2021
Essential Messages from ESC Guidelines

Clinical Practice
Guidelines Committee

CVD Prevention

Guidelines on cardiovascular disease
prevention in clinical practice

ESC
European Society
of Cardiology

Essential Messages

2021 Essential Messages from the ESC Guidelines on cardiovascular disease prevention in clinical practice*

Developed by the Task Force for cardiovascular disease prevention in clinical practice
with representatives of the European Society of Cardiology and 12 medical societies and
with the special contribution of the European Association of Preventive Cardiology (EAPC)

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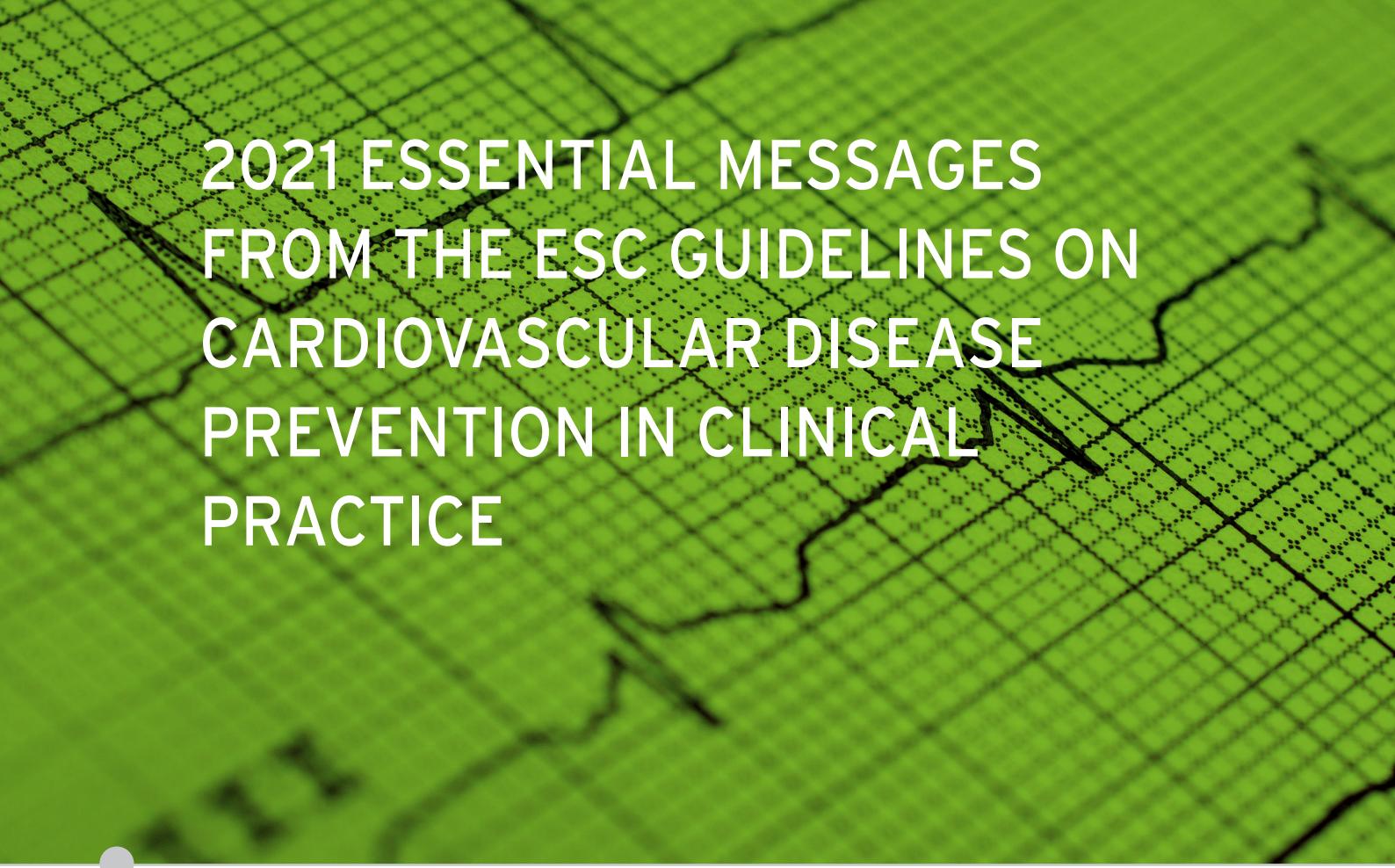
2021 ESSENTIAL MESSAGES FROM THE ESC GUIDELINES ON CARDIOVASCULAR DISEASE PREVENTION IN CLINICAL PRACTICE

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Key messages

Risk factors and risk classification

1. The major risk factors for ASCVD are cholesterol, BP, cigarette smoking, DM, and adiposity.

2. Risk factors are treated in a stepwise approach to reach the ultimate treatment goals in apparently healthy people, patients with established ASCVD, and patients with DM.

3. 10-year CVD risk is estimated in apparently healthy people aged 40–69 years with SCORE2, and in people aged > 70 years with SCORE2-OP.

4. Age-specific 10-year CVD risk thresholds—together with consideration of risk modifiers, frailty, comorbidities, lifetime CVD risk, treatment benefit, polypharmacy, and patient preferences—guide treatment decisions for lipid and BP treatment.

5. There are various options of communicating the (residual) CVD risk, and this should be tailored to the individual patient.

Risk modifiers

6. Psychosocial stress is associated with risk of ASCVD.

7. Current risk scores may under- or overestimate CVD risk in differing ethnic minority groups.

8. CAC scoring is the best-established imaging modality to improve CVD risk stratification.

9. Frailty is a functional risk factor of both CV and non-CV morbidity and mortality.

10. Frailty assessment is not a method to determine eligibility for any particular treatment, but rather serves to build an individualized care plan with predefined priorities.

11. Family history should be enquired about routinely, and a positive family history of premature ASCVD should be followed by comprehensive CVD risk assessment.

12. Current data does not support the use of genomic risk scores in CVD risk assessment in primary prevention.

13. ASCVD development and prognosis are linked to social gradients.

14. Air pollution is strongly associated with ASCVD.

15. Additional circulating and urine biomarkers should not be routinely measured.

16. Assess CVD risk in persons with obesity.

Clinical conditions

17. CKD is an independent risk factor for ASCVD, and ASCVD is the leading cause of death in CKD.

18. A short-term reduction in albuminuria by approximately 30% upon starting RAAS inhibition is associated with improved CV and kidney outcomes.

19. Similarly, SGLT2 inhibitors are associated with long-term benefits in CV and renal risks.

20. AF is associated with an increased risk of death and an increased risk of CVD.

21. Ischaemic HF constitutes the most advanced clinical manifestation of atherosclerosis within the myocardium.

22. The diagnosis of overt HF, as well as asymptomatic presentation with LV dysfunction, increases the risk of CVD events (myocardial infarction, ischaemic stroke, CV death).

23. There is an overlap between cancer and CV risk factors; CV risk in patients with cancer depends on both the CV toxicity of treatments and patient-related factors.

24. Signs or symptoms of cardiac dysfunction should be monitored before, periodically during, and after treatment.

25. Exercise should be strongly advised, in particular aerobic exercise, to prevent cardiotoxicity.

26. COPD is a major risk factor for CVD, especially ASCVD, stroke, and HF.

27. COPD patients are prone to arrhythmias (AF and ventricular tachycardia) and sudden cardiac death.

28. All COPD patients should be investigated for CVD.

29. Common COPD medications are usually safe in terms of CV adverse events.

30. Chronic inflammatory conditions increase CVD risk.

31. Infection with HIV is associated with an increased risk of LEAD and CAD.

32. There is an association between influenza and periodontitis infections and ASCVD.

33. Migraine, particularly migraine with aura, is an independent risk factor for stroke and ischaemic cardiac disease.

34. The risk of ischaemic stroke in subjects with migraine with aura is magnified by the use of combined hormonal contraceptives and cigarette smoking.

35. Non-restorative sleep and a sleep duration that varies significantly up or down from the optimum of 7 h are associated with increased CV risk.

36. Mental disorders are common in the general population (12-month prevalence of 27%) and are associated with excess mortality.

37. The onset of CVD increases the risk of mental disorders by 2.2-fold, leading to a worse prognosis.

38. Some mental disorders–even symptoms of anxiety and depression–are associated with the development of CVD and with a worse prognosis in those with existing CVD (CHD, arterial hypertension, AF, HF).

39. Excess mortality is mainly caused by behaviour-dependent risk factors (e.g. smoking addiction) and an impaired capacity for selfcare (e.g. treatment adherence).

40. NAFLD is associated with other cardiometabolic risk factors. Patients with NAFLD should be evaluated for other cardiometabolic risk factors.

41. Sex-specific conditions:

    i.   Preeclampsia and pregnancy-related hypertension are associated with a higher risk of CVD.
    ii. Polycystic ovary syndrome confers a significant risk for future development of DM.
    iii. ED is associated with future CV events and mortality in men.
    iv. CVD risk should be assessed in men with ED.
    v.   Asking about ED should be a standard procedure in routine CV risk assessment in men.

Risk factors and interventions at the individual level

42. Regular PA is a mainstay of ASCVD prevention.

43. Aerobic PA in combination with resistance exercise and the reduction of sedentary time are recommended for all adults.

44. A healthy diet lowers the risk of CVD and other chronic diseases.

45. A shift from a more animal- to plant-based food pattern may reduce CVD.

46. Achieving and maintaining a healthy weight through lifestyle changes has favourable effects on risk factors (BP, lipids, glucose metabolism) and lowers CVD risk.

47. When changes in diet and PA–as well as other conventional, non-invasive interventions– are unsuccessful, bariatric surgery should be considered for high-risk individuals.

48. Anti-obesity medications with protective ASCVD effects may also be considered.

49. Patients with mental disorders have sharply increased lifestyle risks that need recognition and treatment.

50. Mental healthcare improves stress symptoms and quality of life, reduces the risk of suicide, and may improve CV outcomes.

51. The treatment of ASCVD patients with mental disorders requires interdisciplinary cooperation and communication.

52. Stopping smoking rapidly reduces CVD risk and is the most cost-effective strategy for ASCVD prevention.

53. There is strong evidence for medication-assisted interventions: NRT, bupropion, varenicline, and drugs in combination. The most effective are assistance using drug therapy and follow-up support.

54. Lower is better: the effect of LDL-C on the risk of CVD appears to be determined by both the baseline level and the total duration of exposure to LDL-C.

55. Lowering LDL-C with statins, ezetimibe, and—if needed and cost-effective—PCSK9 inhibitors, decreases the risk of ASCVD proportionally to the absolute achieved reduction in LDL-C.

56. When LDL-C goals according to level of risk cannot be attained, aim to reduce LDL-C by > 50% and then strive to reduce other risk factors as part of a shared decision-making process with the patient.

57. When hypertension is suspected, the diagnosis should be confirmed by repeated office BP measurement at different visits, or ABPM or HBPM.

58. Lifestyle interventions are indicated for all patients with hypertension and can delay the need for drug treatment or complement the BP-lowering effect of drug treatment.

59. BP-lowering drug treatment is recommended in many adults when office BP is > 140/90 mmHg and in all adults when BP is > 160/100 mmHg.

60. BP treatment goals are lower than in the previous ESC CVD prevention guidelines for all patient groups, including independent older patients.

61. Wider use of single-pill combination therapy is recommended to reduce poor adherence to BP treatment.

62. A simple drug treatment algorithm should be used to treat most patients, based on combinations of a reninangiotensin system (RAS) blocker with a CCB or thiazide/thiazide-like diuretic, or all three. Beta-blockers may also be used where there is a guideline-directed indication.

63. Many patients with hypertension will be at sufficient risk to benefit from statin therapy for primary prevention. Antiplatelet therapy is indicated for secondary prevention.

64. A multifactorial approach, including lifestyle changes, is critical in persons with type 2 DM.

65. Management of hyperglycaemia reduces the risk of microvascular complications and, to a lesser extent, the risk of CVD. Glycaemic targets should be relaxed in older adults and frail individuals.

66. New antihyperglycaemic drugs are particularly important for persons with type 2 DM with existing ASCVD and (heightened risk of) HF or renal disease, broadly irrespective of glycaemia levels.

Type 1 diabetes mellitus

67. Intensive management of hyperglycaemia in DM reduces the risk of micro- and macrovascular complications and premature mortality; a target of 6.5-7.5% (48-58 mmol/mol) HbA1c is recommended.

68. Metformin is not recommended in type 1 DM to lower CVD risk.

69. Dapagliflozin has been recommended for use in type 1 DM, although there is an increased risk of diabetic ketoacidosis with such therapies.

70. Targeting other risk factors, in particular smoking, BP, and cholesterol levels, remains an important means to lower CVD risk in type 1 DM.

71. All patients with established ASCVD require some form of antithrombotic therapy.

72. Anti-inflammatory therapy is a promising strategy in CVD prevention.

73. Patients after ACS and/or coronary artery bypass graft/PCI, or with chronic HFrEF, should participate as early as possible in structured, multidisciplinary EBCR and prevention programmes.

74. EBCR and prevention programmes must comply with certain quality standards and be individualized to each patient's profile.

75. Participation and long-term adherence to these programmes has to be encouraged and enhanced. Telerehabilitation and mHealth may help towards achieving this target.

Population-level approaches to cardiovascular disease | Physical activity

76. A significant percentage of the worldwide population, in particular the European population, shows high levels of sedentary behaviour and physical inactivity.

77. The percentage of those exercising at a regular level is greater in men than in women.

78. Global progress to increase PA has been slow, largely due to lack of awareness and investment.

79. The optimal dose of different types of PA for CVD and general prevention is still controversial and subjected to frequent updates. Increasing moderate-to-vigorous PA and reducing sitting time, however, is beneficial and any level of PA is considered better than none.

80. PA for health promotion should be implemented by physicians in the same way as drug prescription and should also be promoted by other healthcare professionals.

81. Population-based interventions are effective in promoting PA for groups based on age, sex, and race, for high-, middle-, and low-income populations, and for different environments (e.g. kindergarten, school, gyms, companies, and worksites in general).

82. Daily PA at school should be practised for at least 3 h/week, and preferably for 60minutes per day.

83. Population-based approaches are complementary to individualcentred interventions.

84. Diet.

85. Structural measures such as changes in agricultural supply chain and food industry, product reformulation, limitations on (digital) marketing to children, taxes on unhealthy foods/nutrients, and consumer-friendly nutrition labelling will improve healthy food choices.

86. Healthy environments in the community, on public transport, at schools, and in workplaces will stimulate a healthier lifestyle.

87. The WHO Global Action Plan for the Prevention and Control of Non-Communicable Diseases 2013–2020 extended to 2025 recommends to develop goals in global, regional, and national agendas. Within the 10 voluntary targets to reach in 2025 is a 30% relative reduction in mean population intake of sodium/salt.

Population-level approaches to cardiovascular disease | Smoking and tobacco use

88. Adolescence is the most vulnerable period for the uptake of smoking, with lifelong consequences.

89. Previous prevention campaigns reduced tobacco use in girls much less than in boys.

90. Teenagers should be informed that smoking is not helpful in weight control.

91. High taxes on all tobacco products is the most effective policy measure to reduce smoking uptake by the young.

92. There should be restrictions on smokeless tobacco due to strong evidence of harm.

93. Also, restrictions on e-cigarettes due to evidence of harm.

94. Plain packaging is effective in reducing the attractiveness of tobacco products.

95. There should be restrictions on advertising, promotion, and sponsorship by the tobacco industry.

96. A goal would be to make a common European decision to achieve a smoking-free Europe by 2030.

Population-level approaches to cardiovascular disease | Alcohol

97. Alcohol intake is associated with increased CV mortality, and alcohol use is the leading risk factor for premature death and disability among people aged 15–49 years.

98. The interventions for addressing the harmful use of alcohol are cost-effective, with a good return (i.e. increasing alcoholic beverage minimum unit pricing and excise taxes, restricting access to alcoholic beverages, and implementing comprehensive restrictions and bans on advertising and the promotion of alcoholic beverages).

99. Healthcare providers may inquire about alcohol intake in every medical evaluation and should inform patients that alcohol is energy-dense: it provides 7 kcal/g and no nutrients.

Population-level approaches to cardiovascular disease | Environment, air pollution, and climate change

100. Air pollution contributes to mortality and morbidity, and specifically increases the risk of respiratory and CV diseases.

101. Environmental exposure has taken on new urgency, as air pollution, in addition to its health effects, has also been ascribed as a major contributor to climate changes, notably through the burning of fossil fuels leading to increasing emissions of carbon dioxide.

Key messages

Risk management of disease-specific cardiovascular disease | Coronary artery disease

102. Multidimensional prevention is crucial for short- and long-term outcomes in CAD.

Risk management of disease-specific cardiovascular disease | Heart failure

103. Patients with HF benefit from multidisciplinary care management programmes.

104. Several neurohormonal antagonists, as well as novel molecules, improve clinical outcomes in symptomatic patients with HFrEF.

Risk management of disease-specific cardiovascular disease | Cerebrovascular diseases

105. Ischaemic events are mainly caused by atherothrombosis, cardioembolism, or small vessel disease, whereas intracerebral haemorrhage is mostly caused by hypertensive angiopathy or cerebral amyloid angiopathy.

106. Platelet inhibitors are recommended for non-cardioembolic events and anticoagulants for cardioembolic events.

107. In patients with a previous stroke or TIA and high BP, BP lowering reduces the recurrence risk.

108. In patients with stroke or TIA, statins prevent CVD and cerebrovascular events.

109. Lower extremity artery disease

110. LEAD is associated with an increased CVD risk.

111. Antiplatelet therapy (alone or in combination with low-dose oral anticoagulation) reduces the risk of adverse limb events and overall CVD risk in patients with LEAD.

112. Smoking cessation and control of other CVD risk factors improve prognosis.

Risk management of disease-specific cardiovascular disease | Chronic kidney disease

113. Hypertension, dyslipidaemia, and DM are prevalent among individuals with CKD and require a high-risk treatment strategy approach.

114. Risk management includes lifestyle, smoking cessation, nutrition, sufficient RAAS blockade, target BP control, lipid management, and−in established CVD−aspirin.

115. A high value is placed on self-management education programmes and team-based integrated care in patients with DM, CKD, and CVD.

Risk management of disease-specific cardiovascular disease | Atrial fibrillation

116. Holistic management of patients with AF improves prognosis and reduces health-related costs.

117. Comprehensive risk-factor modification and targeting underlying conditions reduce AF burden and recurrence.

Risk management of disease-specific cardiovascular disease | Multimorbidity

118. The number of patients with multiple CV and non-CV comorbidities is rapidly increasing.

119. Therapeutic competition should be considered in multimorbid patients, as the treatment of one condition might worsen a coexisting condition.

120. A paradigm shift from disease-focused to patient-centred care for multimorbid CVD patients is recommended..

Main gaps in evidence and areas for future research

CVD risk classification

1. Country-specific risk algorithms for patients with established CVD and people with DM.

2. Formal comparison of effectiveness and cost-effectiveness of CVD risk-guided treatment vs. treatment guided by risk factor level.

3. Comparison of the precision of competing risk-adjusted CVD risk models vs. standard CVD risk models.

4. Incorporating potential risk markers into conventional risk models, such as socioeconomic status and ethnicity.

5. Comparison of treatment benefit-guided strategy vs. risk-guided strategy in reducing risk factor levels and CVD risk.

6. Management of CVD risk in older people (> 85 years) with marked fragility, for whomno data currently exist.

7. Comparison of different methods for the estimation of lifetime CVD risk and lifetime benefit of risk factor treatment.

Risk modifiers | Psychosocial factors

8. More evidence that psychosocial factors improve risk prediction beyond the classical risk-factormodels.

Risk modifiers | Ethnicity

9. Whether recalibration of factors for ethnicity are homogeneous in various European countries.

10. Risks associated with other ethnic backgrounds.

Risk modifiers | Frailty

11. Consensus on a clinically orientated screening tool for frailty to be applied across the spectrum of ASCVD.

12. Quantitative contribution of frailty to the global CVD risk prediction scheme.

13. At which degree of frailty treatment of specific risk factors should be less aggressive.

Risk modifiers | Family history

14. Disentangle the role and (genetic, socioeconomic, etc.) mechanisms of family history on CVD risk.

Gaps in evidence

Risk modifiers | Family history

15. Disentangle the role and (genetic, socioeconomic, etc.) mechanisms of family history on CVD risk.

Risk modifiers | Genetics

16. The potential of polygenic risk scores to complement existing risk scores.

Risk modifiers | Socioeconomic determinants

17. More evidence from different risk regions that the inclusion of socioeconomic factors improves risk prediction beyond classical risk factor models in bothmen and women.

Risk modifiers | Environmental exposure

18. Whether air pollution reclassifies risk in individual patients.

Risk modifiers | Biomarkers

19. Added value of biomarkers in risk classification.

Clinical Conditions | Chronic kidney disease

20. Identification of a good biomarker, besides albuminuria, and perhaps the use of CAC score to subclassify CV risk in CKD.

21. Early and precise identification of progressive CKD with novel biomarkers that are more sensitive than eGFR and albuminuria.

Clinical Conditions | Atrial fibrillation

22. Evaluate the effect of interventions aimed at reducing outcomes beyond stroke.

23. Is AF a causal factor for increased CVD morbidity and mortality?

24. Stroke risk prediction for low-risk AF patients.

25. Emerging evidence suggests that stroke can occur in patients with AF even after sinus rhythm is restored.

Clinical Conditions | Heart failure

26. It remains unknown whether patients with HFrEF of ischaemic origin should have different target LDL-C levels than those recommended for secondary prevention in individuals without HF.

Clinical Conditions | Cancer

27. RCTs using preventive therapy to demonstrate a clear effect on prevention of CV events.

Gaps in evidence

Clinical Conditions | Chronic obstructive pulmonary disease

28. Although common pathophysiological pathways between CVD and COPD are probable, they remain to be clarified.

Clinical Conditions | Inflammatory conditions

29. The optimal way of integrating information on chronic inflammatory conditions into CVD risk assessment.

30. The effect of modern anti-inflammatory drugs on CV risk [e.g. anti-tumour necrosis factor (TNF), interleukin (IL)-1, IL-17, IL-23 biologics].

Clinical Conditions | Infections

31. Large-scale studies to assess the efficacy of influenza vaccination or periodontitis treatment in preventing CVD.

32. The association of infection with HIV and total CVD risk.

Clinical Conditions | Migraine

33. There are no data that allow reliable identification of subgroups of migraineurs at particular high risk (e.g. active migraine, high-frequency auras, young subjects, women).

34. The role of comorbid factors (e.g. patent foramen ovale, thrombophilic factors) is unclear, and at the moment there is no indication to screen or to manage for these factors.

Clinical Conditions | Sleep disorders

35. There is lack of evidence that the inclusion of sleep improves risk prediction.

36. Trials are needed that target the complex pathways linking sleep disturbances with CVD.

Clinical Conditions | Mental disorders

37. The precise mechanism by which mental disorders increase CVD remains uncertain.

38. How the consideration of mental disorders improves CV risk models.

Clinical Conditions | Non-alcoholic fatty liver disease

39. Whether NAFLD increases CV risk beyond traditional risk factors.

Clinical Conditions | Sex-specific conditions

40. The degree to which increased CVD risk associated with several of the female-specific conditions occurs independently of conventional CVD risk factors, although data in women are still underpowered compared to men.

41. Information on whether female-specific conditions improve risk classification.

42. There are insufficient data to draw conclusions on a possible increased risk of hypertension or DM with premature menopause. Studies on the specificities of CVD disease in the transgender population are scarce.

Clinical Conditions | Erectile dysfunction

43. The benefit of routine screening for ED and the most effective tool to assess it are still unclear.

44. The benefit of assessment of subclinical vascular disease in men with ED and low-to-intermediate CVD risk is unclear.

Risk factors and interventions at the individual level | Physical activity and exercise

45. Knowledge of the relative importance of the various characteristics of aerobic PA and resistance exercise, or their combination, on all-causemortality, CV incidence, and mortality.

46. Understanding how sex, age, weight, race/ethnicity, occupation, and socioeconomic status may modify associations between PA and health outcomes.

47. Implementation of strategies to achieve long-term adherence to PA.

48. Evaluation of the effects of eHealth tools in promoting PA.

Risk factors and interventions at the individual level | Nutrition

49. Effective strategies to encourage people to change their diet and to enjoy and maintain a healthy diet.

Risk factors and interventions at the individual level | Body weight

50. Knowledge and implementation of effective lifestyle and medication-assisted strategies to achieve weight loss and maintain a long-term healthy weight.

Risk factors and interventions at the individual level | Mental healthcare and psychosocial interventions

51. The effectiveness of mental healthcare for the prevention of major CVD events.

52. How to implement effective CVD prevention measures in this high-risk population of patients withmental disorders.

Gaps in evidence

Risk factors and interventions at the individual level | Smoking intervention

53. A better understanding of how to incorporate effective smoking cessation into clinical practice.

Risk factors and interventions at the individual level | Lipids

54. Direct empirical evidence for the stepwise approach to treatment intensification from RCTs. The feasibility and effects of reaching LDL-C levels < 1.4 mmol/L (55 mg/dL) needs further investigation, especially in primary care.

55. Particularly among people at low-to-moderate CVD risk, older people, and for newer interventions, more evidence of the effects of lipid-modifying treatments on overall mortality is needed in the formof long-term post-trial follow-up in RCTs.

56. The cost-effectiveness of using lifetime CVD risk and more precise CVD risk scores to target interventions needs further investigation.

57. The value of triglycerides or HDL-C values as a target for therapy.

58. Whether lipoprotein(a) lowering against background statin, ezetimibe and PCSK9i therapy can reduce the risk of ASCVD.

59. Whether functional foods and food supplements with a lipidlowering effect can safely reduce the risk of CVD.

Risk factors and interventions at the individual level | Blood pressure

60. What is the incremental benefit, over CVD risk calculators, of measures of HMOD in reclassifying the CV risk of patients with hypertension?

61. Direct empirical evidence for the stepwise approach to treatment intensification from RCTs.

62. What are the benefits of BP treatment for patients with BP in the high-normal range?

63. More data on the benefits of BP treatment in very old people and the influence of frailty.

64. Effect of single-pill vs. multidrug treatment strategies on adherence to treatment, BP control, and clinical outcomes.

65. Effectiveness of antihypertensive treatment in preventing cognitive dysfunction or dementia.

66. Efficacy and cost-effectiveness of invasive procedures and devices for the treatment of hypertension.

67. Sex-specific BP treatment thresholds for men and women.

Gaps in evidence

Risk factors and interventions at the individual level | Diabetes mellitus

68. More work is needed to develop risk scores for both MACE and HF in type 2 DM.

69. Whether combined SGLT2 inhibitor and GLP-1RA treatments lower MACE or other outcomes beyond either drug alone requires testing.

70. Longer-term safety of newer classes of drug is required.

Risk factors and interventions at the individual level | Antithrombotic therapy

71. The role of antithrombotic therapy in primary prevention in (very) high-risk individuals remains to be established.

Risk factors and interventions at the individual level | Cardiac rehabilitation and prevention programmes

72. The effect and the optimal delivery of EBCR in women, older/frail patients, patients with cardiac implantable electronic devices, after heart transplantation or valve replacement, and in patients with AF, stroke, HFpEF, LEAD, or multiple comorbidities.

73. Alternative and cost-effective models of CR need to ensure participation globally, including low- and middle-income countries.

74. Large RCTs investigating the long-term effects of home-based telerehabilitation and mHealth are needed.

Risk factors and interventions at the individual level | Environment, air pollution, and climate change

75. Individual-level exposure studies are needed to better specify the effect of mitigating measures.

Risk factors and interventions at the individual level | Risk management of disease-specific cardiovascular disease - *Coronary artery disease*

76. The efficacy and safety of aspirin or other antithrombotic therapy in patients without clinical manifestations of CAD–but with atherosclerotic disease identified on imaging, such as CCTA–requires further assessment.

77. The optimal long-term antithrombotic therapy in patients at high risk of ischaemic events is uncertain.

78. Clinical studies comparing the efficacy and safety of P2Y12 inhibitors vs. low-dose rivaroxaban or other factor Xa inhibitors, in combination with aspirin, are warranted to determine which subgroups will derive greater clinical benefit with each strategy.

Risk factors and interventions at the individual level | Risk management of disease-specific cardiovascular disease - *Heart failure*

79. For patients with HFpEF, no specific pharmacotherapy or device implantation has been shown to modify the risk of any CV outcome.

80. Lower dosage of HF treatments in women with HFrEF needs to be addressed, since women were underrepresented in many HF trials.

Risk factors and interventions at the individual level | Risk management of disease-specific cardiovascular disease - *Cerebrovascular disease*

81. The optimal selection of patient for a short course of DAPT.

82. The optimal antihypertensive regimen and target BP.

83. The optimal target level of LDL-C.

84. Optimal treatment for patients with silent cerebrovascular disease.

Risk factors and interventions at the individual level | Risk management of disease-specific cardiovascular disease - *Lower extremity artery disease*

85. The optimal type and potency of antithrombotic therapy in patients with different manifestations of symptomatic or asymptomatic LEAD are partly unclear.

Risk factors and interventions at the individual level | Risk management of disease-specific cardiovascular disease - *Chronic kidney disease*

86. Few CVD trials have a focus on patients with CKD, particularly those with advanced CKD.

87. Additional prospective studies focusing on diagnosis, prevention, and treatment of CAD and CVD are needed in CKD.

Risk factors and interventions at the individual level | Risk management of disease-specific cardiovascular disease - *Atrial fibrillation*
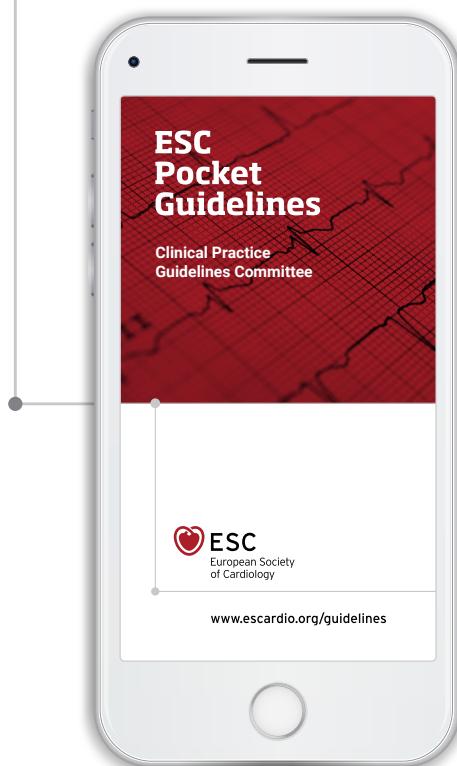
88. The effects of various CV risk factors and comorbidities in AF.

89. Optimal treatment of OSA and its effect on AF progression and symptoms.

Risk factors and interventions at the individual level | Risk management of disease-specific cardiovascular disease - *Multimorbidity*

90. The effect of different clusters or combinations of CV and non-CV comorbidities on CV outcomes.

91. Optimal, pragmatic treatment strategies in patients with CV and non-CV comorbidities, with particular focus on treatment adherence and therapeutic competition.
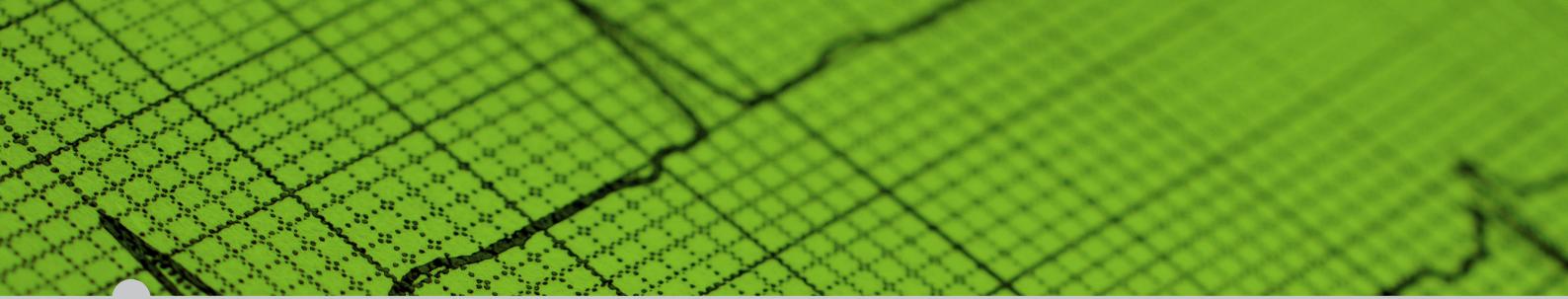
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