WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

Molnupiravir - Novel promising antiviral used in treatment of COVID-19

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ABSTRACT :

A new antiviral medication called molnupiravir has demonstrated encouraging promise in the management of COVID-19. Molnupiravir, a ribonucleoside prodrug, prevents SARS-CoV-2 from replicating and spreading within the host by inserting mistakes into the viral RNA during replication. This study examines the safety profile, clinical trials, mechanism of action, and pharmacodynamics of molnupiravir as a COVID-19 therapy. It has been shown in early clinical trials to be beneficial in lessening the intensity and length of symptoms, particularly when given early in the course of infection. Additionally, although worries about mutagenicity and long-term effects are still being investigated, molnupiravir has been linked to a generally positive safety profile. This study further highlights molnupiravir's special significance in COVID-19, especially in outpatient settings, by contrasting it with other antiviral treatments currently on the market. To properly evaluate its long-term effectiveness, safety, and possible usefulness in conjunction with other antiviral medications, more research is necessary.

Keywords: Molnupiravir, COVID-19, Antiviral medication, Clinical trials,Safety profileOutpatient settings, Long-term effectiveness, Mutagenicity concerns.

1.INTRODUCTION :

1.1Coronavirus: -

Numerous pathogenic microorganisms, such as bacteria, fungi, viruses, protozoa, and helminths, are encountered by the human body. There are several ways in which these microbes might damage human tissues. Because they have the ability to take over our cells' reproductive mechanism, viruses are unique. In order to thrive in different species, they are also always evolving and adapting [1]. The disease known as COVID-19 is brought on by a novel virus known as SARS-CoV-2. The virus was initially brought to the attention of the World Health Organization (WHO) on December 31, 2019, following reports of many cases of "viral pneumonia" in Wuhan, China[2]. Birds and animals can get sick from a class of viruses called coronaviruses (CoVs). A novel coronavirus sparked a worldwide epidemic in 2019. "Coronavirus Disease 2019" or COVID-19 is the term given to this illness by the World Health Organization (WHO). It was given the new moniker SARS-CoV-2 because of the similarities in its RNA structure to the SARS-causing virus. Within the Nidovirales order, this virus is a member of the Orthocoronavirinae subfamily of the Riboviria family[3] .

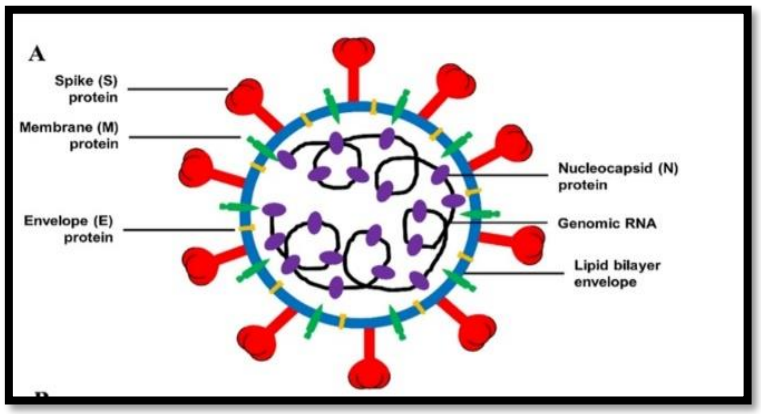

Figure1 schematic structure of coronavirus(22)

1.2 Sign and symptoms of Coronavirus:-

- The most typical COVID-19 symptom is fever.
- The cough is dry.
- The weariness.
- Some people may have other, less frequent symptoms, such as
- A loss of scent or flavor.
- Congestion in the nose.
- Conjunctivitis, commonly referred to as red eyes.
- A sore throat.
- Headache.
- Pain in the muscles or joints.
- Various skin rash kinds
- Nausea or vomiting
- Diarrhea, chills, or lightheadedness

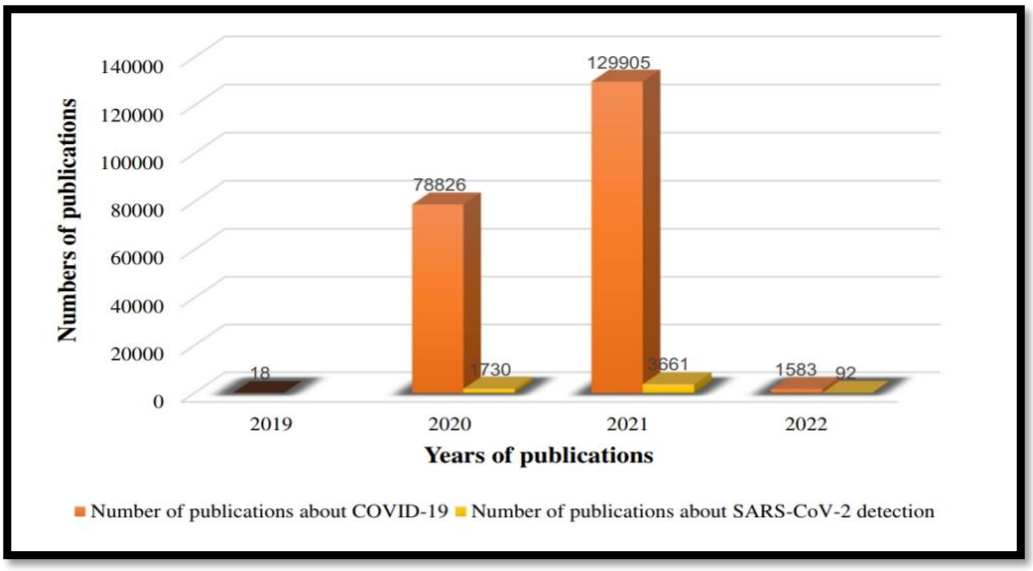

Figure.2 statistics of the number of publication per year related to COVID-19(23)

2. Molnupiravir:

One oral antiviral medication being researched for the treatment of COVID-19 is molnupiravir. It was first created in Atlanta, Georgia, by Drug Innovation Ventures, and then Ridgeback Therapeutics, in collaboration with Merck & Co., USA, purchased it. It was first developed to treat Eastern and Western horse encephalitis viruses, as well as influenza viruses and several encephalitic alpha viruses. In laboratory experiments, the medication demonstrated potent antiviral activity.[4,5]

The FDA approved Molnupiravir for emergency use to treat mild-to-moderate COVID-19 in people who are not in the With little risk of developing medication resistance, molnupiravir is a successful therapy for coronaviruses, including many SARS-CoV-2 variations. 800 mg should be taken orally every 12 hours for five days by adults who are 18 years of age or older. The optimal time to start it is five days after the onset of COVID-19 symptoms. The likelihood of medication interactions is decreased since it is a prodrug, meaning that the body transforms it into its active form. Easy to take at home, molnupiravir doesn't conflict with other long-term drugs.[7]

medical facility The MOVE-OUT trial's Phase II and Phase III investigations provided the basis for this conclusion, demonstrating that the medication reduces the risk of serious illness, hospitalization, or death when administered promptly—that is, within five days of the onset of symptoms. By preventing the virus from growing within the body, it functions. [8]

A culture of human airway cells infected with SARS-CoV-2 (the virus that causes COVID-19) was given molnupiravir in a laboratory investigation. The amount of virus present was decreased and the function of the airway cells was improved by this therapy[9]. Molnupiravir inhibited the replication of SARS-CoV-2 in the Syrian hamster model [11] and human lung-only mice (LoM) [10]. In vivo experiments by Abdelnabi et al. showed a decrease in the viral RNA burden. This research examined the combination of favipiravir and molnupiravir [12]. Molnupiravir kills the virus by increasing the rate of mutations in the SARS-CoV-2 genome [13,14,15].

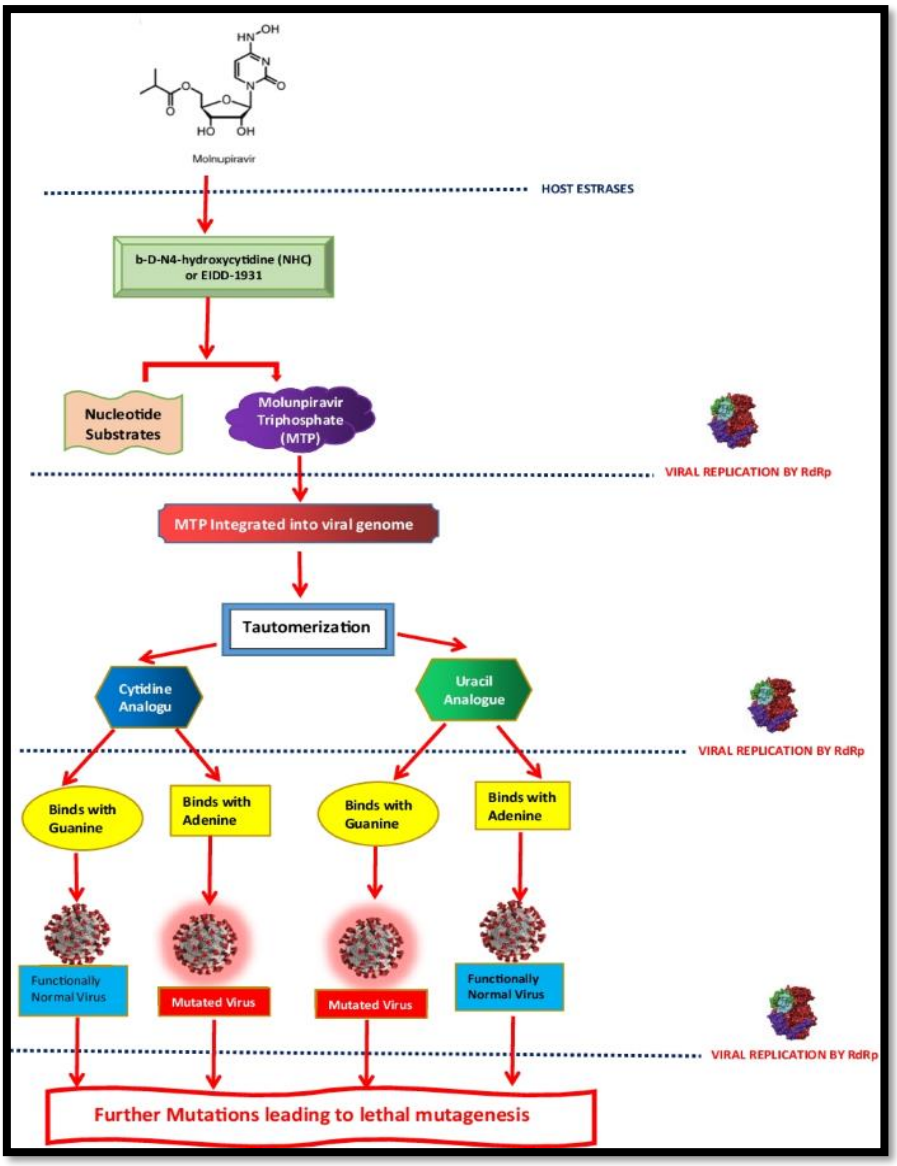

Figure 3 RNA-directed RNA polymerase (RdRp) uses NHC triphosphate as its substrate in place of cytidine and uridine forming stable complexes and forms mutated RNA.(24)

2.1 Structure of Molnupiravir:

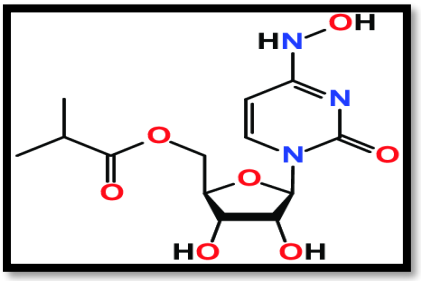

Figure 4. Chemical structure of Molnupiravir

- **IUPAC name:**[(2R,3S,4R,5R)]-3,4-Dihydroxy-5[4-(hydroxyamino)-2-oxopyrimidin-1-yl] oxolan-2-yl] methyl 2-methylpropanoate
- **Molecular formula:** C13H19N3O7
- **Molecular weight:** 329.31g/mol
- **Category:** Antiviral Agent
- **Dose:** 800mg twice a day

- **Description:** Available in white or off-white solid form
- **Storage:** Stored at room temperature between 20C to 25C . Inert atmosphere
- **Solubility:** Soluble in Water and di-methyl sulfoxide (DMSO)
- **Melting point:** 128ºC – 132ºC
- **Boiling point:** 131°C
- **Brand name:** Lagevrio

3. MECHANISM OF ACTION: -

Serving as a ribonucleoside antilog for RNA polymerase, NHC inhibits RdRp as its primary mechanism. The main way that NHC acts as a mutagen is by increasing the frequency of transition mutations (G-to-A and C-to-U), in contrast to Remdesivir, which inhibits RNA replication[16,17]. For NHC-induced RNA mutagenesis, a two-step model is inferred. NHC is phosphorylated by host kinases into its active form, NHC-5′-triphosphate (NHC-TP), after being cleaved in the plasma when it enters the cell[18]. RdRp then incorporates NHC-TP, rather than C or U (with reference to the plus-gRNA template), into the generated minus-gRNA and sub-genomic RNA. Notably, NHC-TP has more competition from C than from U for incorporation. Because of this, the minus-gRNA that contains NHC-TP is causing mutations in the positive-strand RNA products and the development of viruses that are not functional[19].

Prodrug Activation

Molnupiravir is a prodrug of β-D-N4-hydroxycytidine (NHC).
After ingestion, molnupiravir is metabolized into NHC in the body.
NHC is then phosphorylated into its active triphosphate form (NHC-TP).

Oral Administration and Absorption

Molnupiravir (an isopropylester prodrug of β-D-N4-hydroxycytidine (NHC)) is administered orally.
It is absorbed in the gastrointestinal tract and enters the bloodstream.

Hydrolysis to NHC (β-D-N4-hydroxycytidine)

In the body, molnupiravir is hydrolyzed by esterases (enzymes present in tissues and plasma).
This hydrolysis removes the isopropyl ester group, converting molnupiravir into its active metabolite, NHC (β-D-N4-hydroxycytidine).

Phosphorylation to NHC-TP (Active Form)

Inside the host cell, NHC undergoes phosphorylation through the action of cellular kinases in a three-step process:
1. NHC → NHC-monophosphate (NHC-MP) by a nucleoside kinase.
2. NHC-MP → NHC-diphosphate (NHC-DP) by a nucleoside monophosphate kinase.
3. NHC-DP → NHC-triphosphate (NHC-TP) by a nucleoside diphosphate kinase.

The final product, NHC-triphosphate (NHC-TP), is the active antiviral form.

Incorporation into Viral RNA

NHC-TP mimics natural nucleosides (cytidine and uridine) and gets incorporated into the SARS-CoV-2 RNA genome by the viral RNA-dependent RNA polymerase (RdRp).

1.Activation and Cellular Uptake

Molnupiravir is converted into its active form, NHC-triphosphate (NHC-TP), inside the host cell through hydrolysis and phosphorylation.

2. Recognition by Viral RNA Polymerase (RdRp)

NHC-TP mimics the structure of cytidine (C) and uridine (U) nucleotides.
The SARS-CoV-2 RNA-dependent RNA polymerase (RdRp) cannot distinguish NHC-TP from natural nucleotides.
NHC-TP gets incorporated into the growing viral RNA strand during replication.

3. Base Pair Mismatching

After incorporation, NHC can tautomerize between two forms:
Amino form (pairs with Guanosine (G)
Imino form (pairs with Adenosine (A)
This causes base pair mismatching during subsequent rounds of RNA replication.

4. Error Accumulation (Lethal Mutagenesis)

The mismatched base pairs lead to an accumulation of mutations in the viral genome.
With each replication cycle, more errors are introduced, resulting in a phenomenon called "error catastrophe."

5. Loss of Viral Viability

The extensive mutations lead to non-functional viral proteins and defective viral particles.
Eventually, the virus loses its ability to replicate effectively and infect host cells.

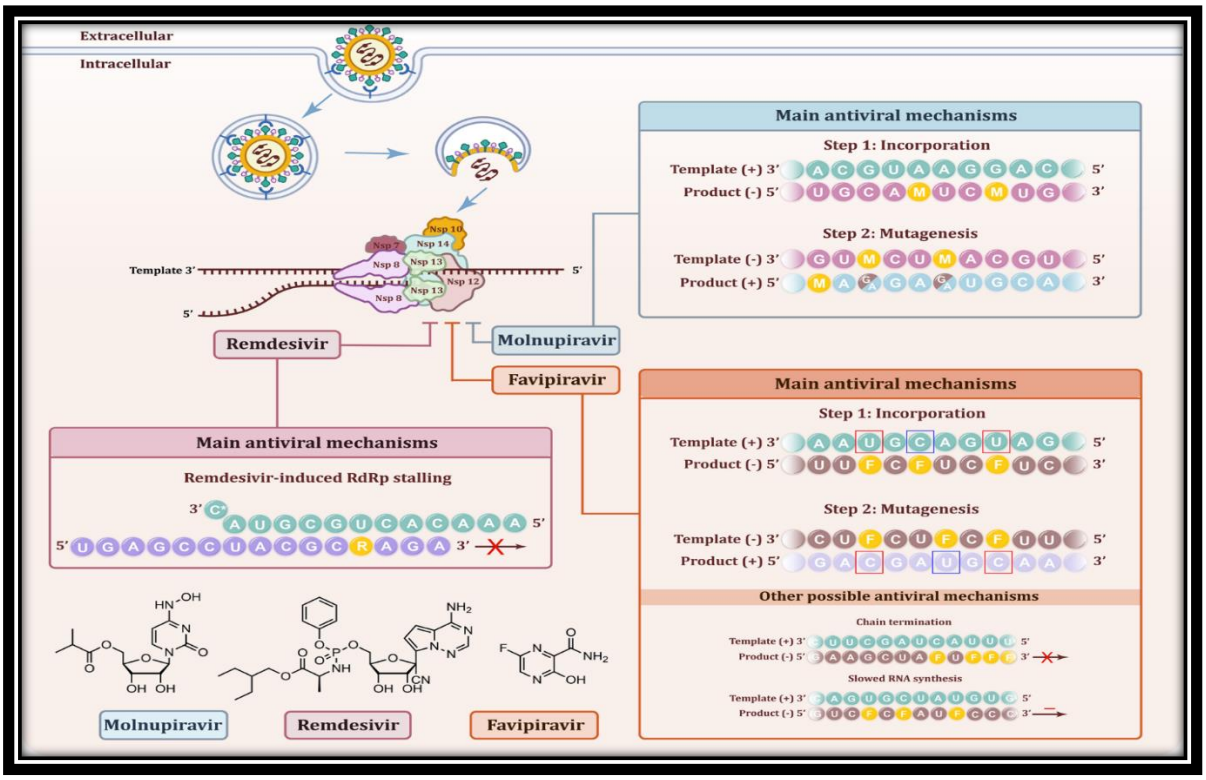

Fig.5: Mechanism of molnupiravir(25)

4. Method of molnupiravir:-

4.1 Concise Synthesis of Molnupiravir

Scale-up from 5 g of uridine

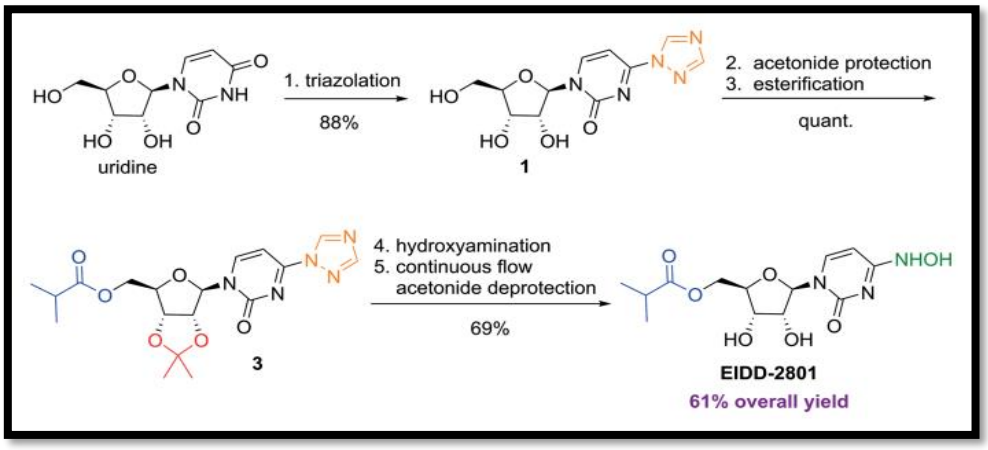

Fig:6. Synthesis Method Of Molnupiravir 1

Synthesis Steps:

1. **Starting Material:** Uridine as the initial nucleoside.
2. **Step 1:** Convert uridine into uridine monophosphate using a phosphate donor(e.g., ATP)

Reaction:

Uridine + ATP —**Uridine Kinase**→ **UMP + ADP**

Uridine Kinase: Catalyzes the phosphorylation of uridine using ATP as the phosphate donor.
ATP (Adenosine Triphosphate): Provides the phosphate group.

1. **Step 2:** Oxidize uridine monophosphate to introduce a hydroxyl group at the 4-position.

   1. Target of Oxidation in UMP

   UMP consists of:

   Uracil (Base)

   Ribose (Sugar)

   Phosphate group at the 5' position of ribose

   Depending on the oxidation conditions, different parts of the molecule can be targeted:

   Ribose Sugar: The hydroxyl (-OH) groups on ribose can be oxidized to carbonyl (-C=O) or carboxylic acid (-COOH) groups.

   Uracil Base: Oxidation can occur at the uracil ring, introducing hydroxyl (-OH) or carbonyl (-C=O) groups.

2. **Step 3:** Catalyze the formation of an ester bond, converting the hydroxylated nucleoside into molnupiravir.

   Step 1: Activation of Isobutyric Anhydride/Chloride

   The isobutyric anhydride or isobutyryl chloride is activated by the base catalyst (e.g., pyridine), facilitating the nucleophilic attack.

   Step 2: Nucleophilic Attack by 5'-Hydroxyl Group

   The 5'-hydroxyl group of NHC (nucleotide analog) acts as a nucleophile and attacks the carbonyl carbon of the isobutyric reagent.

   Step 3: Formation of the Ester Bond

   The reaction forms an ester bond between the 5'-hydroxyl group of NHC and the isobutyryl group.

   Pyridine or TEA neutralizes any acidic byproducts (like HCl if isobutyryl chloride was used).

3. **Purification:** Use chromatography or crystallization techniques to purify molnupiravir.
4. **Final Product:**Molnupiravir as the ester prodrug of β-D-N4-hydroxycytidine (NHC).

2.Enzyme mediated Synthesis

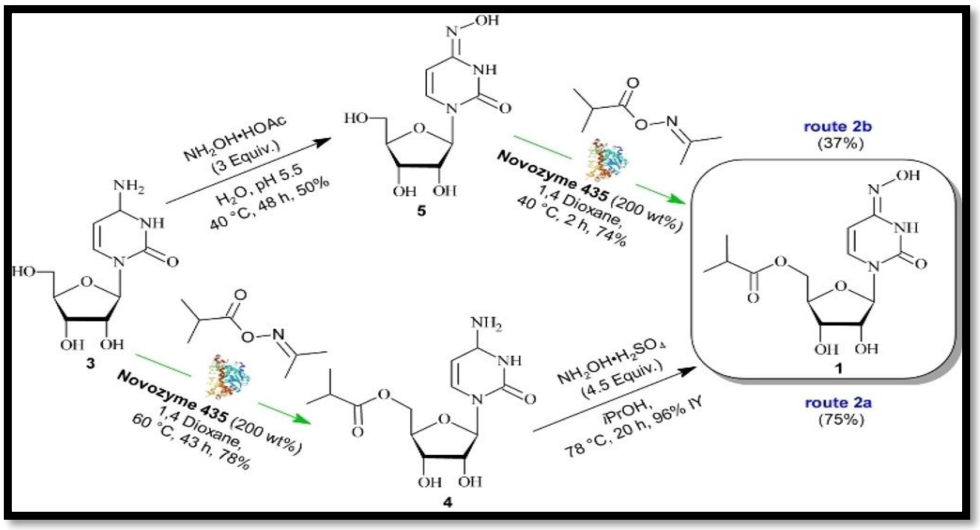

Fig:6. Synthesis Method Of Molnupiravir 2

1. Starting Material

Compound 3 undergoes a reaction with Novozym 435 in 1,4-dioxane at 60 °C for 43 hours, yielding compound 4 with a yield of 78%.

Novozym 435 (CALB) is an enzyme catalyst that facilitates regioselective and enantioselective reactions.

In organic solvents like 1,4-dioxane, lipase enzymes can still maintain their activity while reducing unwanted hydrolysis reactions.

At 60 °C, the enzyme remains stable and active, driving the reaction toward the desired product.

Novozym 435 facilitates the selective acylation of the sugar moiety.

2. Route Divergence: Route 2a and Route 2b

From compound 4, two different reaction pathways are presented:

Route 2a (75% yield)

Compound 4 reacts with $NH_2OH \cdot H_2SO_4$ (hydroxylamine sulfate) in iPrOH (isopropanol) at 78 °C for 20 hours, yielding compound 1 with a high yield of 96%.

1. Role of Hydroxylamine Sulfate ($NH_2OH \cdot H_2SO_4$)

Hydroxylamine sulfate provides hydroxylamine (NH_2OH) under acidic conditions.

Hydroxylamine acts as a nucleophile, typically attacking carbonyl carbons (e.g., ketones, aldehydes, or esters).

The reaction can produce oximes (if attacking aldehydes or ketones) or hydroxamic acids (if attacking esters or amides).

2. Solvent (iPrOH) and Temperature (78 °C)

Isopropanol (iPrOH): Acts as a solvent to dissolve both the reactants and allows efficient mixing and reaction progression.

Scenario 1: Ester to Hydroxamic Acid Conversion

If Compound 4 contains an ester group, hydroxylamine (NH_2OH) can attack the carbonyl carbon of the ester.

1. Nucleophilic Attack:

The lone pair on the nitrogen of NH_2OH attacks the carbonyl carbon of the ester group.

2. Tetrahedral Intermediate Formation:

A tetrahedral intermediate forms, with the carbonyl oxygen becoming an alkoxide.

3. Elimination of Alcohol (iPrOH or R-OH):

The intermediate collapses, releasing the alcohol group and forming a hydroxamic acid.

78 °C: Elevated temperature accelerates the reaction rate, ensuring complete conversion of Compound 4 to Compound 1.

4. Significance of the Reaction

The introduction of a hydroxyimino (C=NOH) or hydroxamic acid (-CONHOH) group often increases biological activity, improves solubility, or modifies reactivity.

The high yield (96%) suggests a clean and regioselective transformation, with minimal side reactions.

5. Final Answer

The reaction of Compound 4 with hydroxylamine sulfate in isopropanol at 78 °C involves a nucleophilic attack by NH_2OH on an electrophilic carbonyl group, leading to either an oxime or hydroxamic acid derivative (Compound 1) in 96% yield.

If you can provide the structure of Compound 4 or more details, I can tailor the mechanism to your specific compound and clarify whether it formed an oxime or a hydroxamic acid

This step involves the formation of an oxime derivative.

Route 2b (37% yield)

Alternatively, compound 4 can be converted to compound 5 using $NH_2OH \cdot HOAc$ (hydroxylamine acetate) in water (pH 5.5) at 40 °C over 48 hours, yielding 50% of compound 5.

Side reactions can be a major cause of inefficiency. In the synthesis of Molnupiravir, if the hydroxylation step or any intermediate is unstable or prone to degradation, unwanted products can form, reducing the desired product yield.

For instance, esterification or transesterification reactions that involve activated reagents (like isobutyric anhydride or isobutyryl chloride) could lead to side reactions (e.g., hydrolysis or formation of unwanted esters).

Catalysts and Reaction Conditions

The enzyme-catalyzed step in Route 2b (if it involves Novozym 435 or a similar lipase) might be less efficient under the specific conditions used (e.g., temperature, solvent, or time). Even though Novozym 435 is a highly effective enzyme, its performance can decrease if reaction conditions are suboptimal.

In Route 2b, the enzyme might be working at a less favorable temperature, leading to longer reaction times or lower conversion rates of intermediates into Molnupiravir. For example, Novozym 435 can be deactivated at higher temperatures or in solvents that are not optimized for the enzyme's stability.

Intermediate Stability

One common challenge in Route 2b could be the instability of intermediates. If intermediate compounds degrade, polymerize, or react undesirably, it could lead to yield loss. For example:

Hydrolyzed intermediates like NHC may degrade during esterification or other modifications, leading to side products or incomplete reactions.

Molnupiravir precursors may be sensitive to certain reaction conditions, resulting in loss of yield during subsequent steps.

Poor Solubility or Reactivity of Intermediates

If intermediates in Route 2b are poorly soluble in the reaction solvent (e.g., 1,4-dioxane, iPrOH, or others), the reactions may proceed with lower efficiency.

Poor solubility can hinder proper mixing of reactants or cause low conversion rates in enzyme-catalyzed reactions. The long reaction time (e.g., 43 hours in one of the steps) may be a response to insufficient reaction progress due to poor solubility or low reactivity of intermediates.

Purification Losses

The purification process in Route 2b could involve complex chromatographic techniques, crystallization, or other methods that result in losses of the desired product. If the product is poorly soluble or similar in properties to side products, significant losses can occur during isolation and purification

steps.

Comparison with Route 2a

Route 2a may be a more efficient pathway due to:

Fewer reaction steps, resulting in higher cumulative yields.

Use of more efficient catalysts, reagents, or reaction conditions that minimize side reactions or maximize the rate of conversion to Molnupiravir.

The 37% yield of Route 2b suggests that this pathway has multiple challenges, including side reactions, enzyme inefficiency, poor solubility, intermediate instability, or purification losses. Route 2a, with a higher overall yield, likely benefits from more efficient reaction steps, better catalysts, fewer side products, or optimized reaction conditions. Improvements in Route 2b could focus on:

Optimizing reaction conditions (temperature, solvent, time) for each step.

Improving catalyst performance, especially in enzymatic steps.

Enhancing intermediate stability and minimizing side reactions.

Better intermediate stability or more robust synthetic steps that lead to higher overall yield.Compound 5 is then treated again with Novozym 435 in 1,4-dioxane at 40 °C for 2 hours, producing compound 1 with a yield of 74%.

The overall yield of Route 2b is 37%, indicating a less efficient pathway compared to Route 2a.

5. Analogue of molnupiravir

5.1 Medical analogue of molnupiravir

1. Favipiravir

Mechanism: A nucleoside analogue that inhibits viral RNA-dependent RNA polymerase (RdRp), leading to errors in viral RNA replication.

Therapeutic Use: Approved for treating influenza and investigated for use against other RNA viruses like Ebola and SARS-CoV-2.

2. Remdesivir

Mechanism: A nucleotide analogue prodrug that incorporates into viral RNA, prematurely terminating RNA synthesis.

Therapeutic Use: Used for treating COVID-19 and effective against a range of RNA viruses.

3.Sofosbuvir

Mechanism: A nucleotide analogue that inhibits hepatitis C virus RNA polymerase.

Therapeutic Use: Approved for chronic hepatitis C infection.

5.2 Explosive analogue of molnupiravir

1. **Chemical Instability Analogy:**

Molnupiravir has reactive chemical groups (like its hydroxylamine moiety) that are essential for its antiviral activity. An "explosive analogue" could refer to a hypothetical modification of its chemical structure, introducing groups that make the molecule thermally or mechanically unstable, potentially leading to explosive properties.

2. **Biological Analogy:**

Molnupiravir acts as a "genetic bomb" for viruses by causing catastrophic mutations in their RNA. The term "explosive analogue" might metaphorically describe an even more potent version of this mutation-inducing mechanism.

3. **Hypothetical Research Context:**

If you're referring to a specific scientific paper or concept, it might describe a compound structurally similar to molnupiravir but with a highly reactive or hazardous functional group, such as azides or nitro groups, which are known to be explosive.

6. Evaluation Parameter of Molnupiravir:

1.Solubility:

Molnupiravir is soluble in Water and organic solvent: -

1. **Water:** Molnupiravir has low to moderate solubility in water. At neutral pH, it dissolves better compared to very acidic or basic conditions.
2. **Organic Solvents:** Soluble in solvents like dimethyl sulfoxide (DMSO) and ethanol.

Limited solubility in non-polar solvents such as hexane.

Buffer Solutions: Its solubility may improve in certain buffer solutions with pH adjustment, typically favoring slightly acidic or neutral conditions.

3. Buffer solubility:

1. **Acidic Buffers (pH < 5):** Molnupiravir shows moderate solubility in acidic conditions. However, extremely acidic pH might reduce solubility due to protonation effects.
2. **Neutral Buffers (pH 6-7):** Molnupiravir has relatively good solubility in this range. Neutral or slightly acidic conditions are often optimal for its dissolution.
3. **Basic Buffers (pH > 7):** Solubility may decrease in basic conditions, as the molecule may not ionize favorably in highly alkaline environments.

2.Stability:

1. Stability in Solid Form

Room Temperature: Molnupiravir is generally stable in its solid form when stored at room temperature under dry conditions.

Light Sensitivity: It is recommended to store it away from direct light to prevent potential photodegradation.

Hygroscopicity: Molnupiravir can absorb moisture from the air, so storage in airtight containers is essential to maintain its stability.

2.Stability in Aqueous Solutions

pH-Dependent Stability:

Acidic pH: Relatively stable in mildly acidic conditions.

Neutral pH (pH 6-7): Generally stable for short periods; however, degradation may occur over time.

Alkaline pH: Stability decreases in basic environments due to hydrolysis.

Temperature Sensitivity: Higher temperatures accelerate degradation in solution, so refrigerated conditions (2-8°C) are preferable for storage of aqueous preparations.

3.Chemical Stability

Molnupiravir undergoes degradation through hydrolysis and is ultimately converted to its active form, β-D-N4-hydroxycytidine (NHC), under physiological conditions.

The hydrolytic stability is influenced by the pH of the medium and the presence of enzymes, such as esterases, which facilitate its conversion to NHC.

5.Storage Recommendations

Solid Form: Store in a cool, dry place, ideally below 25°C, in sealed and light-protected containers.

Aqueous Preparations: Should be prepared fresh or stored under refrigeration for short durations to maintain stability.

4. Shelf Life

Molnupiravir has a defined shelf life in its solid dosage form, typically around 24 months when stored under recommended conditions. However, aqueous solutions are less stable and should be used promptly.

5. Melting point:

The reported melting point of molnupiravir is approximately 128–132°C. This value may vary slightly depending on the purity of the sample and the measurement method used.

6. Boiling point: Molnupiravir, being a complex organic compound, does not have a well-defined boiling point as simple liquids do. Instead, it decomposes before reaching a boiling point, as is common for many pharmaceutical compounds. (Approx.131^{o}c)

7. pKa (acid and base properties):

1. Solubility: The pKa suggests that molnupiravir is more soluble in slightly acidic to neutral environments, while solubility may decrease in highly basic conditions.

2. Stability: pH-dependent degradation is influenced by the ionization state, so the pKa is critical for optimizing formulation and storage conditions.

8. Reactivity:

1. Reactivity in Biological Systems

Hydrolysis to Active Form: Molnupiravir is a prodrug that undergoes enzymatic hydrolysis (primarily by esterases) to form its active metabolite, β-D-N4-hydroxycytidine (NHC). This reaction occurs efficiently in vivo and is crucial for its antiviral activity.

2.Reactivity in Aqueous Solutions

pH Sensitivity:

Stable in mildly acidic and neutral conditions.

Temperature Sensitivity:

Increased reactivity at elevated temperatures due to faster hydrolytic and degradation processes.

1. **Chemical Reactivity**

Ester Group: The ester bond in molnupiravir is reactive and undergoes hydrolysis under physiological conditions to release the active metabolite.

Hydroxyl Groups: These groups may participate in hydrogen bonding, influencing solubility and reactivity in different environments.

2. **Reactivity with Other Substances**

Oxidizing/Reactive Agents: Molnupiravir is relatively stable under normal handling conditions but may degrade in the presence of strong oxidizing agents or under extreme pH conditions.

Compatibility with Excipients: Care is needed in formulation to avoid reactions with excipients that could alter its stability or efficacy.

Brand products:

MOLNUTOR 200

Fig.7:Molnutor 200Capsulkes

- **Medicine Name**: MOLNUTOR-200
- **API**: Molnupiravir
- **Dosage form and steangth:** Capsules – 200mg
- **Storage:** Store below 30ºC
- **Manufactured By:** Torrent Pharmaceutical Ltd.

2.MOLAZ

Fig.7:MOLAZ 200mgCapsules

- **Medicine Name:** MOLAZ
- **API:** Molnupiravir
- **Dosage form and steangth:** Capsules – 200mg
- **Manufactured By:** Azista Pharmaceuticals Ltd.

3.MOLUNAMX

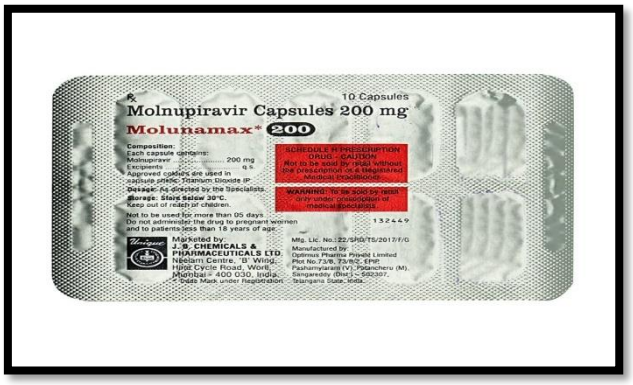

Fig.7:Molnumax-200 Capsules

- **Medicine Name:** Molunamax-200
- **API:** Molnupiravir
- **Storage:** Stored below 30ºC
- **Manufactured By:** J.B. Chemicals & Pharmaceutical Ltd.

4.Movir:

Fig.7: Movir Capsules

- **Medicine Name:** Movir
- **API:** Molnupiravir
- **Dosage form and steangth:** Capsules 200mg
- **Storage:** Stored below 30ºC
- **Manufactured By:** REScuers life Science Ltd.

Result and Discussion:-

After conducting a detailed review of the drug Molnupiravir, I have observed that effective Treatment for Coronavirus (COVID-19). Half-life: Molnupiravir has a short plasma half-life (~0.9 hours), while NHC has a longer half-life (~3–4 hours). It work as the antiviral drug used for threatment of viral infection. In term of physical properties, the Melting Point Of Molnupiravir has 128ºC - 132ºC. It has does not effective Boiling Point. It has suitable and stable for pharmaceutical uses. Molecular formula of Molnupiravir C13H19N3O7. Confirm solubility and suitability for long term storage. Molnupiravir is an oral antiviral medication initially developed for treating influenza and encephalitic alpha viruses. It was later repurposed for COVID-19 treatment due to its potent activity against SARS-CoV-2. It works by introducing lethal mutations into the viral RNA genome, disrupting viral replication and limiting infection progression. Molnupiravir effectively bypasses the proofreading exonuclease (ExoN) activity, which normally corrects RNA errors in coronaviruses.

Conclusion:

Molnupiravir is a notable antiviral agent demonstrating significant promise in treating COVID-19 due to its unique mechanism of action, which involves inducing lethal mutagenesis in the SARS-CoV-2 genome. Clinical trials and studies have highlighted its effectiveness in reducing the severity of symptoms, especially when administered during the early stages of infection, and its suitability for outpatient treatment. The drug's safety profile is relatively favorable, although concerns about long-term effects, particularly mutagenicity, remain areas for further investigation.

Despite its benefits, continued research is crucial to comprehensively evaluate molnupiravir's long-term safety and efficacy, its role in combination therapies, and its comparison with other antiviral treatments. This understanding will optimize its use and address concerns regarding its potential side effects.

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