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Research on the Mechanisms of Mushroom Polysaccharides Against Colorectal Cancer

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Abstract. Colorectal cancer has high occurrence and death rates, and it brings great troubles to medical treatment. Natural polysaccharide active materials are now a hot research topic, for they have special power to fight cancer cells. Mushroom polysaccharides are the main active parts of food and medical mushrooms. They win more and more attention as they work in many ways against colorectal cancer. Some mushroom polysaccharides can work directly on tumor cells. They can make cells die naturally and stop cell division. They also block epithelial–mesenchymal transition (EMT) to slow tumor growth and stop its spread. They can also affect key parts of cancer root cells. They stop these cells from keeping active and prevent cancer from returning completely. Besides, these polysaccharides work better together to fight cancer. They change the immune environment around tumor cells and adjust immune cell states. They help the body release more immune materials and keep different gut bacteria balanced. They also make the gut's protective layer work well and link immune control with environmental improvement. Now, using mushroom polysaccharides in real medical work still has many problems. It is hard for human bodies to absorb them, and it is not easy to produce them in standard ways. The results of this study offer useful ideas for further research on their working principles and future medical use.

Keywords: mushroom polysaccharides, colorectal cancer, antitumor activity, cell apoptosis

1. Introduction

Colorectal cancer is one of the most common cancers around the world. It has high occurrence and death rates, and it greatly harms people's health and lives [1]. Nowadays, doctors mainly use surgery, chemical treatment and radiation therapy to cure colorectal cancer. But these common treatment methods cannot target tumor cells well. They bring serious side effects, and cancer cells will gradually resist the medicine [2]. These problems make the treatment less effective, and patients are hard to recover well. So many researchers are trying to find safe, effective and precise natural drugs to fight cancer, and this has become a key direction in colorectal cancer research.

Mushrooms are natural materials that can be used as food and medicine. They contain active substances called mushroom polysaccharides. These substances cause little harm to the human body. They work well and can fight many kinds of cancers [3]. For these reasons, scientists and medical workers have paid much attention to mushroom polysaccharides. Many studies prove that

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mushroom polysaccharides can slow down the growth of colorectal cancer cells in various ways. They can make cancer cells die naturally [4] and stop the cell growth cycle. They also control the self-cleaning process inside cells. Besides, these substances can fight cancer through ways related to intestines. They protect the inner protective layer of intestines and change the living environment of tumor cells.

Mushroom polysaccharides are useful for treating colorectal cancer, and their chemical structures decide how well they work in the body. This paper sums up the main ways how mushroom polysaccharides act on colorectal cancer cells. They cut off the energy supply of tumor cells, cause tumor cells to die, control the cell self-cleaning process and adjust the functions related to intestines. This study aims to build a good foundation for developing mushroom polysaccharides and putting such natural anticancer substances into real medical use.

2. Research background

2.1. Current incidence and therapeutic challenges of colorectal cancer

Colorectal cancer develops step by step and involves many genes. Its appearance and growth are closely linked with genetic conditions, eating habits and unbalanced intestinal environment [5]. As people's living habits have changed in recent years, more people suffer from colorectal cancer, and the disease also tends to affect younger people [6]. Today, removing tumors through surgery is still the main treatment for colorectal cancer. But for patients in the middle and late stages, surgery alone cannot clear out all tumor tissues. In this case, extra treatments like chemical therapy and radiation therapy are needed. Common cancer drugs such as capecitabine and fluorouracil can kill tumor cells, yet they also cause great harm to normal body cells. Patients may feel sick, vomit or suffer from problems with blood cell growth, which makes their daily life much harder [7]. Besides, long-term chemical treatment will make tumor cells resist the drugs gradually. The treatment effect will get worse, and the cancer may come back and spread to other parts of the body. This trouble has become a big obstacle in the treatment of colorectal cancer [8]. So developing new anticancer drugs that cause little harm and work well, as well as finding more treatment methods, is of great value for medical care and the whole society.

2.2. Research progress and chemical structural characteristics of mushroom polysaccharides

Mushroom polysaccharides can dissolve in water. People extract them from fungi including shiitake, reishi, Inonotus obliquus and chanterelles. They are the main active substances that bring medicinal value to mushrooms. Researchers across the world have carried out more studies on this field since the 1960s. At that time, people first found that lentinan has the effect of fighting tumors. Later studies prove that these polysaccharides can not only stop tumor growth, but also perform many other useful functions. They can adjust immune ability, ease inflammation, remove harmful substances and lower blood sugar levels [9].

The chemical structure of mushroom polysaccharides decides how they work in the body. Polysaccharides from different kinds of fungi have obvious differences, and these differences create their unique functions. Most polysaccharides that can fight tumors share a basic structure. Their main chain is β-(1→3)-D-glucan, and this part is very important for activating immunity and resisting tumors [10]. For example, lentinan is a straight chain of D-glucose, and the units are mainly connected by β-(1→3) bonds [11]. But polysaccharides from reishi have a more complex structure. Besides the main β-(1→3) chains, they also contain β-(1→4) linkages in some parts [12].

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Side chains also affect the actual effects of these polysaccharides. These side chains are mostly connected through β-(1→6) glycosidic bonds. Their length, quantity and arrangement will greatly change the water solubility and overall activity of the substances. Take lentinan for example. Every five glucose units on its main chain connect with two glucose side chains via β-(1→6) bonds [13]. This well-proportioned branch structure lets lentinan dissolve easily and work well against tumors. Generally speaking, too few or too many branches will make the active effects weaker [14]. Molecular weight is another key structural factor. The molecular weight of mushroom polysaccharides is usually from 10^3 to 10^8 Da [15]. If the molecular weight is too high, the polysaccharides will hardly dissolve in water. The human body cannot absorb and use them well, so their functions are limited. On the contrary, polysaccharides with too low molecular weight cannot keep a stable shape. They will fail to combine closely with target parts in cells, and their effects will become much weaker [16].

Besides the main structural features mentioned above, some mushroom polysaccharides also contain a small number of non-glucose units, such as mannose, galactose and xylose. These extra sugar units change the chemical properties and biological functions of polysaccharides, so they work in special ways. Chanterelle branched mannan (CC2a) is a typical example. It is mainly made of mannose units and has a special branched structure. The way it fights tumors is totally different from common β-glucan polysaccharides. It plays an anticancer role mainly by adjusting the production of proteins related to cell death [17].

3. Antitumor mechanisms of mushroom polysaccharides

3.1. Disruption of tumor cell energy metabolism

Tumor cells mainly use anaerobic glycolysis to produce ATP and other substances to support their rapid growth. Mitochondria still play an essential role, for they can supply part of the ATP needed for basic cell activities. Inside cancer cells, mitochondria no longer focus on efficient energy production. They help cells relieve oxidative pressure and complete material synthesis, so tumor cells can survive and keep growing. This proves that mitochondria are still vital to the metabolic activities of cancer cells.

Mushroom polysaccharides can disturb the normal function of mitochondria in tumor cells. Lentinan, for example, has a targeted effect on CD133 positive ($CD133^+$) colon cancer cells. CD133 is a special protein on cell surfaces. It acts as a cancer-related gene and is also a typical marker for cancer stem cells (CSCs) [18]. After $CD133^+$ HCT 116 cells are treated with lentinan, reactive oxygen species (ROS) build up in mitochondria and cause oxidative damage to cells [19]. Lentinan also lowers the activity of genes that control key enzymes in the tricarboxylic acid (TCA) cycle, leading to a large accumulation of intermediate metabolites. Meanwhile, it reduces certain components of the mitochondrial respiratory chain, which directly weakens oxidative phosphorylation [18].

As a result, tumor cells have to rely on low-efficiency anaerobic glycolysis to maintain normal metabolism. This metabolic change not only cuts down ATP production, but also increases the release of lactic acid [20]. The accumulated lactic acid makes the environment around tumors acidic. Such an acidic environment damages tumor cells and the nearby tissues. All these effects work together to slow down the growth and spread of tumors.

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3.2. Induction of tumor cell apoptosis

3.2.1. Activation of the intrinsic mitochondrial apoptotic pathway

Cells have two main ways to carry out programmed death. One relies on mitochondria inside cells, and the other depends on special receptors on cell surfaces. Mitochondria take a leading part in the inner cell death process [21].

Researchers did tests on human colon cancer cells named HCT-116. They found polysaccharides from *Agrocybe cylindracea* can slow down the oxidative phosphorylation process. More reactive oxygen species (ROS) will gather inside cells. The mitochondria will lose their normal electric state and get damaged. All these problems finally cause cancer cells to die. A substance called N-acetylcysteine (NAC) can clear away ROS and stop such changes. This proves the polysaccharides work mainly by breaking the normal function of mitochondria with ROS [22].

Lentinan comes from shiitake mushrooms. It can cause programmed death in HT-29 human colon cancer cells, and this change is closely linked to reactive oxygen species (ROS). After cells are treated with lentinan, the amount of ROS inside cells rises greatly. More ROS will start the cell death process related to mitochondria. It increases the level of Bax protein which causes cell death, and cuts down Bcl-2 protein which protects cells from dying [23]. The changed ratio of these two proteins breaks the normal electric state of mitochondria. The membrane also becomes easier for substances to pass through. Then cytochrome c (CytC) inside mitochondria flows into cell fluid. CytC will activate a group of proteins and start a continuous chain reaction. In the end, cancer cells die completely. The effect becomes stronger when people use more lentinan [21].

Polysaccharides extracted from *Inonotus obliquus* (IOP) work on SW620 human colorectal cancer cells. They raise the level of Bax and activated Caspase-3 proteins that lead to cell death. At the same time, they reduce Bcl-2 protein which prevents cell death. In this way, IOP successfully makes cancer cells die [24].

Chanterelle branched mannan (CC2a) can effectively cause programmed death in LS180 human colon cancer cells. The level of BAX gene does not drop much, but the BCL2 gene and its related protein are largely held back. This makes the ratio of Bax to Bcl-2 rise sharply, which is a clear sign that the mitochondrial cell death pathway is activated. Besides, CC2a treatment breaks more DNA inside LS180 cells. This fact fully shows that CC2a has a strong ability to kill cancer cells [25].

3.2.2. Activation of the extrinsic death receptor apoptotic pathway

There is another important way to cause cell death, which works through cell surface receptors. When tumor necrosis factor receptor 1 (TNFR1) connects with its matching substance TNF-α, this pathway will start to work. But the combination of TNFR1 and TNF-α also makes NF-κB move into the cell nucleus. It will boost the production of substances that stop cell death and hold back Caspase 8. On the whole, this process prevents cells from dying. Whether the cells finally die or not depends on the competition between these two opposite effects.

When HT 29 human colon cancer cells are treated with lentinan, the level of TNF-α rises sharply. This activates the TNF-α/TNFR1 pathway. Meanwhile, lentinan stops NF-κB from moving into the nucleus. So Caspase 8 is turned on, and it further activates Caspase 3 that carries out cell death. In the end, the cancer cells go through programmed death [21].

Research on chanterelle polysaccharide CC2a clearly shows that limiting NF-κB can help trigger cell death. CC2a can stop NF-κB from entering the nucleus and keep it in the cell fluid. In this way, NF-κB can no longer slow down the cell death process. The research did not check the early signals

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of the receptor pathway. Still, the test results show that cell death becomes much stronger while NF-κB loses its activity. This matches the rule that blocking the anti-death effect of NF-κB will make cells tend to die. All these results prove that mushroom polysaccharides can adjust the balance of NF-κB signals and speed up cell death via the receptor pathway [25].

Inonotus obliquus polysaccharides (IOP) can activate two pathways at the same time. One is the NF-κB pathway, which can be seen from the increased phosphorylation of NF-κB p65. The other is the NLRP3 inflammasome, for the levels of ASC, Caspase-1 and NLRP3 all go up. Even so, the inflammatory substances IL-6 and TNF-α in the later stage are reduced. These results mean that IOP can adjust the balance between NF-κB and NLRP3 signals. It changes the working state of the classic pathway and works with other factors to cause cancer cells to die [26].

3.2.3. Cell cycle arrest

Cells grow and multiply as their DNA replicates accurately, and they divide equally under a complete cell cycle. The S phase is the only stage where DNA is produced. The normal running of this stage decides whether cells can enter the G2/M phase and finish cell division. Mushroom polysaccharides can hold back the growth of tumor cells by blocking the S phase of the cell cycle. For example, when HT-29 human colon cancer cells are treated with Agaricus bisporus polysaccharides, the cells can pass the G0/G1 phase normally but get trapped in the S phase. As a result, the number of cells in the S phase rises greatly. This further makes chromatin gather closely, breaks cell nuclei and forms apoptotic bodies, and finally causes cell death [27].

Chanterelle branched mannan (CC2a) also has a strong effect to block the cell cycle. In LS180 cells, CC2a makes most cells stay in the G0/G1 and S phases, and greatly cuts down the number of cells in the G2/M phase. Blocking cells at these two stages directly stops DNA synthesis and cell growth. It also works together with the cell death pathways mentioned above, including the unbalanced ratio of Bax and Bcl-2. In the end, all these changes lead cancer cells to die [25].

3.3. Mechanisms of autophagic cell death induced by mushroom polysaccharides

Autophagy is a common metabolic activity inside cells, and it works like a double-edged sword. Proper autophagy breaks down abnormal proteins and damaged cell parts to help tumor cells stay alive. But if autophagy becomes too strong or out of order, it will break the inner balance of cells and lead to cell death caused by overactive autophagy. Mushroom polysaccharides have unique advantages. They can effectively adjust abnormal autophagy and act on tumor cells through two main ways, which also provides many new directions for cancer treatment.

3.3.1. Induction of excessive autophagy in tumor cells

The whole process of autophagy is known as autophagic flux. It includes several continuous steps: cells form autophagosomes, these structures fuse with lysosomes, and the substances inside cells get broken down. Whether this process runs smoothly directly decides how autophagy affects cell survival. Mushroom polysaccharides can make tumor cells produce excessive autophagy. They work by adjusting key signal paths related to autophagy, increasing proteins that start autophagy, and making the whole autophagic process run completely.

Mammalian target of rapamycin (mTOR) is a major substance that stops autophagy from starting. Its activity directly controls the formation of autophagosomes. When cells have enough growth factors and nutrients, mTOR will be activated. It changes the state of autophagy-related proteins

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ULK1 and ATG13 [28], so autophagy is suppressed. On the contrary, when cells are short of nutrients or oxygen, AMP-activated protein kinase (AMPK) will take effect. It acts as a sensor to check cell energy and blocks the activity of mTOR, so autophagy gets promoted [29].

Two main signal paths can control mTOR, which are the PI3K/Akt/mTOR path and the AMPK/mTOR path. Lentinan can trigger autophagy in HT-29 cells by blocking the PI3K/Akt/mTOR path. Specifically, lentinan reduces the content of phosphoinositide 3-kinase (PI3K) in HT-29 cells. It also stops PI3K from changing the state of its downstream substance Akt. In this way, p-AKT cannot activate mTOR any longer, and autophagy is finally started. The AMPK/mTOR path can also arouse autophagy in tumor cells. Studies show that lentinan greatly increases the level of phosphorylated AMPK (p-AMPK) in HT-29 cells. It restrains mTOR activity and removes its suppression on autophagy, so autophagy takes place in these cells [21].

The formation of autophagosomes also relies on Beclin1, a key protein that helps carry out autophagy. In normal cells, Beclin1 binds with Bcl-2 and Bcl-XL, and this combination keeps autophagy under control. Lentinan can activate c-Jun N-terminal kinase (JNK) in HT-29 cells [30]. It changes the state of Bcl-2 and Bcl-XL, so these proteins can no longer combine with Beclin1. Beclin1 is then released and activates autophagy afterwards [31]. Likewise, extracts from *Ganoderma lucidum* can raise the level of Beclin1, so as to slow down the growth of colorectal cancer cells [32].

3.3.2. Blockade of normal autophagy in tumor cells

Tumor cells rely on normal autophagic activity to survive. This process breaks down abnormal substances inside cells and reuses nutrients to keep metabolism stable. Once this key process is interrupted, harmful materials will build up in tumor cells and finally lead to cell death. Mushroom polysaccharides and their related substances can fight cancer. They affect key steps of autophagy and break the normal autophagic cycle inside tumor cells.

Ganoderma lucidum polysaccharides (GLP) are important active substances. They can block normal autophagy in tumor cells, and plenty of studies have confirmed their working mechanisms. With the help of special detection methods, researchers found that GLP directly prevents autophagosomes from fusing with lysosomes in colorectal cancer (CRC) cells. A great many autophagosomes pile up abnormally, which causes cancer cells to die. The MAPK/Erk signal pathway is activated to bring about such changes [33]. Further research shows that after CRC cells are treated with GLP, the number of dead cells rises obviously. Meanwhile, the levels of autophagy-related proteins p62 and LC3-II also increase. Under this condition, more autophagosomes are produced, yet they cannot be broken down normally. At the same time, MAPK/ERK becomes more active, while mTOR and AMPKα are restrained. These results clearly prove that GLP disturbs autophagic activity by activating the MAPK/ERK pathway [34]. Scientists also carried out tests on CRC cell lines including HT-29 and HCT116. The results show GLP works on autophagy at specific stages. 3-methyladenine can stop autophagy at the early stage, and it will reduce the cell death caused by GLP. On the contrary, chloroquine acts on the late stage and blocks the function of autophagosomes and lysosomes. It can make GLP work better against cancer. These findings further prove that GLP mainly takes effect in the late fusion stage of autophagy. Besides, experiments on living bodies show GLP can effectively slow tumor growth and block autophagy in tumor tissues. It is promising to be used as an autophagy inhibitor for cancer treatment [35].

Besides Ganoderma lucidum polysaccharides, muscarine G is another active substance from mushrooms. It regulates autophagy in a special way. Zhou et al [36]. found that muscarine G has two different effects on autophagy in SW480 colorectal cancer cells. In the early stage of autophagy, it

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helps form autophagosomes and starts the autophagic process. But in the later stage, it stops autophagosomes from combining with lysosomes and blocks the whole autophagic flow. As a result, toxic substances accumulate inside cells, and the growth of SW480 cancer cells is greatly controlled.

3.4. Regulation of intestinal-related mechanisms

3.4.1. Protection of the intestinal mucosal barrier

Mushroom polysaccharides mainly act on the connecting structures of intestinal cells. β-glucans can greatly raise the content of two connecting proteins ZO-1 and Claudin-1. They also push cells to release more secretory immunoglobulin A (SIgA), so the physical protection of the intestinal wall gets stronger [19]. Research shows lentinan (LNT) can increase the level of the connecting protein Occludin. It repairs damaged intestinal tissues and recovers the normal protective function of cell connections in the intestines [37]. Ganoderma lucidum polysaccharides (GLP) also raise Occludin content through the same pathway, which further tightens the links between cells. The increase of these connecting proteins can reduce the penetration of the intestinal wall. It also stops lipopolysaccharide (LPS) from moving across cells, and relieves inflammation all over the body [38]. At the same time, mushroom polysaccharides serve as nutrients for good gut bacteria. They help beneficial bacteria such as *Lactobacillus* and *Bifidobacterium* grow in large numbers, and these bacteria will produce short-chain fatty acids like butyrate. Butyrate acts through the GPR41/43 signaling pathway. It indirectly increases ZO-1 and Claudin-1, and strengthens the protective structure of intestinal cells. In this way, a beneficial cycle is formed between the metabolism of gut bacteria and the protection of the intestinal wall [39].

3.4.2. Regulation of the colorectal tumor microenvironment (TME)

Mushroom polysaccharides can change the working state of immune cells in the environment around tumor tissues. Research proves that lentinan can turn resting M0 macrophages into M1 macrophages which fight against tumors. This change raises the release of IL-12 and TNF-α, and meanwhile restrains M2-type tumor-related macrophages (TAMs) that help tumors grow [39, 40]. Besides, lentinan blocks the IL-6/STAT3 signal pathway. It lowers the activity of this pathway in colorectal cancer cells and eases the immune-suppressed environment caused by inflammation [41]. Tests done outside living bodies show that Ganoderma lucidum polysaccharides can stop NF-κB from being activated. They cut down the production of inflammatory substances like IL-1β and IL-6, so the body can regain the ability to monitor abnormal cells [42]. Polysaccharides extracted from Hericium *coralloides* also work to limit NF-κB signals and further block the inflammatory pathways linked with tumors [43]. By adjusting several signal pathways together, mushroom polysaccharides completely change the tumor environment. It turns from an inflammatory state into an anti-inflammatory one, and changes from suppressing immunity into supporting the body's immune system.

3.4.3. Inhibition of tumor invasion and metastasis

Studies find that polysaccharides taken from mushrooms can directly stop colorectal cancer cells from invading nearby tissues and spreading to other body parts. Researchers have studied Agaricus bisporus and discovered its polysaccharides can greatly slow down the movement and invasion of

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HT-29 cells. These substances raise the content of E-cadherin and cut down Vimentin, so they block the epithelial-mesenchymal transition (EMT) inside tumor cells [44].

Polysaccharides obtained from *Sanghuangporus* can also prevent SW480 and HCT-116 cancer cells from moving and invading, and they work better on SW480 cells. These substances act on the Wnt/β-catenin pathway. They hold back the overactive state of this signal chain, reduce the buildup of β-catenin in cell nuclei and make this protein move to the cell surface. As a result, the follow-up signals of the Wnt pathway become weaker. They also adjust the proteins related to EMT: they increase E-cadherin and reduce N-cadherin and Vimentin. Besides, these polysaccharides lower the content and working ability of matrix metalloproteinase-9 (MMP-9). This stops the breakdown of substances around cells and further keeps cancer cells from invading surrounding tissues [45].

Polysaccharides from Ganoderma lucidum, also known as reishi mushroom, can obviously slow down the movement and invasion of colorectal cancer cells. Results from common cell tests show that their blocking effect is almost as strong as the chemotherapy drug capecitabine [46].

In short, all these results prove that mushroom polysaccharides use several similar ways to fight cancer cells. They adjust the Wnt/β-catenin pathway, reverse the EMT process and restrain MMP-9. These effects work together to stop colorectal cancer cells from moving and invading other parts of the body.

4. Conclusion

In short, mushroom polysaccharides are natural active substances that can fight tumors in many ways. They have unique strengths and great application potential for preventing and treating colorectal cancer. Their main working modes include joint regulation on multiple targets, immune adjustment and intestinal environmental balance. What these substances can do is largely decided by their own chemical structures. Mushroom polysaccharides can control key signal paths like PI3K/AKT, Wnt/β-catenin and NF-κB, so they directly slow down the growth and spread of cancer cells. Meanwhile, they can cause cancer cells to die. They raise oxidative damage triggered by ROS, break the normal function of mitochondria, and start the two major cell death pathways. They can also either block the autophagic process or bring about harmful excessive autophagy. In terms of immunity, mushroom polysaccharides can activate macrophages, natural killer (NK) cells and T cells to regulate the body's immune system. Besides, they act as prebiotics to adjust intestinal bacteria, strengthen the protective layer of intestinal walls and repair the abnormal environment around colorectal tumors. It is worth noting that their effects rely heavily on detailed structural features. Polysaccharides taken from shiitake mushrooms, Ganoderma lucidum, chanterelles and other fungi work in quite different ways and affect distinct signal paths to fight cancer.

Now relevant research is in a key period moving from laboratory experiments to practical medical use. To turn mushroom polysaccharides from natural materials into usable clinical medicines, future research needs to dig deeper into their working mechanisms, improve drug delivery methods, carry out clinical tests and set up unified quality management rules. It is very important to develop tiny capsules and micro-particles that target the colon. Such carriers can help the drugs stay stable in the digestive tract and act more accurately on tumor tissues. It is also necessary to make unified rules for extraction, purification and detection, so that all product batches can keep the same quality. All these steps will greatly help promote the clinical use of mushroom polysaccharides. On the whole, mushroom polysaccharides are valuable natural medical resources for colorectal cancer treatment. They cause little harm to human bodies and can target tumor cells precisely. Their value in cancer prevention, auxiliary treatment and tumor environment adjustment still needs more research and development.

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