ScienceMind

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KING'S College LONDON

STAFF

THIS ISSUE

Dear Reader,

Welcome to the second issue of ScienceMind (previously KCL Pharmacologist). A big thanks to the writing, editing and media teams for helping to contribute to this issue.

If this is your first time reading our magazine...
Science Mind is the award-nominated, student-led science magazine of King's College London, which focuses on reporting recent findings in the main branches of science to students and the wider community. We aim to showcase and develop the written and oral communication skills of students interested in research by concisely explaining complex scientific concepts in the form of lay articles and conducting interviews. Authors can also broaden their knowledge by writing articles for different sectors between issues.

If science communication is a concept that interests you, I encourage you to join the dynamic and constantly expanding team of ScienceMind. We would love to have you on our team!

Yours faithfully,

The Editor-in-Chief
The Founder

CONTENTS

SHALLOW DIVE

NANOMEDICINE LIGANDS-RECEPTOR SYSTEMS FOR ATHEROSCLEROSIS

WRITTEN BY CHETANA PRABHU & CHARLINE HENDRICKX

Atherosclerosis - the primary cause of cardiovascular dysfunction, is a detrimental accumulation of plaque which is made up of lipids and fibrous elements within the arterial wall. An atherosclerotic plaque forms as the result of a flawed immune response and an imbalance of cholesterol metabolism. An increased blood concentration of the low-density lipoproteins leads to its accumulation in the arterial wall through necrosis and calcification and once this plaque ruptures, platelets congregate and start the clotting process forming a thrombus. This thrombus blocks the lumen and leads to ischaemic events.

The risk factors include smoking, diabetes mellitus, obesity and so on. Atherosclerosis can remain asymptomatic in 30 to 40% individuals, while the majority suffer from sudden death or acute coronary syndrome, which is why assessing the vulnerability of the unstable plaque could have great benefits. Recent research undertaken by scientists in Australia aims to collate the choices of ligand-receptor systems used in targeting nanoparticles for diagnosis and treatment of atherosclerosis.

Nanomedicine is the medical usage of nanotechnology/nanoparticles to treat a disease. It involves the production of medicine on nanoscale levels to improve their ability to target specific receptors. They may be organic or inorganic and spherical/cylindrical/plate-like shaped. Nanoparticles are developed to function as drug carriers and are sometimes labelled with binding ligands such as peptides or antibodies that specifically target disease biomarkers, for delivering therapeutics and for diagnostic imaging. An advantage of nanoparticles is that they can carry multiple ligands due to a high surface area to volume ratio and the binding ligands modulate the biodistribution of the nanoparticles.

Vascular targeting (for local delivery of therapeutics) involves usage of one of the earliest known binding ligands- RGD, which when recognised by integrins allows us to carry out thrombus imaging. Due to a wide range of cellular and molecular processes and cell types serving as targeting models, choosing a highly specific ligand-receptor system is essential in developing the favourable nanoparticles for diagnostic and therapeutic purposes.

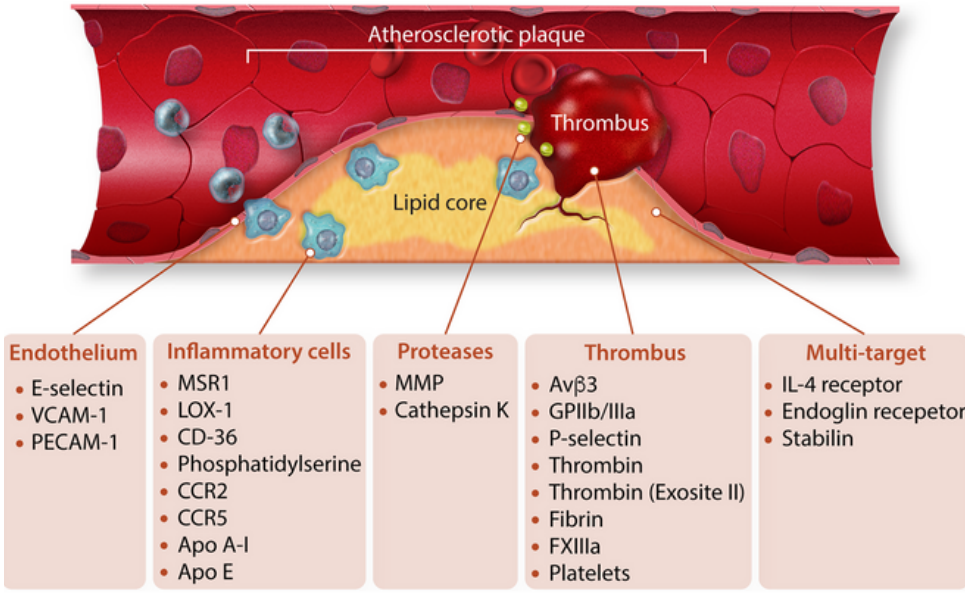

Figure 1. Illustration of the atherosclerotic plaque, depicting the rupturing of the lipid core

Investigations of ligands were conducted to compare their binding affinity and the most studied ligand binding system was an antibody-based system. The system allowed for high affinity and specificity compared to other ligands but caused immunogenic responses due to its size. However, the size of the particle resulted in a loss of affinity. Peptide ligands were therefore studied due to their advantages over the antibody system. Their size and synthetic flexibility alongside the lower production cost made them more favourable than their antibody counterpart. Peptide ligands were also less immunogenic than the antibody system and more easily modified to increase affinity through cyclization.

A novel method was studied where aptamers (oligonucleotides or peptide molecules that bind to a specific receptor) were used as ligands which proved to be more efficacious than the antibody-based system as well. Aptamers did not produce immunogenicity due to their smaller molecular size and were also more rapid to manufacture. Ligand-receptor systems using peptide ligands or aptamers have shown promising results, but there is still a need for more in-depth research of nanoparticle ligand targeting as specific aspects of atherosclerosis change over time due to molecular progression which occurs in atherosclerotic plaques.

ABOUT THE AUTHOR

Chetana Prabhu is a 1st year BSc Pharmacology student. Her current areas of interest include neuroscience, genetics and pharmacology.

Charline Hendrickx is a 3rd year BSc Pharmacology student with an interest in oncology and depression in neuropharmacology.

SHALLOW DIVE

CAN THE NEW TREATMENT FOR ALZHEIMER'S DISEASE BE FOUND IN YOUR KITCHEN CABINET?

WRITTEN BY FATIMAH PATEL

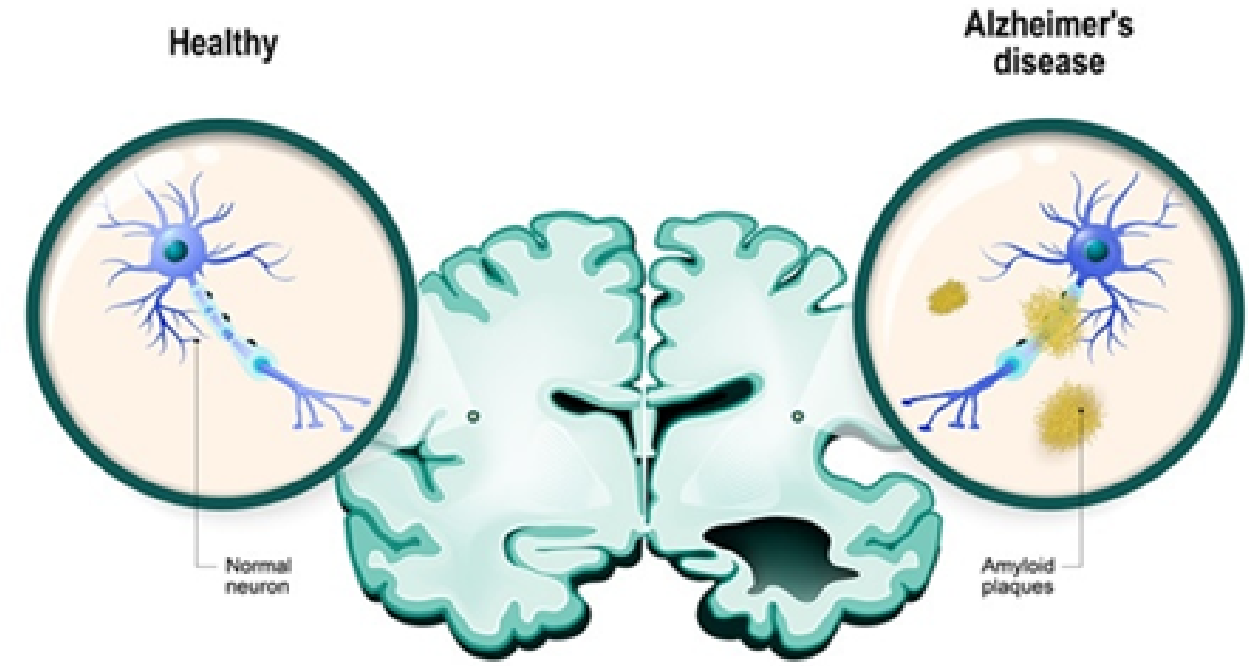

Figure 1. A diagram showing the difference between healthy brains and brains with Alzheimer's Disease.

A 2019 study by Jia et al. proposed that Vitamin D supplements could improve cognitive function by possibly removing toxic proteins in the brain of people with Alzheimer's disease (AD). This remarkable finding can encourage scientists to explore the protective properties of Vitamin D and its role in neurodegenerative diseases. AD is a brain disorder which accounts for approximately 65% of all dementia cases.

According to the NHS, it is associated with a continuous decline in brain functioning which results in cognitive dysfunction and memory loss. Although the exact cause is still unknown, it is thought that the build-up of Amyloid beta protein (Ab) plays a major role in disease progression and pathogenesis. This protein can be produced in the brain of normal aging individuals, however abnormally high levels are found in the brain of AD sufferers.

These Ab proteins then end up clumping together to form 'plaques' in the brain, which are considered to be toxic and destructive to neurones, and additionally they contribute to inflammation. These detrimental effects reduce brain activity and function. Vitamin D is shown to have a wide range of health-related benefits such as protective actions on neurons and hindering inflammation in the brain. There are also links between AD and low levels of Vitamin D, so it's thought that Vitamin D works by lowering how much Ab is in the brain.

The researchers studied whether giving Vitamin D for 12 months would improve cognitive function in individuals with AD, and if this improvement was linked to the effect of vitamin D on Ab levels. It was found that participants taking Vitamin D performed better in cognitive assessments relating to memory and recognition compared to the placebo group. The Vitamin D group also had fewer biomarkers in their blood unlike the placebo group, which meant there was a decrease in the level of neural Ab. These results show that Vitamin D has some beneficial properties for improving brain functioning in AD patients, and it's thought to do this by reducing the abnormally high concentration of Ab in the brain (which means fewer Ab plaques are formed).

The findings can raise inquiries for the exact mechanisms by which Vitamin D can reduce Ab when treating AD. Does Vitamin D only reduce the level of biomarkers in blood (but doesn't affect Ab levels in brain)? Does Vitamin D inhibit Ab production by interactions with proteins in a cascade of reactions? These concepts need to be understood first before reaching a conclusion as to how Vitamin D improves cognitive function.
Nonetheless, the results are still promising as it can be translated into the development of novel therapies for AD.

ABOUT THE AUTHOR

Fatimah Patel is a 3rd year BSc Pharmacology student with a strong interest in neuropharmacology.

"Working as a scientist, one has multiple setbacks. And I always say that you have to move two steps backwards to move one step forward."

PROFESSOR SUSAN BRAIN

Professor Susan Brain is a lecturer and researcher in pharmacology and has worked at King's College London since 1989. She was the first woman to work for the pharmacology department and the first to lead it. Professor Brain is Head of the 'Vascular Biology and Inflammation Section'.

Her most influential contribution to the field was her discovery that the novel peptide CGRP possessed vasodilating properties, this was published in the prestigious journal, Nature in 1985.

Figure 1. Professor Brain awarded the Pharmacology Honorary Fellowship Award by the British Pharmacological Society.

WRITTEN BY AAIMAN BHARMAL & SEAN CRAWFORD

The calcitonin gene-related peptide (CGRP) was initially discovered when Dr Brain was working as a postdoc. CGRP, a protein, is found in certain types of sensory nerves. At this point, Dr Brain and her team realised that CGRP is found abundantly in the cerebrovascular system. With progress in research, it was realised that the peptide may have a role in migraine. This research which was supported by the British heart foundation also looked at the effect of CGRP in diabetes and heart failure. New drugs that have come from discovering the role of CGRP in treating migraine include CGRP antagonists and antibodies which have since been made available to patients. Meanwhile, Dr Brain and her team are investigating CGRP's role as a therapeutic for cardiovascular disease.

In the cardiovascular system, insufficient CGRP is produced naturally to induce a physiological effect. Nevertheless, applying a greater amount of stable forms of CGRP can exert a protective effect.

In her research, her team has used two types of mouse models to study the protective effects of CGRP. The first mouse model is for hypertension. The mice in this experiment were given angiotensin-2 over a period of two weeks by placing a mini-pump in the body of the mice, causing them to become hypertensive. Upon examination, it was observed that the mouse hearts had undergone cardiac remodelling due to inflammation and fibrosis. The hypertensive mice were then treated with CGRP which normalised the blood pressure of the mice and showed protective effects against cardiac remodelling.

Dr Brain's research

The second mouse model is of heart failure induced by surgery. These mice, when treated with CGRP showed that the peptide provided protection against heart failure. Also, researchers working at a pre-clinical level are showing similar results.

Dr Brain's research on TRPV1 and TRP1A has also yielded some significant results

Dr Brain and her team have discovered that the sensory nerves which release CGRP contain abundant Transient Receptor Potential Vanilloid 1 (TRPV1) receptors which are channels that allow the movement of ions. A typical agonist for the TRPV1 receptors is capsaicin which can be found in pepper. She says that if one were to have a hot curry (containing peppers), a hot sensation is seemingly felt due to the activation of TRPV1 receptors by its agonist, capsaicin. This leads to an influx of positive charge into the neuron, depolarising it and opening calcium ion channels. This leads to an influx of positive charge into the neuron, depolarising it and opening calcium ion channels. The subsequent activation of the nerve terminals leads to exocytosis of CGRP and a feeling of hotness. Although the team hasn't gathered much evidence of TRPV1 receptor activation in cardiovascular disease, some arthritis models show a correlation between TRPV1 and the pain resulting from arthritis.

Furthermore, her studies focusing on Transient Receptor Potential Ankyrin 1 (TRPA1) receptors, have shown that they are activated by the agonist cinnamaldehyde and cold temperatures.

In her research she investigates the events that occur in the skin with a fall in temperature. The team has discovered that the skin constricts as a result of TRPA1 activation using a skin model from a mouse. Also, the team detected the release of CGRP from the sensory nerves causing vasodilation as the constricted skin begins to recover. In her most recent work, she has shown that the skin's response to cold seems to have reduced in aged and diabetic rats. Similar observations have been noticed in humans as they age and as a result the usual behavioural responses to cold temperatures such as seeking warmth do not occur.

Additionally, a similar loss of skin sensitivity can also be seen in diabetics. This is due to neuropathy; the nerve damage caused by high blood sugar concentrations. The condition causes painful stimuli to become non-painful due to imbalance caused by the loss of sensory nerves. This can cause ulcers, sores and reduced CGRP secretions. CGRP secretions would otherwise mediate an increase in blood supply and promote wound healing. Notwithstanding the therapeutic potential of CGRP, Professor Brain believes that both CGRP agonists (drugs that cause the release of CGRP) and antagonists (drugs that inhibit the release of CGRP) may play a role in treating Covid-19.

Professor Brain studied pharmacology at Chelsea College which later merged into the present-day King's College London. During this time she undertook a placement at the then Institution of Basic Medical Sciences, Royal College of Surgeons in the Department of Pharmacology. Later, she returned to the institution to complete her Ph.D. and postdoctoral studies.

When CGRP was discovered, Professor Brain worked in a microvascular lab where she and her team studied small blood vessels. Her knowledge of microcirculation and inflammatory mechanisms and her interest in the skin led her to take off on an independent path to study CGRP. She suspects that CGRP may have a role in the reddening and darkening of the skin which is observed in skin diseases like dermatitis. In addition to her role as a researcher, Professor Brain is also an academic. She states that she always loved to research and arrived at a time when she could make a difference. Professor Brain likes to teach and help students develop as they progress through university.

She believes that teaching is a cooperative effort with her students. When she initially took the role of a lecturer, she was the first woman in her department. By the time she became the head of the department, the number of women in the department had increased. As for the present day, she feels that the numbers can still be improved to achieve equal representation in the department. When Dr. Brain is not working in her research, she likes to be outdoors, in her garden, or somewhere close to nature. Had she not been a pharmacologist, she would have enjoyed being a zoologist studying animal behaviour.

Lastly, she mentions that COVID has been a major setback for her and her team of researchers she collaborates with. She believes to have lost vital time but she also believes that it is necessary to have a character that can keep one going in times of adversity. And with optimism she says that the pandemic could bring hope of increased investment into the field of science and create greater job opportunities for budding scientists like us!

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THREADING WATER

COULD DRUGS USED TO REDUCE CHOLESTEROL INNOVATE CANCER THERAPY?

WRITTEN BY RACHEL BRADY & JAMILA CHOUDHURY

Every second, cells within our body divide to create new cells. Various mechanisms control the process of cell division to prevent errors. Over time errors accumulate, which result in damage to the DNA known as mutation. These errors accumulate in our cells over time and become more probable as a result of lifestyle choices, such as smoking or a bad diet. If enough errors build up within a cell's DNA, the cell becomes damaged. These damaged cells divide uncontrollably and form a mass known as a tumour. Malignant tumours are what we refer to as cancer; these cells infiltrate the surrounding tissue in a process called metastasis.

The body's immune system generally targets abnormal cell types; however, cancerous tumours shield themselves against the immune system by activating immune checkpoints. Immune checkpoints prevent the immune system from becoming overactive and damaging healthy cells. Proteins found on the surface of a group of immune cells, called T-cells, interact with signalling proteins that switch off the T-cells. The process of immune checkpoint signalling dampens the immune system. Cancer cells can express signalling proteins to switch off T-cells that would otherwise destroy the tumour. Immune checkpoint therapy is already used to treat skin cancers by inhibiting the ability of the tumour to switch off the immune system. Still, novel research suggests that these drugs could be improved if they were used clinically in conjunction with drugs that modulate intracellular cholesterol levels.

Cholesterol metabolism is known to be critical in specialised T-cells with significant anti-tumour effects.

Studies found that the efficacy of these specialised cells was a result of the control of cholesterol synthesis. Using statins to reduce the levels of cholesterol within these T-cells was shown to improve their anti-tumour action.

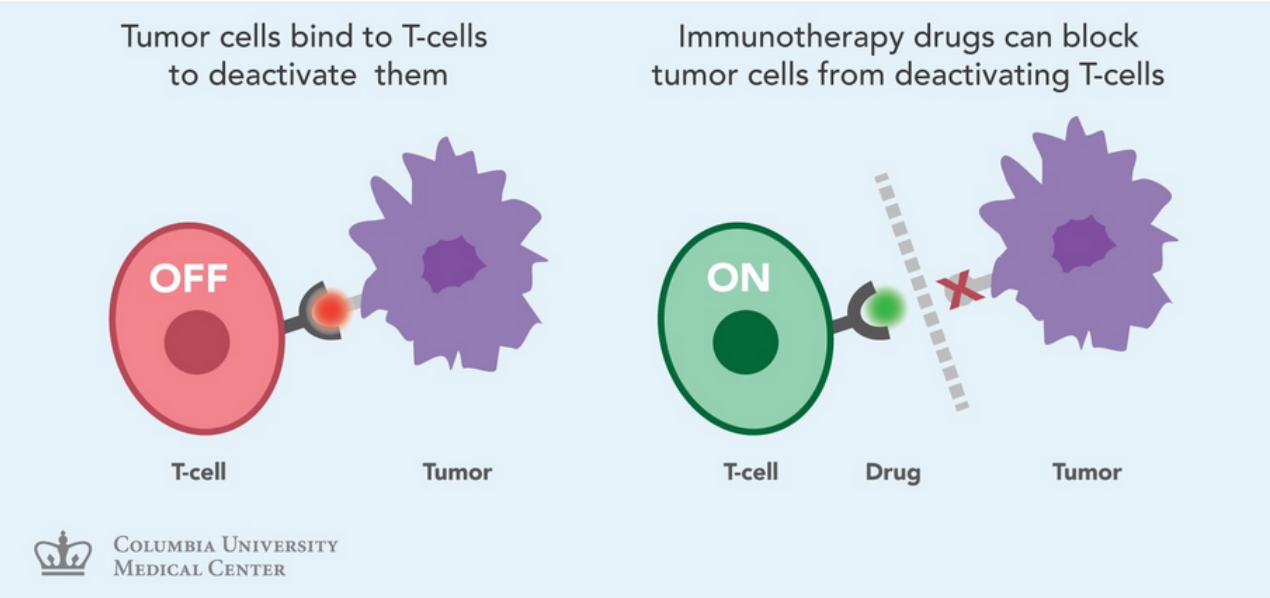

Figure 1. Illustration of the mechanisms of how immunotherapy drugs function

Cholesterol is also essential within cancer cells; its importance has been shown in the movement of the major histocompatibility complex class 1 (MHC 1) around the cell. MHC 1 is vital for tumour recognition by the immune system; it is an antigen-presenting protein that allows T-cells to target foreign cells. Cancer cells can evade the immune system by not expressing MHC 1 on their surface. If MHC 1 is not expressed on tumours, immune cells cannot interact with the tumour and destroy it. PCSK9 is a protein that regulates cholesterol metabolism. Cholesterol is carried in the blood by lipoproteins, and PCSK9 can control the movement of lipoproteins into cells, thereby regulating the cholesterol content of cells.

With the current knowledge indicating the importance of cholesterol metabolism in both T-cells and cancer cells, it was hypothesised that PCSK9 inhibition could positively influence cancer therapy.

The effects of PCSK9 deficiency were assessed on mice with various types of cancer, including skin and breast cancer. The results showed that tumour growth was restricted in PCSK9 deficient mice. The study investigated the reasons for the reduced tumour growth and found that a more significant number of immune cells moved into and targeted the tumours.

PCSK9 deficiency also resulted in increased expression of MHC 1 on tumour cells; this is likely to account for the increased activity of the immune cells on the tumour. It is therefore believed that PCSK9 presence leads to degradation of MHC 1 by directing the protein to the lysosome. PCSK9, therefore, inhibits recognition of the tumour by the immune system.

The findings of this study are very promising for cancer treatment, as drugs used to inhibit PCSK9 are already used in the treatment of high blood cholesterol (hyperlipidaemia). Extensive drug discovery and development is therefore needless, and trials to investigate PCSK9 inhibitor dual therapy with immune signalling treatment in humans are likely to begin shortly.

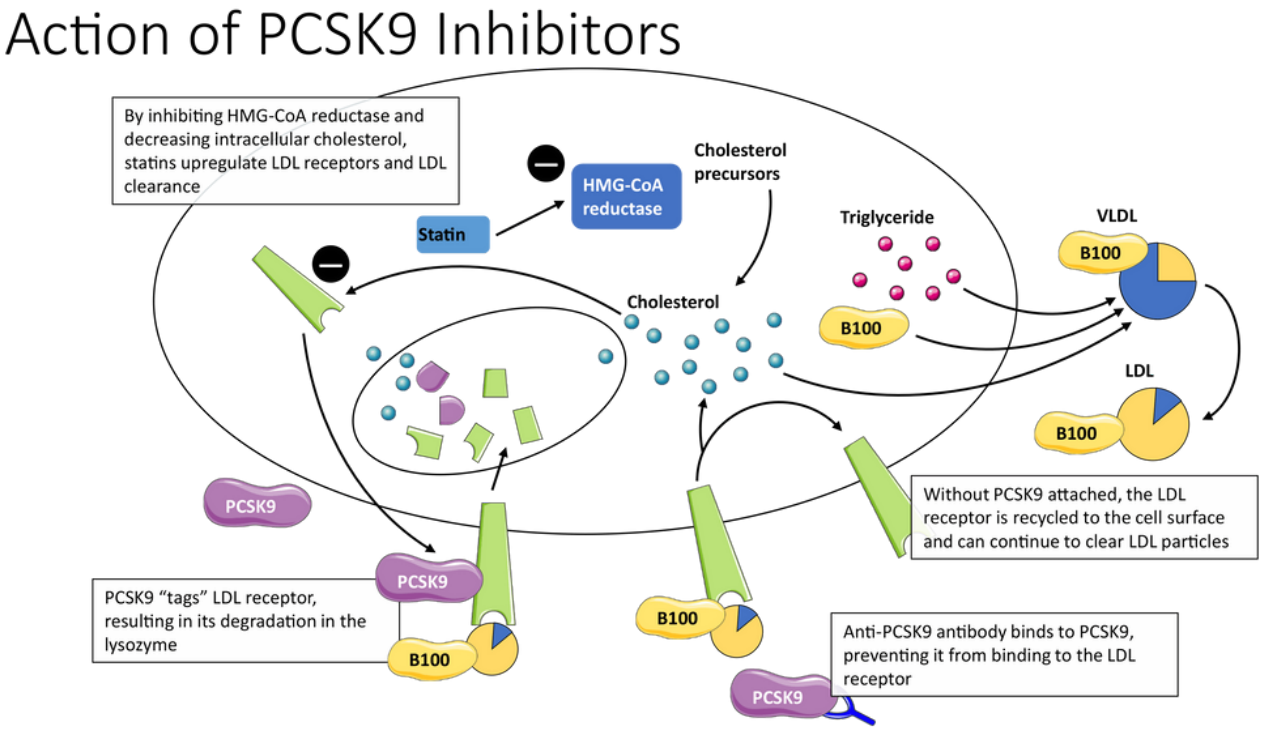

Figure 2. Diagram depicting the action of PCSK9 inhibitors.

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Jamila Choudhury is a 3rd year BSc Pharmacology student, co-author of this article.

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DEEP DIVE

COULD CARDIOVASCULAR DRUGS REDUCE COVID MORTALITY?

WRITTEN BY ZAHRAA BHATTI & KIRA LINKE

The coronavirus disease 2019 (COVID-19) outbreak has been ravaging the world with no effective antiviral drugs discovered yet. The lack of effective antivirals as of November 2020 has subsequently led to continuous clinical trials, to find successful treatments. COVID-19 is an infectious disease caused by Severe Acute Respiratory Syndrome coronavirus 2 (SARS-CoV-2). It's important to understand the structure and mechanisms of COVID-19, in order to reduce mortality and morbidity.

Increased understanding of the virus' entry mechanism has revealed that it exploits physiological pathways of the cardiovascular system, so researchers hypothesise that drugs used to treat cardiovascular disease may show efficacy in treating COVID-19. A literature review published in Elsevier's Pharmacology and Therapeutics, focuses on the renin-angiotensin-aldosterone system (RAAS) pathway, which is crucial in regulating blood pressure, wound healing and inflammation.

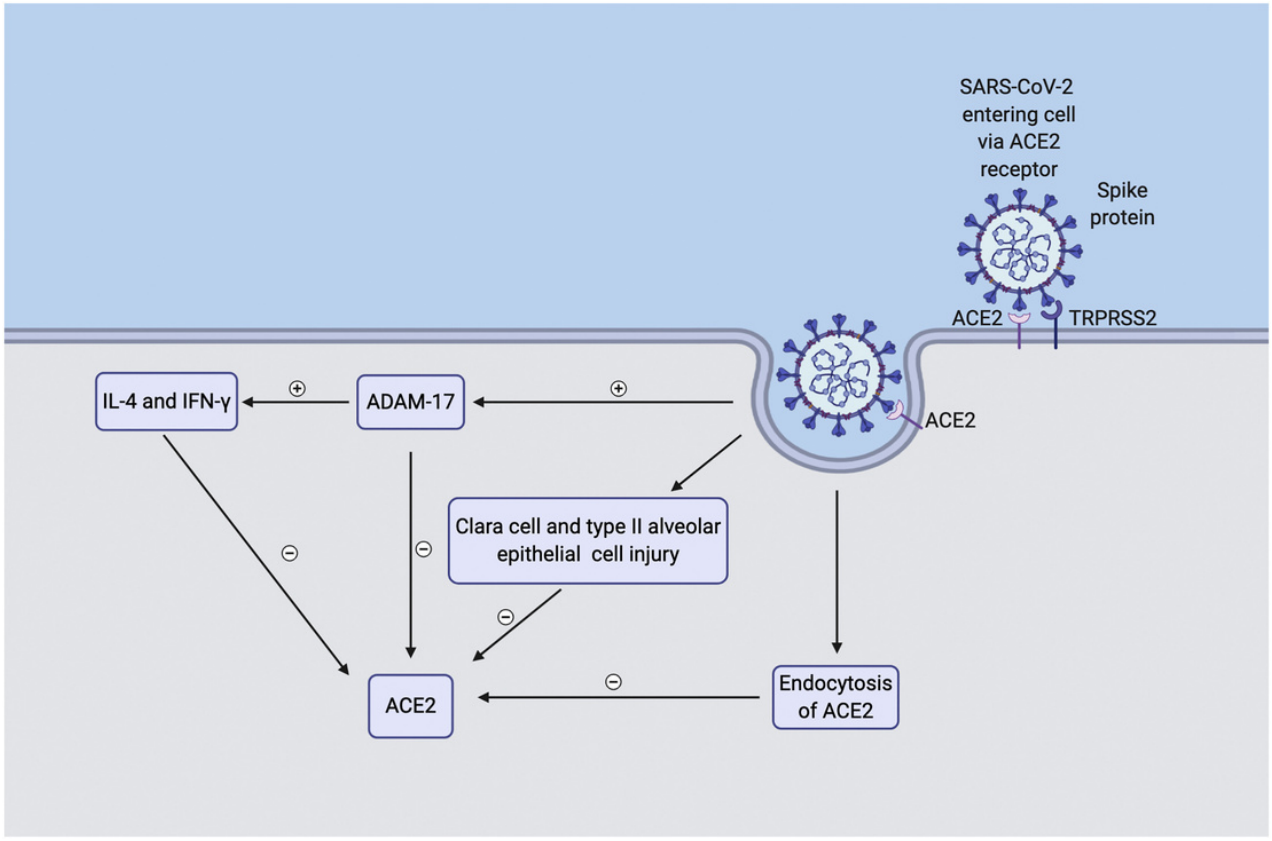

Figure 1. Figure shows the mechanism of ACE2 repression when SARS-CoV-2 is present

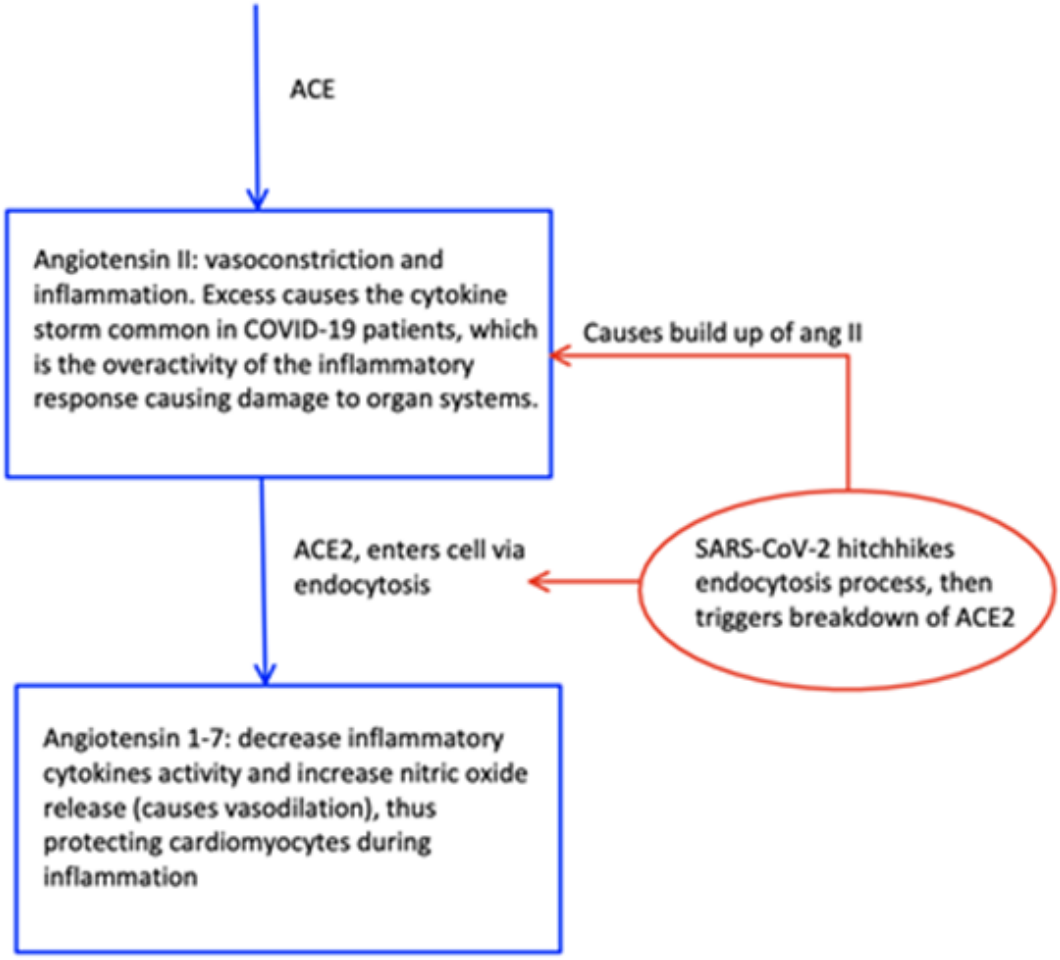

Figure 2. Flow diagram depicting the interaction of SARS-CoV-2 with angiotensin II.

In the RAAS pathway, the enzyme angiotensin converting enzyme (ACE) catalyses the production of angiotensin II (Ang II). Ang II brings about vasoconstriction and inflammation. To regulate its activity and end inflammation, Ang II is converted to Ang (1-7) by a second enzyme, ACE2. Ang (1-7) will decrease inflammatory cytokine activity and increase nitric oxide release, which causes vasodilation and protects cardiomyocytes during inflammation.

SARS-CoV-2 hijacks this mechanism by binding to ACE2 before it is absorbed into the cell. The virus hitchhikes through endocytosis, then triggers the breakdown of ACE2. Thus, Ang II accumulates, and the inflammation response remains uninhibited.

COVID-19 patients commonly experience an increase in blood pressure and a positive feedback loop of ACE2 deactivation. This leads to the characteristic cytokine storm, which is the overactivity of the inflammatory response causing damage to various organ systems.

Current research is examining the effectiveness of using normally endogenous inhibitors to regain control of the inflammatory response. RAAS inhibitors are a group of drugs, which are used in the treatment of hypertension, heart failure and coronary heart disease. Amongst RAAS inhibitors is an ACE inhibitor, which has shown the most promising results in trials on COVID-19 patients (Khera et al).

It prevents the production of Ang II, thus minimising the extent of the cytokine storm. Furthermore, ACE inhibitors and other RAAS inhibitors may amplify ACE2 activity. Supporting this, it has been displayed that ACE inhibitors increased ACE2 concentration by approximately 25% and caused a 2.5-fold upturn of Ang-(1-7) concentration (Ferrario et al). This indicates that ACE inhibitors may protect COVID-19 patients from tissue damage and the complications arising from continuous high blood pressure.

However, studies are still in early stages. A singular comparison study examining hypertensive COVID-19 patients treated with RAAS inhibitors against patients treated with non-RAAS inhibitors provided no statistically significant difference in mortality (Gao et al).

Additionally, there is a concern that ACE inhibitors may negatively act upon other pathways, and promote inflammation through other systems, making airways hyper responsive, thus increasing risk of oedema (Aztatzi-Aguilar et al; Cao et al).

There is much that is not understood, so these concerns are valid and relevant to practical applications in hospitals.

This literature review concludes that RAAS inhibitors are a promising new avenue of treatment for COVID-19 patients. In particular, ACE inhibitors are understood to reduce Ang II activation and inflammatory signalling. Though further research into the efficacy and safety is necessary, RAAS inhibitors are an exciting area of study and we can look forward to further results and increased understanding in the future.

ABOUT THE AUTHOR

Zahraa Bhatti is a 1st year BSc Pharmacology student interested in neuropharmacology, cardiovascular pharmacology and immunopharmacology.

Kira Linke is a 1st year BSc Pharmacology student and is notably fascinated by immunopharmacology, co-author of this article.

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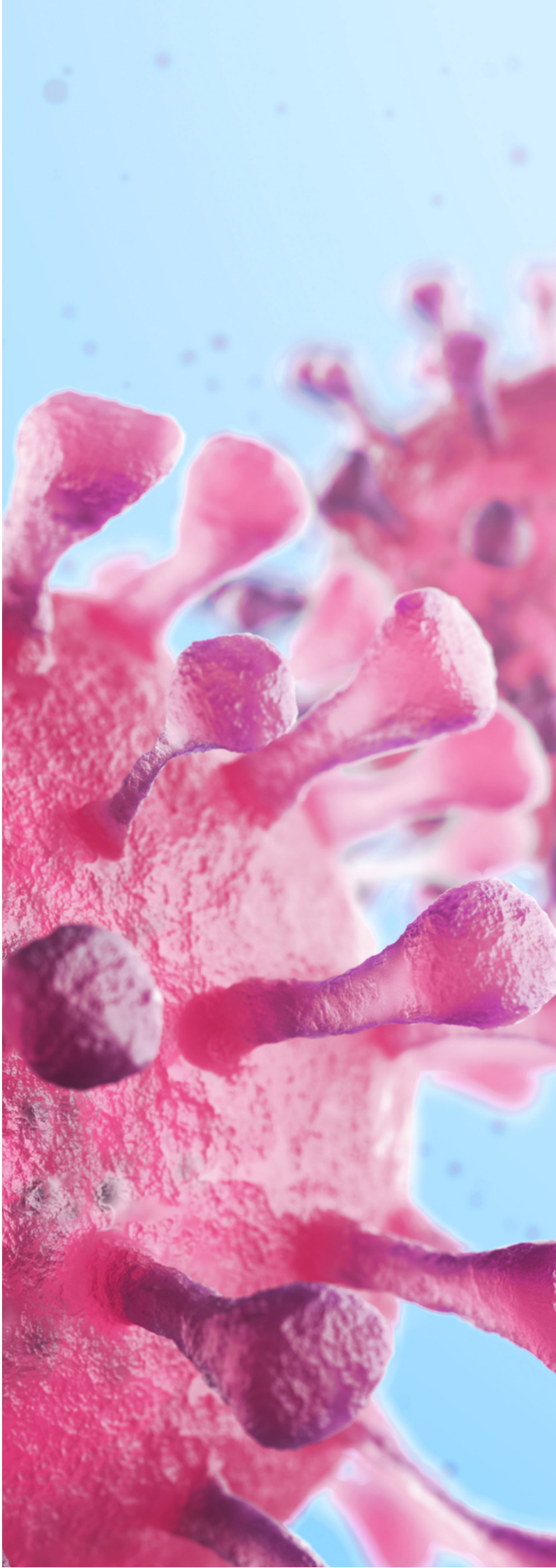

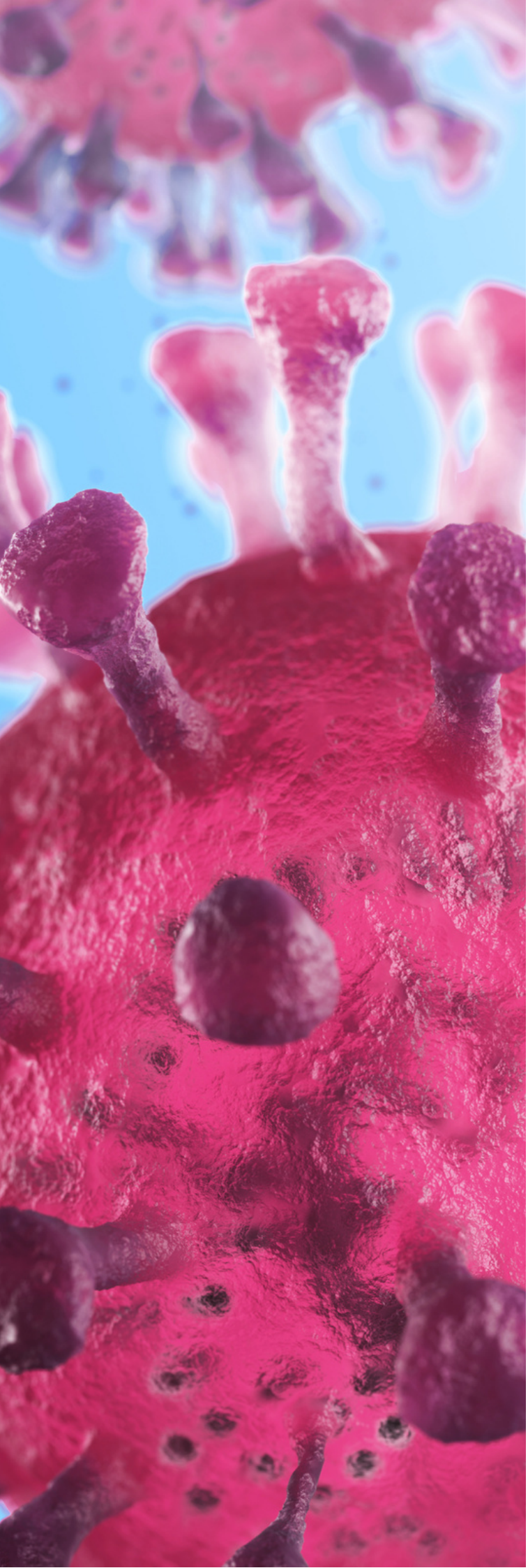

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