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**Original Research Article**

# EXPLORING *HEMIDESMUS INDICUS AS* A HYPOLIPIDEMIC AGENT: A COMPARATIVE STUDY WITH ROSUVASTATIN IN RATS

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**Abstract:**
In the perpetual quest for novel drugs characterized by heightened efficacy and diminished side effects, statins have emerged as potent yet not entirely side-effect-free agents for hyperlipidaemia. Herbal remedies, celebrated for their cost-effectiveness and minimal adverse effects. Among them, *Hemidesmus indicus* (*H. indicus*) stands out due to its recognized hypolipidemic activity and wide prevalence in India. This study aims to scrutinize the efficacy of *H. indicus* as a hypolipidemic agent, comparing it with Rosuvastatin in a cholesterol diet-induced hyperlipidaemia rat model.

**Methods**: Hyperlipidaemia was induced in male albino rats of the Wistar strain during the initial 30-day feeding period, persisting through the subsequent 30-day treatment phase. The aqueous root extract of *H. indicus* (administered at 50 and 100 mg/kg, per oral) served as the test drug during the treatment period, while Rosuvastatin (10 mg/kg, per oral) functioned as the standard drug. Serum lipid profile, atherogenic index, and body weights were assessed on the day preceding the feeding period commencement and on days 0, 15, and 30 of the treatment period. Statistical analysis involved student's unpaired and paired t-tests where applicable.

**Results:** Significant reductions ($p < 0.001$) in TC, TG, LDL-C, and VLDL-C, coupled with substantial elevations ($p < 0.001$) in HDL-C, were observed in both the Rosuvastatin and test groups. However, the Rosuvastatin group exhibited higher percentage reductions in lipid

**ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021**

levels, percentage elevations of HDL-C, and protection from atherosclerosis compared to the test groups.

**Conclusions:** *H. indicus* manifests a distinct hypolipidemic potential. While its effectiveness trails behind Rosuvastatin, its beneficial role as a hypolipidemic agent needs clinical exploration.

**Keywords:** Hypolipidemic, *Hemidesmus indicus*, Rosuvastatin, Blood Cholesterol, Hyperlipidaemia

## 1. INTRODUCTION

Hyperlipidaemia stands as a pivotal precursor to atherosclerosis and its associated conditions, including coronary heart disease (CHD), ischemic cerebrovascular disease, and peripheral vascular disease (1). Decades of research have solidified dyslipidaemia as a paramount risk factor for atherosclerotic cardiovascular disease (CVD) (2). The prevalence of CHD in India has surged significantly over the years, underscoring the urgency for effective interventions (3). Pharmacological interventions, especially statins, have played a pivotal role in combating cardiovascular diseases. While statins exhibit remarkable efficacy and are widely used globally, concerns about statin intolerance and potential side effects, such as diabetes mellitus, cancer, and memory loss, persist (4). Safety issues, including transaminase and creatinine elevation, skeletal muscle pain, and creatine kinase elevation, have sparked a need for alternative hypolipidemic agents with enhanced efficacy and fewer adverse effects (5).

### The Role of Medicinal Plants:

Medicinal plants continue to be integral to healthcare, offering advantages in terms of effectiveness, safety, affordability, and acceptability (6). *Hemidesmus indicus* (*H. indicus*), an Indian medicinal plant entrenched in ayurvedic traditions, spans the tropical regions of India. While known for its blood purifying, health tonic, brain tonic and hepatoprotective properties, its potential as a hypolipidemic agent, particularly in non-diabetic rats, remains underexplored. The plant's cost-effectiveness, widespread availability, and low toxicity further accentuate its appeal (7).

### Objective of the Study:

This study aims to elucidate and evaluate the hypolipidemic efficacy of *H. indicus* in a cholesterol diet-induced hyperlipidaemia rat model. Besides its proven health benefits, the investigation seeks to provide comprehensive insights into the plant's potential as a lipid-lowering agent, addressing the current gap in non-diabetic rat studies.

## 2. MATERIAL AND METHODS

**Study Location:** The current investigation performed in the Animal House, Department of Pharmacology, Oriental College of Pharmacy. The focal point of this research was to appraise the hypolipidemic potential of the aqueous root extract of *H. indicus* in rats exhibiting cholesterol diet-induced hyperlipidaemia.

**ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021**

**Extract Preparation:** Fresh roots of *H. indicus* were meticulously obtained from Munnalal Dawasaaz, Hyderabad, AP, India. The taxonomic estimation was done by Prof. P. Jayaraman, Director, Institute of Herbal Botany, Plant Anatomy & Research Centre, Chennai, TN, India. The isolated roots underwent a meticulous process involving chopping into small pieces, followed by shade drying at room temperature for seven days. The dried roots were then finely powdered. This resultant powder served as the foundation for the preparation of the aqueous extract, employing a heat distillation process (8). The resulting extract received the codename AEHE (Aqueous Extract of *Hemidesmus Indicus*).

**Animal Procurement and Housing:**

Healthy male albino wistar rats, 18 weeks old and weighing 180-200 grams, were sourced from the national institute of biosciences Pune, India. These rats were carefully maintained under optimal laboratory conditions, ensuring a conducive environment for the study.

**Experimental Setup and Animal Handling:**

**Animal Housing Conditions**:

Rats were housed in well ventilated room (temperature 23± 2 $^{0}$C, humidity 45-60% and 12 h light/dark cycle at animal house. All Animals were fed with standard pellet diet and had access to water ad libitum, with the exception of the experimental periods. The institutional animal ethics committee approved the experimental protocol. The research was conducted in strict accordance with the norms established by the Committee for the Purpose of Control and Supervision of Experiment on Animals (CPCSEA) and adhered to the guidelines outlined in the National Institute of Health's "Guide for the care and use of laboratory animals."

**Grouping:**

**Selection and Randomization:**

Following the methodology outlined by Ghule et al (9), thirty healthy male Albino rats, matched in body weight, were chosen. Randomization led to the formation of treatment and control groups, with all rats undergoing a one-week acclimatization phase in the laboratory settings. The rats were then allocated into five groups, each consisting of six animals.

**Study Duration:**

The study spanned over 2 months, encompassing a 30-day feeding period and an additional 30-day treatment period, with continuous feeding throughout (9).

**Dietary and Experimental Phases:**

**Feeding Period:**

**Group I (Normal Control):** Served as the normal control and received standard rat feed pellets throughout.

ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021

**Groups II, III, IV, and V:** Were subjected to a high-fat diet for 30 days during the feeding period, which persisted into the subsequent 30 days of the treatment phase. Rats in these groups had access to food and water ad libitum.

**Treatment Period:**
**Group I (NC):** Normal control receiving normal saline (5ml/kg, per oral) daily for 30 days.

**Group II (Negative):** Hyperlipidaemic control receiving normal saline (5ml/kg, per oral) daily for 30 days.

**Group III (Std):** Standard drug control receiving Rosuvastatin (10 mg/kg, per oral) daily for 30 days.

**Group IV (Test 1):** Test group receiving AEHE (50 mg/kg, per oral) daily for 30 days.

**Group V (Test 2):** Test group receiving AEHE (100 mg/kg, per oral) daily for 30 days.

**Hyperlipidaemia Induction:**
High Cholesterol Diet (HCD): The induction involved a high cholesterol diet comprising deoxycholic acid (5 g), cholesterol (5 g), coconut oil (300 ml), and standard rat feed pellets (700 g). The diet components were meticulously mixed to create a soft mixture, which was administered to rats during the specified periods.
**Dietary Administration:** Homogeneous Cake Supply: To ensure uniformity, homogeneous cakes were diligently prepared and daily provided to rats in each cage in ample quantities (10).
**Monitoring and Calculation of Body Weight:**
Body weights were precisely recorded on the day before the commencement of the feeding period and on days 0, 15, and 30 of the treatment phase (9).
The total weight gain was computed as follows:
Total weight gain on day 30 = Final body weight–Initial body weight
**Drug Treatment:** In the treatment period, test groups received a daily single dosage of AEHE dissolved in normal saline (5 ml/kg), administered orally through an oral gavage procedure for 30 days. Control groups were administered normal saline alone. The standard group received orally dissolved rosuvastatin at a dosage of 10 mg/kg/day in 5 ml/kg normal saline. The selected doses for AEHE and rosuvastatin (Reddy labs Pvt Ltd, Hyderabad) were determined based on previous studies demonstrating hypolipidemic activity (7,10,11). All doses were administered between 10-11 am.
**Blood Sampling and Serum Lipid Analysis:**
**Sampling Procedure:**
Blood samples were consistently collected within a one-hour window between 9:00 am and 10:00 am. Fasted blood samples, obtained under light ether anaesthesia through retro-orbital puncture, were collected on the day preceding the feeding period and on days 0, 15, and 30 of the treatment period (9).

**ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021**

**Lipid Profile Analysis**:

Serum lipid profiles were assessed on the specified days. After allowing blood samples to clot for 30 minutes, serum was separated via centrifugation at 3,000 rpm for 5 minutes using a centrifuge and transferred to sterile 1.5 mL centrifuge tubes. Commercial kits and an autoanalyzer (Lablife Robochem, RFCL Ltd.) were utilized following manufacturer directions for analyzing serum total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL-C), low-density lipoproteins (LDL-C), and very low-density lipoproteins (VLDL-C). The estimation of triglycerides, total cholesterol, and HDL-C employed enzymatic glycerol-3-phosphate oxidase (GPO-ESPAS) and Cholesterol oxidase/peroxidase (CHOD-PAP) methods (12). VLDL-C was calculated as one-fifth the level of TG using the empirical equation of Friedwald (13). LDL-C levels were determined using the Friedewald method, subtracting HDL-C from TC.

**Calculation of Lipoprotein Levels and Indices:**

*VLDL and LDL Calculation:*

$VLDL = TG / 5$

$LDL = TC - (HDL + VLDL)$

**Percentage Change Calculation***:* Percentage change from initial values (day 0 of the treatment period) for serum lipid levels and body weights on days 15 and 30 were calculated using the formula:

**Atherogenic Index (AI) Calculation:**

Atherogenic index (AI) was calculated as AI=(total serum cholesterol/total serum HDL)(14)

Percentage Protection from Atherosclerosis Calculation:

Protection (%)=(difference in AI between control and treated groupAI of control)×100Protect ion (%)=(AI of control difference in AI between control and treated group)×100

**Statistical Analysis:**

Data Presentation: Results were expressed as mean ± standard deviation (SD) for each group, with n=6.

**Statistical Tests:** Statistical differences between control and treatment groups were assessed using student's unpaired and paired t-tests, as applicable.

**Significance Levels:** Values were considered significant at $p < 0.05$ and $p < 0.01$; highly significant at $p < 0.001$.

## 3. RESULTS

**Effects of Aqueous Root Extract of *H. Indicus* and Rosuvastatin on Rats:**

Administration Regimen: Aqueous root extract of *H. Indicus* (50/100mg/kg, p.o. once daily) and rosuvastatin (10 mg/kg, per oral, once daily) were administered, and the following effects were observed.

**Effect on Serum Lipid Levels:**

*Comparison with Normal Group:* On day 0 of the treatment period, the hyperlipidemic control group, rosuvastatin group, and test groups exhibited a significant increase ($p<0.001$*p*$<0.001$) in total cholesterol, triglyceride, LDL-C, VLDL-C levels and a significant decrease ($p<0.05$*p*$<0.05$) in HDL-C levels (Table 1).

ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021

**Table 1: Serum lipid levels and atherogenic index (AI) in various groups on Day 0, 15 and 30**

| Groups | Day | C (mg/dl) | TG (mg/dl) | HDL-C (mg/dl) | VLDL-C (mg/dl) | LDL-C (mg/dl) | AI |
|---|---|---|---|---|---|---|---|
| **Normal Control** | 0 | 103.93±14.17 | 84.32±8.12 | 40.84±2.91 | 17.66±1.63 | 47.41±16.13 | **2.61±0.52** |
| | 15 | 103.92±13.57 | 78.50±8.71 | 41.27±2.18 | 16.89±1.74 | 46.71±11.63 | **2.53±0.30** |
| | 30 | 101.77±8.94 | 78.49±7.99 | 40.93±4.81 | 15.36±1.21 | 43.49±10.53 | **2.51±0.41** |
| **Negative Control** | 0 | 291.80±18.89 | 212.18±15.96 | 41.29±2.91 | 42.63±3.18 | 205.47±19.18 | **6.59±0.93** |
| | 15 | 293.04±18.9 | 210.97±11.35 | 38.77±4.38 | 42.38±2.27 | 205.69±21.01 | **6.88±0.97** |
| | 30 | 279.23±15.14 | 209.81±9.52 | 39.01±4.43 | 41.57±1.91 | 197.16±18.37 | **6.84±0.71** |
| **STD** | 0 | 291.28±13.86 | 215.57±10.32 | 42.29±4.97 | 43.12±2.06 | 205.88±12.87 | **6.98±0.82** |
| | 15 | 197.53±15.67 | 158.27±16.01 | 51.78±2.33 | 32.65±3.20 | 98.62±15.00 | **3.03±0.38** |
| | 30 | 168.33±15.73 | 116.05±11.08 | 54.97±4.10 | 24.41±2.23 | 71.70±15.11 | **2.27±0.23** |
| **TEST-1** | 0 | 294.67±16.41 | 203.15±12.52 | 42.14±7.17 | 40.84±2.50 | 212.70±19.67 | **7.01±1.23** |
| | 15 | 273.29±13.2 | 187.82±13.87 | 49.49±4.82 | 37.37±2.75 | 187.44±14.66 | **5.65±0.55** |
| | 30 | 254.50±12.91 | 168.70±15.34 | 54.30±2.60 | 34.94±3.07 | 168.26±14.41 | **4.72±0.30** |
| **TEST-2** | 0 | 296.80±14.26 | 201.42±13.28 | 38.12±4.31 | 41.48±2.66 | 217.19±14.19 | **7.68±0.96** |
| | 15 | 246.08±19.90 | 171.03±16.16 | 51.49±2.47 | 35.01±3.23 | 157.59±19.03 | **4.69±0.48** |
| | 30 | 222.95±19.48 | 145.50±11.28 | 61.57±3.08 | 28.30±2.25 | 132.31±19.54 | **3.61±0.43** |

Data expressed as Mean+SD of 6 observations. Student's unpaired t-test:
* Values are statistically highly significant at $p<0.001$when compared to normal control (N)
# Satistically highly significant at $p<0.001$when compared to hyperlipidemic control (H)

ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021

**Table 2: Mean percentage change (%) from day 0 values of serum lipid levels in different groups on day 15 and 30.**

| | Day | Mean percentage change (%) | | | | |
|---|---|---|---|---|---|---|
| | | C | TG | HDL-C | VLDL-C | LDL-C |
| **Normal Control** | 15 | 0.22 | 4.53 | 1.43 | 4.61 | **4.63** |
| | 30 | 3.01 | 7.68 | 0.03 | 7.67 | **1.26** |
| **Negative Control** | 15 | 0.56 | 6.67 | 8.11 | 0.31 | **0.13** |
| | 30 | 3.84 | 2.28 | 5.36 | 2.27 | **3.49** |
| **STD** | 15 | 31.81 | 25.67 | 22.58 | 25.67 | **52.95** |
| | 30 | 41.15 | 44.75 | 34.59 | 44.77 | **65.05** |
| **TEST-1** | 15 | 7.83 | 8.48 | 14.55 | 8.47 | **12.73** |
| | 30 | 12.16 | 16.83 | 26.74 | 16.86 | **18.92** |
| **TEST-2** | 15 | 17.79 | 16.06 | 36.21 | 16.04 | **27.37** |
| | 30 | 26.22 | 26.62 | 57.11 | 27.63 | **39.99** |

Data expressed as mean of 6 observations

**Table 3: Atherogenic index (AI) in different groups onday 30.**

| Groups | Atherogenic index | Protection (%) |
|---|---|---|
| **Normal Control** | 2.51 | - |
| **Negative Control** | 7.3 | - |
| **STD** | 3.02 | **59.55** |
| **TEST-1** | 4.81 | **34.74** |
| **TEST-2** | **3.61** | **51.84** |

Data expressed as mean of 6 observations

**Table 4: Weight gain in different groups on day 30 ascompared to day 0.**

| Groups | Weight gain (gm) |
|---|---|
| **Normal Control** | **6.67±2.57** |
| **Negative Control** | **14.17±3.77 *** |
| **STD** | **7.6±2.71 #** |
| **TEST-1** | **10.1±4.46 @** |
| **TEST-2** | **9.17±3.75 $** |

Data expressed as Mean+SD of 6 observations. Student's unpaired t-test:* Highly significant difference when compared to normal control (N), $p<0.01$.#Highly significant difference when

**ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021**

compared to Hyperlipidemic control, $p<0.01$.@ No significant difference when compared to Hyperlipidemic control, $p>0.05$.
Comparison with Hyperlipidaemic Group: On day 30 of the treatment period, the Rosuvastatin group, Test group A, and Test group B displayed a significant decrease ($p<0.001p<0.001$) in total cholesterol, triglyceride, LDL-C, VLDL-C levels and a significant increase ($p<0.001p<0.001$) in HDL-C levels (Table 1).

**Comparison with Day 0 of Treatment Period (Intragroup):** A highly significant decrease in total cholesterol, triglyceride, LDL, VLDL levels, and a significant increase ($p<0.001p<0.001$) in HDL-C levels were observed within each group by day 30 of the treatment period (Table 1, Figure 3).
These findings suggest a positive impact on lipid levels, showcasing the potential of *H. Indicus* and Rosuvastatin in mitigating hyperlipidaemia.

**Percentage Change in Lipid Levels:**
**Observations on HDL Levels:**
A notable increase in HDL levels was evident in the rosuvastatin group and both test groups on day 15 ($p<0.001$ $p<0.001$) and day 30 ($p<0.001$ $p<0.001$) within the same group

**Comparative Analysis on Day 30:**
When the test groups were compared with the rosuvastatin group on day 30, they exhibited a relatively lower percentage reduction in serum total cholesterol, triglyceride, LDL-C, and VLDL-C. However, it is noteworthy that test group B demonstrated a greater percentage change in certain lipid levels.
These results indicate varied effects on lipid profiles, with some distinctions between the test groups and the rosuvastatin group, particularly notable in the case of test group B.

**Effect on Weight Gain:**
**Day 30 Observations:** On day 30, the hyperlipidaemia group displayed a significant increase ($p<0.01p<0.01$) in weight gain, whereas both the rosuvastatin group and test group B exhibited a significant decrease ($p<0.001p<0.001$) in weight gain. Notably, test group A showed no significant decrease ($p>0.05p>0.05$) in weight gain (Table 4).

## 4. DISCUSSION

**Nutritional Role in Hyperlipidaemias and Atherosclerosis:** The study emphasizes the established connection between nutrition and the development of hyperlipidemias and atherosclerosis. The hyperlipidemic diet employed in this study, comprising deoxycholic acid, cholesterol, coconut oil, and chow, is recognized for inducing atherogenic changes in the lipoprotein profile, elevating LDL-C levels, and reducing HDL-C levels(1).
*Hypercholesterolemia Induction Mechanism:* The mechanism involves the interference of chow-based diets supplemented with cholesterol and sodium cholate, inducing hypercholesterolemia in rodents. The addition of cholic acid contributes to this process by

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increasing cholesterol absorption and suppressing cholesterol 7α-hydroxylase activity, subsequently limiting cholesterol excretion. Cholic acid's emulsifying property enhances cholesterol absorption (15,16,17,18).

**Comparative Analysis of Rosuvastatin and H. indicus Extract (AEHI)***:* While both rosuvastatin and AEHI exhibited significant lipid-lowering effects and increased HDL-C levels, AEHI demonstrated a lesser percentage change in lipid parameters, except for HDL-C, compared to rosuvastatin. This discrepancy may be attributed to the distinct mechanisms of hypolipidemic action between *H. indicus* and rosuvastatin. Unlike statins, *H. indicus* is not known to impact de novo cholesterol synthesis, which statins regulate by blocking the HMG-CoA reductase enzyme. Rosuvastatin, known for its efficacy in lowering LDL-C, operates through this mechanism, reporting reductions of up to 63% with a daily dose of 40 milligrams (19).

**Mechanism of AEHI on Serum Cholesterol:** The exact mechanism by which AEHI reduces serum cholesterol remains unclear. However, the observed increase in HDL-C levels is proposed as a potential mechanism for **AEHI** lipid-lowering effect. Elevated HDL-C facilitates the transport of triglycerides or cholesterol, contributing to the decrease in serum lipid levels by AEHE

**Limitations and Future Directions:** This study lays the groundwork for understanding the hypolipidemic potential of *H. indicus*. However, further investigations are required to elucidate the precise mechanisms underlying its action on serum cholesterol and to explore its potential in clinical applications.

The study suggests that AEHI observed increase in HDL-C levels may be linked to the mobilization of cholesterol from peripheral cells to the liver through the action of Lecithin Cholesterol Acyl Transferase, a key player in the 'reverse cholesterol transport' pathway. Flavonoids present in *H. indicus* root extracts are known to enhance LCAT activity, facilitating the incorporation of free cholesterol into HDL and subsequently increasing HDL-C levels. reducing total cholesterol. Tannins are reported to boost endothelium-bound lipoprotein lipase activity, leading to triglyceride hydrolysis.

**Comparison with Rosuvastatin:** Biochemical estimations reveal that both AEHI and rosuvastatin increase protective HDL-C levels while decreasing atherogenic LDL-C and VLDL-C levels. Although AEHI demonstrates a greater elevation of HDL-C compared to rosuvastatin, the percentage protection from atherosclerosis remains similar. This is advantageous, particularly in treating hypercholesterolemia among populations, such as Indians, where low HDL-C is prevalent. AEHI significant reduction in total cholesterol, LDL-C, and atherogenic index without causing mortality or adverse effects in rats underscores its efficacy as a hypolipidemic agent.

*Considerations on Atherosclerosis Predict

**ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021**

**The compelling evidence of AEHI hypolipidemic and cardioprotective potential**, combined with its traditional use as a cardioprotective agent, strongly advocates for its clinical testing in the treatment of hyperlipidaemia and associated cardiovascular disorders. The findings from this study provide a robust foundation for further exploration of *H. indicus*'s therapeutic applications, holding promise for its potential integration into clinical practices.

Further clinical trials and research are essential to validate and expand upon these initial positive results, ensuring a more comprehensive understanding of its therapeutic benefits and safety in human applications.

## 5. CONCLUSION

Clinical Potential of H. indicus: The findings strongly indicate that Hemidesmus indicus (*H. indicus*) possesses definite hypolipidemic, cardioprotective, and antiatherosclerotic potential. Its traditional application aligns with the observed effects on lipid profiles, highlighting its suitability for clinical testing in the treatment of hyperlipidaemia and associated cardiovascular disorders.

*Scientific Basis for Traditional Use:* The study provides a valid scientific basis for considering *H. indicus* for clinical benefits in cardiovascular disease treatment in India. The observed hypolipidemic and cardio-protective effects supports the traditionally claimed benefits of *H. indicus*.

Future Directions: While the present study supports the potential therapeutic role of *H. indicus*, further investigations are deemed necessary to strengthen these findings. Future studies should include extensive case-control studies to document its therapeutic application in human beings, providing a more comprehensive understanding of its clinical efficacy and safety profile.

In summary, the study underscores the promising role of *H. indicus* in addressing hyperlipidaemia and associated cardiovascular concerns, paving the way for future research and potential integration into clinical practices.

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Ethical approval: The study received approval from the Institutional Ethics Committee.

**ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021**

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**ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021**


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