State-of-the-Art Review

Summarizing 2019 in Cardiovascular Prevention using the Johns Hopkins Ciccarone Center for the Prevention of Cardiovascular Disease's 'ABC's Approach

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ABSTRACT

In 2019, Preventive Cardiology welcomed many exciting discoveries that improve our ability to reduce the burden of cardiovascular disease (CVD) nationwide. Not only did 2019 further clarify how various environmental exposures and innate and acquired risk factors contribute to the development of CVD, but it also provided new guidelines and therapeutics to more effectively manage existing CVD. Cardiovascular disease prevention requires the prioritization of early and effective detection of CVD in order to implement aggressive lifestyle and pharmacologic therapies, which can slow, halt, or even reverse the progression of the disease. To help streamline and simplify the process of CVD prevention, The Johns Hopkins Ciccarone Center for the Prevention of Cardiovascular Disease has historically adopted an 'ABC' approach, which focuses on optimizing individual CVD risk through lifestyle, behavioral, and pharmacologic interventions. Given the practice changing research and innovation from the past year, this article intends to summarize the Ciccarone Center's key takeaways from CVD prevention in 2019 utilizing our expanded 'ABC' approach.

1. Introduction

A strong focus on the prevention of cardiovascular disease (CVD) has been critical in attempts to dethrone CVD as the leading cause of death nationwide. The year of 2019 was an exciting one for the field of Preventive Cardiology, with the release of the American College of Cardiology (ACC)/American Heart Association (AHA) Guideline for the Primary Prevention of CVD in March 2019 [1], and the release of the European Society of Cardiology (ESC)/European Atherosclerosis Society (EAS) and ESC/European Association for the Study of Diabetes (EASD) Guidelines in September 2019 ahead of print [2,3]. The year of 2019 brought many exciting advancements that furthered our understanding of how various exposures and risk factors contribute to the development of CVD, as well as many promising therapeutics.

Inspired by an informative review summarizing 2019 as the Year in Cardiovascular Prevention, from a European standpoint [4], we at The Johns Hopkins Ciccarone Center for the Prevention of Cardiovascular Disease have formulated our own summary. This review consolidates and highlights many of the key messages in preventive cardiology from 2019

using the Ciccarone Center's expanded 'ABC's approach (Fig. 1).

1.1. Assessment of atherosclerotic cardiovascular disease (ASCVD) risk

For patients with no established ASCVD, the first step in primary prevention is estimating ASCVD risk. In the United States, this is done using the ACC/AHA risk estimator, based on the Pooled Cohort Equations (PCE). This resource provides an estimated 10-year risk for ASCVD using individual demographics, risk factors and objective laboratory measurements.

With risk estimated, clinicians and patients can determine which lifestyle and pharmacologic strategies are recommended for ASCVD risk reduction. It is important to highlight known limitations of the current ASCVD risk prediction model, namely that it is often imprecise and is focused primarily on relatively short-term risk assessment [5]. Should a decision regarding pharmacologic strategy remain uncertain, shared decision making, as well as ancillary data, such as risk-enhancing factors or coronary artery calcium (CAC) scoring, can help clarify a path forward.

Echoing the recent 2019 ACC/AHA Primary Prevention of

Cardiovascular Disease Guideline, the European societies also encourage the inclusion of lifetime risk in order to implement preventive strategies earlier in ASCVD pathogenesis [1–3]. The ACC/AHA Guideline offered a lifetime risk score estimate for those aged 20–59 years using the PCE, while Ray et al. highlighted a Lifetime-perspective CardioVascular Disease (LIFE-CVD) model that attempts to estimate the treatment-effects of cholesterol, blood pressure, and antithrombotic therapy and smoking cessation in healthy individuals [6]. There are several online calculators, which can be accessed at http://tools.acc.org/ASCVD-Risk-Estimator-Plus/or www.U-Prevent.com, that should further inform shared decision making during the clinician-patient discussion surrounding 10-year and lifetime risk for ASCVD.

Risk estimates can be refined by utilizing risk-enhancing factors such as elevated triglycerides or inflammatory markers, metabolic syndrome, and pregnancy related history; if there is still uncertainty after examining risk enhancing factors, the appropriate test is a CAC score. A CAC score is the best "tie-breaker" when doubt remains about estimated ASCVD risk and the benefits of pharmacologic therapy such as statins. CAC has recently been incorporated into not only the 2019 ACC/AHA Prevention Guidelines but also the 2019 ESC/EAS and ESC/EASD Guidelines [1–3].

1.2. Aspirin

Aspirin therapy, which was previously considered a cornerstone for the primary prevention of ASCVD, is now recommended only in select adults 40–70 years of age who are at higher ASCVD risk but at low risk of bleeding. While the 2019 ACC/AHA Primary Prevention Guideline give a Class IIb recommendation for low dose daily aspirin, the ultimate decision is based on a clinician-patient risk discussion where additional risk factors such as current smoking, very high LDL-C levels with statin intolerance, 10-year ASCVD risk >20%, and CAC score >100 can be considered [1].

A recent study by Cainzos-Achirica et al. using data from the Multi-Ethnic Study of Atherosclerosis determined the role of CAC to personalize the allocation of aspirin for the primary prevention of ASCVD [7]. They determined that aspirin therapy was indicated based on a number needed to treat being lower than number needed to harm analysis in individuals with CAC ≥100 independent of their ASCVD risk strata.

There was also a clear signal for harm from aspirin therapy in those individuals with CAC = 0. This data suggests that a more personalized approach to aspirin therapy allocation is possible with a CAC score compared to the PCE.

1.3. Anti-inflammatory

While inflammation clearly plays an important role in the pathobiology of ASCVD, the role of anti-inflammatory pharmacotherapy for the primary prevention of ASCVD is uncertain. An elevated level of high-sensitivity C-Reactive Protein (hsCRP) was considered a risk modifier by both the United States and European prevention guidelines [1–3]. Suppressing inflammation in patients with high ASCVD risk was promising in the Canakinumab Anti-Inflammatory Thrombosis Outcomes Study (CANTOS) but was limited by relatively high rates of adverse effects [8]. To further evaluate the role of anti-inflammatory agents in ASCVD prevention, methotrexate and colchicine were studied in the Cardiovascular Inflammation Reduction Trial (CIRT) [9] and Colchicine Cardiovascular Outcomes Trial (COLCOT) [10], respectively, which were both published in 2019.

In CIRT, patients with previous myocardial infarction (MI) or multivessel coronary artery disease randomized to low dose methotrexate did not benefit from reduced ASCVD outcomes but did experience more adverse events [9]. In the COLCOT trial, patients with recent MI randomized to low dose colchicine experienced a 23% risk reduction in cardiovascular endpoints including revascularization at the expense of a <1% higher prevalence of diarrhea and pneumonia [10]. The search for an effective and safe anti-inflammatory pharmacologic agent for the primary prevention of ASCVD is still ongoing. However, the anti-inflammatory effects of a healthy lifestyle—such as regular physical activity, following a healthy diet, maintaining an optimal weight, and avoidance of tobacco products—cannot be emphasized enough.

1.4. Body weight

Achieving a healthy body weight (body mass index (BMI) >18 to ≤25, waist circumference <35 inches in women; <40 inches in men) by
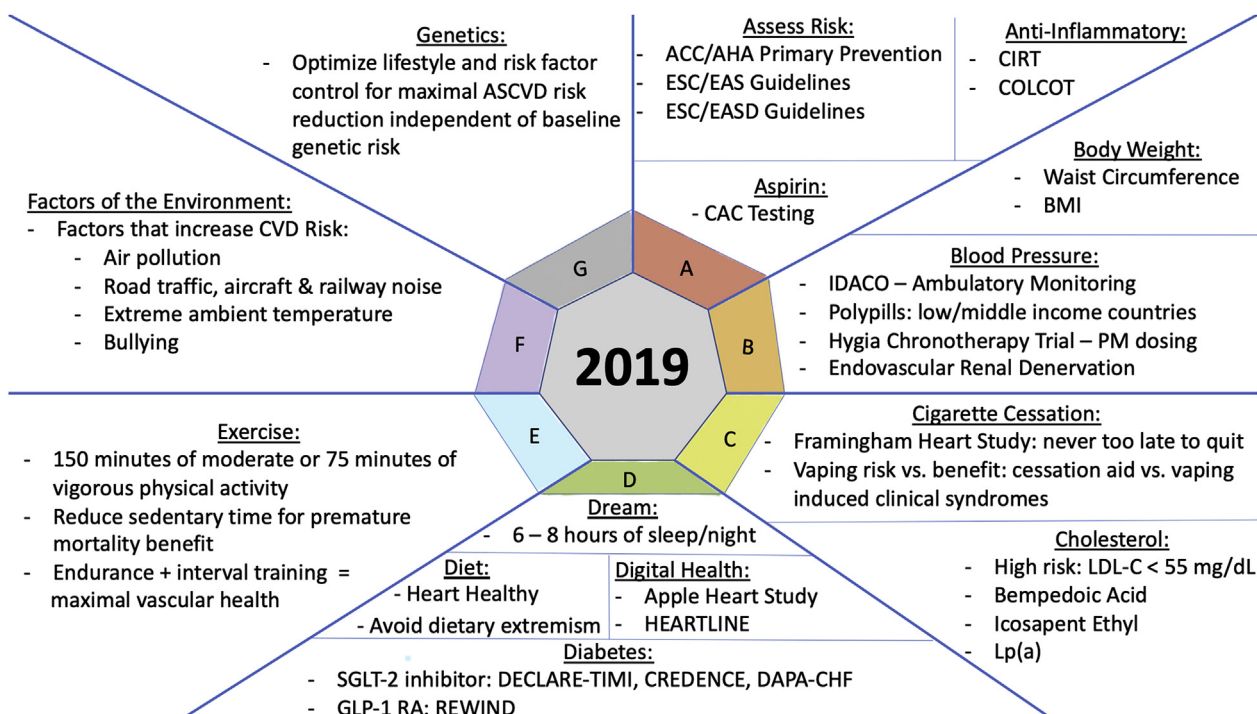

Fig. 1. Johns Hopkins Ciccarone Center for the Prevention of Cardiovascular Disease 'ABCDEFG' Approach to 2019 in Cardiovascular Prevention.

adhering to a heart healthy diet and daily physical activity can further optimize ASCVD risk factor profiles.

In 2019 in the *European Heart Journal,* Chen et al. further described the importance of body weight on ASCVD risk by focusing specifically on the distribution of fat [11]. Independent of body fat mass, higher truncal (abdominal/visceral) adiposity was associated with higher ASCVD risk compared to other patterns of fat distribution.

These data highlight the limits of using BMI, which only accounts for body weight and height. Given that anthropometric measurements are commonly incorporated into ASCVD risk factor profiles, this data and other data demonstrating the increased metabolic activity of truncal adiposity favor the routine use of waist circumference as a surrogate for truncal adiposity [12].

The first step for achieving weight loss in a patient with obesity should include counseling, caloric restriction, healthful nutrition and physical activity. When additional weight loss is required despite initiating this approach, clinicians should determine whether the patient meets the appropriate criteria to consider recommendations for pharmacologic therapies or bariatric surgery. In individuals with Class I obesity and adequate glycemic control, trialing FDA approved medications for the treatment of obesity including liraglutide, orlistat, phentermine/topiramate, or naltrexone/buproprion can be considered. When individuals have Class III obesity (BMI >40) or have Class II obesity (BMI >35) and at least one or more obesity-related co-morbidities, including diabetes mellitus, hypertension, or hyperlipidemia, bariatric surgery, such as gastric sleeve, Roux-en-Y, or biliopancreatic diversion with duodenal switch is an additional approach that can be considered after careful selection guided by a multidisciplinary team [13].

1.5. Blood pressure

Controlling blood pressure remains tantamount in reducing the burden of ASCVD. It is recommended that most patients maintain a blood pressure less than 130/80 mmHg [1]. The foundation of blood pressure management remains aggressive lifestyle modifications, as well as anti-hypertensive medications.

With the ubiquity of home blood pressure machines, when and how to treat hypertension is evolving. Based on data from the International Database of Ambulatory Blood Pressure in relation to Cardiovascular Outcome (IDACO) investigators in 2019, there appears to be a greater use of ambulatory blood pressure monitoring assessments based on data in a heterogeneous patient population [14]. This strategy helps limit the effect of 'white coat hypertension' and also allows treatment decisions to be based on 24 hour and nighttime systolic blood pressures, which are both associated with greater risk of death and CVD events.

The World Health Organization has recently pushed to incorporate multidrug combinations as the initial step in treatment for those individuals with high blood pressure, especially in low to middle income countries where this approach of polypills vs. usual care demonstrated cost-effectiveness [15]. In addition, based on data from the 2019 Hygia Chronotherapy trial, ≥ 1 anti-hypertensive medications should probably be administered at bedtime rather than the morning for better blood pressure control and lower sleep time blood pressure [16].

In addition to lifestyle modifications and pharmacotherapy, recent trials using endovascular renal denervation aim to use radiofrequency, ultrasound or alcohol chemical ablation to control high blood pressure through interruption of the renal sympathetic nervous system signaling. The Global SYMPLICITY Registry demonstrated sustained reductions in blood pressure over 3 years of follow-up after radiofrequency ablation without evidence of higher adverse safety outcomes or renal dysfunction [17]. Additional studies aim to evaluate additional modalities to achieve renal denervation in patients with resistant hypertension or tachycardia [18].

1.6. Cholesterol

Low-density lipoprotein cholesterol (LDL-C) is causally associated with ASCVD risk and lowering LDL-C improves ASCVD outcomes. In patients at very high ASCVD risk, the 2018 AHA/ACC Cholesterol Guideline recommends considering ezetimibe or a PCKS9 inhibitor if the patient has an LDL-C level greater than 70 mg/dL despite maximally tolerated statin therapy [19] and the 2019 ESC/EAS Dyslipidemia Guideline recommends achieving a goal LDL-C level of less than 55 mg/dL [2].

After optimizing lifestyle modifications, statins are first line therapy for LDL-C reduction in those with elevated ASCVD risk. Over the last few years, the use of PCSK9 monoclonal antibodies and ezetimibe has increased when statins are not tolerated or are insufficient in reducing LDL-C levels.

Favorable genetic variants that mimic ATP citrate lyase, an enzyme that works upstream of HMG-CoA reductase, demonstrated effective reduction of LDL-C levels and as a result CVD risk. Bempedoic acid, a novel therapy that inhibits ATP citrate lyase, recently gained FDA approval for use to lower LDL-C levels. Two studies published in 2019, the CLEAR HARMONY and CLEAR WISDOM trials, demonstrated that addition of bempedoic acid to maximally tolerated statin decreases LDL-C by ~15–17% and also reduced hsCRP [20,21]. Additional studies assessing how bempedoic acid affects ASCVD outcomes are ongoing. The greatest use of bempedoic acid will likely be when it is used in a combination pill with ezetimibe in order to achieve about a 35–40% LDL-C reduction.

The 2019 ACC/AHA Guideline for the Primary Prevention of CVD also highlights that persistently elevated triglycerides, elevated apolipoprotein B (apoB), and elevated lipoprotein(a) [Lp(a)] are risk-enhancers that if present should move an individual into a higher risk category that would favor more intensive preventive treatments [1].

Evidence from Mendelian randomization analyses show that the clinical benefit of lipid lowering is proportional to the absolute change in ApoB-containing lipoproteins [22]. In the Reduction of Cardiovascular Events with Icosapent Ethyl-Intervention Trial (REDUCE-IT), icosapent ethyl, which is a highly purified eicosapentaenoic acid ethyl ester, lowered triglycerides and resulted in a 25% reduction in the initial adverse cardiovascular event in patients with hypertriglyceridemia already on statin therapy [23]. In a follow-up paper published in 2019, the authors also demonstrated that icosapent ethyl reduced total ASCVD events (including recurrent events) by 30% [24]. Of note, none of the other omega-3 preparations, or triglyceride lowering therapies to date have shown similar benefits.

Based on prior studies estimates, this effect was greater than what would be expected by reduction in ApoB alone and an analysis of REDUCE-IT showed that the benefit of icosapent ethyl was independent of baseline or on-treatment triglyceride levels.

In a study of 65,000 participants with measured Lp(a), higher levels (>93 mg/dL vs. <10 mg/dL) were associated with a higher risk of CVD (50% increase) and all-cause mortality (20% increase). The authors believe that this excess risk of elevated Lp(a) is driven by corresponding low LPA KIV-2 number of repeats and not Lp(a) cholesterol content [25]. In a recent clinical trial published in early January 2020 in the *New England Journal of Medicine,* treatment with an antisense oligonucleotide therapy was able to reduce Lp(a) levels by up to 80% among patients with established CVD and Lp(a) $\geq$60 mg/dL [26]; a larger cardiovascular outcome study is currently underway.

1.7. Cessation of smoking

Cigarette smoking is one of the leading causes of preventable death nationwide. There are an abundance of data highlighting the beneficial health effects of smoking cessation and clinicians regularly counsel and provide resources to patients to aid in quitting. Once motivational interviewing for cigarette cessation is initiated, additional treatment

options should include nicotine replacement and pharmacotherapy such as varenicline or bupropion [1].

A recent study published in 2019 from the Framingham Heart Study over a 25-year period found that quitting smoking within 5 years was associated with a ~40% lower risk of incident CVD compared with current smokers [27], which highlights the fact that it is never too late to quit.

Unfortunately, while smoking rates are declining, the use of e-cigarettes is increasing [28]. The long-term effects of 'vaping' are unknown; however, clinicians have documented emerging clinical syndromes that are possibly the result of e-cigarette toxin exposure [29]. Continuing to educate all individuals, especially teenagers who have adopted 'vaping', of data like that from Duncan et al. [27] will be critical in controlling the long-term detrimental effects of smoking on ASCVD risk.

1.8. Diabetes

Diabetes is associated with both microvascular and macrovascular complications. In addition to our traditional approach to improve glycemic control, newer classes of medications provide opportunities for improved control of diabetes-related comorbidities including ASCVD, heart failure (HF) and chronic kidney disease (CKD).

Based on this growing body of literature, the American Diabetes Association recommends assessing an individual's risk for ASCVD, CKD and HF when stepping up therapy from lifestyle interventions, metformin or both. In patients with inadequately controlled diabetes despite maximum dose metformin and aggressive lifestyle modifications, if HF or CKD risk predominates, the next modality added should be an SGLT-2 inhibitor. This is based on data from the CANVAS, EMPAREG, DECLARE-TIMI, and CREDENCE trials, with the DECLARE-TIMI [30] and CREDENCE [31] trials reported in 2019.

DECLARE-TIMI randomized individuals with diabetes and established ASCVD or multiple ASCVD risk factors to dapagliflozin or a placebo. The minority of individuals had established ASCVD (~41%) and therefore this was largely a primary prevention trial. After ~4 years, individuals who received dapagliflozin had a 17% relative reduction in CVD mortality or hospitalizations for HF [30]. A 2019 meta-analysis of SGLT-2 inhibitors not only demonstrated a decrease in HF hospitalizations and CVD deaths in individuals with ASCVD, but also a slowing of the progression of kidney disease independent of CVD status [32].

While other SGLT-2 inhibitor trials showed renal benefit, CREDENCE was the first trial specifically designed to study whether SGLT-2 inhibitors preserve renal function in individuals with albuminuric CKD and diabetes. Individuals receiving canagliflozin had both 30% reductions in the primary renal outcome and 20% and 29% reductions in major adverse cardiovascular outcomes and HF hospitalizations, respectively [31].

Given the interplay of HF and these newer medications, investigators sought to understand their role in patients without diabetes. Most recently the DAPA-CHF trial aimed to assess the benefit of dapagliflozin on the composite outcome of CVD mortality or worsened HF in participants with a reduced ejection fraction, regardless of diabetes status [33].

Over 18 months, there was a 26% reduction in the composite endpoint with dapagliflozin, which was similar in the subgroup of those with or without diabetes. The EMPEROR-Preserved and DELIVER trial are scheduled to be completed over the next 1–2 years and will investigate the effect of empagliflozin and dapagliflozin, respectively, in patients with HF but a preserved ejection fraction on either CVD mortality or worsened HF events.

When ASCVD risk predominates it is recommended to initiate therapy with a GLP-1 receptor agonist such as liraglutide (LEADER trial) [34], semaglutide (SUSTAIN-6 trial) [35], albiglutide (HARMONY trial) [36], and dulaglutide (REWIND trial) [37], which have demonstrated CVD mortality benefits.

In the REWIND trial 9,901 participants who had a prior CVD event (~30%) or ASCVD risk factors were given either dulaglutide or placebo;

individuals randomized to dulaglutide had a 12% reduction in the composite outcome of CVD, non-fatal MI, or non-fatal stroke [37]. They also found a 15% reduction in the risk of having a composite adverse renal outcome. Given the low rates of prior CVD events in enrolled individuals, this was another largely primary prevention trial.

1.9. Diet

Currently, the ACC/AHA strongly recommends at risk individuals adhere to a heart healthy diet, which encourages intake of vegetables, fruits, legumes, nuts, whole grains, and fish to optimize CVD risk factor profiles [1]. Historically, nutritional studies are difficult to interpret as researchers are often unable to control all of the necessary factors required to conclude a cause-effect relationship [38]. For these reasons, guidelines tend to emphasize the foods with good nutritional value and low caloric density and recommend minimizing consumption of trans and saturated fats, foods with high levels of cholesterol, sodium, and refined carbohydrates, including sugar sweetened beverages [39].

In 2019, red meat and carbohydrates were again studied in an attempt to clarify their effect on CVD-specific and overall mortality, respectively. While observational data suggest a carcinogenic effect of red meat [40], possibly through its systemic effect on a microbiome-dependent metabolite (trimethylamine N-oxide, TMAO), systematic reviews concluded that there was a low predictive certainty on red meat consumption and CVD mortality [41].

Within the span of two years, data from both the Prospective Urban Rural Epidemiology (PURE) study and the National Health and Nutrition Examination Survey (NHANES) provided contradictory conclusions associating the amount of carbohydrate intake and CVD risk [42,43]. In an earlier 2019 issue of *European Heart Journal*, de Souza et al. practically summarize their assessment of this conflicting data by stating that any diet focused on one macronutrient may adversely impact health. Instead, we should emphasize nutritional variety and everything in moderation to avoid dietary extremism [44].

1.10. Digital health and devices

The widespread incorporation of mobile devices and wearables into clinical practice provides new opportunities for ASCVD detection, monitoring and management [45]. Digital health allows both patients to participate actively in their health and for clinicians to engage with patients via innovative virtual pathways (ex. video visits, remote monitoring, passive and active data sharing). Overall it broadens clinicians' reach with regards to prevention, diagnosis, and treatment.

In 2019, the Apple Heart Study recruited 419,297 individuals without self-reported atrial fibrillation to study the accuracy of a smartwatch-based screening tool to detect irregular pulse intervals. While only 2,161 individuals (0.52%) received a notification for an irregular pulse, the positive predictive value of an irregular pulse notification was 0.84 for this study population [46]. This was the first large-scale virtual study demonstrating atrial fibrillation detection with the Apple Watch, however given the majority of these patients were healthy and predominantly young participants (average participant was 41 years of age) there was an opportunity for further study of this technology among older and higher risk individuals.

To address this gap in knowledge the HEARTLINE study (in collaboration with Apple) intends to enroll 150,000 participants 65 years of age and older in whom the risk of developing atrial fibrillation is higher and may benefit from a health-focused engagement program. This program, which includes an iPhone application ("app") paired with the electrocardiogram sensor of the Apple Watch, will provide notifications when an irregular rhythm is identified. Researchers hope to identify those at-risk individuals that would benefit from continuous noninvasive monitoring to detect incident atrial fibrillation and modalities to support adherence to anticoagulation medication for stroke prevention [47]. Once identified, clinician-patient shared decision making can occur to

determine the appropriate steps for reducing the CVD risk associated with atrial fibrillation.

1.11. Dream (sleep)

Sleep deprivation is a well-known risk factor for CVD. In a 2019 *Nature* article, McAlpine et al. identified a neuro-immune axis that links sleep with atherosclerosis. Based on animal data, disturbances in this system are thought to explain the relationship between sleep deprivation and increased CVD risk [48]. In addition, based on data from the PURE study, which followed 100,000 participants for ~8 years, 6–8 h of nightly sleep is associated with the lowest risk of death and major CVD events independent of individual demographic characteristics, lifestyle behaviors or health status [49]. Given the limitations of observational studies, clinicians are unable to translate this data into definitive guidance for patients regarding sleep and CVD risk. However, this data can be used to promote healthy sleep patterns for possible CVD benefit.

1.12. Exercise

The 2019 ACC/AHA Prevention Guideline for physical activity recommends greater than 150 minutes of moderate intensity or 75 minutes of vigorous intensity physical activity each week [1]. Currently only a minority achieve this goal, while the others spend the majority of their day being sedentary. The ACC/AHA Guideline emphasizes that any physical activity, even if less than the recommended amount, should be encouraged to reduce CVD risk. Additionally, the ACC/AHA Guideline states that it is reasonable to also minimize overall sedentary behavior, in addition to achieving recommended physical activity levels [1].

Ekelund et al. performed a systemic review and meta-analysis published in 2019 studying the effect of dose-responses in physical activity and sedentary time and all-cause mortality. They found that engaging in any physical activity, independent of the intensity, reduces risk for premature mortality. In addition, minimizing sedentary time also has a positive effect on premature mortality risk [50].

The 2019 ACC/AHA Prevention and 2018 Physical Activity Guidelines recommend both endurance and resistance training, respectively, to obtain the maximum benefit of physical activity [1,51]. Werner et al. attempted to elucidate the role of endurance, interval and resistance training on telomere length/telomerase activity to further clarify the optimal combination of these various types of physical activity on CVD health. They concluded that endurance and interval training, but not resistance training, had positive effects on circulating blood cells cellular senescence and regenerative capacity, which are critical for maintaining vascular health [52].

1.13. Factors of the environment

Air pollution is a well-known risk factor for mortality, with the majority attributable to CVD causes [53]. However, a 2019 study highlights other environmental exposures that negatively affect CVD risk. Individual exposure to road traffic, aircraft and railway noise are each associated with excess mortality from MI independent of air pollution [54]. In addition, changes to the climate, which can introduce new extremes of temperature, has significant implications for countries like Kuwait where researchers have found an association between extreme ambient temperature and CVD mortality [55].

In another 2019 study, using large cohort studies in Sweden and Denmark, researchers documented harmful effects of bullying on CVD health. After adjustment, being a victim of bullying at work was associated with a 59% increased risk of ASCVD [56].

1.14. Genetics

Genetic factors have a significant impact on cardiovascular health and predict ASCVD outcomes. Longitudinal studies such as the UK Biobank shed light on the lifetime effects of genetic risk factors. In his pivotal 2019 review, Ference et al. demonstrated that a 1 mmol/L (39 mg/dL) and a 10 mmHg reduction in genetically determined LDL-C and systolic blood pressure, respectively, correlated with an 80% lower risk of CVD [57]. Studies like this have huge implications for future ASCVD prevention trials as these data suggest that earlier, more modest control of ASCVD risk factors have significant, lasting effects on ASCVD lifetime risk.

Each individual has a predetermined genetic vulnerability with variable adaptive or maladaptive ability. Over time, individuals will face exposures, which can be causal factors or enhancers of CVD. Based on the exposure magnitude and duration, each individual will accrue a certain cumulative burden of ASCVD risk. However, even in those with the highest genetic risk, the implementation a healthy lifestyle alone was associated with a 46% lower relative risk of coronary heart disease [58].

2. Conclusion

Optimizing preventive measures to preserve health is critical to delay any major adverse cardiovascular events. When these adverse events occur, the focus transitions to preservation of health through aggressive risk factor modifications in an attempt to improve the quality and duration of life.

Based on Johns Hopkins Ciccarone Center for the Prevention of Cardiovascular Disease 'ABC's approach to preventive cardiology, prioritization of both inherited and lifestyle factors is critical to optimize individual ASCVD risk. Early identification of increased ASCVD risk and subsequent implementation of aggressive lifestyle and pharmacologic therapies can help slow the progression of genetically determined ASCVD.

Adhering to this expanded 'ABC' approach provides clinicians and patients a framework for systematically optimizing an individual's cardiovascular risk with lifestyle, behavioral and pharmacologic interventions.

Declaration of competing interest

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