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Cell Membranes as Therapeutic Targets: Implications in Infectious and Metabolic Disorders

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Abstract

This is a review of the crucial role played by cell membranes in health and disease with respect to their contribution to infectious and metabolic diseases. Dysfunction of membranes, such as membrane transporter changes and changes in lipid content, has been associated with a variety of diseases, including diabetes, cardiovascular disease, and cancer. The present therapeutic approaches are focused on attacking membrane constituents by disrupting the entry of pathogens or dysfunctional membrane proteins, which have potential as therapies for infections, metabolic disorders, and cancer. Examples are SGLT2 inhibitors to treat diabetes and CCR5 inhibitors to treat HIV. Moreover, nanotechnology, gene editing, and individualized treatment are giving new prospects for more specific and efficient treatment. The applications of membrane-targeted therapies are enormous, and future studies are bound to allow therapeutic agents to be delivered more effectively and produce better treatment results. This paper brings to light the present methods as well as the future perspectives of the membrane-targeted therapies and provides an insight into the potential of the same in the treatment of complex diseases.

Keywords: Ccr5-inhibitors, cell membranes, gene editing, membrane dysfunction, membrane transporters, metabolic disorders, nanotechnology, personalized medicine, SGLT2-inhibitors, therapeutic strategies

INTRODUCTION

All living cells possess cell membranes that are very important features of the cell and make an effective selective barrier that facilitates cell integrity and the movement of substances in and out of the cell [1]. These membranes consist of a lipid bilayer intertwined with proteins and carbohydrates, which play crucial roles in the maintenance of cellular homeostasis, cell–cell communication, and other significant roles [2]. The lipid bilayer also provides the cell with a semipermeable compartment, which allows the cell to have varying internal environments, and cellular recognition, the membrane proteins are also implicated in the vital processes of nutrient uptake, waste removal, signal transduction, and cellular recognition [3]. Membrane carbohydrates bind to either proteins or lipids, and they assist in cell–cell, cell immunity, and cell recognition. The cell membrane is not only essential in supporting the structure but also in health, nutrient absorption along the gut, signal transmission inside the nerve cells, as well as immune response to pathogens is dependent on the cell membrane. Also, cell division, cell migration, as well as tissue repair require membrane dynamics, and so do normal cellular processes and tissue homeostasis [4]. However, membrane malfunction has been linked to diseases of different diseases, specifically infectious diseases and metabolic diseases. The pathogens that typically enter the host cells using the components

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of the membranes and infect them are viruses, bacteria, and fungi. Viruses are an example: HIV and influenza have membrane receptors to form an entrance, and bacterial toxins may lead to altering the membrane permeability and causing an aberrant cellular response, leading to the development of the disease [5]. In metabolic disorders, such as diabetes and cardiovascular diseases, membrane receptor or transporter malfunction, e.g., dysfunction of insulin receptor in diabetes or lipid transporters in atherosclerosis, can play a role in the development of a disease by affecting cellular communication and nutrient transport. Abnormal membrane proteins in cancer can lead to immune surveillance evasion or the acquisition of the capability to spread cancer cells to other body parts [6]. This is equal to the importance of the therapeutic application of cell membranes. Viral entry blocking drugs, receptor modulating drugs, or transporter dysfunction corrective drugs are some of the promising therapies for various diseases because they are membrane-targeted. An example is an antiretroviral drug that increases the CCR5 receptor and blocks HIV infection, such as maraviroc and a sodium–glucose cotransporter 2 (SGLT2) drug that inhibits the excess glucose uptake in the kidney. More detailed therapies of infections, metabolic diseases, cancer, and even neurodegenerative diseases are also emerging through research in membrane biology and therapeutic development, which target the cell membrane components of the disease [7]. The field of nanomedicine and gene editing technologies keeps enhancing the ability to preferentially address the molecules bound to the membrane and offers a chance to discover more personalized and effective medications with fewer side effects [8]. There is a high probability of discovering new therapies because research efforts continue to elucidate the complexity of membrane structures and their interactions with pathogens, drugs, and signaling pathways, offering new hope for treating various difficult health disorders. Cell membrane complexes are significant in medical science and in improving therapeutic outcomes in most diseases. The focus on membrane-targeted intervention has become one of the priorities of modern medicine [9].

CELL MEMBRANES IN INFECTIOUS DISEASES

The membrane proteins, receptors, ion channels, and transporters are required for the entry of pathogens. To serve as an example, HIV uses the CD4 receptor, and the co-receptors of CCR5 or CXCR4 of T- helper cells, to fuse with the host cell membrane, thus allowing invasion of the virus cells and their reproduction [10]. Similarly, the influenza virus binds to sialic acid residues in the glycoproteins on the cell surface of the respiratory epithelial cells, which induces the fusion and internalization. Bacteria also adhere and invade using the host cell membrane. Helicobacter pylori also attaches to the membranes of the gastric epithelial cell, and Streptococcus pneumoniae attaches to the membranes of the respiratory cells with the assistance of surface proteins [11]. Once in the host, the pathogens seize the organelles and the system of trafficking within the cell that is enclosed in a membrane to facilitate replication. Viruses like vesicular stomatitis virus (VSV) invade through endocytic routes to replicate, and bacteria like Salmonella invade using the host cell membrane to assemble structures to assist them in survival and reproduction. The fact that cells are dependent on these membranes is a testimony to the relevance of cell membranes as a therapeutic approach [12]. Antiviral agents that inhibit entry of pathogens, such as enfuvirtide, which inhibits HIV fusion, or oseltamivir, which inhibits viral release, directly affect the elements of the membrane on which the infection resides. In addition, the HIV invasion has been inhibited by means of blocking host cell receptors, e.g., CCR5, with the assistance of maraviroc [13]. The other possible solution is to target the bacterial adhesins, which allow bacteria to adhere to the host cells, thus reducing the number of infections such as colonization of Helicobacter pylori. The other potential option of hindering the entry of pathogens is by disrupting the establishment of lipid rafts within the membrane, which is a significant aspect of the membrane receptor clustering [8]. Moreover, the lipid composition of the membranes may change to make them watertight to the entry of pathogens, another way of preventing the infection. Membrane proteins on pathogens, e.g., the proteins developed against the spike protein of SARS-CoV-2, have been demonstrated to be useful in preventing the entry of viruses into host cells by using monoclonal antibodies. These therapeutic measures emphasize the role of the cell membrane in the cell entry and replication of pathogens and demonstrate that therapeutic interventions targeting the membrane can be designed to provide a cure to numerous infectious diseases [14]. Table 1 summarizes

the membrane proteins that can be important in the entry of the pathogens, whereas Figure 1 summarizes pathogen–membrane interaction schematically, indicating how the pathogens utilize the membrane components to enter and replicate.

Table 1. Membrane proteins involved in pathogen entry.

Pathogen	Membrane protein target	Mechanism of entry
HIV	CD4, CCR5, CXCR4	Fusion with the host cell membrane, viral entry.
Influenza	Sialic acid residues	Binding to glycoproteins on the host cell membrane, fusion.
Helicobacter pylori	Glycoproteins	Adhesion to gastric epithelial cells.
Streptococcus pneumoniae	Surface proteins	Adhesion to epithelial cells in the respiratory tract.
Salmonella	Epithelial cell receptors	Internalization via membrane ruffling.
Vesicular Stomatitis Virus (VSV)	Endocytic pathways	Membrane fusion and release of the viral genome.
SARS-CoV-2	ACE2, TMPRSS2	Binding and fusion with the host cell membrane.

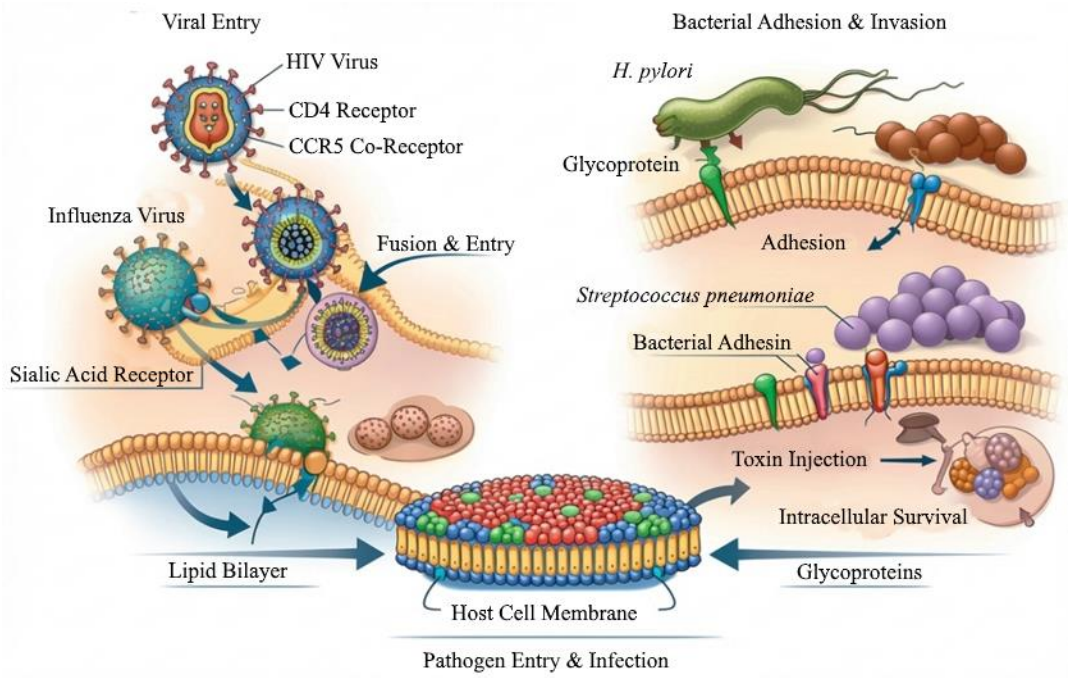

Figure 1. Pathogen–membrane interaction.

CELL MEMBRANES IN METABOLIC DISORDERS

The cell membranes play an important role in the maintenance of the metabolic processes, and impairments in the membrane can cause various metabolic disorders. Membrane proteins, especially transporters, take part in the flow of key molecules like glucose, lipids, and ions in and out of the cells, and failure of them may cause disease [15]. In metabolic diseases like type 2 diabetes, obesity, and cardiovascular diseases, the impairment of membrane transport has a huge role in the pathophysiology of these diseases. In diabetes type 2 diabetes, e.g., the insulin receptor, which is a protein bound to the membrane, becomes unresponsive to insulin, causing it to be insulin-resistant [16]. The resistance affects the absorption of glucose into the cells and leads to the rise of blood sugar. On the same note, in obesity, the changes in the activity of membrane transporters that control lipid metabolism and energy storage may help accumulate fat and disrupt metabolic regulation. Additionally, in cardiovascular conditions, lipid composition abnormalities and malfunction of lipid carriers cause cholesterol accumulation in blood vessels, causing atherosclerosis and other complications. Insulin-regulated glucose uptake requires membrane transporters, such as GLUT4, a glucose transporter occurring in muscle and fat cells [6]. The dysfunction of the GLUT4 in patients

with type 2 diabetes leads to the inability to absorb glucose and helps to increase the level of glucose in the blood. The other important transporter in metabolic regulation is the sodium–glucose cotransporter 2 (SGLT2), which is engaged in the reabsorption of glucose in the kidneys. Empagliflozin is an inhibitor of this transporter that is used to treat type 2 diabetes by inhibiting glucose reabsorption and enhancing urine glucose excretion, leading to a decrease in blood glucose levels [17]. The disturbance of these transporters not only interferes with the normal metabolic activities but also worsens the manifestation and course of the metabolic diseases. Besides, lipid metabolism transporters, including ATP-binding cassette (ABC) transporters, are also critical in cholesterol and phospholipid transport [18]. The unusual functioning of these transporters may result in the buildup of lipids in cells and tissues, which may cause such conditions as non-alcoholic fatty liver disease (NAFLD) and atherosclerosis. Malfunctioning of the ion transporters that control the movements of sodium, potassium, and calcium across the membranes is also involved in metabolic disorders. As an illustration, changes in the function of sodium–potassium pumps may interfere with homeostasis in cells, causing hypertension and propagating the onset of heart diseases [19]. It is important to understand how membrane transporters are involved in regulating metabolism so as to develop specific therapies to restore normal functioning of the same. Suppressing or regulating the action of the maladaptive transporters can be used as an encouraging option in the treatment of metabolic diseases. An example is insulin-sensitizing drugs, such as metformin, that enhance glucose uptake by cells, and the use of SGLT2 inhibitors that are used to regulate glucose through the inhibition of glucose transporters in the kidneys [20]. Since research has advanced, there is a lot of potential in the use of therapies that can specifically target membrane transporters that play a role in the regulation of metabolism in treating diseases like type 2 diabetes, obesity, and cardiovascular diseases. Table 2 describes the major membrane transporters that are used in metabolic diseases, and Figure 2 shows how the malfunctioning of the transporters causes abnormal glucose, lipid, and ion homeostasis, resulting in the development of a disease [21].

Table 2. Major Membrane Transporters in Metabolic Disorders and Therapeutic Targets.

S.N.	Transporter / Receptor	Location	Function	Associated Disorder	Therapeutic Target / Drug	Mechanism of Action
1	GLUT4 (Glucose Transporter Type 4)	Muscle & adipose tissue	Insulin-mediated glucose uptake	Type 2 Diabetes Mellitus	Metformin, Insulin sensitizers	Enhances glucose uptake and improves insulin sensitivity
2	SGLT2 (Sodium–Glucose Cotransporter 2)	Kidney (proximal tubules)	Glucose reabsorption from urine	Type 2 Diabetes Mellitus	Empagliflozin, Dapagliflozin	Inhibits glucose reabsorption → increases urinary glucose excretion
3	Insulin Receptor	Cell membrane (various tissues)	Regulates glucose metabolism	Insulin resistance, Diabetes	Insulin therapy, sensitizers	Activates glucose uptake pathways
4	ABC Transporters (ATP-binding cassette)	Liver, intestine, macrophages	Lipid and cholesterol transport	Atherosclerosis, NAFLD	Statins (indirect), lipid modulators	Regulate cholesterol efflux and lipid homeostasis
5	Na^+/K^+ ATPase Pump	All cells	Maintains ion balance (Na^+/K^+ gradient)	Hypertension, cardiovascular diseases	Cardiac glycosides (indirect)	Alters ion transport affecting cardiac function
6	Calcium Channels (Ca^{2+} transporters)	Cardiac & smooth muscle cells	Regulates muscle contraction	Hypertension, heart disease	Calcium channel blockers	Reduce Ca^{2+} influx → vasodilation
7	Lipid Transporters (e.g., FAT/CD36)	Adipose & muscle tissue	Fatty acid uptake	Obesity, metabolic syndrome	Experimental inhibitors	Reduce lipid accumulation

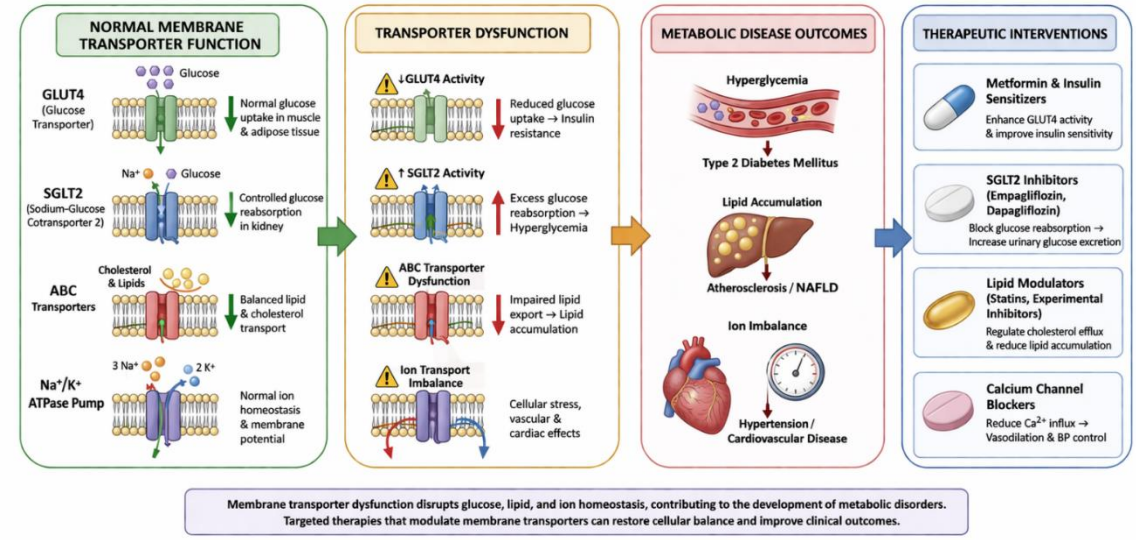

Figure 2. Membrane Transporter Dysfunction and Metabolic Disease Progression: Therapeutic Targets for Restoring Cellular Homeostasis.

THERAPEUTIC STRATEGIES TARGETING CELL MEMBRANES

Intervention on cell membranes by therapeutic means has gained a center-stage in medical research which has brought promising solutions to a range of diseases through the manipulation of cell membrane structure and functions [22]. Membrane disruption and inhibition apply to alter or specifically target the lipid bilayer, membrane proteins or lipid microdomains, such as lipid rafts, to cure diseases such as infections, cancer, and metabolic diseases. Antimicrobial peptides (AMPs) can be used as an example: they disrupt microbial membranes to make pathogens more vulnerable to immune surveillance, or nanocarriers based on lipids to deliver drugs can be used in cancer therapy, targeting membrane receptors or transporters [22]. In metabolic diseases, such as type 2 diabetes, the SGLT2 inhibitors prevent the reabsorption of glucose in the kidney, which regulates the level of blood sugar, and in HIV treatment, CCR5 inhibitors suppress the entry of the virus attacking membrane receptors. In the future, nanotechnology and gene editing, such as CRISPR-Cas9, have tremendous potential of providing more accurate and individualized membrane-targeted therapy. Scientists are also researching how to alter membrane lipid composition to remedy dysfunction in such diseases as Alzheimer's [23]. These therapies are in the process of being developed and are expected to be more effective therapies with fewer side effects due to their specific targeting of the underlying membrane processes in disease, thus this can be seen as a new frontier in personalized medicine and offer more specific and effective medicines to a large number of conditions.

CONCLUSION

To sum up, cell membranes play an important role in keeping the cells functional and are also significant contributors to many diseases such as infections, metabolic diseases, and cancer. The existing methods of therapeutic intervention of cell membranes involve interfering with or blocking cell membrane constituents, including proteins, lipids, and receptors, to prevent the entry of pathogens, to regulate metabolism, or to prevent the growth of cancer cells. Antimicrobial peptide usage, membrane receptor inhibitors, and lipid-based nanoparticles are some of the strategies that have demonstrated effectiveness in enhancing the selectivity and efficacy of treatment, besides reducing side effects. As an example, membrane transporter and receptor inhibitors, such as SGLT2 in diabetes and CCR5 in HIV, have been shown to be clinically effective. Also, nanotechnology and gene editing technologies, such as CRISPR, are promising a more specific and personalized treatment, as it is possible to modify the functions of a membrane at the molecular level. With the continued development of membrane biology, in the future, more advanced and targeted therapies will be developed that extend past mere membrane-shaking and altering their functionality, and that will provide hope to more accurate curing

of diseases such as cancer and neurodegenerative disorders. The possibilities that exist in terms of membrane-targeted therapies are enormous, and new and more tailored forms of therapeutic options are coming.

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