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REVIEW

# Endothelial progenitor cells and coronary artery disease: Current concepts and future research directions

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# Abstract

Vascular injury is a frequent pathology in coronary artery disease. To repair the vasculature, scientists have found that endothelial progenitor cells (EPCs) have excellent properties associated with angiogenesis. Over time, research on EPCs has made encouraging progress regardless of pathology or clinical technology. This review focuses on the origins and cell markers of EPCs, and the connection between EPCs and coronary artery disease. In addition, we summarized various studies of EPC-capturing stents and EPC infusion therapy, and aim to learn from past technology to predict the future.

Key Words: Endothelial progenitor cells; Coronary disease; Endothelial progenitor-cell capture stents; Endothelial progenitor-cell infusion; Clinical application

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**Core Tip:** The development of clinical applications of endothelial progenitor cells (EPCs) has progressed in recent decades. In this review, we summarize and discuss the origins and antibody markers of EPCs and the clinical effects of EPC stents and infusion. We hope to predict future clinical uses of EPCs.

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# INTRODUCTION

The development of clinical applications of endothelial progenitor cells (EPCs) has progressed over the years, with identification of antibody markers of EPCs and with clinical study of EPC stents and EPC infusion. The core application depends on the excellent properties associated with angiogenesis of EPCs. We mainly discuss the applications of EPCs in coronary artery disease and EPC application and summarized various studies of EPC-capturing stents and EPC infusion therapy, focusing on the mechanisms involved.

# INTRODUCTION TO EPCS

Decades ago, scientists found that endothelial cells (ECs) could proliferate and migrate to ischemic tissues or tumors and promote angiogenesis. Identifying markers of circulating cells that promote new blood vessel development has been a research challenge in recent years. For example, Flamme *et al*[1] and Weiss *et al*[2] explored the origin and development of hematopoietic cells and ECs, and they found that hematopoietic cells and EPCs are derived from a common precursor and share some markers during embryonic development. In 1997, EPCs were first isolated and cultured from peripheral blood by Asahara *et al*[3]. They are positive for both hematopoietic stem cell (HSC) markers like CD34 and CD133 and endothelial marker proteins like vascular endothelial growth factor receptor (VEGFR)2. CD34 is expressed on virtually all normal hematopoietic progenitor cells. Steen et al[4] identified CD34 as an HSC marker on human peripheral blood monocytes in 1998. The researchers suggested that human peripheral blood mononuclear cells could be expanded in vitro as pluripotent stem cells and differentiate into cells of distinct lineages and transplantation candidates. Gehling et al[5] found that CD133-positive cells formed new blood vessels in mice, which indicated that CD133-positive cells have the potential to differentiate into ECs. CD146, a transmembrane immunoglobulin mainly expressed at the intercellular junctions of ECs, by vascular smooth muscle cells (VSMCs), and pericytes[6], is involved in cell-cell adhesion, angiogenesis, and monocyte transmigration, CD146 includes three forms, lgCD146, shCD146, and sCD146. Jouve et al[7] found that CD146 expression reflected alteration of vascular permeability, and was significantly increased and accompanied by release of cell adhesion molecules when endothelial dysfunction was present. A reduction in permeability was observed in CD146-deficient mice[7]. In addition, CD146 is expressed in late EPCs[8-10], which enhances the angiogenic properties, endothelial function, and reduces neointimal formation by EPCs[8]. Factors that identify EPCs may reflect changes in not only cell number but also cell function. However, EPC nomenclature still lacks concordance in the biomedical field<sup>[11]</sup>, and no single surface marker has been reported to specifically identify EPCs.

Studies have shown that EPCs come from various sources, and the most representative cells are HSCs from bone marrow. In recent years, Yang *et al*[12] examined the importance of CD34 as a progenitor cell marker and studied the origin of progenitor cells. CD34 cells derived from mouse bone marrow had increased adhesion, homing capacity, and angiogenesis. Additional EPC characteristics have been identified, including angiogenesis that promotes blood vessel growth, differentiation to both hematopoietic and endothelial phenotypes, mobilization and adhesion to the walls of blood vessels, and survival and homing capacity[13]. In recent years, scientists have found that vascular progenitor cells are derived from the vessel itself; human umbilical vein ECs (HUVECs) and human aortic ECs derived from vessel walls have high proliferative potential [14], and have become the basis for vascular transplantation technology. Transplanted EPCs are capable of enhancing neovascularization in different tissues and the vasculature[15]. In addition, EPCs found in the bone marrow and the adventitia of arteries, have provided new ideas for the treatment of cardiovascular diseases[16,17]. Progenitor cells from different sources have been shown to migrate into blood vessels and induce the proliferation of blood vessels in corresponding tissues[18], and increasing the number of EPCs enhances neovascularization [19]. From those we guess there might be some dissimilarities among various types of EPCs, and recent years transcriptome analysis has showed some evidence to confirm predictions. Abdelgawad et al<sup>[20]</sup> reported that genes involved with angiogenic potential such as BMP2, 4, and ephrinB2 were highly expressed in EPCs. The expression of neuropilin-1 and vascular endothelial growth factor (VEGF)-C were significantly upregulated in EPCs and HUVECs. Other genes such as Notch1, MIR21



and platelet/endothelial cell adhesion molecule-1 (PECAM-1) were also differentially expressed in EPCs of various origins. Single-cell RNA sequencing also revealed interesting results. CD163 and CD115 are considered to be markers of early EPCs, and CD36 might be a marker of late EPCs. Two other EPC-related gene markers, PLAUR and NOTCH2 are highly regulated in EPCs and peripheral blood mononuclear-cell monocyte subcompartment. The genes influenced the hematopoietic activity and migration of cells[21,22]. The findings may help to identify novel EPC markers and increase the yield of EPCs thus promoting the clinical application of cell therapy.

However, the supportive effect of EPCs on growth is not the only effect. For example, endothelial dysfunction is a crucial step in the pathology of atherosclerosis, and the secretion of reactive oxygen species (ROS) by dysfunctional ECs accelerates the progression of vascular inflammation[23]. In addition, inflammatory cytokines, such as interleukin (IL)-6, IL-8 and IL-1 $\alpha$ , produced by cells induce inflammation, and inflammatory cytokines and the senescence-associated secretory phenotype contribute to cell aging<sup>[24]</sup>. On the other hand, the inflammatory environment promotes the migration and angiogenic functions of EPCs[25], and we must consider the influence of inflammation and immunity on EPC transplantation therapy. Studies have found that (1) EPCs suppress T cell proliferation and modulate T cell differentiation into less proinflammatory and active phenotypes; and (2) Tumor necrosis factor (TNF)a interacts with TNF receptor (TNFR)2 to enhance the immunosuppressive and antiinflammatory effects of EPCs. TNFa is a proinflammatory cytokine that regulates both pro- and anti-angiogenic activity [26,27], and binding with the TNFR1/TNFR2 transmembrane receptors has different immunomodulatory effects of EPCs on T cell immunity[28]. A study has shown that endothelial colony-forming cells (ECFCs) reduced the production of T cell proinflammatory cytokines and the immunosuppressive effect depended on the TNF/TNFR2 axis. Other studies have shown that EPCs from cord blood or from adult peripheral blood have different influences on regu-lating the immunosuppression of T cells[29]. Co-culture of human aortic endothelial cells, and ECFCs isolated from umbilical cord blood (CB-ECFCs) and from adult peripheral blood (APB-ECFCs) were less susceptible to immune rejection. The TNFα-TNFR2 axis was found to be important in the ECFC immunomodulatory effect. TNFR2 agonists enhanced the anti-inflammatory activity ECFCs, but antagonists inhibited that function. Such different mechanisms could help to choose ideal EPCs to avoid immune rejection and tolerate allogenic responses.

Recent progress has been made in the therapeutic applications of EPCs. Tateishi-Yuyama et al[30] reported that the local transplantation of bone marrow cells improved limb ischemia[31]. In 2012, Donndorf et al[32] injected CD133-labeled bone marrow cells into the myocardium by to verify the ability of progenitor cells to regenerate ischemic tissues and induce angiogenesis. In recent years, bioscaffolds have been used for vascular repair. Human adventitial ECs can proliferate on pepsindigested porcine adventitial and porcine small intestinal submucosal extracellular matrix (ECM) bioscaffolds in response to basic fibroblast growth factor (FGF)2[33]. Acellularized scaffolds can also induce angiogenesis by mediating the adhesion and chemotaxis of EPCs to growth factors such as platelet-derived growth factor (PDGF), VEGF and hypoxia-inducible factor[34]. In the heart, a cell-free engineered scaffold promoted revascularization in ischemic myocardial tissue, and expression of the S100 protein marker indicated nerve fiber regeneration[35]. Stem cell transplantation or scaffolds that capture stem cells, have great therapeutic potential to improve transplanted cell function and have applications in ischemic necrotic tissue and the myocardium in acute myocardial infarction (AMI). The effects are associated with activation of growth factors, and involve cell migration, transplanted cell proliferation, new matrix deposition, and the production of signaling molecules. The mechanisms will be discussed in detail.

# EPCS AND CORONARY ARTERY DISEASE

# Pathological processes in coronary atherosclerosis

Coronary artery disease (CAD) is a major public health issue and has been the leading cause of mortality and morbidity worldwide in recent years[36]. Study of coronary artery structure and cell physiology has led to considerable progress in the treatment of atherosclerosis. EC damage is an important step in the pathology of atherosclerosis [37]. Mechanical injury or inflammation. cause subendothelial VSMCs of the artery to proliferate and produce a large amount of ECM during the initial stage of intimal thickening[38]. Then, low-density lipoprotein and cholesterol combine in regions

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prone to atherosclerosis, which are mainly located at vessel branches and curves disturbed by irregular low wall shear stress<sup>[39]</sup>. Inflammatory cells such as macrophages enter the arterial wall in response to stimulation and phagocytose oxidized lipids to form foam cells[40], which is a key step in the pathological process of coronary atherosclerosis.

# Mechanisms of EPC proliferation and migration in CAD-associated angiogenesis

EPCs cause rapid healing by proliferation, migration, and adhesion to the sites of blood vessel damage in CAD[41]. The molecular mechanisms may include (1) VEGFstimulated migration of EPCs from the bone marrow to the damaged vasculature[42]. The migration of EPCs was first discovered by Asahara et al[3], but the exact mechanism is still unclear. In recent years, scientists have shown that VEGF acts via the PI3K/AKT signaling pathway to promote EPC mobilization[43]. Differential gene expression analysis showed that high levels of PI3K and AKT increased VEGF expression and induced angiogenesis in EPCs[44,45]. The P38-MAPK pathway was also associated with the differentiation and mobilization EPCs from the bone marrow, which is the main source[46,47]. In terms of signal transmission, P38-MAPK is a downstream component of VEGFR2 signaling (the dominant component of the VEGF family that regulates angiogenesis)[44]. Through p38-MAPK, VEGF2 promotes the proliferation and migration of EPCs by regulating the expression of the activators urokinase plasminogen, serine protein kinases, and threonine protein kinases[48,49], thus strengthening angiogenesis. (2) Some noncoding small RNAs, such as microRNAs also have an important role in EPC mobilization. For example, microRNA-221 (miR-221), miR-222, and miR-206 are involved in EPC-mediated promotion of angiogenesis by influencing VEGF expression[50-52]. The molecular mechanism involves binding to the 3'-UTRs of downstream protein-coding mRNAs, thus modulating the growth and differentiation of EPCs[52]. (3) When hypoxia and ischemia injure blood vessels, endothelial nitric oxide synthase (eNOS) activates the release of proangiogenic factors and induces the migration and proliferation of EPCs[53,54]. eNOS is one of the markers used to identify EPCs in humans[55]. PCR and western blotting have shown that CXCR4 activates the PI3K/Akt/eNOS signal transduction pathway to promote the phosphorylation of eNOS and then stimulate the migration of EPCs[56,57]. Nitric oxide (NO) is produced by eNOS uncoupling[58]. Ischemia in mice was improved when the mice were fed arginine, a substrate of NOS[59]. NO-sensitive guanylyl cyclase participated by cGMP-dependent mechanisms, which revealed the role of NO in EPC proliferation[60]. NADPH oxidase 4 can impair the function of EPCs. NADPH acts as a substrate and is crucial for maintaining cellular redox homeostasis[61]. Oxidative stress can cause endothelial cell injury and EPC dysfunction[62], which promotes pathological changes in coronary heart disease. The main mechanisms involve the expression of NOX and high levels of ROS. Patients with upregulated NOX and ROS have decreased EPCs, and it was shown that the migration and adhesion of EPCs was reduced[63,64].

# STUDIES OF EPCS IN AMI

AMI is one of the most dangerous events associated with coronary heart disease[36]. Because of massive ischemia and necrosis of the myocardium, it difficult to resolve ischemia and revascularization except by surgery and percutaneous coronary intervention (PCI)[65,66]. However, there are some limitations. Not all patients are qualified for surgery, and poor prognosis and clinical events after surgery are still challenging for clinicians<sup>[67]</sup>. Therefore, we need an effective therapeutic strategy for conditions that surgery cannot address. EPCs can proliferate, migrate, and adhere to tissues. In ischemic tissue, EPCs can differentiate into corresponding ECs[1]. Vasculogenesis and myogenesis have been described in heart tissues after EPC infusion in a canine model of AMI [67]. In addition, the expression of VEGF was upregulated, and the EPCs differentiated into myocardial cells. The evidence suggests that EPC infusion could enhance neovascularization after AMI. In a mouse model of AMI, injecting EPCs enhanced myocardial healing after AMI and reduced the formation of lymphatic vessels, which may decrease inflammation and myocardial remodeling[68]. However, in an in vitro study, the results were not ideal, and the effects of EPCs on AMI patients were attenuated compared with those in the healthy group. Many factors, like lifestyle habits and drug treatment affect the process and impact treatment to different degrees [69]. Smoking and drinking may cause EC damage and angiotensin converting enzyme inhibitors or angiotensin receptor inhibitors can increase the activity of EPCs.



Therefore, clinical applications require additional clinical data to support safety and effectiveness.

Scientists have conducted several clinical trials to demonstrate the efficacy of EPCs in the treatment of ischemic cardiac tissue [70-80] (Table 1). First, scientists performed several imaging evaluations to assess the migratory and regenerative capacities of EPCs in the ischemic myocardium after progenitor cell therapy [70-72]. The efficacy of progenitor cell therapy reduced infarct size and improved left ventricle function. In addition, the cellular mechanisms associated with EPC therapy were initially explored. The migratory capacity of EPCs toward the target tissue relied on their homing capacity, and some chemoattractants, such as SDF and VEGF, were involved in the homing signaling pathway that recruited circulating EPCs and enhanced repair mechanisms after ischemia [71,72]. However, the scientific community awaits the results of clinical trials to assess safety and efficacy. A few years later, a study that used EPC infusion to treat idiopathic pulmonary arterial hypertension and reported the EPC therapy had patient benefits<sup>[73]</sup>. Cell infusion increased the distance walked in 6 min by 42.5 m (95% confidence interval 28.7-56.3, P = 0.001) compared with conventional therapy, and improved in pulmonary artery pressure and cardiac output, with no adverse events, suggesting feasibly and safety. A study of EPC treatment of AMI focused on bone marrow-derived CD34+ cells and showed that a 3% improvement in ejection fraction (EF) occurred in the treatment group after the infusion of EPCs[74]. CD34+ cell homing was observed. Another clinical trial showed that a certain number of CD34 cells may increase the EF and reduce the infarct size in AMI<sup>[75]</sup>. In the study, an overall improvement in LVEF of approximately 5.0% was reported. Angina and heart failure improved at the 12-mo follow-up (all P < 0.001), and the survival rate at the 18.5-mo follow-up was 94.7% (n = 36). The evidence supports the safety and efficacy of EPC therapy. PECAM-1, also called CD31, is a vascular cell adhesion and signaling molecule that is expressed on the surface of human granulocytes, monocytes, and platelets[76]. In experimental animal models, CD31+ EPCs therapy enhanced perfusion and reduced apoptosis in the healing myocardium<sup>[78]</sup>, and immune system stimulation of increased anti-inflammatory cytokine may predict fewer adverse events<sup>[79]</sup>. In another mouse model, abnormal proliferation was not observed after EPC transplantation[80]. Whether various types of EPCs are available for intravenous therapy remains unclear. Long-term prognosis and safety also need further investigation.

# **EPC-CAPTURE STENTS**

# Clinical applications of EPC-capture stents

EPC-capture stents are stainless steel devices that are coated with monoclonal antibodies such as CD133, CD34 and CD146 and are associated with a decreased incidence of restenosis and thrombosis[81-83]. Monoclonal CD34 antibodies bind to EPCs in the peripheral blood and promote healing [84], by migration and proliferation. The promotion of EPC colonization in the stent accelerates re-endothelialization and revascularization of the stented segment[85], which leads to decreased rates of restenosis and thrombosis after PCI. Studies of EPC stents coated with different types of antibodies are shown in Table 2, and described below.

The advantage of EPC stents coated with monoclonal CD34 antibodies is that CD34 enhances stent endothelialization, thus enhancing the adhesion and proliferation of EPCs. Studies of endothelialization in different stents has shown that CD34 antibodies stents increased endothelial coverage,  $97 \pm 3\%$  in anti-CD34 antibody stents,  $95 \pm 4\%$  in hyaluronan-chitosan-anti-CD34 antibody and sirolimus-eluting stents, and  $74 \pm 8\%$  in sirolimus-eluting stents[86]. A clinical study of CD34 antibody-coated stents in a group of 100 patients reported major adverse cardiac events (MACE), a composite of cardiac death, myocardial infarction, and emergency cardiac surgery, in 15.6% of patients at 12 mo and 16.6% at 24 mo. The target vessel failure rate, a composite of revascularization, recurrent infarction, or cardiac death of the target vessel) was 14.6% at 12 mo and 24 mo[87]. In another study, 2279 patients were treated with EPC stents and grouped into low bleeding risk (LBR) and intermediate-to-high bleeding risk (IHBR) groups. The rate of 1-year target lesion failure (TLF) was 4.1% in the IHBR and 2.6% in the LBR groups. The AMI rates were 1.8% in the IHBR and 1.1% in the LBR groups, and the incidence of stent thrombosis was 1.2% in the IHBR and 0.6% in the LBR groups [88], which showed a higher 1-year TLF rates. Animal studies of CD133 combination stents mainly investigated restenosis and endothelialization, and two compared CD133 with CD34. Wu et al [89] reported that the time of cell adhesion was longer and EPC capture



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# Table 1 Clinical studies of endothelial progenitor cell therapy

Ref.	Article	Species	EPCs category	Result
Britten <i>et a</i> l[71], 2003	Infarct remodeling after intracoronary progenitor cell treatment in patients with acute myocardial infarction (TOPCARE-AMI): Mechanistic insights from serial contrast-enhanced magnetic resonance imaging	Human	CD34, CD45, CD133	The progenitor cell therapy could rescue dysfunctional myocardium early after AMI
Döbert <i>et al</i> <b>[72]</b> , 2004	Transplantation of progenitor cells after reperfused acute myocardial infarction: Evaluation of perfusion and myocardial viability with FDG-PET and thallium SPECT	Human	BMCs and EPCs	The EPC therapy could increase myocardial viability
Wang et al[ <mark>73</mark> ], 2007	Transplantation of autologous endothelial progenitor cells may be beneficial in patients with idiopathic pulmonary arterial hypertension: A pilot randomized controlled trial	Human	Peripheral blood EPCs	Infusion of EPCs seemed to be feasible and safe, and might have beneficially affect to AMI patients
Dedobbeleer <i>et al</i> [74], 2004	Myocardial homing and coronary endothelial function after autologous blood CD34+ progenitor cells intracoronary injection in the chronic phase of myocardial infarction	Human	CD34	The safety and homing ability of EPCs are proved in both acute and chronic conditions
Flores-Ramírez et al[77], 2010	Intracoronary infusion of CD133+ endothelial progenitor cells improves heart function and quality of life in patients with chronic post-infarct heart insufficiency	Human	CD133	The EPCs therapy had improved the heart function of patients
Dubois <i>et al</i> [ <mark>78</mark> ], 2010	Differential effects of progenitor cell populations on left ventricular remodeling and myocardial neovascularization after myocardial infarction	Pig	CD31, CD90, CD29, CD44, CD45	Infusion of late-outgrowth EPCs could improve myocardial infarction remodeling
Lee <i>et al</i> [70], 2015	Intracoronary transfusion of circulation-derived CD34+ cells improves left ventricular function in patients with end-stage diffuse coronary artery disease unsuitable for coronary intervention	Human	CD34	CD34+ cell therapy was safe and efficacious in improving heart function
Sung et al[ <mark>75</mark> ], 2018	Five-year clinical and angiographic follow-up outcomes of intracoronary transfusion of circulation-derived CD34+ cells for patients with end-stage diffuse coronary artery disease unsuitable for coronary intervention-phase 1 clinical trial	Human	CD34	CD34+ cell therapy might contribute to improving left ventricular function, heart failure, and amelioration of left ventricular remodeling
Shen <i>et al</i> [ <mark>80</mark> ], 2018	Induced pluripotent stem cell-derived endothelial progenitor cells attenuate ischemic acute kidney injury and cardiac dysfunction	Mouse	CD31	EPC therapy may reduce the effect of cardiomyocyte apoptosis and cardiac dysfunction
Lee <i>et al</i> [79], 2019	Clinical assessment of intravenous endothelial progenitor cell transplantation in dogs. cell transplant	Dog	CD105, CD31 and CD144	Dogs with EPC transplantation have reduced platelets, increased VEGF, and increased IL-10
Angulski <i>et al</i> [81], 2019	systemic infusion of expanded CD133+ cells and expanded CD133+ cell-derived EVs for the treatment of ischemic cardiomyopathy in a rat model of AMI	Rat	CD133	Not significant effect was observed in this experiment

AMI: Acute myocardial infarction; BMCs: Blood mononuclear cells; EPCs: Endothelial progenitor cells; EV: Extracellular vesicle; SPECT: Single photon emission computed tomography; TOPCARE-AMI: Transplantation of Progenitor Cells and Regeneration Enhancement in AMI; VEGF: Vascular endothelial growth factor.

> was improved with anti-CD133 antibody-coated stents compared with the anti-CD34coated stents. Li et al[90] reported that anti-CD133 antibody-coated stents enhanced endothelialization more than anti-CD34-coated stents. In-stent restenosis was investigated by Wawrzyńska et al[91], and confocal images of ECs and VSMCs showed that the anti-CD133 antibody stent accelerated re-endothelialization and inhibited the proliferation of VSMCs. The overall results indicate that anti-CD133 antibody stents potentially avoided thrombosis and reduce restenosis in. A recent study by Park et al [83] focused on CD146. CD146, which is related to endothelial lineages, such as late EPCs and outgrowth ECs. CD146 stents had a higher cell capture efficiency than CD133 stents. The CD144 stent was expected to have synergistic effects in suppressing restenosis.

> Because of the diversity of antibody markers for EPCs, there are various types of EPC-capture stents. The choice for clinical treatment depends on the cell capture efficiency and endothelialization function to prevent in-stent restenosis. Anti-CD34



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### Table 2 Clinical outcomes with endothelial progenitor cell stents Patients, Ref. Article Inclusion criteria Major clinical outcomes n Sung et al[75], Five-yr clinical and angiographic follow-38 Death from any cause/major adverse Five-yr clinical outcomes: 2018 cardiac and cerebrovascular event/target Noncardiovascular death: 13.2% up outcomes of intracoronary transfusion of circulation-derived CD34+ cells for vessel revascularization/newly onset Cardiovascular death: 7.9%. Acute patients with end-stage diffuse coronary atrial fibrillation myocardial infarction: 7.9%. Newly artery disease unsuitable for coronary onset atrial fibrillation: 2.6% intervention phase 1 clinical trial Sarno et al[85], Real-life clinical outcomes with 42357 Clinical presentation/lesion One-vr outcomes: Restenosis: 1.1%; 2017 everolimus eluting platinum chromium Restenotic lesion: 3.8%; Death: 5.2% characteristics stent with an abluminal biodegradable polymer in patients from the Swedish coronary angiography and angioplasty registry (SCAAR) den Dekker et al Final results of the HEALING IIB trial to 100 Angiographic features/ MACCE rate Two-yr clinical outcomes: MACCE: [87], 2011 16.6%, MI: 5.2%, TLR clinically evaluate a bio-engineered CD34 driven: 11.5%, TVF: 14.6%, Stent antibody-coated stent (Genous stent) thrombosis: 3.1% designed to promote vascular healing by capture of circulating endothelial progenitor cells in CAD patients One-yr TLF/target lesion 1-vear COMBO stent outcomes stratified 2279 One-vr outcomes: TLF: 6.7%, Chandrasekhar et al[88], 2020 by the PARIS bleeding prediction score: revascularization/ST/major adverse Cardiac death: 2.4%, MI: 2.9%, TLR: From the MASCOT registry 3.1% , Stent thrombosis: 1.8%cardiac events

MACCE: Major adverse cardiac and cerebrovascular events; MI: Myocardial infarction; TLR: Target lesion revascularization; TVF: Target vessel failure; TLF: Target lesion failure.

> antibody stents are commonly used in the clinic, but studies have shown that CD133 and CD146 have a better potential to stimulate angiogenesis and prevent restenosis, which may reduce the incidence of clinical events. Larg multicenter randomized controlled trials are needed to standardize and verify the therapeutic applications before clinical application is feasible.

# EPC-mediated reduction in thrombus formation may be an advantage of EPC-

# capture stents

The incidence of clinical events can be reduced by EPC capture, but there are still no significant differences between EPC-capture and traditional drug-eluting stents (DES). That may be related to the characteristics of EPCs, which can promote the repair of the vascular endothelium and also influence thrombus propagation[92,93]. Platelets can bind to bone marrow-derived CD34+ cells and recruit the cells to the vascular wall during vascular injury; the chemokine SDF-1 $\alpha$  and GPIIb integrin mediate the process. Platelets also adhere to the vessel wall, forming a thrombus. SDF-1 and VEGF recruit EPCs, resulting in vascular repair and remodeling[94]. EPCs participate in the resolution of thrombosis together with multiple chemokines. VEGF, SDF1, and PDGF are involved in EPC migration[95-97]. EPCs integrate into the damaged endothelium and repair injured vessels. New vessels are formed, and vascular endothelial monolayers are integrated. In that way, EPCs significantly drive the development of new vascular channels in thrombi[98], and neovascularization is a significant marker to indicate thrombus resolution and recanalization<sup>[99]</sup>. Second, NO affects the activation, adhesion, and aggregation of platelets, ultimately preventing thrombosis [100]. Finally, the integrity of vascular ECs can prevent thrombosis[101]. When new damage occurs to the lining of blood vessels, ECs are recruited to accelerate repair of the damage, thus significantly reducing the incidence of thrombi.

The curative effect of EPC-capture stents leads a better result than that of bare metal stents or DESs containing antiproliferative drugs, such as sirolimus, which confirms the original hypothesis that stents that reduce the rates of clinical events such as LST, MI and thrombosis extend survival [102,103]. The clinical trial results may have been slightly skewed by the interference of age, medication use and implantation time[104, 105]. Age may be associated with increased rates of heart failure and AMI. The risk of late stent thrombosis increases with time, and patients who are treated with drugs under a physician's guidance may have decreased risks of clinical events. However, those factors did not have absolute statistical significance. Clinical outcomes are affected by clinical and technical factors, mental health, and ethnic origin. Long-term

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follow-up to show the impact of clinical events and the associated risk factors is also lacking

# CONCLUSION

The results of recent EPC studies has been encouraging, regardless of the CAD pathology or vascular repair technology. Clinical manipulation of EPCs still needs to be practiced, and the possibility of using drugs to promote vascular repair needs to be further explored. Antibody-coated stents have also been successfully used, and it is unclear whether additional antibodies can be used for treatment. Various antibodies, including CD34, CD133 and CD146 have had unique results in animal experiments, but it is unclear which has the best potential for EPC capture efficiency. Can the stent structure be improved to reduce the incidence of acute thrombosis and late clinical events? In basic research, we found high adhesion, homing capacity, and angiogenic abilities of EPCs, and more study of the mechanisms are needed to understand and improve the understanding of EPCs. All the challenges need to be solved. The optimal patients for EPC-capture stents and relevant risk assessments also need to be established, and perhaps we need a large clinical study to study that.

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