Introduction

As one of the most deadly diseases in the world, the number of deaths due to cardiovascular disease (CVD) has accounted for 31% of all deaths in the world. Atherosclerosis is a chronic inflammatory disease caused through imbalance of lipid metabolism and poor immune response. Coronary atherosclerotic heart disease (CADs), which originates from the peripheral coronary arteries of the heart, is a typical type of cardiovascular disease. This is a disease in which atherosclerotic plaques on the vascular wall cause vascular stenosis, which interferes with the continuous blood supply of the coronary arteries, resulting in insufficient blood supply or ischemia to the heart, and ultimately causing cardiac dysfunction.

With the continuous development of biomedical materials, implant materials such as artificial blood vessels, tissue-engineered blood vessels, and cardiovascular...
stents\textsuperscript{10–13} have become important choices for the clinical treatment of CADs.

However, the risk of restenosis\textsuperscript{14–16} and late thrombosis\textsuperscript{17–19} in the lesion site after implantation is increased, which severely limits the application of implant materials. First, the adsorption of plasma proteins on the surface of the material occurs within seconds of contact between the material and blood. The platelets and other blood cells in the plasma are interact with the protein adsorption layer, the platelets are activated, and the fibrinogen converts to fibrin, which stimulates a large number of activated platelets to aggregate and lead to the formation of acute thrombosis.\textsuperscript{20} Then the implantation of the material induces damage to the vascular endothelial layer and activates the surrounding ECs. These activated ECs and platelets release a large number of chemokines and inflammatory factors, which induce inflammatory cells to aggregate toward the lesion and trigger an inflammatory response,\textsuperscript{20,21} which peaks within 2 days.\textsuperscript{22} In addition, clinical research data show that the peak of intimal hyperplasia is within half a year after implantation.\textsuperscript{23} However, about 2 weeks after implantation, the rapid proliferation of smooth muscle cells (SMCs) will also occur, and the cell proliferation tends to be stable after 4 weeks.\textsuperscript{24,25} and this early rapid proliferation of SMCs is also closely related to the occurrence of restenosis in late stages. In order to overcome these complications after material implantation, people have been exploring more effective solutions.

The natural vascular endothelial layer has excellent anti-coagulation and anti-intimal hyperplasia abilities, which is the key to maintaining the steady state of the environment between the blood vessel wall and the blood.\textsuperscript{26–28} ECs are a type of multilateral flat cells with jagged edges, located between the blood and the media of the blood vessel wall\textsuperscript{29} (Figure 1). ECs prevent coagulation and thrombosis by secreting or expressing biological factors (such as Nitric Oxide (NO),\textsuperscript{30} Prostaglandin I\textsubscript{2} (PGI\textsubscript{2}),\textsuperscript{31} tissue factor pathway inhibitor (TFPI).\textsuperscript{32}) On the other hand, ECs can shield the stimulating contact of growth factors in the blood to SMCs, and avoid the transformation of SMCs phenotype from contraction type to synthesis.
type. Synthetic SMCs secrete a large amount of extracellular matrix and invade the endometrial layer to form excessive endometrial hyperplasia, at the same time, the biological factors (such as NO) secreted by ECs can prevent the abnormal proliferation of SMCs, thereby preventing vascular stenosis. It is precisely these abilities that ECs possess to maintain the homeostasis of the vascular wall microenvironment.

Based on the above studies, the formation of a complete endothelial layer on the surface of cardiovascular materials is widely regarded as a more ideal treatment method. In early studies, researchers directly planted ECs on the material surface to form a complete endodermis layer to achieve the endothelialization of the material surface. This method is intuitive, simple, and operable in vitro, so it has achieved certain success. However, direct planting of cells has more stringent requirements for materials and cells, which led to a higher failure rate and longer planting time, so this idea was gradually abandoned. With extensive development and in-depth exploration of research, people’s understanding about endothelialization of implant materials has been further improved, and the surface modification of materials for promoting endothelialization has gradually attracted attention. Through the biofunctional design of the material surface, the ability of surface to promote ECs adhesion and proliferation was strengthened, and the process of surface endothelialization was accelerated. In addition, stem cells with endothelial differentiation ability such as endothelial progenitor cells (EPCs) and mesenchymal stem cell (MSCs) have gradually entered the field of researchers in recent years, providing new modification ideas for materials based on in situ induced endothelialization in vivo. Endothelialization-promoting modification on the surface of materials induces ECs proliferation, migration and adhesion, or promotes the homing and directional differentiation of stem cells. The strategies are mainly divided into three categories: Surface design based on material surface science method, Surface design based on bioactive molecules, and Surface design based on biological function intervention and feedback (Figure 2). Based on the reports of vascular implants in recent years, this paper reviews the research on endothelialization of materials by surface modification, and discusses the advantages and challenges of current surface modification methods, in order to provide new reference ideas for endothelialization design of vascular implants.

**Surface design based on material surface science method**

In the normal vascular wall, the main function of ECs is to maintain vascular homeostasis and avoid atherosclerosis caused by abnormal changes in blood environment. Therefore, ECs are very sensitive to the abnormal

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**Figure 2. Design strategy for material surface endothelialization.**
hemodynamic changes in the blood environment, and the changes in the physicochemical properties of the material surface can also affect the function of ECs. The endothelialization of the surface can be effectively promoted by controlling the changes in the physicochemical properties or microstructure of the material surface.

**Surface microstructure**

In the normal physiological environment, the function of ECs depends largely on their interaction with extracellular matrix, while extracellular matrix (ECM) exhibits micron or nano-scale microstructure characteristics in physical structure, and cells are very sensitive to this structure. Thus microstructure can effectively regulate the morphology of ECs on the surface of the material, and promote the secretion of extracellular matrix, thereby endowing the surface with better biocompatibility. Periodic pattern with a certain interval distance is a common modification method of material surface microstructure, which can effectively promote the migration and adhesion of ECs. Some grooved structures can effectively promote ECs to arrange along the direction parallel to the pattern, and have stronger migration ability.

Different roughness has distinct effects on cell activity and behavior. Due to the difference in the average size of ECs and SMCs, their adaptability to the roughness is significantly different. Comparing the ratio of cells adhered to the same roughness range, it was found that the proportion of ECs increased greatly as the roughness increased. Thus the microstructure size is also an important factor affecting ECs behavior. The microstructure of suitable size (15 μm) can keep the ECs in the form of a fused monolayer on the surface, which can effectively promote the ordered arrangement of ECs on the surface of the material. When the size of the microstructure is in the micron level, the large size of the pattern (7656 μm²) makes the cells “free expansion” across the region, and the smaller (1781 μm²) makes the cells “limited mobility.” This shows that when the area of the micropattern is less than the maximum area of the cell spreading, the cell shape is related to the size and shape of the pattern. Of course, in addition to regular structures, Random structures have the ability to regulate cell behavior at micro-nano scale. Wu et al. enhanced the adhesion of ECs on the material surface by constructing a random pattern with controllable size on the nonmetallic material surface, and avoided the interference of wall shear stress on the attached ECs. Metal materials such as titanium can use physical vapor deposition-thermochemical or anodic oxidation methods to construct microstructures with certain roughness on the surface. By adjusting the roughness, the protein adsorption, blood compatibility, and cell behavior on the material surface were affected to selectively promote the growth of EC and avoid restenosis after implantation.

Due to the close interaction between cells and materials, cells can make corresponding stress responses to the surface morphology of materials through actin polymerization, and this reaction can not only be reflected in ECs and SMCs, but also the differentiation and migration of stem cells. The multi-layer structure of the vascular implant uses the directional microgrooves between the biomimetic layers to promote the directional growth and migration of ECs, and better mimic the arrangement of natural vascular cells. Vascular implant materials containing microchannel structures both highly interconnected and circumferentially oriented can not only effectively affect the morphology, arrangement, and phenotypic changes of SMCs, but also improve the directional deposition of ECM on the surface of the material and the enrichment and polarization of macrophages, promote cell infiltration and accelerate the process of endothelialization.

**Physicochemical properties**

The adhesion and migration of cells on the surface of the material depend on the adsorption of plasma protein and cell secretory protein on the surface of the material, and the physical and chemical properties of the surface of the material affect the type, conformation, and adsorption amount of the surface protein. Grafting hydrophilic substances or UV irradiation can enhance the hydrophilicity of the material surface and promote the adhesion and proliferation of cells on the surface. Plasma treatment can significantly improve the hydrophilicity of metal surface. After plasma activation, the concentration of C-O group on the material surface increases, and the proliferation and migration of ECs are enhanced. It is noteworthy that due to the water barrier effect, the binding form of water molecules on the hydrophilic surface has a significant effect on the adhesion of cells. Chemically bound water molecules cannot provide the required binding site density for ECs, but physically bound water molecules can provide it. In addition, the surface charge can control protein (such as Fibronectin) adsorption, thereby regulating cell behavior, thereby enhancing cell adhesion and migration.

The stiffness and the magnetic of the surface can directly or indirectly affect the adhesion and proliferation of ECs on the material. Being anchorage-dependent, the adhesion and proliferation of ECs were inhibited on soft surface, while promoted on higher stiffness surface. But the ECs monolayer on the soft surface showed higher endothelial function than that on the hard surface. Chang
et al. used this discovery to construct a Vascular endothelial growth factor (VEGF)-loaded poly (l-lysine)/hyaluronic acid (PLL/HA) material surface, and then control the stiffness of the material surface by changing the crosslinking degree. The results showed that VEGF could effectively promote ECs adhesion on the material surface. The gene expression of ECs on soft surface (200 kPa) was significantly increased compared with that on hard surface (430 kPa), and the cell activity was significantly enhanced. The magnetic properties of materials can also significantly affect cell behavior. Under the action of 300 mT external magnetic field, the magnetized material surface can achieve high adsorption of magnetically labeled endothelial progenitor cells within 3 h. At the same time, due to the weakening edge effect, cells can be uniformly covered on the surface of the material. And Zhang et al.’s study shows that cell adhesion is also significantly related to the oscillation frequency of the magnetic field, and can effectively promote cell growth under low frequency magnetic field (0.1 Hz), and has long-term stability.

Whether microstructure or surface physicochemical properties, the surface modification methods based on material surface science focus on the interaction between materials and cell behavior, which makes the related modification methods can effectively promote cell adhesion and directional migration on the material surface. Cell adhesion and directional migration can rapidly form an endothelial layer on the surface of the material, which is the basis for rapid endothelialization and long-term stability of the vascular graft after implantation.

**Surface design based on bioactive molecules**

The excellent endothelial cell compatibility on the surface of vascular implant materials is the basis of vascular regeneration. The surface can create a good regeneration micro-environment for the migration and adhesion of cells, which is conducive to the process of endothelialization of the material. In order to improve the cytocompatibility of the material surface, the surface of vascular implant material is usually modified based on bionics methods, that is, immobilize natural or synthetic biological functional molecules on the surface of biological materials to construct natural physiological microenvironment, such as heparin, fucoidan, hyaluronic acid, ECM protein, and peptide. Based on the characteristics and advantages of heparin and hyaluronic acid, many researchers have carried out a large number of related studies and have made important progress. This article will also describe them separately.

**Heparin**

Heparin is a commonly used anticoagulant in clinic. It is often used to modify the surface of vascular implants and has anti-inflammatory function. Heparin also has the ability to protect ECs and resist intimal hyperplasia. Covalent binding is a common modification method. Heparin can be introduced into the surface of materials by EDC/NHS crosslinking reaction. Negatively charged heparin can also be introduced into the surface of the material by electrostatic interaction in the form of layer-by-layer self-assembly. However, compared with covalent modification methods, biomolecules assembled by electrostatics are often less stable, and uncontrolled molecular bursts occur in dynamic environments. Therefore, the characteristics of heparin with negative charge can be used to form nanoparticles with positively charged biological molecules to control its stability. For example, Song et al. used the specific binding of nerve growth factor (NGF) and heparin and the electrostatic interaction between heparin and chitosan to design a heparin/chitosan nanoparticle loaded with NGF to accelerate the endothelialization of the material surface. In addition to chitosan, poly-L-lysine is also a biomolecule that can be electrostatically bound to heparin to form nanoparticles. It is worth noting that too high density of heparin can also reduce ECs growth and enhance SMCs proliferation. Therefore, heparin content on the surface of the material may be the primary consideration in the process of surface endothelialization.

**Hyaluronic acid**

Hyaluronic acid (HA) is a linear polysaccharide composed of D-glucuronic acid and N-acetylglucosamine distributed in extracellular matrix, which has high biocompatibility with cells. As the main component of ECM, HA has excellent functions of promoting endothelial cell proliferation and anticoagulation, so it is often used for the modification of vascular materials and bone materials.

Some polymeric materials usually do not have good endothelial cell compatibility, so HA can be introduced into polymer materials by simple electrospinning and chemical crosslinking, which can improve the blood compatibility of the surface and promote the endothelialization of the material. For example, Dimitrievska et al. covalently grafted thiol-modified sodium hyaluronate onto the surface of decellularized scaffold materials with thiol-reactive properties, and cross-linked through the disulfide bond between HA to form a uniform thin layer on the surface of the material. In addition, the deposition of dopamine on the surface of the material can also graft HA onto the metal material, which can promote the endothelialization of the material surface and improve the corrosion resistance of the metal material.

In addition, some studies have found that the biological characteristics of hyaluronic acid are related to its relative molecular weight. Low molecular weight HA promotes inflammatory response and SMCs proliferation.
Jiang et al.’s study, they found that HA with a molecular weight of 100kDa confers anti-inflammatory, anti-proliferative, and reendothelializing properties to the scaffold. Although high molecular weight HA can be grafted on the surface, high molecular weight HA can be decomposed into low molecular weight hyaluronic acid in vivo, which is not conducive to long-term implantation of materials. At the same time, too high molecular weight HA can also inhibit the proliferation of ECs. Therefore, HA is often used in conjunction with other biomolecules to overcome the adverse effects of HA. For example, ECs-affinity peptides (RGD and YIGSR) were grafted onto HA hydrogel (100–150 kDa) by click chemistry with single or orthogonal density gradients, the modified material surface showed synergistic effect on cell adhesion and demonstrated the specific competitive adhesion of ECs. Low molecular weight HA (5 kDa) promoted cells adhesion and proliferation through EDC/NHS cross-linking Type I collagen. HA (4 kDa) hydrogel scaffold formed by calcium ion crosslinking of sodium alginate (SA) can provide excellent microenvironment for stem cells. This scaffold has a high ratio of water content and a slow degradation rate, exhibits a porous structure suitable for stem cell loading and good rheological behavior, which are helpful for stem cell differentiation. In vivo experiments further demonstrated that this modified material could protect human umbilical cord mesenchymal stem cells (hUC-MSCs) and maintain their cellular viability.

**ECM protein**

The adhesion, migration, and proliferation of ECs largely depend on the interaction between cells and the surrounding ECM proteins, so ECM proteins are also widely used in the surface modification of vascular materials to promote endothelialization. Fn is a common ECM protein, which can effectively improve the adhesion ability of ECs and EPCs as a protein coating on the surface of materials. Plasma can be used to treat the material surface to introduce fibronectin, but the differences in parameters will lead to great changes in the affinity, conformation, and direction of adsorbed Fn, which in turn significantly affects endothelial cell-material and cell-cell interactions. Surface deposition of dopamine can also effectively graft Fn onto the material to promote surface endothelialization. In addition to fibronectin, other proteins such as serum human laminin (Ln) and collagen have also been gradually applied to promote endothelialization on the surface of materials.

There are three kinds of collagen commonly used: type I collagen and type III collagen distributed in the media of blood vessel, type IV collagen distributed in the intima of blood vessels. Type I collagen can provide a good micro-environment for cell migration and differentiation. Compared with simple physical coating, the type I collagen structure on the material surface introduced by covalent crosslinking is more stable. Considering that type III collagen had platelet binding sites, Yang et al. prepared a new recombinant human type III collagen (hCOLIII), which retained the fragment that promoted cells adhesion and bypassed the platelet activation sequence containing hydroxyproline. Type IV collagen was fixed on the surface of the polymer material to construct a biomimetic vascular basement membrane. The results of cell experiments showed that the modified material surface promoted the proliferation of ECs and inhibited platelet adhesion.

**Peptide**

Specific amino acid sequences can mediate cell adhesion, proliferation, and differentiation by binding to transmembrane protein integrin family receptors or other receptors. Therefore, in recent years, more and more studies have focused on the extraction or synthesis of amino acid sequences that play a key role, and the application of these polypeptide molecules in the surface modification of vascular implants to promote the endothelialization of such materials (Table 1).

ECs are surrounded by an ECM composed of type IV collagen, Ln, and heparin sulfate polysaccharide (HSP), which is filled with collagen fibers and glycoproteins. ECM triggers intracellular signaling pathways through the interaction between its receptors and integrins on ECs, thereby regulating the differentiation, proliferation, and migration of ECs. Therefore, biomimetic-based surface modification is intended to provide a good growth micro-environment for cells. The physiological behavior of ECs was regulated by constructing biomimetic ECM microenvironment or specializing a binding site, so as to achieve the effect of material surface endothelialization.

**Surface design based on biological function intervention and feedback**

After the material is implanted into the blood vessel, the induction of endothelial regeneration in situ on the surface of the material is called in situ endothelialization. In situ endothelialization is mainly aimed at two types of cells: ECs and EPCs. For the former, on the one hand, it promotes the migration and proliferation of cells in the contact area between the material and the blood vessel to the material surface, and on the other hand, it attracts and induces the adhesion of circulating ECs (eECs) in the blood. The latter is a precursor cell derived from bone marrow, distributed in the blood, and has the potential to differentiate into mature ECs. EPCs are specifically captured on the surface of the material and induced to differentiate to achieve surface endothelialization in situ. Therefore, different researchers intervene the biological
function of the two types of cells by functional factors, antibodies and gases on the surface of the implant materials, respectively/jointly. Using specific positive feedback, ECs/EPCs in vivo are induced to proliferate and differentiate on the surface of the material to achieve in situ endothelialization of the material in vivo.

### Functional factor

VEGF is a key factor in angiogenesis, which can promote the migration and proliferation of ECs and the proliferation of EPCs. The surface of the material is loaded with VEGF in the form of physical coating or electrostatic deposition for orderly release in the body, which can significantly improve the in-situ endothelialization ability of the vascular implant material. Min et al. used collagen to construct biomimetic renal vascular 3D stent, and then VEGF is introduced into the surface of the material by grafting heparin on the stent to prepare a vascular implant material with VEGF controlled release ability. Due to the short half-life of VEGF in the blood environment, early burst release can increase the content of VEGF in the environment, so as to achieve rapid endothelialization. Although early burst release can increase the content of VEGF in the environment to achieve rapid endothelialization, studies have shown that high concentrations of VEGF may inhibit the migration of ECs.

The introduction of VEGF on the surface by covalent immobilization can effectively inhibit the endocytosis of VEGF by cells, prolong the action time of VEGF, and control the local VEGF concentration, so as to better promote cell adhesion, proliferation, and directional differentiation on the surface of the material. By depositing dopamine on the surface, VEGF is grafted onto the material by covalent binding of dopamine to VEGF. By the interaction of dopamine-heparin and heparin-VEGF, VEGF was immobilized on the surface of the material to prepare a bio-functional coating with specific capture of EPCs and enhanced ECs activity to achieve in situ endothelialization of the material surface. Since Fc fusion protein can promote the expression of VEGF function at low concentration, VEGF-Fc was synthesized by genetic engineering, and then VEGF was introduced into the material surface by using the high binding force

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**Table 1.** Endothelial cells affinity peptides and their modification methods.

| Peptide | Pathway | Modification method | Finding |
|---------|---------|---------------------|---------|
| RGD     | Promote the adhesion and migration of ECs through integrin recognition. | Covalent grafting<sup>109</sup> Click chemistry<sup>114</sup> Layer-by-layer self-assembly<sup>115</sup> | 1. That the structure and density distribution of RGD peptides on the material surface are closely related to their functions. The cyclic RGD can improve the biocompatibility of the material surface, which may be related to the more similar conformation of cyclic RGD and natural ligands. 2. In addition, the integrin receptor α<sub>IIIb</sub>β<sub>3</sub> corresponding to RGD also exists on platelets, so the surface of the material modified with RGD peptide may also mediate platelet adhesion. Therefore, in the use of RGD modification, in addition to considering the conformation and density of RGD peptide, combined with anticoagulant substances is also a factor to be considered. |
| REDV    | Specifically bind to integrin α<sub>4</sub>β<sub>1</sub> on the ECs. | Covalent grafting<sup>118-120</sup> Thiol-ene click reaction<sup>122</sup> EDC/NHS cross-linking<sup>123</sup> | 1. The C-terminal and N-terminal closure of REDV peptide does not affect the role of REDV, so REDV has a variety of derivatives. 2. In order to achieve better endothelialization in vivo, the combination of REDV peptides with other biological factors is also essential. Such as NO, gene, hyaluronic acid, and VE-cadherin antibody. |
| YIGSR   | Binding to laminin receptors to regulate ECs migration<sup>129</sup> | Covalent grafting<sup>129</sup> Direct solid-state synthesis extension of amino terminal<sup>130</sup> Layer-by-layer self-assembly by electrostatic interaction<sup>131</sup> | The introduction of YIGSR not only promoted the proliferation of ECs, but also promoted the proliferation of smooth muscle cells<sup>130</sup> |
| CAG     | A tripeptide of type IV collagen derived from extracellular matrix<sup>132</sup> | Covalent grafting by dopamine<sup>133</sup> Thiol-ene click chemistry<sup>135</sup> | CAG has strong affinity for ECs and inhibits the adhesion and proliferation of smooth muscle cells<sup>132,134</sup> |
between Fc and hydrophobic surface. VEGF on the material surface enhanced the expression of PI3K and MAPK by activating vascular endothelial growth factor receptor-2 (VEGFR2), and promoted the adhesion and proliferation of ECs, thus achieving rapid vascularization in vascular implant materials.

Because stromal cell-derived factor-1 (SDF-1) and its receptor CXCR4 play an important role in EPC mobilization, migration and homing, they can be used to mediate EPC mobilization and homing of endogenous EPCs to implant materials.

SDF-1 was introduced into the surface of small-diameter implant materials to promote EPCs recruitment, migration, and proliferation of mature endothelial cells and ECs. Shafiq et al. constructed SDF-1 release surface on PCL-collagen implant material, which enhanced the recruitment of surface stem cells, accelerated the surface endothelialization and promoted angiogenesis through SDF-1 release. Liu et al. constructed a novel SDF-1 α/ laminin-loaded nanoparticle coating on the surface of 316L SS to improve the endothelialization ability of the material surface. The modified material can effectively inhibit the adhesion and activation of platelets on the material surface, and induce the migration of ECs and the aggregation of EPCs. Gao et al. increased the free radicals on the surface of expanded polytetrafluoroethylene (ePTFE) by plasma immersion ion implantation, and realized the grafting of heparin, SDF-1 α, and CD47 onto the material surface. Immobilized SDF-1 α could effectively capture EPCs under flow conditions and enhance EPCs activity on the surface of materials.

Other functional factors that affect endothelialization are listed in Table 2.

### Table 2. Other factors that promote endothelialization and their modification methods.

| Base material | Factor | Modification method | Conclusion |
|---------------|--------|---------------------|------------|
| ePTFE         | Fibroblast growth factor (FGF) | Non-covalent binding | Anticoagulation, promoting ECs adhesion and proliferation |
| ePTFE         | Selenocystamine (SeCA) | Covalent binding | Promoted human coronary artery endothelial cell (HCAEC) growth and proliferation while preventing platelet adhesion |
| 316 L stainless steel | H₂S | Layer-by-layer self-assembly | Anti-inflammatory, anticoagulant, and promoting ECs proliferation |
| Nitinol stent | Citric acid | Spray | Targeted promotion of ECs adhesion, migration, and proliferation |
| 316 L stainless steel | Nucleic acid aptamers | Electrostatic effect | Anti-inflammatory, promoting ECs adhesion, proliferation, and migration |
| 316 L stainless steel | Exosomes | | Capture EPCs and promote endothelialization |
| Polyurethane (PU) | Gallic acid | Sandwich coating | Selectively promoting ECs proliferation and inhibiting SMCs growth |
| 316 L stainless steel | Chondroitin sulfate | Covalent binding | Better blood compatibility and endothelialization |
| Glass | Big Endothelin-1 (bigET-1) | Amide bond | Promoting specific binding of ECs |

**Antibody**

EPCs and ECs express some specific markers, such as CD31, VEGFR-2, CD133, and CD34. The surface of the material can increase the adhesion and proliferation of ECs or EPCs by introducing these common or specific antibodies to promote in situ endothelialization of the material.

Anti-CD31 antibodies can specifically induce ECs binding, so that anti-CD31 modified materials can effectively promote the adhesion of ECs on the surface and enhance their activity. Through EDC/NHS cross-linking reaction, antibodies such as CD31 or VEGFR-2 can be grafted onto the surface of the material, specifically capturing circulating EPCs and ECs to promote in situ endothelialization of the material surface. VEGFR-2 is expressed on the surface of ECs, eECs, and EPCs, but its effect on eECs is about 20 times higher than that on EPCs. Therefore, the recombinant anti-VEGFR-2 antibody fragment was grafted onto the aminated surface. In vivo experiments proved that the modified material has the potential to accelerate the surface endothelialization in situ. CD133 and CD34 are proteins specifically expressed by EPCs, but the non-specific binding of CD34 to other cells will interfere with their specific binding to EPCs, so CD133 is a more specific EPCs marker. After sulhydrylation of the material surface, the anti-CD-133 antibody was fixed by disulfide bond formed by sulphydryl and cysteine. After oxidation treatment of anti-CD133 antibody, it can also introduce thiol or amino functionalized...
The modified material surface can effectively capture EPCs and reduce the growth of SMCs. CD34 antibody is the most widely studied antibody in the field of endothelialization. Fixation of CD34 antibody on the surface of biological materials can promote the adhesion of EPCs and the formation of endothelium. CD34 monoclonal antibody was introduced into the material surface by EDC/NHS cross-linking with the surface-encapsulated ECM. Anti-CD34 antibody stimulated the adhesion and proliferation of EPCs on the material surface, and improved the endothelialization ability and long-term patency rate of graft. Anti-CD34 antibodies can also be covalently grafted onto a hydroxyl-terminated ePTFE surface with a lubricant injection layer by silanization, and the introduction of antibody enhanced the ability of the material surface to capture ECs specifically. At present, the CD34 antibody stent (COMBO) has been clinically approved, and the clinical results have shown a relatively good target disease vascular recanalization rate. But there are still certain risks, it may be related to the tendency of the captured EPCs to differentiate into SMCs and precursor inflammatory cells, thereby causing the proliferation of the neointima.

**Table 3. Different ways of NO and their modification methods.**

| Nitric oxide source | Nitric oxide donor/catalyst | Modification method | Finding |
|---------------------|---------------------------|---------------------|---------|
| Exogenous           | S-Nitroso-N-acetyl-DL-penicillamine (SNAP) | Wicking effects | The release rate of NO in this design method usually depends on the load of NONOates or RSNOs. In addition, limited donors cannot achieve long-term release of NO, and the unstable release rate will lead to high concentrations of NO, which is not conducive to the construction of endothelial microenvironment. |
|                     | S-nitrosoglutathione (GSNO) | Blend | |
|                     | Diazeniumdiolates (NONOates) | Covalent bonding | |
| Endogenous          | Copper                     | Nanoparticle | In addition to Cu, there are other metal elements involved in the catalytic release of NO, such as Au. |
|                     |                            | Chelate | |
|                     |                            | Covalent bonding by polydopamine | |
|                     |                            | Layer-by-layer assembly | |
|                     | Organic sulfur             | Covalent bonding thiol-containing cysteine | This design can control the release of NO by adjusting the proportion of each component and the loading sequence. |
|                     |                            | Electrostatic interaction | |
|                     | Selenium compounds         | Covalent bonding selenized cysteamine | |
|                     |                            | Selenocystamine (SeCA) bound to the dopamine framework | |
|                     |                            | Cu-catechol-SeCA framework | |
|                     |                            | Amide coupling chemical covalent reaction | |

**Conclusion**

Table 4 is a summary of different material surface endothelialization designs in recent years, including key design ideas, the advantages of different design strategies, and their disadvantages.

Since interventional surgery has been used to treat cardiovascular disease, vascular grafts have been key to the success of this treatment modality. In previous treatments, either abnormal proliferation after metal stent implantation or late restenosis caused by drug-eluting stents can cause serious complications for long-term treatment of vascular grafts. Tissue engineered blood vessels can cause adverse cardiac events due to insufficient compliance or strength. Therefore, it is generally recognized that most vascular grafts can only “restore” the blood supply function of blood vessels. But to “repair” damaged blood vessels requires the participation of cells, especially endothelial cells.

Endothelialization is a key factor for the long-term effect of implant materials. The delay of endothelialization on the surface of the material is the main cause of restenosis and advanced thrombosis after implantation. When the implant material is implanted, the incomplete endothelialization of

**NO**

As a gas signal molecule, NO is an important factor in maintaining the steady state of vascular microenvironment. NO promotes the growth of ECs while inhibiting platelet activation, leukocyte-endothelial adhesion, and SMCs proliferation, thereby reducing intimal proliferation and inflammation to maintain vascular homeostasis. The research results in recent years are shown in Table 3.
Table 4. The key, advantages, and disadvantages of different surface modification methods.

| Surface design based on material surface science method | Surface design based on bioactive molecules | Surface design based on biological function intervention and feedback |
|--------------------------------------------------------|-------------------------------------------|---------------------------------------------------------------|
| Key                                                   | Advantages                                | Disadvantages                                               |
| Induction of cell behavior by material surfaces        | Simpler design                            | Lack of biologically induced differentiation ability         |
|                                                      | Rapid realization of endothelial cell adhesion and directional migration on material surfaces |                                                      |
|                                                      | Enhance the activity of ECs and promote cell proliferation, so as to achieve rapid cell adhesion and migration on the surface of the material. | In vitro culture takes a long time |
|                                                      |                                          | There are problems such as promoting platelet aggregation, promoting abnormal proliferation of SMCs, and inducing inflammation. |

the material surface at the early stage of implantation will activate the platelets in the blood, trigger the related coagulation mechanism, and form acute thrombosis in the short term. Inflammatory cells infiltrate into the vascular wall, and the growth factors in the blood environment stimulate the excessive proliferation of SMCs to cause vascular stenosis. These factors in turn affect the adhesion and proliferation of ECs on the surface of the material. With the prolongation of implantation time, the delayed endothelialization of the implant surface further accelerates this trend, eventually leading to restenosis and late thrombosis.

Based on this point of view, most researchers have done a lot of research on vascular grafts to achieve the regeneration of endothelial cells on the material surface through different surface modification methods (microstructure, physicochemical property, bionics and biological factors, etc.). These studies based on the interaction of materials with cells have achieved surface endothelialization in vitro or in vivo to varying degrees, but the repair of blood vessels is a complex process, which consists of three stages: In the early stage of implantation, the vascular implant may not provide an ideal platform for endothelial cells to homing, adhere and proliferate due to the coverage of platelets and coagulation proteins; Good cell viability is the basis for the rapid proliferation of implanted mid-stage ECs; In long-term implantation, good histocompatibility is central to maintaining the homeostasis of vascular graft endothelialization.

Endothelialization is the most ideal design idea and final effect of vascular implant materials, it is undeniable that the endothelialization process of vascular implants after implantation is not necessarily or completely around ECs. The complete remodeling of vascular wall structure is affected by many factors, such as the mechanical properties of materials, hemodynamics, plasma protein adhesion, and drug effects. Therefore, it is determined that the endothelialization design strategy should be a holistic consideration of the post-implantation pathological environment rather than surface modification under the influence of a single factor. This means that not only the effectiveness, feasibility, and time-sequence of the modification method need to be considered, but also the impact of the material surface on the entire vascular microenvironment, including cells, blood, and tissues (Figure 3):

Implant material-Blood: After implantation, the material first faces the blood environment. Therefore, vascular graft must have good blood compatibility. This requires vascular implant materials to inhibit the activation of platelets and thrombin on the one hand and avoid acute thrombosis caused by the generation and aggregation of plasma fibrinogen. On the other hand, it is to avoid further local vascular inflammation caused by monocyte chemotactic factors and adhesion molecules. Inflammation will aggravate endothelial dysfunction, which is not conducive to endothelialization of the material surface. Therefore, the material surface endothelialization design through a variety of means, such as surface physicochemical properties change, heparinization, and NO controlled release, which can effectively improve the blood compatibility of the material.

Implant material-Cells: The endothelialization of vascular implant materials is based on ECs. The normal ECs on the surface of the material can not only stabilize the interaction between the material and the blood environment, but also regulate the phenotypic changes of SMCs and inhibit restenosis caused by abnormal proliferation. It is worth noting that although the inhibition of SMCs...
activity has become an important consensus in endothelialization design, it is undeniable that the normal growth of SMCs is also of positive significance for the improvement of vascular structure in vascular remodeling. Therefore, in the process of material surface endothelialization, SMCs should focus on “inhibiting abnormal proliferation” rather than “inhibiting proliferation.”

Implant material-Tissue: Cell-ECM interaction is the basis of cell function regulation. After anticoagulation and promotion of cell adhesion in the early stage of implantation, whether a complete layer of ECs with normal function can be formed on the surface of the material, the key point is whether the material surface can continuously provide an in vivo microenvironment suitable for cell growth. Although ECs will release ECM outside after adhering/migrating to the surface of the material, the pre-built microenvironment of the material through antibodies, peptides, or (continuous and stable release) functional factors are easier for cells to activate in the early stage of adhesion and to rapidly proliferate and stabilize after long-term implantation. It is worth noting that the exposure of extracellular matrix can stimulate the initiation of coagulation pathway in vivo, so when the material surface has good histocompatibility, it should also actively avoid the possible decline in blood compatibility.

Therefore, a reasonable endothelialization design strategy needs to meet the following requirements (Figure 4):
1. In the early stage of implantation, the surface of the material should inhibit coagulation and inflammation, while promoting the migration of ECs to the surface of the material to construct a biomimetic microenvironment of normal blood vessels.
2. In the middle stage of implantation, the proliferation of surface ECs should be enhanced to accelerate surface endothelial regeneration, so as to inhibit the abnormal proliferation of SMCs.
3. Long-term implantation should provide a good microenvironment for cell growth.

How to construct a reasonable and selective surface that can induce the migration and proliferation of vascular endothelial cells will still be an important research direction for the surface endothelialization of vascular implants in the future. Researchers should take into account the temporal interaction between the material and the vascular environment during implantation, and design a modified method for three stages. Therefore, the ideal scheme to achieve rapid endothelialization of the material surface should not only be based on a single cell factor for material design, but also consider the correct construction of vascular microenvironment on the material surface, so as to guide the healthy repair of vascular tissue.

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References
1. Cicha I and Alexiou C. Cardiovascular applications of magnetic particles. J Magn Magn Mater 2021; 518: 167428.
2. Lacy M, Atzler D, Liu R, de Winther M, Weber C and Lutgens E. Interactions between dyslipidemia and the immune system and their relevance as putative therapeutic targets in atherosclerosis. Pharmacol Ther 2019; 193: 50–62.
3. Libby P, Loscalzo J, Ridker PM, et al. Inflammation, immunity, and infection in atherothrombosis JACC review topic of the week. J Am Coll Cardiol 2018; 72: 2071–2081.
4. Jang SR, Kim JI, Park CH and Kim CS. Development of Y-shaped small diameter artificial blood vessel with controlled topography via a modified electrospinning method. Mater Lett 2020; 264: 127113.
5. Zhang Y, Liu Y, Jiang Z, et al. Poly(glycerol sebacate)/silk fibroin small-diameter artificial blood vessels with good elasticity and compliance. Smart Mater Med 2021; 2: 74–86.
6. Kuang H, Wang Y, Shi Y, et al. Construction and performance evaluation of Hep/silk-PLCL composite nanofiber small-caliber artificial blood vessel graft. Biomaterials 2020; 259: 120288.
7. Jiang W-C, Hsu W-Y, Ao-leong W-S, Wang CY, Wang J and Yet SF. A novel engineered vascular construct of stem cell-laden 3D-printed PGSA scaffold enhances tissue revascularization. Biofabrication 2021; 13: 045004.
8. Manjua AC, Cabral JMS, Portugal CAM and Ferreira FC. Magnetic stimulation of the angiogenic potential of mesenchymal stromal cells in vascular tissue engineering. Sci Technol Adv Mater 2021; 22: 461–480.
9. Wang J-N, Kan C-D, Lin S-H, Chang KC, Taso S and Wong TW. Potential of autologous progenitor cells and decellularized porcine artery matrix in construction of tissue-engineered vascular grafts. Organogenesis 2021; 17: 72–84.
10. Ahuja R, Kumari N, Srivastava A, et al. Biocompatibility analysis of PLA based candidate materials for cardiovascular stents in a rat subcutaneous implant model. Acta Histochem 2020; 122: 151615.
11. Vishnu J, Calin M, Pilz S, et al. Superhydrophobic nanostructured surfaces of beta Ti 29Nb alloy for cardiovascular stent applications. Surf Coat Technol 2020; 396: 125965.
12. Wang J, Qian H-L, Chen S-Y, et al. miR-22 eluting cardiovascular stent based on a self-healable spongy coating inhibits in-stent restenosis. Bioact Mater 2021; 6: 4686–4696.
13. Yue R, Niu J, Li Y, et al. In vitro cytocompatibility, hemocompatibility and antibacterial properties of biodegradable Zn-Cu-Fe alloys for cardiovascular stents applications. Mater Sci Eng C 2020; 113: 111007.
14. Caracciolo PC, Diaz-Rodriguez P, Ardao I, et al. Evaluation of human umbilical vein endothelial cells growth onto heparin-modified electrospun vascular grafts. Int J Biol Macromol 2021; 179: 567–575.
15. Yan S, Xu Y, Lin Y-J, et al. Ethanol-lubricated expanded-polytetrafluoroethylene vascular grafts loaded with eggshell membrane extract and hepatic for rapid endothelialization and anticoagulation. Appl Surf Sci 2020; 511: 145565.
16. Zhao Y, Shirasu T, Yodsamit N, et al. Biomimetic, ROS-detoxable nanoclusters-a multimodal nanoplatfor for anti-restenotic therapy. J Control Release 2021; 338: 295–306.
17. Park S, Lee H, Kim H-E, Jung HD and Jang TS. Bifunctional detonable nanoclusters-a multimodal nanoplatform for anti-restenotic therapy. J Control Release 2021; 396: 125965.
18. Wang J, Qian H-L, Chen S-Y, et al. miR-22 eluting cardiovascular stent based on a self-healable spongy coating inhibits in-stent restenosis. Bioact Mater 2021; 6: 4686–4696.
19. Zhang Z-Q, Yang Y-X, Li JA, Zeng RC and Guan SK. Advances in coatings on magnesium alloys for cardiovascular stents - a review. Bioact Mater 2021; 6: 4729–4757.
20. Repsold L and Joubert AM. Platelet function, role in thrombosis, inflammation, and consequences in chronic myelo-proliferative disorders. *Cells* 2021; 10: 15.

21. Conte M, Petraglia L, Poggio P, et al. Inflammation and cardiovascular diseases in the elderly: the role of epicardial adipose tissue. *Front Med* 2022; 9: 844266.

22. Li JJ, Fang C-H, Jiang H, Huang CX, Hui RT and Chen MZ. Frequency and time course of reocclusion and restenosis in coronary artery occlusions after balloon angioplasty versus wikit stent implantation: results from the Mayo-Japan investigation for Chronic Total Occlusion (MAJIC) trial. *Am Heart J* 2004; 147: E9.

23. Geary RL, Kohler TR, Vergel S, Kirkman TR and Clowes AW. Time course of flow-induced smooth muscle cell proliferation and intimal thickening in endothelialized baboon vascular grafts. *Circ Res* 1994; 74: 14–23.

24. Hanke H, Strohschneider T, Oberhoff M, Betz E and Karsch KR. Time course of smooth muscle cell proliferation in the intima and media of arteries following experimental angioplasty. *Circ Res* 1990; 67: 651–659.

25. Brandtner AK, Lehner GF, Pircher A, Feistritzer C and Joamnidis M. Differential procoagulatory response of micro-vascular, arterial and venous endothelial cells upon inflammation in vitro. *Thromb Res* 2021; 205: 70–80.

26. Vanhoutte PM, Shimokawa H, Feletou M and Tang EH. Endothelial dysfunction and vascular disease - a 30th anniversary update. *Acta Physiol* 2017; 219: 22–96.

27. Ammann KR and Slepian MJ. Vascular endothelial and smooth muscle cell galvanotactic response and differential migratory behavior. *Exp Cell Res* 2021; 399: 112447.

28. Zhuang Y, Zhang C, Cheng M, et al. Challenges and strategies for in situ endothelialization and long-term lumen patency of vascular grafts. *Bioact Mater* 2021; 6: 1791–1809.

29. Li P, Jin D, Dou J, et al. Nitric oxide-releasing poly(e-caprolactone)/S-nitrosylated keratin biocomposite scaffolds for potential small-diameter vascular grafts. *Int J Biol Macromol* 2021; 189: 516–527.

30. Russell-Puleri S, dela Paz NG, Adams D, et al. Fluid shear stress induces upregulation of COX-2 and PGI(2) release in endothelial cells via a pathway involving PECAM-1, PI3K, FAK, and p38. 2017; 312: H485–H500.

31. Motawei SM, Sudha T, Yalcin M, Godugu K and Mousa SA. Lead-induced endothelial cell dysfunction: protective effect of sulfated non-anticoagulant low molecular weight heparin. *Toxicol Environ Health Sci* 2021; 13: 123–131.

32. Ashraf JV and Al Haj Zen A. Role of vascular smooth muscle cell phenotype switching in arteriogenesis. *Int J Mol Sci* 2021; 22: 10585.

33. Tsukada J, Wolf F, Vogt F, et al. Development of in vitro endothelialized drug-eluting stent using human peripheral blood-derived endothelial progenitor cells. *J Tissue Eng Regen Med* 2020; 14: 1415–1427.

34. Chang K-B, Shen C-C, Hsu S-H, et al. Functionalized collagen-silver nanocomposites for evaluation of the biocompatibility and vascular differentiation capacity of mesenchymal stem cells. *Colloids Surf A Physicochem Eng Asp* 2021; 624: 126814.

35. Sun Y, Zhang B and Xia L. Effect of low wall shear stress on the morphology of endothelial cells and its evaluation indicators. *Comput Methods Programs Biomed* 2021; 208: 106082.

36. Nguyen DT, Smith AF and Jiménez JM. Stent strut streaming and thickness reduction promote endothelialization. *J Roy Soc Interface* 2021; 18: 20210023.

37. Liu Y, Zhang J, Li S and Xia H. Photopolymerization strategy for the preparation of small-diameter artificial blood vessels with micro-nano structures on the inner wall. *Biomed Opt Express* 2021; 12: 5844–5854.

38. Anzai H, Watanabe T, Han X, et al. Endothelial cell distributions and migration under conditions of flow shear stress around a stent wire. *Technol Health Care* 2020; 28: 345–354.

39. Han C, Luo X, Zou D, et al. Nature-inspired extracellular matrix coating produced by micro-patterned smooth muscle and endothelial cells endows cardiovascular materials with better biocompatibility. *Biomater Sci* 2019; 7: 2686–2701.

40. Schieber R, Lasserre F, Hans M, et al. Direct laser interference patterning of CoCr alloy surfaces to control endothelial cell and platelet response for cardiovascular applications. *Adv Healthc Mater* 2017; 6: 1700327.

41. Cortella LRX, Cestari IA, Guenther D, Lasagni AF and Cestari IN. Endothelial cell responses to castor oil-based polyurethane substrates functionalized by direct laser ablation. *Biomater Sci* 2017; 12: 065010.

42. Kang I-G, Park C-I, Seong Y-J, Lee H, Kim HE and Han CM. Bioactive and mechanically stable hydroxyapatite patterning for rapid endothelialization of artificial vascular graft. *Mater Sci Eng C* 2020; 106: 110287.

43. Purnama A, Furlan V, Dassi D, et al. Laser surface texturing of SS316L for enhanced adhesion of HUVECs. *Surf Eng* 2020; 36: 1240–1249.

44. Zhao Y, Sun Y, Lan W, et al. Self-assembled nanosheets on NiTi alloy facilitate endothelial cell function and manipulate macrophage immune response. *J Mater Sci Technol* 2021; 78: 110–120.

45. Zhou K, Li Y, Zhang L, et al. Nano-micrometer surface roughness gradients reveal topographical influences on differentiating responses of vascular cells on biodegradable magnesium. *Bioact Mater* 2021; 6: 262–272.

46. Chan Y, Kealey CP, Levi DS, et al. An in vivo pilot study of a microporous thin film nitinol-covered stent to assess the effect of porosity and pore geometry on device interaction with the vessel wall. *J Biomater Appl* 2017; 31: 1196–1202.

47. Wu M, Panduranga MK and Carman GP. Proliferation of human aortic endothelial cells on nitinol thin films with varying hole sizes. *Biomed Opt Express* 2018; 20: 25.

48. Bitö K, Hasebe T, Maegawa S, et al. Micropatterning of a 2-methacryloyloxyethyl phosphorylcholine polymer surface by hydrogenated amorphous carbon thin films for endothelialization and antithrombogenicity. *Acta Biomater* 2019; 87: 187–196.

49. Wu X, Moimas S, Hopf R, et al. A free-form patterning method enabling endothelialization under dynamic flow. *Biomaterials* 2021; 273: 120816.
51. Cherian AM, Joseph J, Nair MB, Nair SV, Maniyal V and Menon D. Successful reduction of neointimal hyperplasia on stainless steel coronary stents by titania nanotexturing. *ACS Omega* 2020; 5: 17582–17591.

52. Pan C, Hu Y, Gong Z, et al. Improved blood compatibility and endothelialization of titanium oxide nanotube arrays on titanium surface by zinc doping. *ACS Biomater Sci Eng* 2020; 6: 2072–2083.

53. Zhang Y, Wang X, Zhang Y, et al. Endothelial cell migration regulated by surface topography of poly(epsilon-caprolactone) nanofibers. *ACS Biomater Sci Eng* 2021; 7: 4959–4970.

54. Abagnale G, Sechi A, Steger M, et al. Surface topography guides morphology and spatial patterning of induced pluripotent stem cell colonies. *Stem Cell Reports* 2017; 9: 654–666.

55. Chen X, Yao Q, Liu S and Hu Q. An integrated strategy for designing and fabricating triple-layer vascular graft with oriented microgrooves to promote endothelialization. *J Biomed Mater Res A* 2021; 36: 297–310.

56. Wu P, Wang L, Li W, et al. Construction of vascular graft with circumferentially oriented microchannels for improving artery regeneration. *Biomaterials* 2020; 242: 119922.

57. Luo X, Yang P, Zhao A, et al. The self-organized differentiation from MSCs into SMCs with manipulated micro/nano two-scale arrays on TiO2 surfaces for biomimetic construction of vascular endothelial substratum. *Mater Sci Eng C* 2020; 116: 111179.

58. Liu T, Liu S, Zhang K, Chen J and Huang N. Endothelialization of implanted cardiovascular biomaterial surfaces: the development from in vitro to in vivo. *J Biomed Mater Res A* 2014; 102: 3754–3772.

59. Le ANM, Tran NMP, Phan TB, Tran PA, Tran LD and Nguyen TH. Poloxamer additive as luminal surface modification to modulate wettability and bioactivities of small-diameter polyurethane/polyurethane electrospun hollow tube for vascular prosthetic applications. *Mater Today Commun* 2021; 26: 101771.

60. Tateshima S, Kaneko N, Yamada M, Duckwiler G, Vinuela F and Ogawa T. Increased affinity of endothelial cells to NiTi using ultraviolet irradiation: an *in vitro* study. *J Biomed Mater Res A* 2018; 106: 1034–1038.

61. Tan X, Gao P, Li Y, et al. Poly-dopamine, poly-levodopa, and poly-norepinephrine coatings: comparison of physicochemical and biological properties with focus on the application for blood-contacting devices. *Bioact Mater* 2021; 6: 285–296.

62. Shim JW, Bae I-H, Park DS, et al. Hydrophilic surface modification of coronary stent using an atmospheric pressure plasma jet for endothelialization. *J Biomater Appl* 2018; 32: 1083–1089.

63. Krüger-Genre A, Dietze S, Yan W, et al. Endothelial cell migration, adhesion and proliferation on different polymeric substrates. *Clin Hemorheol Microcirc* 2018; 70: 511–529.

64. Chang H, Zhang H, Hu M, et al. Stiffness of polyelectrolyte multilayer film influences endothelial function of endothelial cell monolayer. *Colloids Surf B Biointerfaces* 2017; 149: 379–387.

65. Zhang L, Wei F, Bai Q, et al. Oscillating magnetic field regulates cell adherence and endothelialization based on magnetic nanoparticle-modified bacterial cellulose. *ACS Appl Mater Interfaces* 2020; 12: 52467–52478.

66. Zhang H, Chang H, Wang LM, et al. Effect of polyelectrolyte film stiffness on endothelial cells during endothelial-to-mesenchymal transition. *Biomacromolecules* 2015; 16: 3584–3593.

67. Chen J, Wang S, Wu Z, Wei Z, Zhang W and Li W. Anti-CD34-Grafted magnetic nanoparticles promote endothelial progenitor cell adhesion on an iron stent for rapid endothelialization. *ACS Omega* 2019; 4: 19469–19477.

68. Ye L, Takagi T, Tu C, Hagiwara A, Geng X and Feng Z. The performance of heparin modified poly(ε-caprolactone) small diameter tissue engineering vascular graft in canine—a long-term pilot experiment in vivo. *J Biomed Mater Res A* 2021; 109: 2493–2505.

69. Yao Y, Zaw AM, Anderson DEJ, Hinds MT and Yim EKF. Fucoidan functionalization on poly(vinyl alcohol) hydrogels for improved endothelialization and hemocompatibility. *Biomaterials* 2020; 249: 120011.

70. Liu Y, Stadler FJ, Fang J and Galluzzi M. Hyaluronic acid-functionalized poly-lactic acid (PLA) microfibers regulate vascular endothelial cell proliferation and phenotypic shape expression. *Colloids Surf B Biointerfaces* 2021; 206: 111970.

71. Yu C, Guan G, Glas S, Wang L, Li Z and Turng LS. A biomimetic basement membrane consisted of hybrid aligned nanofibers and microfibers with immobilized collagen IV and laminin for rapid endothelialization. *Bio Des Manuf* 2021; 4: 171–189.

72. Liu Y, Munisso MC, Mahara A, Kambe Y and Yamakoa T. Anti-platelet adhesion and in situ capture of circulating endothelial progenitor cells on ePTFE surface modified with poly(2-methacryloyloxyethyl phosphorylcholine) (PMPC) and hemocompatible peptide 1 (HCP-1). *Colloids Surf B Biointerfaces* 2020; 193: 111113.

73. Xing Y, Gu Y, Guo L, et al. Gelatin coating promotes in situ endothelialization of electrospun polycaprolactone vascular grafts. *J Biomed Sci Polym Ed* 2021; 32: 1161–1181.

74. Chen J, Wang H, Gao C, et al. Tetramethylpyrazine alleviates LPS-induced inflammatory injury in HUVECs by inhibiting Rho/ROCK pathway. *Biochem Biophys Res Commun* 2019; 514: 329–335.

75. Liu W, Li Y, Wu Z, et al. Heparin alleviates LPS-induced endothelial injury by regulating the TLR4/MyD88 signaling pathway. *Exp Ther Med* 2021; 22: 1397.

76. Zhu T, Gu H, Zhang H, et al. Covalent grafting of PEG and heparin improves biological performance of electrospun vascular grafts for carotid artery replacement. *Acta Biomater* 2021; 119: 211–224.

77. Wang D, Wang X, Li X, Jiang L, Chang Z and Li Q. Biologically responsive, long-term release nanocoating on an electrospun scaffold for vascular endothelialization and heparin in vascular tissue engineering. *Microvasc Res* 2020; 131: 104027.
improvement of blood compatibility and biocompatibility. *Int J Biol Macromol* 2019; 127: 159–168.

80. Song H, Wu T, Yang X, et al. Surface modification with NGF-loaded chitosan/heparin nanoparticles for improving biocompatibility of cardiovascular stent. *Stem Cells Int* 2021; 2021: 9941143.

81. Liu T, Li X, Liu S, et al. Surface modification with micropatterned heparin/poly-L-lysine nanoparticles to direct platelet and endothelial cell behavior. *J Biomater Tissue Eng* 2017; 7: 962–968.

82. Yu C, Yang H, Wang L, et al. Surface modification of polytetrafluoroethylene (PTFE) with a heparin-immobilized extracellular matrix (ECM) coating for small-diameter vascular grafts applications. *Mater Sci Eng C* 2021; 128: 112301.

83. Bae I-H, Jeong MH, Kim JH, et al. The control of drug release and vascular endothelialization after hyaluronic acid-coated paclitaxel multi-layer coating stent implantation in porcine coronary restenosis model. *Korean Circ J* 2017; 47(1): 123–131.

84. Li M, Zhang X, Jia W, et al. Improving in vitro biocompatibility on biomimetic mineralized collagen bone materials modified with hyaluronic acid oligosaccharide. *Mater Sci Eng C* 2019; 104: 110008.

85. Luo X, Han C, Yang P, et al. The co-deposition coating of collagen IV and laminin on hyaluronic acid pattern for better biocompatibility on cardiovascular biomaterials. *Colloids Surf B Biointerfaces* 2020; 196: 111307.

86. Busch R, Strohbach A, Rethfeldt S, et al. New stent surface materials: the impact of polymer-dependent interactions of human endothelial cells, smooth muscle cells, and platelets. *Acta Biomater* 2014; 10: 688–700.

87. Dimitrievska S, Wang J, Lin T, et al. Glycolalcalyx-like hydrogel coatings for small diameter vascular grafts. *Adv Funct Mater* 2020; 30: 1908963.

88. Li JA, Chen L, Zhang XQ and Guan SK. Enhancing biocompatibility and corrosion resistance of biodegradable Mg-Zn-Y-Nd alloy by preparing PDA/HA coating for potential application of cardiovascular biomaterials. *Mater Sci Eng C* 2020; 109: 110607.

89. Qin K, Wang F, Simpson RML, et al. Hyaluronan promotes the regeneration of vascular smooth muscle with potent contractile function in rapidly biodegradable vascular grafts. *Biomaterials* 2020; 257: 120226.

90. Li J, Wu F, Zhang K, et al. Controlling molecular weight of hyaluronic acid conjugated on amine-rich surface: toward better multifunctional biomaterials for cardiovascular implants. *ACS Appl Mater Interfaces* 2017; 9: 30343–30358.

91. Jiang T, Xie Z, Wu F, et al. Hyaluronic acid nanoparticle composite films confer favorable time-dependent biofunctions for vascular wound healing. *ACS Biomater Sci Eng* 2019; 5: 1833–1848.

92. Kang L, Jia W, Li M, et al. Hyaluronic acid oligosaccharide-modified collagen nanofibers as vascular tissue-engineered scaffold for promoting endothelial cell proliferation. *Carbohydr Polym* 2019; 223: 115106.

93. Wang C, Hao H, Wang J, et al. High-throughput hyaluronic acid hydrogel arrays for cell selective adhesion screening. *J Mater Chem B* 2021; 9: 4024–4030.

94. Jia W, Li M, Kang L, Gu G, Guo Z and Chen Z. Fabrication and comprehensive characterization of biomimetic extracellular matrix electrospun scaffold for vascular tissue engineering applications. *J Mater Sci* 2019; 54: 10871–10883.

95. Zhang K, Shi Z, Zhou J, et al. Potential application of an injectable hydrogel scaffold loaded with mesenchymal stem cells for treating traumatic brain injury. *J Mater Chem B* 2018; 6: 2982–2992.

96. Daum R, Visser D, Wild C, et al. Fibronectin adsorption on electrospun synthetic vascular grafts attracts endothelial progenitor cells and promotes endothelialization in dynamic in vitro culture. *Cells* 2020; 9: 778.

97. Wacker M, Riedel J, Walles H, et al. Comparative evaluation on impacts of fibronectin, Heparin-Chitosan, and albumin coating of bacterial nanocellulose small-diameter vascular grafts on endothelialization in vitro. *Nanomater* 2021; 11: 952.

98. Daum R, Mrsic I, Hutterer J, et al. Fibronectin adsorption on oxygen plasma-treated polyurethane surfaces modulates endothelial cell response. *J Mater Chem B* 2021; 9: 1647–1660.

99. Montaño-Machado V, Noël C, Chevallier P, et al. Interaction of phosphorylcholine with fibroconnectin coatings: surface characterization and biological performances. *Appl Surf Sci* 2017; 396: 1613–1622.

100. Jin Z, Yan X, Liu G and Lai M. Fibronectin modified TiO2 nanotubes modulate endothelial cell behavior. *J Biomol Appl* 2018; 33: 44–51.

101. Liu S, Hu Y, Tao R, et al. Immobilization of fibroconnectin-loaded polyelectrolyte nanoparticles on cardiovascular material surface to improve the biocompatibility. *Biomed Res Int* 2019; 2019: 5478369.

102. Li P, Li X, Cai W, et al. Phospholipid-based multifunctional coating via layer-by-layer self-assembly for biomedical applications. *Mater Sci Eng C* 2020; 116: 112377.

103. Bedair TM, Min JJ, Park W, Park BJ, Joung YK and Han DK. Covalent immobilization of fibroblast-derived matrix on metallic stent for expeditious re-endothelialization. *J Ind Eng Chem* 2019; 70: 385–393.

104. Yang L, Wu H, Lu L, et al. A tailored extracellular matrix (ECM) - mimetic coating for cardiovascular stents by step-wise assembly of hyaluronic acid and recombinant human type III collagen. *Biomaterials* 2021; 276: 121055.

105. Ardila DC, Liou J-J, Maestas D, et al. Surface modification of electrospun scaffolds for endothelialization of tissue-engineered vascular grafts using human cord blood-derived endothelial cells. *J Clin Med* 2019; 8: 185.

106. Yu C, Xing M, Sun S, Guan G and Wang L. In vitro evaluation of vascular endothelial cell behaviors on bio-mimetic vascular basement membranes. *Colloids Surf B Biointerfaces* 2019; 182: 110381.

107. Castellanos MI, Mas-Moruno C, Grau A, et al. Functionalization of CoCr surfaces with cell adhesive peptides to promote HUVECs adhesion and proliferation. *Appl Surf Sci* 2017; 393: 82–92.

108. Wetzel R, Hauser S, Lin W, et al. Screening arrays of laminin peptides on modified cellulose for promotion of adhesion of primary endothelial and neural precursor cells. *Adv Biol Sci* 2021; 5: 1900303.

109. Zhu T, Yu K, Bhutto MA, et al. Synthesis of RGD-peptide modified poly (ester-urethane) urea electrospun nanofibers
as a potential application for vascular tissue engineering. *Chem Eng J* 2017; 315: 177–190.

110. Antonova LV, Silnikov VN, Sevostyanova VV, et al. Biocompatibility of small-diameter vascular grafts in different modes of RGD modification. *Polymers* 2019; 11: E174.

111. Hao D, Fan Y, Xiao W, et al. Rapid endothelialization of small diameter vascular grafts by a bioactive integrin-binding ligand specifically targeting endothelial progenitor cells and endothelial cells. *Acta Biomater* 2020; 108: 178–193.

112. Karimi F, McKenzie TG, O’Connor AJ, Qiao GG and Heath DE. Nano-scale clustering of integrin-binding ligands regulates endothelial cell adhesion, migration, and endothelialization rate: novel materials for small diameter vascular graft applications. *J Mater Chem B* 2017; 5: 5942–5953.

113. Xiao Y and Truskey GA. Effect of receptor-ligand affinity on the strength of endothelial cell adhesion. *Biophys J* 1996; 71: 2869–2884.

114. Sivkova R, Táborská J, Reparaz A, et al. Surface design of antifouling polymer constructs bearing biofunctional peptides for tissue regeneration applications. *Int J Mol Sci* 2020; 21: 6800.

115. Kout F, Liu C, Wang L, Yasin A, Li J and Guan G. Surface modification of citric Acid/RGD multilayers on Mg-Zn-Y-Nd Alloy via Layer-by-Layer Self-assembly for promoting surface bio-compatibility. *Adv Mater Interfaces* 2021; 8: 2002241.

116. Mi HY, Jing X, Thomson JA and Turng LS. Promoting endothelial cell affinity and anti-thrombogenicity of polytetrafluoroethylene (PTFE) by mussel-inspired modification and RGD/heparin grafting. *J Mater Chem B* 2018; 6: 3475–3485.

117. Ohya Y, Nishimura K, Sumida H, et al. Cellular attachment behavior on biodegradable polymer surface immobilizing endothelial cell-specific peptide. *J Biomater Sci Polym Ed* 2020; 31: 1475–1488.

118. Wen C, Zhang J, Li Y, et al. A zwitterionic hydrogel coated titanium surface with high-efficiency endothelial cell selectivity for rapid re-endothelialization. *Biomater Sci* 2020; 8: 5441–5451.

119. Wu Y, Yu C, Xing M, Wang L and Guan G. Surface modification of polyvinyl alcohol (PVA)/polyacrylamide (paam) hydrogels with polydopamine and REDV for improved applicability. *J Biomed Mater Res B Appl Biomater* 2020; 108(1): 117–127.

120. Chen L, Li J, Wang S, et al. Surface modification of the biodegradable cardiovascular stent material Mg-Zn-Y-Nd alloy via conjugating REDV peptide for better endothelialization. *J Mater Res* 2018; 33: 4123–4133.

121. Veiseh M, Veiseh O, Martin MC, Asphahani F and Zhang M. Short peptides enhance single cell adhesion and viability on microarrays. *Langmuir* 2007; 23: 4472–4479.

122. Zhou F, Jia X, Yang Y, et al. Peptide-modified PELCL electrospun membranes for regulation of vascular endothelial cells. *Mater Sci Eng C* 2016; 68: 623–631.

123. Peng X, Wang X, Cheng C, et al. Bioinspired, artificial, small-diameter vascular grafts with selective and rapid endothelialization based on an amniotic membrane-derived hydrogel. *ACS Biomater Sci Eng* 2020; 6: 1603–1613.

124. Li X, Liu J, Yang T, et al. Mussel-inspired “built-up” surface chemistry for combining nitric oxide catalytic and vascular cell selective properties. *Biomaterials* 2020; 241: 119904.

125. Wen M, Yan H, Shi X, et al. Modulation of vascular endothelial cells under shear stress on electrospun membranes containing REDV and microRNA-126. *Int J Polym Mater Polym Biomater* 2021; 70: 1090–1099.

126. Zhou J, Wang M, Wei T, et al. Endothelial cell-mediated gene delivery for in situ accelerated endothelialization of a vascular graft. *ACS Appl Mater Interfaces* 2013; 13: 16097–16105.

127. Luo Y, Huang S and Ma L. Zwitterionic hydrogel-coated heart valves with improved endothelialization and anti-calci-fication properties. *Mater Sci Eng C* 2021; 128: 112329.

128. Liu X, Yu K, Cheng S, et al. Ulvan mediated VE cadherin antibody and REDV peptide co-modification to improve endothelialization potential of bioprosthetic heart valves. *Mater Sci Eng C* 2012; 112337.

129. Tang D, Chen S, Hou D, et al. Regulation of macrophage polarization and promotion of endothelialization by NO generating and PEG-YIGSR modified vascular graft. *Mater Sci Eng C* 2018; 84: 1–11.

130. Onak Pugat G, Gökmen O, Çevik ZBY and Karaman O. Role of functionalized self-assembled peptide hydrogels in vitro vasculogenesis. *Soft Matter* 2021; 17: 6616–6626.

131. Zeng Z, Hu C, Liang Q, Tang L, Cheng D and Ruan C. Coaxial-printed small-diameter polyelectrolyte-based tubes with an electrostatic self-assembly of heparin and YIGSR peptide for antithrombogenicity and endothelialization. *Bioact Mater* 2021; 6: 1628–1638.

132. Kanie K, Narita Y, Zhao Y, et al. Collagen type IV-specific tripeptides for selective adhesion of endothelial and smooth muscle cells. *Biotecnol Bioeng* 2012; 109: 1808–1816.

133. Zhao J, Bai L, Ren XK, et al. Co-immobilization of ACH(11) antithrombotic peptide and CAG cell-adhesive peptide onto vascular grafts for improved hemocompatibility and endothelialization. *Acta Biomater* 2019; 97: 344–359.

134. Kuwabara F, Narita Y, Yamawaki-Ogata A, et al. Novel Small-caliber vascular grafts with trimeric peptide for acceleration of endothelialization. *Ann Thorac Surg* 2012; 93: 156–163.

135. Xie J, Shen K, Zheng H, Yao Y, Chen Y and Gao C. Grafting of CAG peptides and (polyethylene glycol) on unsaturated polyurethane films to promote selective adhesion and migration of urethral epithelial cells. *J Mater Chem B* 2021; 9: 6201–6211.

136. Wang X, Fang F, Ni Y, et al. The combined contribution of vascular endothelial cell migration and adhesion to stent re-endothelialization. *Front Cell Dev Biol* 2021; 9: 641382.

137. Mahara A, Kitagawa K, Otaka A, Nakaoki T, Ishihara K and Yamaoka T. Impact of REDV peptide density and its linker structure on the capture, movement, and adhesion of flowing endothelial progenitor cells in microfluidic devices. *Mater Sci Eng C* 2021; 129: 112381.

138. Nuttita K, Samandari M, Endo Y, et al. In vivo printing of growth factor-eluting adhesive scaffolds improves wound healing. *Bioact Mater* 2022; 8: 296–308.

139. Huang C-C, Tseng T-T, Liu S-C, et al. S1P increases VEGF production in osteoblasts and facilitates endothelial progenitor cell angiogenesis by inhibiting miR-16-5p expression via the c-Src/FAK signaling pathway in rheumatoid arthritis. *Cells* 2021; 10: 2168.

140. Wang Y, Wu T, Zhang J, Feng Z, Yin M and Mo X. A bilayer vascular scaffold with spatially controlled release
of growth factors to enhance in situ rapid endothelialization and smooth muscle regeneration. *Mater Des* 2021; 204: 109649.

141. Wang D, Wang X, Zhang Z, et al. Programmed release of multimodal, cross-linked vascular endothelial growth factor and heparin layers on electrospun polycaprolactone vascular grafts. *ACS Appl Mater Interfaces* 2019; 11: 32533–32542.

142. Min S, Cleveland D, Ko IK, et al. Accelerating neovascularization and kidney tissue formation with a 3D vascular scaffold capturing native vascular structure. *Acta Biomater* 2021; 124: 233–243.

143. Tan J, Cui Y, Zeng Z, et al. Heparin/poly-l-lysine nanoplatform with growth factor delivery for surface modification of cardiovascular stents: the influence of vascular endothelial growth factor loading. *J Biomed Mater Res A* 2020; 108: 1295–1304.

144. Lee SJ, Kim ME, Nah H, et al. Vascular endothelial growth factor immobilized on mussel-inspired three-dimensional bilayered scaffold for artificial vascular graft application: in vitro and in vivo evaluations. *J Colloid Interface Sci* 2019; 537: 333–344.

145. Sun A, Huang X, Jiao Y, Wang X and Wen J. Construction of biological factor-coated stent and its effect on promoting endothelialization. *Mater Sci Eng C* 2021; 122: 111943.

146. Duan Y, Yu S, Xu P, et al. Co-immobilization of CD133 antibodies, vascular endothelial growth factors, and REDV peptide promotes capture, proliferation, and differentiation of endothelial progenitor cells. *Acta Biomater* 2019; 96: 137–148.

147. Smith RJ Jr, Yi T, Nasiri B, Breuer CK and Andreadis ST. Implantation of VEGF-functionalized cell-free vascular grafts: regenerative and immunological response. *FASEB J* 2019; 33: 5089–5100.

148. Xu K, Zhong X, Cie J, et al. Enhanced vascularization of PCL porous scaffolds through VEGF-Fc modification. *J Mater Chem B* 2018; 6: 4474–4485.

149. Wang X, Jiang H, Guo L, et al. SDF-1 secreted by mesenchymal stem cells promotes the migration of endothelial progenitor cells via CXCR4/P13K/AKT pathway. *J Mol Histol* 2021; 52: 1155–1164.

150. Shafiq M, Kong D and Kim SH. SDF-1α peptide tethered polyester facilitates tissue repair by endogenous cell mobilization and recruitment. *J Biomed Mater Res A* 2017; 105: 2670–2684.

151. Sugimura Y, Chekhoeva A, Oyama K, et al. Controlled autologous recellularization and inhibited degeneration of decellularized vascular implants by side-specific coating with stromal cell-derived factor 1α and fibronectin. *Biomed Mater* 2020; 15: 035013.

152. Wang W, Liu D, Li D, et al. Nanofibrous vascular scaffold prepared from miscible polymer blend with heparin/stromal cell-derived factor-1 alpha for enhancing antiangiogenesis and endothelialization. *Colloids Surf B Biointerfaces* 2019; 181: 963–972.

153. Shafiq M, Zhang Q, Zhi D, et al. In situ blood vessel regeneration using SP (Substance P) and SDF (Stromal cell-derived factor)-1α peptide eluting vascular grafts. *Arterioscler Thromb Vasc Biol* 2018; 38: E117–E134.

154. Liu T, Wang X, Tang X, et al. Surface Modification with ECM-Inspired SDF-1α/Laminin-Loaded nanocoating for vascular wound healing. *ACS Appl Mater Interfaces* 2017; 9: 30373–30386.

155. Gao A, Hang R, Li W, et al. Linker-free covalent immobilization of heparin, SDF-1α, and CD47 on PTFE surface for antithrombogenicity, endothelialization and anti-inflammation. *Biomaterials* 2017; 140: 201–211.

156. Wang D, Xu Y, Wang L, et al. Long-term nitric oxide release for rapid endothelialization in expanded polytetrafluoroethylene small-diameter artificial blood vessel grafts. *Appl Surf Sci* 2020; 507: 145028.

157. Lu B, Han X, Zhao A, et al. Intelligent H2S release coating for regulating vascular remodeling. *Bioact Mater* 2021; 6: 1040–1050.

158. Ceresnakova M, Murray D, McGourtY KD, et al. Citric acid functionalized nitinol stent surface promotes endothelial cell healing. *J Biomed Mater Res A* 2021; 109: 1549–1559.

159. Deng J, Yuan S, Li X, et al. Heparin/DNA aptamer co-assembled multifunctional catecholamine coating for EPC capture and improved hemocompatibility of vascular devices. *Mater Sci Eng C* 2017; 79: 305–314.

160. Hou YC, Li JA, Zhu SJ, et al. Tailoring of cardiovascular stent material surface by immobilizing exosomes for better pro-endothelialization function. *Colloids Surf B Biointerfaces* 2020; 189: 110831.

161. Das A, Ahmad Sheikh P and Kumar A. A coaxially structured trilayered gallic acid-based antioxidant vascular graft for treating coronary artery disease. *Eur Polym J* 2021; 143: 112033.

162. Zou D, Li J, Kou F, Luo X and Yang P. Reveal crucial subtype of natural chondroitin sulfate on the functionalized coatings for cardiovascular implants. *J Mater Sci Technol* 2021; 91: 67–77.

163. Lee S, Ganesan R, Krüger-Engage A, et al. Substrate-enzyme affinity-based surface modification strategy for endothelial cell-specific binding under shear stress. *Clin Hemorheol Microcirc* 2020; 75: 85–98.

164. Huang Y-H, Xu Q, Shen T, Li JK, Sheng JY and Shi HJ. Prevention of in-stent restenosis with endothelial progenitor cell (EPC) capture stent placement combined with regional EPC transplantation: an atherosclerotic rabbit model. *Cardiol J* 2011; 26: 283–291.

165. West-Livingston L, Ju YM, Lee H, Geary RL, Atala A and Lee SJ. Antibody-conjugated electrospun vascular scaffolds to enhance in situ endothelialization. *ACS Appl Bio Mater* 2020; 3: 4486–4494.

166. Diaz-Rodriguez S, Rasser C, Mesnier J, et al. Coronary stent CD31-mimetic coating favours endothelialization and reduces local inflammation and neointimal development in vivo. *Eur Heart J* 2021; 42: 1760–1769.

167. Paprocka M, Kraskiewicz H, Paprocka M, et al. Increased endothelialization and recruitment. *J Biomed Mater Res B Appl Biomater* 2021; 109: 1549–1559.

168. West-Livingston L, Ju YM, Lee H, Geary RL, Atala A and Lee SJ. Antibody-conjugated electrospun vascular scaffolds to enhance in situ endothelialization. *ACS Appl Bio Mater* 2020; 3: 4486–4494.

169. Wawrzyńska M, Kraskiewicz H, Paprocka M, et al. Increased endothelialization and recruitment. *J Biomed Mater Res B Appl Biomater* 2021; 109: 1549–1559.

170. Wawrzyńska M, Kraskiewicz H, Paprocka M, et al. Increased endothelialization and recruitment. *J Biomed Mater Res B Appl Biomater* 2021; 109: 1549–1559.
170. Wawrzyńska M, Duda M, Wysokińska E, et al. Functionalized CD133 antibody coated stent surface simultaneously promotes EPCs adhesion and inhibits smooth muscle cell proliferation: A novel approach to prevent in-stent restenosis. *Colloids Surf B Biointerfaces* 2019; 174: 587–597.

171. Song C-L, Li Q, Yu YP, et al. Study of novel coating strategy for coronary stents: simultaneous coating of VEGF and anti-CD34 antibody. *Rev Bras Cir Cardiovasc* 2015; 30: 159–163.

172. Chen L, He H, Wang M, Li X and Yin H. Surface coating of polytetrafluoroethylene with extracellular matrix and anti-CD34 antibodies facilitates endothelialization and inhibits platelet adhesion under shear stress. *Tissue Eng Regen Med* 2017; 14: 359–370.

173. Badv M, Alonso-Cantu C, Shakeri A, Hosseinidoust Z, Weitz JJ and Didar TF. Biofunctional lubricant-infused vascular grafts functionalized with silanized Bio-Inks suppress thrombin generation and promote endothelialization. *ACS Biomater Sci Eng* 2019; 5: 6485–6496.

174. de Winter RJ, Chandrasekhar J, Kalkman DN, et al. 1-Year clinical outcomes of all-comer patients treated with the dual-therapy COMBO stent primary results of the COMBO collaboration. *JACC Cardiovasc Interv* 2018; 11: 1969–1978.

175. Kerkmeijer LSM, Chandrasekhar J, Kalkman DN, et al. Final five-year results of the REMEDEE Registry: real-world experience with the dual-therapy COMBO stent. *Catheter Cardiovasc Interv* 2021; 98: 503–510.

176. Blessing R, Ahoopai M, Geyer M, et al. The bioengineered combo dual-therapy CD34 antibody-covered sirolimus-eluting coronary stent in patients with chronic total occlusion evaluated by clinical outcome and optical coherence tomography imaging analysis. *J Clin Med* 2020; 10: E80.

177. Wessely R. New drug-eluting stent concepts. *Nat Rev Cardiol* 2010; 7: 194–203.

178. Förstermann U, Xia N and Li H. Roles of vascular oxidative stress and nitric oxide in the pathogenesis of Atherosclerosis. *Circ Res* 2017; 120: 713–735.

179. Garcia V and Sessa WC. Endothelial NOS: perspective and recent developments. *Br J Pharmacol* 2019; 176: 189–196.

180. Chen S-Y, Wang J, Jia F, et al. Bioinspired NO release coating enhances endothelial cells and inhibits smooth muscle cells. *J Mater Chem B* 2022; 10: 2454–2462.

181. Rao J, Pan Bei H, Yang Y, Liu Y, Lin H and Zhao X. Nitric oxide-producing cardiovascular stent coatings for prevention of thrombosis and restenosis. *Front Bioeng Biotechnol* 2020; 8: 578.

182. Zhao Q, Fan Y, Zhang Y, Liu J, Li W and Weng Y. Copper-based SURMOFs for nitric oxide generation: hemocompatibility, vascular cell growth, and tissue response. *ACS Appl Mater Interfaces* 2019; 11: 7872–7883.

183. Wo Y, Brisbois EJ, Wu J, et al. Reduction of thrombosis and bacterial infection via controlled nitric oxide (NO) release from S-Nitroso-N-acetylcysteine (SNAP) impregnated carboxil intravascular catheters. *ACS Biomater Sci Eng* 2017; 3: 349–359.

184. Douglass M, Hopkins S, Pandey R, Singha P, Norman M and Handa H. S-nitrosoglutathione-based nitric oxide-releasing nanofibers exhibit dual antimicrobial and antithrombotic activity for biomedical applications. *Macromol Biosci* 2021; 21: e2000248.

185. Zhu T, Gao W, Fang D, et al. Bifunctional polymer brush-grafted coronary stent for anticoagulation and endothelialization. *Mater Sci Eng C* 2021; 120: 111725.

186. Tran DL, Le Thi P, Lee SM, Hoang Thi TT and Park KD. Multifunctional surfaces through synergistic effects of heparin and nitric oxide release for a highly efficient treatment of blood-contacting devices. *J Control Release* 2021; 329: 401–412.

187. Wan X, Liu P, Jin X, et al. Electrospun PCL/keratin/aumps mats with the catalytic generation of nitric oxide for potential of vascular tissue engineering. *J Biomed Mater Res A* 2018; 106: 3239–3247.

188. Wang L, Xin X, Li P, et al. Stepwise immobilization of keratin-dopamine conjugates and gold nanoparticles on PET sheets for potential vascular graft with the catalytic generation of nitric oxide. *Colloids Surf B Biointerfaces* 2021; 205: 111855.

189. Lyu N, Du Z, Qiu H, et al. Mimicking the nitric oxide-releasing and glycoalyx functions of endothelium on vascular stent surfaces. *Adv Sci* 2021; 8: e2101788.

190. Fan Y, Zhang Y, Zhao Q, et al. Immobilization of nano Cu-mofs with polydopamine coating for adaptable gas-otransmitter generation and copper ion delivery on cardiovascular stents. *Biomaterials* 2019; 204: 36–45.

191. Yang Y, Gao P, Wang J, et al. Endothelium-mimicking multifunctional coating modified cardiovascular stents via a stepwise metal-catechol-(amine) surface engineering strategy. *Research* 2020; 2020: 1–20.

192. Jiang L, Yao H, Luo X, et al. Polydopamine-modified copper-doped titanium dioxide nanotube arrays for copper-catalyzed controlled endogenous nitric oxide release and improved re-endothelialization. *ACS Appl Bio Mater* 2020; 3: 3123–3136.

193. Yang L, Li L, Wu H, Zhang B, Luo R and Wang Y. Catechol-mediated and copper-incorporated multilayer coating: an endothelium-mimetic approach for blood-contacting devices. *J Control Release* 2020; 321: 59–70.

194. Zhang B, Yao R, Hu C, et al. Epigallocatechin gallate mediated sandwich-like coating for mimicking endothelium with sustained therapeutic nitric oxide generation and heparin release. *Biomaterials* 2021; 269: 120418.

195. Ramachandran B and Muthuvijayan V. Cysteine immobilisation on the polyethylene terephthalate surfaces and its effect on the haemoocompatibility. *Sci Rep* 2019; 9: 16694.

196. Luo R, Zhang J, Zhuang W, et al. Multifunctional coatings that mimic the endothelium: surface bound active heparin nanoparticles with in situ generation of nitric oxide from nitrosothiols. *J Mater Chem B* 2018; 6: 5582–5595.

197. Jin S, Huang J, Chen X, et al. Nitric oxide-generating anti-platelet polyurethane surfaces with multiple additional bio-functions via cyclodextrin-based host-guest interactions. *ACS Appl Bio Mater* 2020; 3: 570–576.

198. Yang Z, Yang Y, Zhang L, et al. Mussel-inspired catalytic selenocystamine-dopamine coatings for long-term generation of therapeutic gas on cardiovascular stents. *Biomaterials* 2018; 178: 1–10.

199. Li X, Shen F, Wang K, et al. Endothelial mimetic multifunctional surfaces fabricated via polydopamine mediated copper immobilization. *J Mater Chem B* 2018; 6: 7582–7593.

200. Qiu H, Qi P, Liu J, et al. Biomimetic engineering endothelium-like coating on cardiovascular stent through heparin.
and nitric oxide-generating compound synergistic modification strategy. *Biomaterials* 2019; 207: 10–22.

201. Cornelissen A, Guo L, Fernandez R, et al. Endothelial recovery in bare metal stents and drug-eluting stents on a single-cell level. *Arterioscler Thromb Vasc Biol* 2021; 41: 2277–2292.

202. Máirtín ÉO, Concannon J, Parry G and McGarry JP. A mechanistic analysis of delamination of elastic coatings from the surface of plastically deformed stents. *Int J Solids Struct* 2021; 224: 111051.

203. Maleckis K, Kamenskiy A, Lichter EZ, Oberley-Deegan R, Dzenis Y and MacTaggart J. Mechanically tuned vascular graft demonstrates rapid endothelialization and integration into the porcine iliac artery wall. *Acta Biomater* 2021; 125: 126–137.

204. Qian Y and Yuan G. Research status, challenges, and countermeasures of biodegradable zinc-based vascular stents. *Acta Metallurgica Sin* 2021; 57: 272–282.

205. Richter A, Li Y, Rehbock C, et al. Triple modification of alginate hydrogels by fibrin blending, iron nanoparticle embedding, and serum protein-coating synergistically promotes strong endothelialization. *Adv Mater Interfaces* 2021; 8: 2002015.

206. Zhang J, Li G, Man J, et al. Mechanism of anti-proteins adsorption behavior on superhydrophobic titanium surface. *Surf Coat Technol* 2021; 421: 127421.

207. Jin Y, Zhu Z, Liang L, et al. A facile heparin/carboxymethyl chitosan coating mediated by polydopamine on implants for hemocompatibility and antibacterial properties. *Appl Surf Sci* 2020; 528: 146539.

208. Elmaggar MA, Han DK and Joung YK. Nitric oxide releasing lipid bilayer tethered on titanium and its effects on vascular cells. *J Ind Eng Chem* 2019; 80: 811–819.

209. Chen G, Xu H, Wu Y, et al. Myricetin suppresses the proliferation and migration of vascular smooth muscle cells and inhibits neointimal hyperplasia via suppressing TGFBR1 signaling pathways. *Phytomedicine* 2021; 92: 153719.

210. Yuan Y, Khan S, Stewart DJ and Courtman DW. Engineering blood outgrowth endothelial cells to optimize endothelial nitric oxide synthase and extracellular matrix production for coating of blood contacting surfaces. *Acta Biomater* 2020; 109: 109–120.