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# Lifestyle and prognosis after percutaneous coronary intervention: from revascularization to long-term management

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Percutaneous coronary intervention, secondary prevention, residual cardiovascular risk, lifestyle intervention, cardiac rehabilitation

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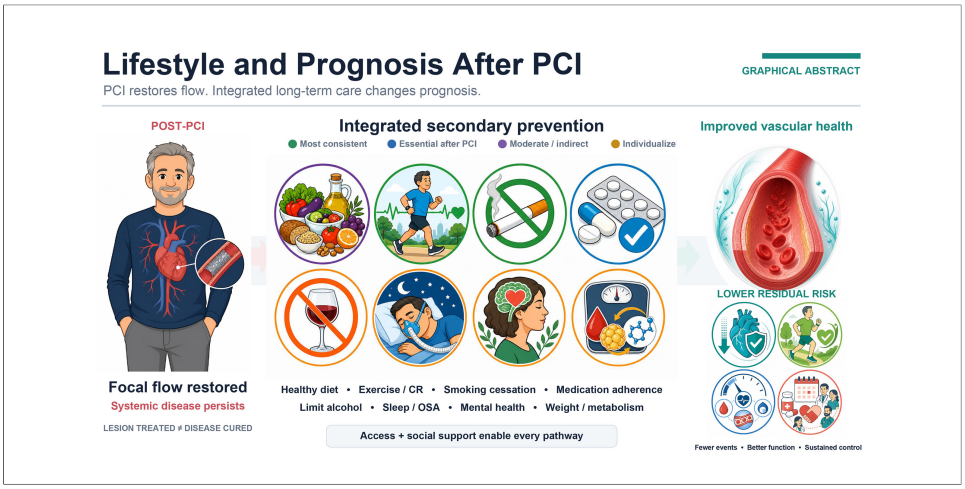


## Abstract

Percutaneous coronary intervention (PCI) restores coronary blood flow but does not stop systemic atherosclerosis or remove the risks associated with untreated lesions, residual ischemia, medication non-adherence, and adverse health behaviors. This narrative review examines how diet, exercise, smoking, alcohol, sleep, psychological health, body weight, social conditions, and medication adherence affect prognosis after PCI. We distinguish randomized from observational evidence and indicate whether findings come directly from PCI populations or are extrapolated from broader coronary disease. The most consistent evidence supports exercise-based cardiac rehabilitation and smoking cessation. A Mediterranean diet has randomized support in secondary prevention, but direct evidence after PCI is limited to a small, short-term biomarker trial. Evidence for alcohol, sleep, psychological interventions, weight management, and digital care is less certain and is often observational or indirect. Lifestyle care should be combined with appropriate lipid-lowering, antithrombotic, anti-inflammatory, and cardiometabolic treatment. We provide a

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practical follow-up framework from discharge onward and identify research gaps in intervention timing, comparative dietary strategies, cost-effectiveness, and delivery in settings with limited resources.

## INTRODUCTION

Over the past three decades, the global burden of ischemic heart disease (IHD), particularly coronary artery disease, has remained substantial despite major advances in prevention and treatment[1-3]. In 2021, approximately 254.3 million people were living with IHD worldwide; IHD accounted for 8.99 million deaths and 188.4 million disability-adjusted life-years[1-3]. Against this background, percutaneous coronary intervention (PCI) has expanded rapidly and continued to evolve technically[4]. PCI can restore epicardial blood flow without open surgery and is widely used for acute coronary syndromes (ACS) and selected chronic coronary syndromes[5-7]. More than 600,000 PCI procedures are performed annually in the United States, and procedural activity also remains high across Europe and Asia[8,9].

Despite these advances, patients who undergo PCI remain exposed to substantial residual cardiovascular risk. Contemporary trials and registries continue to report clinically meaningful rates of major adverse cardiovascular events despite new-generation drug-eluting stents and secondary-prevention treatment[10,11]. This persistent risk reflects progression of untreated plaques, incomplete revascularization, residual ischemia, microvascular dysfunction, inflammation, and lipid abnormalities[12,13]. Rehospitalization, repeat revascularization, prolonged pharmacotherapy, and long-term follow-up also impose a substantial economic burden[14-16]. Reducing recurrent events after PCI therefore remains an important clinical priority.

Lipid-lowering and antiplatelet therapy are essential after PCI, but they do not eliminate recurrent risk. Many patients remain vulnerable because medication adherence is suboptimal and atherosclerosis is a systemic disease. In a contemporary US cohort of adults with prior myocardial infarction, only 36.5% received the full combination of guideline-recommended secondary-prevention medicines for which they were eligible[17]. Population-level data after PCI also show incomplete achievement of guideline-recommended lipid levels[18]. Residual inflammatory and triglyceride risk may also persist despite well-controlled low-density lipoprotein cholesterol[19]. Lifestyle factors directly shape this remaining risk through their effects on inflammation, thrombogenicity, endothelial function, metabolic health, and long-term adherence. Accordingly, guidelines from the European Society of Cardiology (ESC) and the American College of Cardiology/American Heart Association (ACC/AHA) place lifestyle modification and cardiac rehabilitation at the center of secondary prevention[20-22]. However, those documents address broad chronic coronary disease populations and provide limited space for the post-PCI transition.

This review therefore addresses a focused question: which lifestyle recommendations are supported by direct post-PCI evidence, which rely on extrapolation from broader coronary populations? We examine diet, exercise-based cardiac rehabilitation, smoking, alcohol, medication adherence, sleep, psychological health, body weight and metabolic status, and social conditions.

## METHODS AND APPROACH TO EVIDENCE APPRAISAL

We searched PubMed/MEDLINE and the reference lists of major cardiovascular guidelines and systematic reviews for English-language publications from January 2015 through July 2026. Search terms covered PCI, coronary revascularization, secondary prevention, residual risk, diet, cardiac rehabilitation, physical activity, smoking, alcohol, sleep, depression, obesity, metabolic syndrome, medication adherence, digital health, and social determinants. Earlier landmark studies were included when they remained important to current practice.

We gave priority to PCI-specific randomized trials, prospective cohorts, registries, and meta-analyses. When direct evidence was unavailable, we used studies of myocardial infarction or established coronary disease and stated that the evidence was indirect. Because this is a narrative review, we did not perform formal study selection, risk-of-bias scoring, or meta-analysis. The interpretive summaries in the table and figure are descriptive rather than formal Grading of Recommendations Assessment, Development and Evaluation (GRADE) assessment. They consider study design, risk of bias, consistency, directness to PCI, precision, effect size, and dose-response.

## DIET AFTER PCI

Diet is a key determinant of long-term prognosis after PCI, as it shapes the persistent metabolic and inflammatory milieu following revascularization. Dietary patterns are more clinically relevant than isolated nutrients because foods are consumed in combinations that jointly influence low-density lipoprotein cholesterol (LDL-C), triglycerides, blood pressure, glycemia, body weight, inflammation, and satiety[23]. In CORonary Diet Intervention With Olive Oil and Cardiovascular PREVention (CORDIOPREV), 1,002 patients with established coronary heart disease were randomized to a Mediterranean or low-fat diet and followed for a median of 7 years; the Mediterranean strategy reduced the primary cardiovascular outcome [adjusted hazard ratio (HR), 0.72; 95% confidence interval (CI), 0.54-0.96][24]. This is the strongest head-to-head dietary evidence in secondary prevention, but it was a single-center, open-label study; 82.5% of participants were men, and the cohort was not restricted to recent PCI. The more direct trial enrolled only 120 patients recently treated with coronary stents and followed them for 3 months. Mediterranean and low-fat diets both improved fatty-acid profiles, with larger increases in n-3 fatty acids under the Mediterranean diet, but no clinical outcome was evaluated[25]. Thus, PCI-specific support is biologically coherent but based on a small surrogate-endpoint trial rather than demonstrated reductions in stent thrombosis or recurrent events.

Evidence for the Dietary Approaches to Stop Hypertension (DASH) diet, healthful plant-forward, Portfolio, and lower-carbohydrate patterns is more indirect. DASH lowers blood pressure in feeding trials, and healthful plant-based or Portfolio scores are associated with lower coronary risk, but the relevant studies largely involve general or primary-prevention populations[26-28]. Lower-carbohydrate studies use inconsistent thresholds, frequently lack information on replacement nutrients, and show that outcomes differ when carbohydrates are replaced by unsaturated plant fats rather than saturated fat or animal foods[29,30]. No adequately powered trial has compared Mediterranean, DASH, plant-forward, and high-quality lower-carbohydrate strategies after PCI. Accordingly, the Mediterranean pattern has the best overall secondary-prevention support, while selection among other patterns should be driven by hypertension, diabetes, hypertriglyceridemia, renal function, cultural acceptability, affordability, and food access rather than claims of equivalent post-PCI outcome benefit. Across patterns, reducing ultra-processed foods is a reasonable common target, although supporting outcome data remain observational and susceptible to residual socioeconomic and healthy-user confounding[31,32].

## PHYSICAL ACTIVITY AND EXERCISE-BASED REHABILITATION AFTER PCI

Exercise-based cardiac rehabilitation (CR) has the broadest randomized evidence among the interventions reviewed. A meta-analysis of 85 randomized trials involving 23,430 patients with myocardial infarction, angina, coronary artery bypass grafting (CABG), or PCI and a median follow-up of 12 months found reductions in cardiovascular mortality [risk ratio (RR), 0.74; 95%CI, 0.64-0.86], myocardial infarction (RR, 0.82; 95%CI, 0.70-0.96), and hospitalization (RR, 0.77; 95%CI, 0.67-0.89), but not all-cause mortality (RR, 0.96; 95%CI, 0.89-1.04) or repeat PCI (RR, 0.84; 95%CI, 0.69-1.02)[33]. The pooled population was clinically heterogeneous; many trials predated contemporary PCI and pharmacotherapy, follow-up was usually short,

and low-risk men in high-income settings were overrepresented. PCI-specific cohort studies generally favor CR, but referral, socioeconomic, functional, and healthy-adherer selection can exaggerate outcome differences. Evidence is therefore more consistent for CR in coronary disease overall than for PCI-specific hard outcomes.

Aerobic activity remains the foundation of CR, with progressive resistance training added in clinically stable patients[20,34]. Standard goals of at least 150 min of moderate aerobic activity weekly are useful long-term targets, but they should not be applied as a fixed immediate post-procedure prescription. Early planning should account for ACS versus elective PCI, radial versus femoral access, access-site complications, left ventricular function, residual or staged lesions, ischemic symptoms, frailty, and previous activity. Cardiopulmonary exercise testing is particularly valuable when symptoms, ventricular dysfunction, or residual ischemia make intensity uncertain[34,35]. Persistent angina during rehabilitation should prompt clinical reassessment rather than automatic exercise withdrawal or escalation.

Center-based and home-based CR show broadly similar effects on exercise capacity and health-related quality of life in selected stable patients, but comparative trials are not powered to establish equivalent effects on mortality or stent-related events, and digitally enabled programs may exclude people with limited connectivity or literacy[20,36,37]. Consequently, home or hybrid delivery should be presented as an access-enhancing alternative with risk-based supervision, not as proof that all delivery models are interchangeable. The important implementation target is automatic referral before discharge followed by documented initiation and completion, because an unacted referral does not deliver the trial-level benefit.

## SMOKING AND SMOKING CESSATION AFTER PCI

Smoking is consistently associated with recurrent ischemic events and stent thrombosis after PCI, although evidence for clinical outcomes is observational because smoking status cannot be randomized[38-43]. Smokers in PCI cohorts are often younger and have fewer recorded comorbidities, which can create a misleading "smoker's paradox" in incompletely adjusted analyses. Self-reported smoking, relapse, differences in treatment, and healthy-adherer bias further limit causal interpretation.

The biological link is well established: smoking promotes platelet activation and heightened thrombogenicity after PCI[38]. Smokers in PCI cohorts are often younger and have fewer recorded comorbidities, which can produce an apparent "smoker's paradox" in incompletely adjusted analyses[39]. A Korean PCI cohort found a dose-response association between pack-years and major adverse cardiac and cerebrovascular events (MACCE)[42]. Because exposure and later smoking behavior were measured observationally, residual confounding remains possible. The findings support early cessation but do not justify a fixed threshold beyond which vascular injury is considered irreversible.

Smoking cessation is associated with lower long-term risk after PCI. In the Korean cohort, people who quit after smoking < 20 pack-years had risks close to persistent non-smokers, whereas heavier former smokers retained greater residual risk[42]. Another contemporary stenting cohort linked sustained cessation with lower 10-year all-cause and cardiovascular mortality[43]. These studies support quitting as early as possible, but the size of benefit should not be read as a randomized treatment effect because relapse and healthy-adherer bias cannot be excluded.

Effective cessation usually requires repeated behavioral support and, when appropriate, medication. In the 302-patient Evaluation of Varenicline (Champix) in Smoking Cessation for Patients Post-Acute Coronary Syndrome (EVITA) trial, varenicline increased 24-week abstinence from 32.5% to 47.3%, although the trial was not restricted to PCI and was not powered for cardiovascular events[44]. In a cohort of more than 27,000

hospitalized patients with coronary disease, early nicotine replacement therapy was not associated with higher short-term mortality or a longer hospital stay[45]. Mobile interventions may help with abstinence and adherence, but they have generally not been tested for their effects on post-PCI clinical events[46,47]. Treatment should begin during hospitalization when possible and should include early follow-up for relapse[48,49].

## ALCOHOL CONSUMPTION AND CARDIOVASCULAR OUTCOMES AFTER PCI

Alcohol is the area in which observational and other evidence conflict most clearly. In a Korean insurance cohort of 77,409 PCI recipients followed for 4.0 years, both within-guideline and above-guideline drinkers had lower recorded MACCE risks than non-drinkers (adjusted HRs, 0.84 and 0.83)[50]. However, intake was self-reported during a health examination within 1 year after PCI. Patients who were too ill or disadvantaged to attend were less likely to be included, and the non-drinker group may have included former drinkers who stopped because of illness.

The apparent J-shaped association should not be interpreted as proof that alcohol is protective. People who continue to drink may differ from abstainers in health, diet, income, and treatment adherence. Sick-quitter bias, healthy-user bias, changes in intake, reverse causation, and residual confounding are difficult to remove. Mendelian-randomization studies found no protective threshold for blood pressure or coronary disease[51,52], and a meta-analysis designed to reduce former-drinker and study-quality bias found no significant mortality benefit with low-volume intake[53]. No randomized trial has assigned alcohol consumption after PCI.

Patients who do not drink should not be advised to start for cardiovascular protection. Those who drink should avoid binge drinking and should consider reduction or abstinence, especially when they have atrial fibrillation, hypertension, cardiomyopathy, liver disease, problematic use, or important bleeding risk[54-58]. Current evidence does not identify a dose that improves prognosis after PCI. Alcohol use should be reviewed again during follow-up because intake may change after discharge.

## MEDICATION ADHERENCE AND GUIDELINEDIRECTED MEDICAL THERAPY AFTER PCI

The long-term benefit of PCI also depends on adherence to indicated guideline-directed medical therapy (GDMT), particularly antiplatelet and lipid-lowering treatment and, when clinically indicated, renin-angiotensin-system inhibitors, beta-blockers, and other cardioprotective medicines[20,59]. Medication adherence is not a lifestyle factor in the usual sense, but it is a modifiable behavior with immediate importance after stent implantation.

Adherence declines over time, even in clinical trials. In contemporary revascularization trials, use of an antiplatelet agent, beta-blocker, and statin fell from 67% at 1 year to 53% at 5 years. Fewer than 40% remained on all three drugs plus an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker[60].

Common barriers include limited health literacy, the belief that PCI has cured the disease, polypharmacy, complex dosing, adverse effects, medication cost, limited insurance coverage, fragmented follow-up, and poor communication[59,60]. These barriers often cluster with social disadvantage. As a result, observational links between adherence and outcomes may partly reflect healthy-adherer bias or differences in contraindications.

In the functionally complete revascularization cohort, adherence to GDMT declined from 61.2% at 1 month to 35.3% at 3 years. After propensity-score matching, sustained GDMT was associated with lower risks of 3-year MACCE (adjusted HR, 0.52; 95%CI, 0.36-0.75) and ischemia-driven revascularization (adjusted HR, 0.14; 95%CI, 0.07-0.28)[61]. Because treatment was not randomized and adherence changed over time, these

results cannot be interpreted as purely causal. Even so, uninterrupted antiplatelet therapy during stent healing has a clear procedure-specific role. Medication access, understanding of missed doses, and a plan for bleeding should therefore be confirmed before discharge.

The appropriate combination and duration of medication should be reviewed over time. Recent evidence questions routine long-term beta-blocker therapy after myocardial infarction when left ventricular ejection fraction is preserved and there is no other indication; benefit is clearer when systolic function is reduced or mildly reduced[59,62]. Adherence should therefore mean taking medicines that remain indicated, not continuing every discharge medicine indefinitely without review.

Practical measures include medication reconciliation, plain-language counseling, simpler regimens, review of affordability, pharmacist support, nurse-led follow-up, and digital reminders suited to the patient's access and literacy[59,63]. Evidence for individual implementation strategies is heterogeneous, and nurse-led post-PCI studies remain few[63]. Programs should measure access, persistence, bleeding, adverse effects, and clinical outcomes rather than app use alone.

Medication adherence should be managed together with lifestyle care. The immediate priorities are uninterrupted antiplatelet treatment and reliable access to prescribed medicines. Later visits should address adverse effects, regimen complexity, cost, and whether each medicine remains indicated.

## OTHER LIFESTYLE DETERMINANTS OF OUTCOME

### Sleep duration, sleep quality and circadian disruption

Evidence on sleep after PCI is mainly prognostic rather than interventional. Sleep health includes duration, quality, regularity, timing, and sleep disorders[64,65]. Poor sleep may increase sympathetic activity, inflammation, insulin resistance, obesity, endothelial dysfunction, and blood-pressure variability. These mechanisms are plausible, but they do not show that improving sleep prevents post-PCI events.

Obstructive sleep apnea (OSA) is particularly relevant because intermittent hypoxia and oxidative stress may increase vascular and thrombotic risk. A meta-analysis of 9 PCI cohorts involving 2,755 participants found that OSA was associated with major adverse cardiovascular events (MACE) (RR, 1.96; 95%CI, 1.36-2.81; $I^2$ = 54%)[66]. The studies used different definitions and outcomes, and obesity and other comorbidities may have confounded the association. The review did not show that OSA treatment reduces cardiovascular events after PCI.

Sleep assessment should focus on symptoms and established clinical indications. Screening is reasonable for patients with suggestive symptoms, obesity, resistant hypertension, or atrial fibrillation, followed by diagnostic testing and standard treatment. Randomized post-PCI trials are still needed to determine whether sleep interventions reduce clinical events.

### Psychological health and social support

Depression is associated with poorer medication adherence, lower participation in CR, and worse outcomes after PCI. Meta-analyses of PCI cohorts reported higher risks of MACE (approximately RR, 2.10) and all-cause mortality (approximately RR, 1.76)[67]. However, studies differed in screening tools, timing, follow-up, and adjustment, and part of the association may reflect disease severity or social disadvantage.

Randomized evidence for reducing cardiovascular events is limited and is not specific to PCI. In a single-center trial of 300 patients with depression after ACS, 24 weeks of escitalopram was associated with fewer long-term MACE events than placebo (40.9% *vs.* 53.6%; HR, 0.69) over a median of 8.1 years[68]. This

supports appropriate treatment of depression but does not show that every psychological intervention prevents cardiovascular events.

Validated screening can identify patients who may benefit from psychotherapy, collaborative care, social support, or medication chosen for psychiatric need and cardiovascular safety. Future PCI-specific trials should report mental health, CR participation, and cardiovascular outcomes separately.

### Body weight and metabolic health

Body weight and metabolic syndrome are related to glucose control, blood pressure, lipids, inflammation, and physical function, but post-PCI outcome evidence is mainly observational[69,70]. Among patients with ACS undergoing PCI, metabolic syndrome was associated with a 22% higher risk of MACCE and a 43% higher risk of recurrent myocardial infarction, although definitions and adjustment methods varied[69].

The reported "obesity paradox" after PCI may be explained partly by reverse causation, smoking, frailty, illness-related weight loss, treatment selection, and the inability of body mass index to separate fat from lean mass[70]. Management should focus on excess adiposity, fitness, diet quality, and metabolic risk while avoiding unintended weight loss and loss of muscle.

The Semaglutide Effects on Cardiovascular Outcomes in People with Overweight or Obesity (SELECT) trial provides broader secondary-prevention evidence. Semaglutide reduced MACE in patients with obesity and established cardiovascular disease without diabetes (HR, 0.80; 95%CI, 0.72-0.90)[71]. However, SELECT was not limited to post-PCI patients. Cost, long-term continuation, gastrointestinal adverse effects, and integration with lifestyle care all affect its use in practice.

### Social and structural determinants of sustainable lifestyle change

Lifestyle change after PCI is strongly influenced by the conditions in which patients live. Low income, limited health literacy, medication costs, work and caregiving demands, transport, distance, language, cultural preferences, digital access, and the availability of CR can all affect participation. In a 2026 English cohort of 421,281 patients referred to CR after myocardial infarction, PCI, or CABG, 62.7% had undergone PCI. Only 53% started CR, and 77% of those who started completed it. Compared with the most affluent areas, residence in the most deprived areas was associated with higher odds of non-completion among White European patients [odds ratio (OR), 1.63; 95%CI, 1.57-1.68] and South Asian patients (OR, 1.41; 95%CI, 1.27-1.57). South Asian participants were more likely to start but less likely to complete CR than White European participants (adjusted OR for completion, 0.78; 95%CI, 0.75-0.82)[72].

Social needs should be assessed before discharge and reviewed again during follow-up, rather than being labeled as poor motivation. Practical responses include affordable and simpler treatment, automatic CR referral, flexible scheduling, transport or social-work support, culturally and linguistically appropriate education, and home-based or hybrid CR when attendance is difficult. Telephone and non-digital options should remain available so that digital care does not widen existing inequalities[22,72-74].

A comparative appraisal of the representative evidence, its applicability to post-PCI care, and its limitations is presented in Table 1[75,76].

## INTEGRATED SECONDARY PREVENTION AFTER PCI

Residual risk after PCI has several components. Lipid-related risk includes LDL-C, lipoprotein(a), and plaque progression. Inflammatory risk may persist despite lipid control. Thrombotic risk depends on the stent, lesion, and patient. Ischemic risk includes incomplete revascularization, untreated epicardial disease,

**Table 1. Comparative appraisal of evidence across lifestyle, adherence, and access domains after PCI**

| Domain | Representative evidence | Key findings | Applicability and limitations for post-PCI care | Interpretation and practical message |
|---|---|---|---|---|
| Diet | CORDIOPREV RCT: 1,002 patients with stable coronary disease, median 7 years[24]; recent-stenting RCT: 120 patients, 3 months[25] | Mediterranean vs low-fat diet: adjusted HR 0.72 (95%CI, 0.54-0.96)[24]; the stenting trial assessed fatty-acid biomarkers, not clinical events[25] | CORDIOPREV was not limited to PCI; the PCI trial was small, short, open-label, and based on surrogate outcomes | The Mediterranean pattern has the strongest secondary-prevention support, but direct post-PCI outcome evidence remains limited |
| Exercise/CR | Meta-analysis of 85 RCTs involving 23,430 patients with mixed coronary disease, median 12 months[33] | CV mortality RR 0.74; MI RR 0.82; hospitalization RR 0.77; no significant reduction in all-cause mortality or repeat PCI[33] | Mixed diagnoses and treatment eras; short follow-up; low-risk men and high-income settings overrepresented | Evidence is most consistent for coronary disease overall; direct PCI-specific evidence for clinical outcomes remains less complete |
| Smoking cessation | PCI cohort and long-term stenting studies of smoking exposure and cessation[42,43]; EVITA RCT in 302 patients with ACS[44] | Cessation was associated with lower long-term risk[42,43]; varenicline increased 24-week abstinence from 32.5% to 47.3%[44] | Clinical outcomes were observational; smoking and relapse were often self-reported; EVITA was not PCI-specific or powered for events | Begin cessation support early and combine repeated follow-up with medication when appropriate; outcome effect estimates remain largely observational |
| Alcohol | PCI cohort: 77,409 patients, 4.0-year follow-up[50]; Mendelian-randomization studies[51,52]; mortality meta-analysis[53] | The PCI cohort suggested a J-shaped association (adjusted HRs 0.84 and 0.83 *vs.* non-drinkers)[50]; genetic and bias-adjusted analyses did not confirm protection[51-53] | No post-PCI RCT; self-report, sick-quitter bias, healthy-user bias, selection, reverse causation, and residual confounding | Evidence is uncertain; do not recommend starting alcohol for protection, avoid binge drinking, and individualize reduction or abstinence |
| Medication adherence | Revascularization trial adherence analysis[60]; post-revascularization cohort[61]; guideline and implementation sources[59,63] | After propensity-score matching, sustained GDMT was associated with lower 3-year MACCE (adjusted HR, 0.52; 95%CI, 0.36-0.75)[61] | Adherence was not randomized and changed over time; healthy-adherer and contraindication bias remain possible | Confirm access and understanding, protect early antiplatelet adherence, and review indications over time; outcome estimates are observational |
| Sleep/OSA | Meta-analysis of 9 PCI cohorts involving 2,755 patients[66]; broader sleep-health statements and reviews[64,65] | OSA was associated with MACE: RR 1.96 (95%CI, 1.36-2.81; $I^2$ = 54%)[66]; benefit of treatment after PCI is unproven | Observational prognosis; different definitions and outcomes; confounding by obesity and comorbidity | Screen and treat for established clinical indications, while explaining that event reduction after PCI has not been demonstrated |
| Mental health | Meta-analyses of PCI cohorts[67,75]; RCT in 300 patients with depression after ACS, median 8.1 years[68] | Depression was associated with MACE RR approximately 2.10 and mortality RR approximately 1.76[67]; escitalopram HR 0.69[68] | Screening and follow-up varied; observational associations may be confounded; the RCT was single-center and not PCI-specific | Identify and treat depression for clinical need; evidence that psychological treatment reduces post-PCI events remains indirect |
| Weight/metabolism | Post-PCI metabolic-syndrome cohort[69]; obesity-paradox appraisal[70]; SELECT RCT in broader cardiovascular disease[71] | Metabolic syndrome was associated with 22% higher MACCE and 43% higher recurrent MI risk[69]; semaglutide HR 0.80 in SELECT[71] | No post-PCI lifestyle weight-loss outcome trial; SELECT was not PCI-specific; BMI-based obesity paradox is prone to bias | Focus on adiposity, fitness, diet quality, and metabolic risk rather than BMI alone; direct post-PCI intervention evidence remains limited |
| Access/social factors | National CR cohort of 421,281 referred patients[72]; global CR implementation evidence[73,76]; social-determinant review[74] | Only 53% started CR and 77% of starters completed it; completion differed by deprivation and ethnicity[72] | Observational implementation evidence; health systems, culture, and available services differ across settings | Assess barriers and provide flexible, affordable, culturally appropriate, and non-digital options; implementation evidence is observational |

PCI: Percutaneous coronary intervention; HR: hazard ratio; CI: confidence interval; RR: risk ratio; ACS: acute coronary syndromes; OSA: obstructive sleep apnea; CR: cardiac rehabilitation; CORDIOPREV: CORonary diet intervention with olive oil and cardiovascular PREVention; MI: myocardial infarction; EVITA: evaluation of Varenicline (Champix) in smoking cessation for patients post-acute coronary syndrome; RCT: randomized controlled trial; GDMT: guideline-directed medical therapy; MACCE: major adverse cardiac and cerebrovascular events; MACE: major adverse cardiovascular events; SELECT: semaglutide effects on cardiovascular outcomes in people with overweight or obesity; BMI: body mass index.

vasomotor abnormalities, and coronary microvascular dysfunction. Cardiometabolic risk includes obesity, diabetes, hypertension, and kidney disease[12,13,77-81]. Lifestyle care can improve several of these areas, but it cannot replace targeted medication or investigation of recurrent symptoms.

Modern secondary prevention combines lifestyle and adherence support with treatment directed at the patient's remaining risks. In the Further Cardiovascular Outcomes Research With PCSK9 Inhibition in Subjects With Elevated Risk (FOURIER) trial, adding evolocumab to statin therapy reduced cardiovascular events (HR, 0.85; 95%CI, 0.79-0.92)[82]. Low-dose colchicine reduced events after myocardial infarction in the Colchicine Cardiovascular Outcomes Trial (COLCOT) and in chronic coronary disease in LoDoCo2 (HRs, 0.77 and 0.69)[83,84]. Semaglutide reduced MACE in SELECT (HR, 0.80)[71]. These trials support treatment of lipid, inflammatory, and cardiometabolic risk, but none tested a complete post-PCI lifestyle program.

Antithrombotic treatment is especially important after PCI. Dual antiplatelet therapy (DAPT) intensity and duration should balance ischemic and bleeding risks, particularly after complex PCI. Depending on the patient, options may include prolonged DAPT, P2Y12-inhibitor monotherapy, or dual-pathway inhibition[59,79,85]. Lifestyle counseling should reinforce adherence and bleeding surveillance but should not replace an individualized antithrombotic plan.

Delivery also matters. Automatic CR referral, early follow-up, multidisciplinary care, and home-based or hybrid programs may improve access. However, CR capacity remains limited in many countries, and digital programs may widen inequalities unless telephone and other low-technology options are retained[72-74,76,86]. The proposed phase-specific lifestyle and secondary-prevention framework is summarized in Table 2[87]. The relationships among residual-risk pathways, evidence across domains, and the phased management approach are summarized in Figure 1.

## PERSPECTIVES

Future studies should test a clear post-PCI care pathway rather than another general education program. One practical design would start before discharge with confirmation of DAPT access and smoking treatment, provide follow-up at 7-14 days, begin CR and dietary support within 2-4 weeks, review cardiometabolic targets at 3 months, and reassess symptoms, bleeding, relapse, and social barriers through 12 months. Studies should distinguish ACS from elective PCI, radial from femoral access, complete from incomplete revascularization, and center-based from home-based care. Outcomes should include MACE, bleeding, angina, physical function, medication adherence, CR completion, cost, and equity.

Artificial intelligence (AI)-supported coaching, wearable-guided rehabilitation, digital monitoring, and remote follow-up should be tested prospectively before routine use. Studies should define how data will change care and should address privacy, safety, and unequal digital access. Economic analyses should include staff time, devices, travel, patient time, readmissions, and quality-adjusted life-years. In low- and middle-income settings, research should prioritize feasible approaches such as automatic referral, task sharing, culturally appropriate diets, telephone support, and non-digital options[72-74,76,86,88].

## CONCLUSION

PCI restores coronary blood flow but does not cure systemic atherosclerosis or remove the risks from untreated lesions, residual ischemia, medication non-adherence, and adverse health behaviors. A PCI-focused approach is useful because early recovery involves access-site healing, time-sensitive DAPT adherence, possible staged or incomplete revascularization, and a risk that symptom relief will be mistaken for cure.

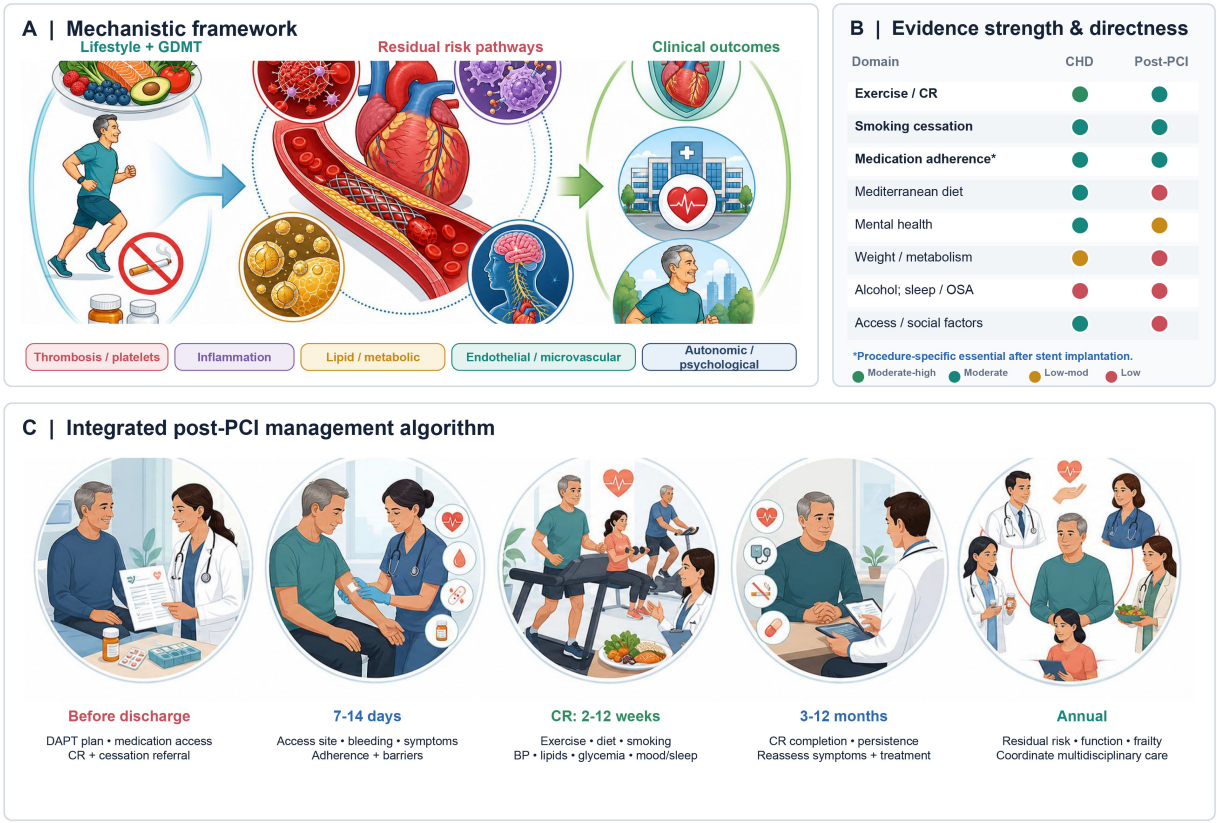


**Figure 1.** Lifestyle pathways, evidence strength, and integrated post-PCI care. (A) Mechanistic framework linking lifestyle interventions and guideline-directed medical therapy to thrombotic, inflammatory, lipid/metabolic, endothelial/microvascular, and autonomic/psychological residual-risk pathways and subsequent clinical outcomes; (B) Descriptive comparison of the strength and directness of evidence across lifestyle-related domains in broader coronary heart disease and post-PCI populations. These comparisons reflect study design, consistency, directness, precision, and risk of bias and should not be interpreted as a formal GRADE assessment; (C) Integrated management algorithm outlining key priorities before discharge, at 7-14 days, during cardiac rehabilitation, at 3-12 months, and during annual follow-up. Illustrative elements in Figure 1 were initially generated using ChatGPT Images 2.0, powered by GPT Image 2 (OpenAI, San Francisco, CA, USA), and were subsequently reviewed, edited, and assembled by the authors using Microsoft PowerPoint 2024 and Adobe Illustrator 2026. BP: Blood pressure; CHD: coronary heart disease; CR: cardiac rehabilitation; DAPT: dual antiplatelet therapy; GDMT: guideline-directed medical therapy; OSA: obstructive sleep apnea; PCI: percutaneous coronary intervention; GRADE: grading of recommendations assessment, development and evaluation.

**Table 2. Proposed guideline-informed lifestyle and secondary-prevention framework after PCI**

| Follow-up stage | What to assess | Main actions |
|---|---|---|
| Before discharge | ACS or elective PCI; access site; ventricular function; residual or staged disease; bleeding risk; medication access; smoking, mood, sleep, and social barriers | Explain that PCI treats a lesion but does not cure atherosclerosis. Reconcile medicines; provide written DAPT, missed-dose, and bleeding instructions; refer to CR and smoking-cessation support[20-22,59,79,87] |
| Early (7-14 days) | DAPT access and adherence; bleeding; access-site recovery; angina or dyspnea; smoking relapse; transport, cost, and digital barriers | Address cost and adverse effects; start activity that is safe for the access site and clinical status; assess recurrent symptoms; confirm a feasible CR plan[20,22,59,79,87] |
| CR phase (2-12 weeks) | CR participation and exercise capacity; diet; smoking; blood pressure, lipids, glycemia, weight and waist; mood and sleep symptoms | Individualize aerobic and resistance exercise; agree on realistic dietary goals; reinforce cessation; treat cardiometabolic and psychological problems when indicated[20-23,33,34,64-68,75] |
| 3-12 months | CR completion; return to smoking or harmful drinking; medication persistence; persistent symptoms; ischemic and bleeding risk; social needs | Review goals and medicine indications; reassess the cause of angina or dyspnea and the antithrombotic plan; add multidisciplinary, home-based, or hybrid support when needed[20,22,59,72-74,79,85,87] |
| Long term (annual) | All lifestyle domains; medication persistence; lipids and other residual risks; physical function and frailty; progression of untreated disease; structural barriers | Coordinate cardiology, primary care, CR, pharmacy, nutrition, and mental-health care. Use digital monitoring only when an abnormal result leads to a defined clinical response[20-22,59,72-74,76,78-80,82-84,86,87] |

PCI: Percutaneous coronary intervention; ACS: acute coronary syndromes; DAPT: dual antiplatelet therapy; CR: cardiac rehabilitation.

The strength of evidence varies across lifestyle domains. Exercise-based CR and smoking cessation have the most consistent support. A Mediterranean diet has randomized secondary-prevention evidence, but direct post-PCI outcome data are limited. Evidence for other dietary patterns, alcohol, sleep, psychological interventions, weight loss, and digital care is less certain. The best post-PCI care combines realistic lifestyle support with individualized management of lipid, inflammatory, thrombotic, ischemic, and cardiometabolic risk.

## DECLARATIONS

### Authors' contributions

Conceived the study: Chen Z, Wang Y, Ma J, Desjardins C
Wrote the first draft of the review: Wang Y, Ma J
Prepared the figures for the manuscript: Hu Y
All authors critically revised the manuscript and approved the final manuscript.

### Availability of data and materials

Not applicable.

### AI and AI-assisted tools statement

During the preparation of this manuscript, the AI tool ChatGPT (version GPT-5.6 Thinking, released 2026-07-09) was used solely for language editing. ChatGPT Images (version 2.0, powered by GPT Image 2, released 2026-04-21) was used to generate the figure based on author-defined scientific content and instructions. The authors subsequently reviewed, verified, and edited all AI-assisted text and visual content to ensure scientific accuracy, appropriate representation, and consistency with the manuscript. These tools did not influence the study design, data collection, analysis, interpretation, or the scientific content of the work. All authors take full responsibility for the accuracy, integrity, and final content of the manuscript.

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### Conflicts of interest

All authors declared that there are no conflicts of interest.

### Ethical approval and consent to participate

Not applicable.

### Consent for publication

Not applicable.



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