise classes, gyms, and cardiac rehabilitation programs.

A higher level of physical activity is linked with improved lipids/lipoproteins, blood pressure, glycemic control, inflammation, platelet aggregation, fibrinolytic activity, autonomic and psychological measures, and endothelial function.[51] Physical activity also plays a role in body weight regulation and the maintenance of a healthy body weight.[52] Sedentary behaviors are associated with dysregulated appetite and energy intake, and achieving energy balance may be facilitated by greater physical activity.[53] Shook et al[53] reported that in a cohort of young adults, the threshold for achieving energy balance occurred at an activity level equivalent to 7116 steps/day.

Prospective cohort studies show that individuals with the highest levels of physical activity have an approximate 30–40% reduction in the relative risk of developing CVD, after adjustment for potential confounders.[54] In a prospective cohort analysis of 15 486 individuals with CHD from 39 countries, a graded decrease in mortality was observed with increased amounts of self-reported exercise. Each doubling of exercise volume was associated with a 10% reduction in total mortality (adjusted HR 0.90; 95% CI: 0.87 to 0.93).[55]

Physical activity should be assessed and promoted by clinicians.[56] Behavioral strategies such as goal-setting and coaching, in combination with pedometers and other wearable activity monitors that enable self-monitoring and provide direct user feedback, are recommended to increase physical activity.[57] Recent systematic reviews suggest physical activity apps with or without wearable connections modestly increase physical activity (in terms of daily steps, minutes of exercise or reducing sedentary behavior), particularly in the short-term (up to 3 months).[58,59] Clinicians can leverage the widespread availability of virtual physical activity classes and coaching to improve patient self-management related to physical activity.

Smoking cessation

Despite steady declines, 15% of US adults still smoke cigarettes.[60] Tobacco product use is higher in males (26%), adults with a General Education Development Certificate (41%), those with lower household income (< $35,000/year; 26%), individuals covered by Medicaid (28%), uninsured (30%), or those with serious psychological stress (37%).[60] Whether cigarette smoking has increased during the COVID-19 pandemic is not known, but greater daily stress is positively related to cigarette consumption in smokers.[61]

Basic science and translational studies show that substances in cigarette smoke have numerous pro-atherogenic effects, and that cigarette smoking is associated with endothelial dysfunction and occult atherosclerotic CVD (ASCVD).[62,63] Epidemiologic evidence shows that cigarette smoking is associated with a doubling of the relative risk of ASCVD events, which is multiplied in the presence of each additional risk factor.[63]

These findings are particularly relevant since thrombotic events, including Type 1 myocardial infarction, are part of the pathophysiology of COVID-19,[64] and cigarette smoking is associated with more serious COVID-19 outcomes.[6]

Cessation of smoking was associated with a 36% reduction in the relative risk of total mortality and a 32% reduction in the relative risk of myocardial infarction in individuals with CHD in a systematic review of 20 cohort studies.[65] Similar benefits of smoking cessation have been observed in older (>65 years) adults.[66] Smoking cessation significantly improves endothelial function,[67] which may in part explain the CVD benefits.

The 5A model is recommended for engaging patients in smoking cessation.[68-70] Motivational interviewing approaches increase the likelihood of smoking cessation by 26% compared to brief advice or usual care. Delivery of motivational interviewing by a primary care physician (RR 3.49, 95% CI 1.53, 7.94) or in a short session (<20 minutes; RR 1.69, 95% CI 1.34, 2.12) further increases the effectiveness.[71] Patients should be encouraged to attend a behavioral support program or utilize community-based support (e.g., telephone Quitline, smokefree.gov, becomeanex.org).

Social engagement and stress reduction

Approximately 31% of US adults experience an anxiety disorder in their lifetime.[72] The 2017 Stress in America survey, conducted on behalf of the American Psychological Association, stated that 75% of adults self-reported experiencing at least one symptom of stress in the previous month.[73] Those with CVD and lower socioeconomic status are disproportionately affected by anxiety and stress, which is linked to health disparities.[74] This burden is likely to be increased by the pandemic, now viewed as a form of mass traumatic stress[75] with the potential for large impacts on CVD risk.

Stress leads to an allostatic stress response that involves a number of pathways, including stimulation of stress hormones, inflammation, endothelial dysfunction, thrombosis, and vascular hyperreactivity.[76] Chronic stress results in long-term elevation of cortisol and other stress response hormones that trigger inflammatory responses in endothelial cells leading to a cascade of events resulting in vasculature injury, fibrosis, and stiffening, which increase blood pressure and the risk of thrombosis.[76] Both acute and chronic stress have been linked to increases in hypertension, arrhythmias, sudden cardiac death, angina, myocardial infarction,[77] and stress-induced cardiomyopathy.[78] Stress may also reduce adherence to medications and healthy lifestyle practices.[79] Despite the increased societal stress in response to the pandemic, the frequency of acute myocardial infarction and cardiac catheterization laboratory activations declined in the early phase of the pandemic,[80] for reasons that are unclear.

Interventions for stress management include screening via validated tools embedded in electronic health records, as well as positive psychology and guided mindfulness practices. These include biobehavioral therapies such as yoga, Tai Chi, and Transcendental Meditation, which have been associated with improvements in risk factors for CVD in small studies.[81] The Centers for Disease Control website outlines interventions for reducing psychological stress during the COVID-19 pandemic.[82]

Loneliness and poor social support are also prevalent risk factors for CVD. One-third of people in industrialized countries and 40% of US adults report loneliness,[83] although this number is likely to increase during the pandemic. A number of meta-analyses have shown that loneliness and social isolation are associated with increased risk of CVD and mortality.[85-87] A meta-analysis of 16 prospective cohort studies showed poor relationships were associated with a 29% and 32% relative risk increase in CHD and stroke, respectively.[85] A recent prospective analysis from the English Lon-

gitudinal Study of Ageing reported that per one-point increase in loneliness the risk of CVD-related hospital admission was increased by 8% (95% CI 1.03, 1.14) after adjustment for demographics and traditional CVD risk factors.[88] The highest level of loneliness was associated with a 48% higher risk of CVD-related hospitalization compared to the lowest level of loneliness.

Despite strong evidence linking social isolation and loneliness with increased risk of CVD, the underlying mechanisms are currently unclear, although as depicted by Holt-Lunstad and Smith there are a number of direct and indirect pathways by which social connections may influence CVD and these factors are likely interrelated.[89] For example, social connection (or lack thereof) affects psychological factors such as stress, depression, and resilience that directly affect CVD risk factors such as inflammation and blood pressure; however, social connection may also affect adherence to medications and lifestyle recommendations, further affecting these CVD risk factors. Thus, reducing loneliness is likely to reduce CVD risk. Quick and valid screening tools for loneliness and isolation exist in most electronic health records.[90] At present, evidence suggests that efforts to strengthen existing family relationships are more effective than clinical interventions delivered by hired personnel.[89]

Effects of lifestyle and other biologic factors on immune function

In addition to their impacts on CVD risk factors and outcomes, healthy diets, physical activity, smoking cessation, social engagement, and stress reduction affect measures of immune system function that may be clinically relevant during the COVID-19 pandemic.

Nutrition

Nutritional status is a key modulator of immune function.[91,92] Zabetakis et al[92] found moderate-quality evidence that systemic markers of immune function are influenced by diet. Undernutrition is linked to immunosuppression[91,92,93]; however, overnutrition resulting in overweight increases chronic inflammation, depresses normal immune function, and promotes autoreactivity,[93] which may be factors linking obesity to poorer COVID-19 outcomes.

Trace elements including iron, zinc, and selenium, and vitamins A, C, D, E, and B vitamins (B-6, B-12, and folic acid) also have immunomodulatory roles, and deficiency of these trace elements adversely effects immune function[94] (Table 1). Retrospective analyses have shown low vitamin D status is associated with greater COVID-19 burden.[95,96] An analysis of 190 000 patients from 50 countries showed that the severe acute respiratory disease coronavirus 2 (SARS-CoV-2) positivity rate was higher in patients with circulating 25-hydroxyvitamin D concentrations in the deficient range (< 20 ng/mL; 12.5%, 95% CI 12.2-12.8%) versus patients with adequate (30–34 ng/mL; 8.1%, 95% CI 7.8–8.4%) or high (≥ 55 ng/mL; 5.9%, 95% CI 5.5–6.4%) circulating 25-hydroxyvitamin D concentrations.[95] In addition, 25-hydroxyvitamin D concentrations were higher in patients from predominantly white non-Hispanic zip codes versus patients from predominately black non-Hispanic or Hispanic zip codes; in all of these racial/ethnic groups, a strong association between the SARS-CoV-2 positivity rate and 25-hydroxyvitamin D concentrations was observed. However, at present it is unclear whether this is a causal association; research examining the prognosis of patients with low 25-hydroxyvitamin D concentrations and the efficacy of vitamin D repletion on COVID-19 is warranted. In the meantime, obtaining vitamin D through safe sun exposure or intake of fortified foods or supplementation may assist with achieving adequacy.

Table 1: The effect of trace element and vitamin deficiency on immune function[94]

Nutritional deficiency	Immune system effects
Vitamin C	Disrupts cell signaling and epigenetic regulation
Iron	Impairs Th1 cellular immunity
Zinc	Impairs T-cell maturation and zinc-dependent redox reactions
Selenium	Impairs redox reactions (eg, glutathione peroxidase)
Vitamin A (as retinoic acid)	Impairs humoral Th2 cell responses and compromises intestinal immune function
Vitamin D	Disrupts differentiation of regulatory T-cells
Vitamin E	Inhibits production of naive T-cells
B vitamins	Impairs cytotoxic cellular immunity and disrupts T-cell responses

Notes: Th1, T helper type 1; Th2, T helper type 2

Other micronutrients (magnesium and copper) and marine-derived long-chain omega-3 fatty acids also impact immune function.[91] Specifically, omega-3 fatty acids have anti-inflammatory effects, magnesium plays a role in leukocyte activation, and copper enhances macrophage function and natural killer cell activity.[97] In addition, fiber may modulate immunity by reducing bacterial translocation across the gut barrier.[92]

To ensure nutritional needs are met, a high-quality diet appropriate for caloric needs should be consumed such as the Healthy US-Style, Healthy Mediterranean-Style, and Healthy Vegetarian diets, as well as the Dietary Approaches to Stop Hypertension diet.[39,41,42] If nutrient needs are not met through dietary sources, nutrient supplementation may be indicated; this should be done in consultation with a primary care physician.

Physical activity

There is a longstanding link between physical activity and immune function.[98] Moderate and vigorous aerobic exercise (a threshold of 60% of oxygen uptake and heart rate reserve for durations of < 60 minutes) are associated with increases in tissue macrophage activity, circulating immunoglobulins, anti-inflammatory cytokines, neutrophils, natural killer cells, cytotoxic T-cells, and immature B cells.[99,100] With repeated exercise, transient increases in selective lymphocyte subsets occur that enhance immune-surveillance and reduce inflammation[100]; these effects may be augmented in individuals with obesity.[101]

Regular moderate exercise was reported to reduce the duration and severity of upper respiratory infections (URI) by 40–50% in a meta-analysis of randomized controlled trials.[102] Similarly, a meta-analysis of epidemiologic studies showed a 28% weighted mean reduction in URI with high versus low physical activity.[102] However, paradoxically, prolonged high-intensity exercise is associated with worsened measures of immune function,[103] inflammation, and oxidative stress.[104] Therefore, regular moderate exercise can be recommended as an intervention for maintaining healthy immune function.

Smoking cessation

Cigarette smoking is associated with chronic immune changes in lung tissue, even in the absence of manifest lung disease. Induced sputum, expired breath condensate, bronchoalveolar lavage secretions, and lung tissue biopsy specimens from smokers show evidence of chronic inflammation.[105] In addition, neutrophil infiltration, phenotypic changes in macrophages, and unfavorable changes in the ratio of CD4/CD8 T-cells are observed. Neutrophil counts in sputum gradually decrease after smoking cessation.

Notably, smokers are twice as likely to contract viral illnesses, and to have more severe manifestations versus non-smokers.[106] Smokers had higher rates of death during the MERS-CoV outbreak.[107,108] Together, this evidence supports intensive smoking cessation efforts in the era of COVID-19.

Social engagement and stress reduction

Social isolation and loneliness, potential consequences of social distancing, are associated with physiological stress. Acute psychological stress enhances some pathways for adaptive immunity, while down-regulating others; however, chronic stress adversely affects immune function.[109] Loneliness is associated with increased blood levels of C-reactive protein, interleukins, and other pro-inflammatory molecules.[110] Biological links were demonstrated in a small but pioneering study showing that measures of loneliness were associated with genome-wide changes in transcription pathways that regulate glucocorticoid and other inflammatory responses.[111]

Social engagement has a role in symptom severity with viral infections. In a study of 276 healthy adults exposed to the rhinovirus, those with only 1 to 3 social interactions every 2 weeks were 4.2 times more likely to develop severe URI symptoms than those with 6 or more interactions.[112] In a similar study, following exposure to 5 types of respiratory viruses, a significant dose-response increase in rates of infection was observed with higher degrees of psychological stress, including loneliness.[113] These findings support counseling patients to maintain social engagement via phone and digital technologies. In addition, mindfulness-based stress reduction practices have been associated with reductions in levels of inflammatory cytokines.[114]

Racial and socioeconomic factors

Higher rates of COVID-19-related death among African Americans persist after adjusting for clinical factors and social disadvantage,[3,7] suggesting that other factors may confer risk. Differences in immune responses could contribute. African Americans versus those of European ancestry have been shown to manifest differences in transcriptional responses to bacterial challenges that translate into stronger inflammatory responses and faster bacterial clearance.[115] While conferring a selective advantage in the past, theoretically this difference could increase the severity of inflammation and cytokine storms in response to viruses with widespread targets in the body, like COVID-19. These biologic differences are amplified by stress, unequal access to health care, racial discrimination, and other social determinants of health.[74] In particular, racial discrimination is a stressor known to adversely affect the cardiovascular and immune systems, which has been comprehensively reviewed previously.[76,116] These differences are a strong impetus for improving lifestyle factors that impact immune and CVD health in African Americans, and for increasing equity in CVD health.[117]

Call to action

It is well-established that a healthy lifestyle characterized by a healthy dietary pattern, maintenance of a healthy body weight, regular physical activity, avoidance of cigarette smoking, and minimization of chronic stress assist in CVD prevention and management.[12,13] However, only 0.1% of US adults aged ≥ 20 years have ideal health behaviors defined as meeting AHA recommendations for physical activity, diet, BMI, and nonsmoking.[9] The onset of COVID-19 has further demonstrated the detrimental health effects of unhealthy lifestyle behaviors that increase CVD risk factor burden. Individuals with CVD or CVD risk factors have a greater likelihood of adverse outcomes from COVID-19, including death.[1] Herein we have described how improving modifiable

lifestyle-related behaviors lowers risk of CVD and improves immune function. Thus, lifestyle improvement may also reduce the severity of COVID-19 in affected individuals because of mitigation of CVD risk. However, at present, research examining effects of lifestyle behavior modification on COVID-19-related outcomes is not available. It is critical that research efforts are directed at examining the potential for healthy lifestyle-focused interventions to improve COVID-19 outcomes.

Conclusions

Existing CVD and CVD risk factors, as well as social disadvantage, are associated with increased severity and mortality from COVID-19. Clinical attention has focused on acute interventions for COVID-19; however, reducing upstream risks associated with poor outcomes must occur in parallel. This is particularly urgent because risk factors associated with greater COVID-19 severity are prevalent, and the pandemic has negatively impacted lifestyle and socioeconomic factors that directly affect these risk factors. Numerous evidence-based interventions for improving lifestyle factors exist, as well as frameworks for behavioral counseling in primary care settings (eg, the 5A model). Importantly, these interventions share strong commonalities and have a generally short time-to-benefit. Concordant evidence shows that a healthy dietary pattern appropriate for caloric needs is associated with lower risk of CVD and better immune function. Similarly, engagement in regular physical activity, avoidance of smoking, stress reduction, and social engagement positively impact immune function and CVD risk. If clinicians motivate only a small percentage of at-risk adults to improve these lifestyle factors, the population effects on cardiovascular risk and related healthcare costs will be substantial, and positive COVID-19-related outcomes may also be realized. Research examining how interventions aimed at improving lifestyle behaviors affect COVID-19 outcomes is urgently needed.

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