Manitoba Prostate Cancer SUPPORT GROUP

Newsletter

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Medical Advisors

Paul Daeninck M.D.
Medical Oncologist

Darrel Drachenberg M.D. Urologist

Arbind Dubey M.D.
Radiation Oncologist

Piotr Czaykowski M.D.
Medical Oncologist

Thanks!

Public meetings cancelled until further notice

Covid is still with us. Be patient. It will end.

The bad news is that it'll likely be a few more months before the Covid crisis ends. The good news is that we are now entering the vaccination phase, which will bring the pandemic to a close. In the meantime stay safe.

Watch our newsletter for updates on this changing situation. We will be resuming our regular public meetings as soon as the Covid situation allows.

We wish all of you a happy new year.

The board

Manitoba Prostate Cancer Support Group

Thought of The Day

"Life's challenges are not supposed to paralyze you, they're supposed to help you discover who you are."

- Bernice Johnson Reagon

BEST WISHES FOR A HEALTHY, HAPPY AND SUCCESSFUL

The Manitoba Prostate Cancer Support Group offers support to prostate cancer patients but does not recommend any particular treatment modalities, medications or physicians ; such decisions should be made in consultation with your doctor.

MPCSG – active since 1992.

HSF1 Inhibition Directly Targets Treatment-Resistant Prostate Cancer

Scientists at Duke University School of Medicine in Durham, North Carolina, have developed a small-molecule inhibitor of the cellular stress-protective transcription factor, heat shock factor 1 (HSF1), which showed developmental promise against treatment-resistant prostate cancer and other cancers.

The well-characterized small molecule Direct Targeted HSF1 InhiBitor (DTHIB) may also be a useful research tool for investigating the regulation and role of HSF1 in basic stress biology and in cancer, the study authors reported in the December 16, 2020, edition of Science Translational Medicine (STM).

"This study describes the first direct inhibitor of human HSF1, which has been validated for efficacy in animal cancer models," said study leader Dennis Thiele, who was a professor in the Department of Pharmacology and Cancer Biology at Duke University when the study was performed.

Thiele collaborated with Jiaoti Huang, chair professor of the Department of Pathology at Duke and a leader in prostate cancer translational research and drug development, to test HSF1 inhibitors in prostate cancer cells and animal models.

Thiele is currently chief scientific officer at Sisu Pharma, a Chapel Hill, NC-based biotechnology start-up launched to develop the HSF1 inhibitor technology developed at Duke.

Various cancers use the HSF1 protein to drive their proliferation, invasion and metastasis, with nuclear HSF1 abundance being prognostic for cancer severity, treatment resistance and reduced disease-free survival.

"Besides our current work, previous studies have demonstrated that high levels of HSF1, particularly HSF1 localized to the cancer cell nucleus, are predictive of aggressive, treatment-resistant prostate cancer and shortened patient survival," said Thiele.

In prostate cancer, the HSF1 gene has been shown to be amplified, and nuclear HSF1 abundance markedly increased, particularly in untreatable neuroendocrine prostate cancer (NEPC).

Development and progression of prostate cancer requires androgen receptor (AR) signaling, which activates expression of the genes underlying prostate cancer biology.

AR antagonists and androgen deprivation by castration are well-established prostate cancer therapies, but virtually all patients eventually become therapy-resistant and progress to castration-resistant prostate cancer (CRPC) or NEPC.

"Moreover, in some patients the disease will recur as highly lethal NEPC, which does not express the AR and is resistant to AR-targeted therapies," Thiele told BioWorld Science.

"Therefore, direct inhibition of the HSF1 pathway might allow us to treat AR-dependent and AR-independent treatment-resistant prostate cancer patients who have exhausted all other therapeutic options."

HSF1 inhibitors

Despite genetic validation of HSF1 as a therapeutic cancer target, with HSF1 genetic knockout or knockdown studies having shown efficacy in multiple animal cancer models, as yet no selective small-molecule HSF1 inhibitors have been developed or validated for clinical use.

Although HSF1 pathway inhibitors have been identified and evaluated in cellular and mouse xenograft cancer models, they either act indirectly in the HSF1 pathway or have an unknown mechanism of action.

In their new STM study, the authors used multiple biochemical experiments to show that the HSF1 inhibitor, DTHIB, binds directly to the HSF1 DNA binding genomic domain and selectively stimulate degradation of nuclear HSF1.

"In normal cells, most HSF1 is found in the cytoplasm, but in prostate cancer cells and tissues, it mostly accumulates in the cell nucleus, where it regulates the expression of genes driving prostate growth and metastasis," explained Thiele.

However, when Thiele and his team treated prostate cancer cells with DTHIB and separated them into their cytoplasmic and nuclear compartments, "cytoplasmic HSF1 levels remained unperturbed, but active nuclear HSF1 levels were rapidly degraded."

Importantly, the researchers then demonstrated that DTHIB robustly inhibited the HSF1 cancer gene signature and prostate cancer cell proliferation in mice.

"Many of the mouse prostate cancer tumor studies have been conducted in mice with a compromised immune system to prevent rejection of implanted human tumors," noted Thiele.

"In these mice bearing human treatment-resistant prostate cancer tumors, DTHIB treatment completely halted tumor growth and resulted in an approximately 40% tumor shrinkage."

However, "in mice with a functioning immune system, tumors regressed to almost undetectable

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levels, suggesting that the immune system, combined with HSF1 inhibition, contributes significantly to preventing prostate cancer progression," he said.

"Finally, DTHIB-treated mice slowed the progression of prostate cancer expressing AR-v7, a form of the AR resistant to treatment with Xtandi (enzalutamide), with a tumor sample analysis having shown inhibition of the HSF1 cancer gene signature."

Moreover, DTHIB was shown to act independently of the AR and to attenuate tumor progression potently in four treatment-resistant prostate cancer animal models, including highly aggressive NEPC, in which it caused profound tumor regression.

"NEPC typically lacks AR expression and is thus not responsive to androgen deprivation therapy or AR-antagonists," Thiele told BioWorld Science.

"DTHIB administration in two mouse NEPC models resulted in significant inhibition of tumor growth," he said. "Because HSF1 is a well-validated target known to support multiple pathways for cancer growth, survival and metastasis, this suggests HSF1 inhibitors are a promising therapeutic approach for NEPC."

Regarding safety, he said, "no mice treated with DTHIB, at or exceeding the concentrations effective against prostate cancer, showed signs of toxicity, although systematic dosing studies must be conducted to evaluate the impact of administering DTHIB, or other HSF1 inhibitors."

However, "while the first-generation human HSF1 inhibitors discovered at Duke are very promising, considerable additional preclinical development is needed to optimize them for use in humans and testing in clinical trials," said Thiele.

"Because this scale of drug development activities is beyond the scope of Duke University, Sisu Pharma, has licensed the university's HSF1 inhibitor technology for further development for the treatment of PCa and other cancers." (Dong, B. et al. Sci Transl Med 2020, 12: eabb5647).

By John Fox December 18, 2020

Source: www.bioworld.com/articles/501556-hsf1-inhibition-directly-targets-treatment-resistant-prostate-cancer

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What is Active Surveillance?

Active Surveillance is the process of a patient and their doctor working together to actively monitor PCa without intrusive treatments. Some physicians will have a predetermined program to manage low-risk cancer that hasn't spread outside of the prostate. For men with low-risk and some intermediate risk cancers, Gleason 6-7, a biopsy is required to determine this score. The cancer usually grows slowly and will not require aggressive treatments. If the PSA rises suddenly, then additional action could be required.

Active Surveillance has received increasing acceptance among PCa patients and health professionals. Fifteen years ago, most men who were diagnosed with PCa had traditional treatment, including prostatectomy, radiation, cryotherapy and other approaches designed to destroy or remove what is typically a slow-growing cancer. About 10 years ago, it is believed that fewer than 10% of men selected AS in the U.S. Now over 50% of diagnosed men opt for AS, and the number is increasing and in some areas much higher. Two-thirds of men

diagnosed with early-stage prostate cancer will never need aggressive therapies. They can live with their cancer and will die from another cause.

Therefore, it is more important than ever to follow the development of AS: who is a candidate for AS, what is the latest technology to evaluate the PCa status and how to live with AS.

What is a Proactive Surveillance approach?

Men may consider a Proactive Surveillance approach. Proactive means before. The patient can take charge of his own health before any treatments and can incorporate food plans for life, exercise, faith, positive attitude and appropriate supplements to prepare the body for any changes in PCa. There are tests such as a 4K score test and Select MDX test that can determine the aggressiveness of a cancer. Once cancer is determined, then a 3T mpMRI can be used to locate cancer. With this information, the patient can decide to have a targeted biopsy.

Source: aspatients.org /what-is-active-surveillance

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Can Curcumin Prevent or Treat Prostate Cancer?

Aggressive prostate cancer cannot be treated effectively by current medical or surgical therapies of radical prostatectomy, chemotherapy, radiotherapy, or androgen deprivation therapy.

Hormone-dependent prostate cancer responds well to androgen deprivation. In aggressive cancer, however, this therapy is actually counterproductive as it exerts positive selection pressure on the clones of androgen-independent cells, promoting their survival and metastasis.

Thus, new approaches must be used to treat tumors that are hormone-independent or to prevent hormone-sensitive tumors from transforming into hormone-refractory ones. The slow pace at which malignant transformation of healthy prostatic tissue occurs makes primary prevention an important strategy in prostate cancer eradication.

What is curcumin?
Curcumin is a plant polyphenol found in spices like turmeric, which are used in Asian cuisine as well as an anti-inflammatory and cleansing agent. Some studies show that the compound called curcumin diferuloyl methane can block certain chemical pathways that stimulate bone cells to form secondary cancer deposits in hormone-refractory prostate cancers.

How does curcumin act?
The mechanism of action of curcumin in prostate cancer is manifold. One mechanism if inhibition of cell signaling pathways. Preliminary laboratory studies point to some possible mechanisms by which curcumin may affect prostate cancer.

Interplay of TGF-β and BMP-7

TGF-β (transforming growth factor-β) decreases E-Cadherin in the tumor cells and promotes de-differentiation, which causes the epithelial-mesenchymal transition (EMT) to invasive and metastatic prostate cancer. However, if BMP-7 interacts with TGF-β, E-Cadherin expression is increased to promote differentiation and inhibit tumor growth. Curcumin has been shown to upregulate BMP-7.

Curcumin is chemical produced by some plants. It is the principal curcuminoid of turmeric.

PPAR-γ upregulation

Curcumin is an anti-inflammatory molecule that upregulates peroxisome proliferator activated receptor-γ (PPAR-γ), which promotes BMP-7 expression. Thus, it may also interrupt the effects of TGF-β.

Wnt inhibition

Curcumin can also suppress or reduce signaling by Wnt pathways. It also reduces the bone formative activity of prostate cancer cells. This may be explained by its activation of the PPAR-γ pathway, which blocks the communication between signaling pathways that promote bone synthesis and those involving TGF-β.

Increased levels of BMP-7 also stimulate the formation of brown adipose tissue (brown fat, a special form of fat cell), which prevents the proliferation of tumor cells. Activation of the Wnt pathway promotes differentiation into osteoblasts, but also prevents fat cell formation. On the other hand, the PPAR-γ pathway promotes BMP-7 expression and therefore brown fat formation, while repressing osteoblast synthesis. Curcumin thus shifts the differentiation pathway towards brown fat.

Curcumin also reduces pro-inflammatory adipokine expression, driving down levels of tumor necrosis factor-α (TNF-α), and monocyte chemoattractant protein (MCP-1), but activating the anti-inflammatory adipokine called adiponectin. Thus it prevents white fat synthesis while promoting anti-oxidant activity within fatty tissue.

Changing the environment

The changing environment of fat cell differentiation can restrict metastatic prostate cancer cells to interrupt interactions with osteoblasts and osteoclasts at bony metastatic sites.

Curcumin also suppresses the release of parathyroid hormone-related protein (PTHrP), a molecule that promotes osteolysis mediated by TGF-β.

Role of curcumin in the chemoprevention of prostate cancer
Curcumin has been shown to have antiproliferative, antioxidant, and anti-carcinogenic effects. Curcumin reduces the expression of androgen receptors and also inhibits binding of androgen receptors to the prostate-specific antigen (PSA) gene to decrease PSA expression in hormone-dependent cells. This could inhibit tumor progression to hormone-independent status.

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Curcumin may also suppress a homeobox gene of the NK class that is involved in both normal and tumorous prostate growth.

Curcumin also inhibits EGFR signals, such as HER2, which mediate tumor cell proliferation and the expression of aggressive phenotypes. It can also inhibit cyclins involved in the cell cycle, which mediate prostate cancer cell proliferation. This could arrest tumor growth and promote apoptosis, stopping the cell cycle in G2/M phase.

Curcumin also targets cancer stem cells which are responsible for the initial growth of the tumor and for treatment failure. Additionally, it acts against miRNAs which target both the tumor suppressor genes and oncogenes, preventing tumor initiation.

Curcumin suppresses NF-κB expression by preventing IκBα phosphorylation and it suppresses phosphorylated Akt kinase in prostate cancer cells. Akt is a central molecule in tumor formation, where it is often constitutively activated, and the inhibition of these factors causes the downregulation of anti-apoptotic Bcl-2 proteins, promoting apoptosis.

Curcumin also prevents angiogenesis, which normally occurs in response to vascular endothelial growth factor secretion and encourages tumor metastasis.

Prevention of prostate cancer metastasis

Curcumin reduces the expression of some matrix metalloproteinases (MMPs) which are important in facilitating tumor invasion and spread.

Curcumin also suppresses the crosstalk between prostate cancer cells and surrounding stromal or osteoblasts in bone to prevent metastasis.

Curcumin promotes apoptosis of the prostate cancer cells both through the mitochondrial and the receptor-mediated pathways. This makes TNFα- related apoptosis-inducing ligand (TRAIL) an attractive choice for immunotherapy for advanced cancers, that is, in both hormone-dependent and refractory prostate cancers. Curcumin sensitizes the cancer cells to the activity of TRAIL and thus induces apoptosis.

Since it might be capable of arresting osteolysis in metastatic bone lesions of prostate cancer, it could be used alone or as an adjuvant with bisphosphonates in order to prevent bone loss, thus reducing the dose of the latter and thereby the incidence of complications like osteonecrosis of the jaw. Secondly, curcumin could be combined with other activators of the PPAR-γ pathway to increase BMP-7 levels.

Summary

Curcumin has multiple modes of action that may be useful in the development of prostate cancer treatments. However, more work is needed to improve the absorption and slow the metabolism of curcumin to improve bioavailability.

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By Dr. Liji Thomas, MD

Reviewed by Dr. Jennifer Logan, MD, MPH

Source: www.news-medical.net/health/Can-Curcumin-Prevent-or-Treat-Prostate-Cancer.aspx

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Plant-based Diets Protect Against Prostate Cancer

Animal-based foods, such as milk and cheese, increase the risk for cancer, according to a review published in the Journal of the American Osteopathic Association.

Researchers reviewed almost 50 publications that assessed diet and cancer risk and observed a protective effect from vegan diets with increased consumption of vegetables, legumes, and tomato products while higher intakes of dairy products increased the risk for prostate cancer.

References

Shin J, Millstine D, Ruddy B, Wallace M, Fields H. Effect of plant- and animal-based foods on prostate cancer risk. J Am Osteopath Assoc. Published online October 21, 2019.

Source: www.pcrm.org/news/health-nutrition/plant-based-diet-protects-against-prostate-cancer

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Combining Biopsy Methods Yields Most Accurate Prostate Cancer Dx

MRI-targeted plus systematic biopsy reduces over- and under-treatment
This article was initially published on March 4, 2020. As prostate cancer, and particularly concern about optimal methods for obtaining biopsies, remains a significant interest among BreakingMED users, we are republishing it as part of our year-end retrospective series on clinical news of 2020.

Combining traditional prostate cancer biopsy — systematic biopsy — with MRI-targeted biopsy provided the most accurate results, pinpointing more clinically relevant cancers and correctly identifying low-risk lesions, researchers from the National Institutes of Health reported.

Michael Ahdoot, MD, of the Urologic Oncology Branch at the National Institutes of Health Clinical Center in Bethesda, Maryland, and colleagues, wrote that the results of the Trio Study, which they reported in The New England Journal of Medicine, "should reduce the risk of both overtreatment and undertreatment out of fear of misdiagnosis."

The researchers noted that systematic biopsy requires 12-core samples and is associated with both grade misclassification and missed diagnoses, while MRI-targeted biopsy "requires 12 fewer biopsy cores and leads to 5% fewer diagnoses of clinically insignificant cancers."

But in Trio Study, Ahdoot and colleagues found that "the omission of systematic biopsy would lead to missing 1.9% more grade group 3 cancers and 5.8% more grade group 2 cancers in our study population. More important, among the patients in whom prostate cancer is diagnosed, the use of MRI-targeted biopsy alone leads to high diagnostic uncertainty, since this method used in isolation is associated with a 30.9% rate of any upgrading of the cancer group and an 8.7% rate of upgrading the cancer to a clinically significant grade group on whole-mount histopathological analysis."

The take-home message on biopsy technique, the Trio investigators concluded, is not either/or, but both.

The Trio Study was actually a substudy of a larger NIH clinical trial looking at the use of tracking devices during invasive procedures. The researchers enrolled 2,103 men who had MRI-visible prostate lesions to undergo both MRI-targeted and systematic biopsy.

Among the findings:

1,312 men had cancer diagnosed using both biopsy methods.
404 of those men underwent radical prostatectomy.
Fewer grade 1 cancers (P <0.001) were detected by MRI-targeted biopsy alone than by systematic biopsy.
Detection of grade 3 (P =0.004), grade 4 (P <0.001) and grade 5 cancers (P =0.003) was significantly higher with MRI-targeted biopsy.
"Combined biopsy led to cancer diagnoses in 208 more men (9.9%) than with either method alone and to upgrading to a higher grade group in 458 men (21.8%)."
If systematic biopsy were omitted, "8.8% of clinically significant cancers (grade group ≥3) would have been misclassified."
The researchers noted that among "2,103 patients who underwent the two biopsy methods, prostate cancer was diagnosed in 1,104 patients (52.5%) with systematic biopsy alone and in 1,084 patients (51.5%) with MRI-targeted biopsy alone."

Importantly, when MRI-targeted biopsy was added to systematic biopsy, 208 more cancers were diagnosed, 59 of which were clinically relevant disease. "The addition of MRI-targeted biopsy led to a reduction of 60 patients (from 454 to 394) who were classified as having clinically insignificant (grade group 1) cancer," they wrote. "Specifically, 134 men in whom grade group 1 cancer was diagnosed on systematic biopsy were upgraded to grade group 2 or higher on MRI-targeted biopsy. Simultaneously, MRI-targeted biopsy led to 74 new grade group 1 cancer diagnoses among men in whom no cancer was detected on systematic biopsy, which led to a net reduction of 60 patients with a grade group 1 cancer diagnosis."

Ahdoot and colleagues noted a number of limitations of their study, including the fact that it was conducted at a single institution where most of the practitioners are more experienced than those found in typical community settings. And, because the study focused only on patients with MRI-visible lesions, the findings may not be applicable to patients who have normal MRI findings.

The authors concluded that the findings "suggest that combined biopsy provides improved diagnostic accuracy over either systematic or MRI-targeted biopsy alone and better predicts the results of final histopathological analysis."

Peggy Peck, Editor-in-Chief, BreakingMED

Dec 30, 2020

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Source: https://www.physiciansweekly.com/combining-biopsy-methods-yields-most-accurate-prostate-cancer-dx/

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Novel Deep-Learning Algorithm Shows Promise in Identifying High Grade Prostate Cancer

Deep-learning algorithms could be used to alert pathologists to suspicious areas of cancer burden prior to clinical assessment, according to Nitin K. Yerram, MD.

A novel deep learning algorithm appeared to accurately identify cancer on whole mount prostate pathology, potentially alerting pathologists to suspicious areas of cancer burden prior to clinical assessment, according to study results presented at Society of Urologic Oncology (SUO) 21st Annual Meeting.

"Accurate Gleason Grade grouping determination at time of prostate cancer diagnosis is vital for patients' decision making to pursue either surveillance, definite treatment, or adjuvant therapy. However, intercarrier variability in the grading of pathologic specimens remains a significant issue," Nitin K. Yerram, MD, a urologic oncology fellow at the National Cancer Institute, said during a virtual presentation.

"Given that deep learning algorithms are being developed to help augment and standardize detection and grading for prostate cancer, our group has utilized AI algorithms to provide a solution to similar problems in bladder cancer pathology and prostate cancer imaging," he added.

The researchers used previous studies to create a novel algorithm that was developed to help identify and grade prostate cancer on whole mount pathology slides.

The algorithm was previously trained for detection and grading of prostate cancer using publicly available datasets, totaling over 9000 biopsies, tissue microarrays, and surgical sections. Additionally, a small number of segmented whole mount prostatectomy slides were utilized for detection task only.

"The algorithm was designed to be agnostic of tissue source operating on patches abstracted from each image," Yerram explained. "Each patch is representing 100 microns by 100 microns, or 200 by 200 pixels at 20x. Each patch is then run through our algorithm. These patches are spatially recreated to produce a probability map where each patch is assigned either a value of zero or 1, 1 being the highest likelihood of cancer."

Burden of each foci within each slide were marked by ink under microscope and mapped digitally for quantitative comparison.

Congruent lesions >2 mm2 area were considered positive for deep learning-based detection.

"At the NCI, we sought to evaluate the congruence of the AI algorithm with pathologist annotated regions from routine clinical practice," Yerram said, adding that the researchers then chose to convert the probability map into a detection mask, "which identifies high risk areas defined as congruent areas of high probability in a set pixel size."

For statistical analysis, they then compared the detection rate to pathologist annotation. A true positive was called as any detection within a prospect annotation. Once the detected areas of probability that are considered cancer were found, they ran it through the grading algorithm. Each patient is given a prediction: either Gleason Grade 3, 4, or 5. Similarly, with the detection map, the researchers were able to visually look at probability maps for each one of these Gleason scores.

The algorithm was applied to 50 patients who underwent radical prostatectomy between 2008 and 2018 with available digitized whole-mount pathology and foci-level annotations of disease burden. In total, 24 patients had Gleason Grade 1 or 2 and 26 had Gleason Grade 3, 4, or 5 on surgical pathology.

Patient level detection accuracy of cancer or no cancer was 96%, with 2 patients classified as negative for cancer. However, these 2 patients had Grade 1 and 2 disease, indicating that no high-grade disease was missed, Yerram noted.

On annotated foci identification, the algorithm showed a 77.6% sensitivity, with a median rate of 1 false-positive per patient (range, 0-20). Positive predictive value was 38.4%.

"Our program performed quite well in the grading of cancer. Overall, a higher percent of the tumor grade and burden was detected in patients with Grade 3-5 disease versus patients with Grade 1-2 disease," Yerram concluded.

"Our algorithm demonstrated excellent accuracy in identifying cancer on whole mount pathology," he added. "Now, there is room for improvement in the first level accuracy, and our positive predictive value needs to be improved, but that will continue to be improved upon with further refinement. Systems such as this, we envision, can be used to alert pathologists to suspicious areas of cancer burden prior to clinical assessment. And lastly, spatial assessment of grade distribution can be achieved with further refinement of our algorithm."

Kristie L. Kahl December 4, 2020

Reference:

Yerram N, Harmon S, O'Connor L, et al. Application of Deep Learning Detection and Grading System for Identification of Clinically Significant Prostate Cancer on Whole Mount Pathology. Presented at: Society of Urologic Oncology 21st Annual Meeting; December 3, 2020. Abstract 3.

Source: www.cancernetwork.com/view/novel-deep-learning-algorithm-shows-promise-in-identifying-high-grade-prostate-cancer

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Our public meetings will not resume until the covid-19 restrictions are lifted.

Watch this space for information on the latest status.

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