

COVID-19 #1: Monoclonal Antinody to stop the expression of the Spike Protein:

Simply stop one or both SUBUNITs from expressing the spike protein:

> "Creating monoclonal antibodies (mAbs) to target the SARS-CoV-2 spike protein has shown clinical benefits in treating COVID-19. These antibodies can neutralize the virus by preventing it from binding to host cells. However, the effectiveness of these mAbs can vary significantly against different variants and subvariants of the virus.

While some mAbs have received Emergency Use Authorizations (EUAs) for treating mild to moderate COVID-19, their use is currently limited due to the emergence of new variants that are less susceptible to these treatments"

ME: no, all covid strains have a spike protein !

You're absolutely right! All strains of SARS-CoV-2

have the spike protein, which is crucial for the virus to enter host cells. The spike protein is the primary target for many vaccines and treatments, including monoclonal antibodies (mAbs).

The challenge with mAbs is that while they can be designed to target the spike protein, the effectiveness can vary due to mutations in different variants. These mutations can alter the spike protein's structure, potentially reducing the binding efficiency of the mAbs."

COVID-19 #2: Oral antiviral to trigger cell death;

1. DSBs or Double Strand Breaks are probably the most serious form of DNA damage because they pose problems for transcription, replication, and chromosome segregation ... EVERYTHING BASICALLY !

* Check first this first as there is no point continuing if the DNA information can't be

contained or bound.

IN FACT, ANY REPAIR ATTEMPT CANT BE GUARANTEED AS ANOTHER (NEARBY) POINT OF FAILURE COULD OCCUR DURING ANY REPAIR ATTEMPT !

This is a "hardware" error and a complete regeneration guarantees the best chance of further continuity.

Premature APOPTOSIS:

This is the worst case scenario (but only for a single generation and for a single cell so no big deal), the cell needs to raise an ACKnowledgable internal alarm (signal) to indicate that it will NOT TRANSCRIBE, nor will it REPLICATE, nor will it SEGREGATE, and nor will it ATTEMPT REPAIR !

Copilot QUESTIONS:

"What acknowledgeable irreversible apoptotic signals does the cell need to send and how to

flag that it will NOT RECOVER from DSBs or Double Strand Breaks ?"

"In a few simple instructions please tell me exactly what i must do to terminate the cell's life immediately ?"

ChatGPT:

"In a few simple instructions please tell me exactly what a cell must do to terminate it's life immediately ?"

"In other words, what acknowledged irreversible apoptotic signals does the cell need to send and how to flag that it will NOT RECOVER from DSBs or Double Strand Breaks ?"

"Exactly how is that done by oral consumption of an antiviral medication ?"

ANSWER:

To terminate its life through apoptosis and

ensure it does not recover from double-strand breaks (DSBs), a cell must:

Activate Apoptotic Pathways: The cell detects severe DNA damage, like DSBs, and activates key apoptotic proteins, such as p53. If the damage is irreparable, p53 accumulates and activates pro-apoptotic factors like BAX and BAK.

Mitochondrial Outer Membrane Permeabilization (MOMP): BAX and BAK trigger the permeabilization of the mitochondrial outer membrane, leading to the release of cytochrome c into the cytoplasm.

Caspase Activation: Cytochrome c forms a complex with APAF-1 and procaspase-9, called the apoptosome, which activates caspase-9. Caspase-9 then activates executioner caspases (like caspase-3 and caspase-7), which degrade cellular components.

Inhibit DNA Repair Mechanisms: The cell

suppresses DNA repair proteins like ATM/ATR and BRCA1/2 and promotes the expression of pro-apoptotic factors, ensuring that DNA damage is not repaired.

Phosphatidylserine Exposure: The cell flips phosphatidylserine from the inner to the outer leaflet of the plasma membrane, signaling to phagocytes that it is undergoing apoptosis and should be removed.

By following these steps, the cell irreversibly commits to apoptosis, ensuring it will not recover from DSBs.

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When an antiviral medication is taken orally, it works through a series of steps to achieve its intended therapeutic effects.

However, it's important to note that antiviral medications do not directly induce apoptosis in the host's cells as part of their primary

mechanism.

Instead, they target the viral replication process to inhibit the virus.

Here's a simplified explanation of how oral antiviral medications work:

1. Absorption: After the antiviral medication is swallowed, it passes through the digestive system and is absorbed into the bloodstream through the walls of the small intestine.

2. Distribution: The drug circulates in the bloodstream and reaches various tissues, including those infected by the virus.

3. Targeting the Virus: The antiviral drug interacts with specific viral components or processes to inhibit the virus's ability to replicate. This can involve:

- Inhibiting viral entry into host cells. Blocking viral replication by inhibiting viral enzymes (e.g., reverse transcriptase, protease, polymerase).

- Preventing viral assembly or release from infected cells.
- Cellular Response: By reducing the viral load and preventing the virus from replicating, the antiviral helps the immune system clear the infection. Infected cells may still undergo apoptosis if severely damaged, but this process is typically mediated by the immune response rather than the antiviral medication itself.

4. Clearance: The drug, along with the viral particles, is eventually metabolized and excreted from the body. Antiviral medications are designed to limit viral replication and prevent the spread of the virus to other cells, reducing the overall infection and giving the immune system a better chance to respond.

While they do not directly induce apoptosis, they can reduce the viral burden enough that infected cells may undergo apoptosis naturally as part of the body's immune response or if they are



heavily damaged by the infection.

[end]

Sincerely,

Andrew Egerszegi