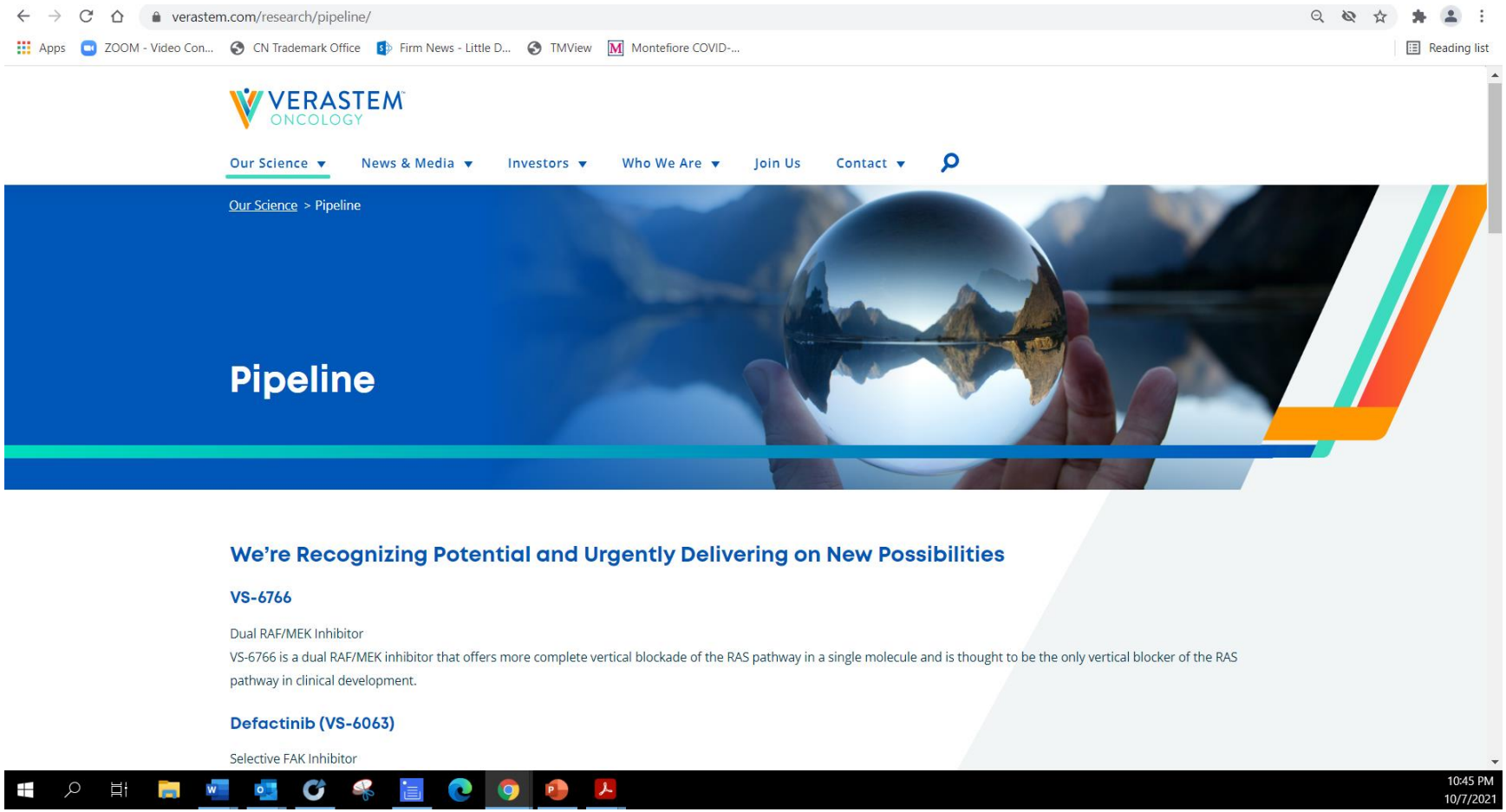
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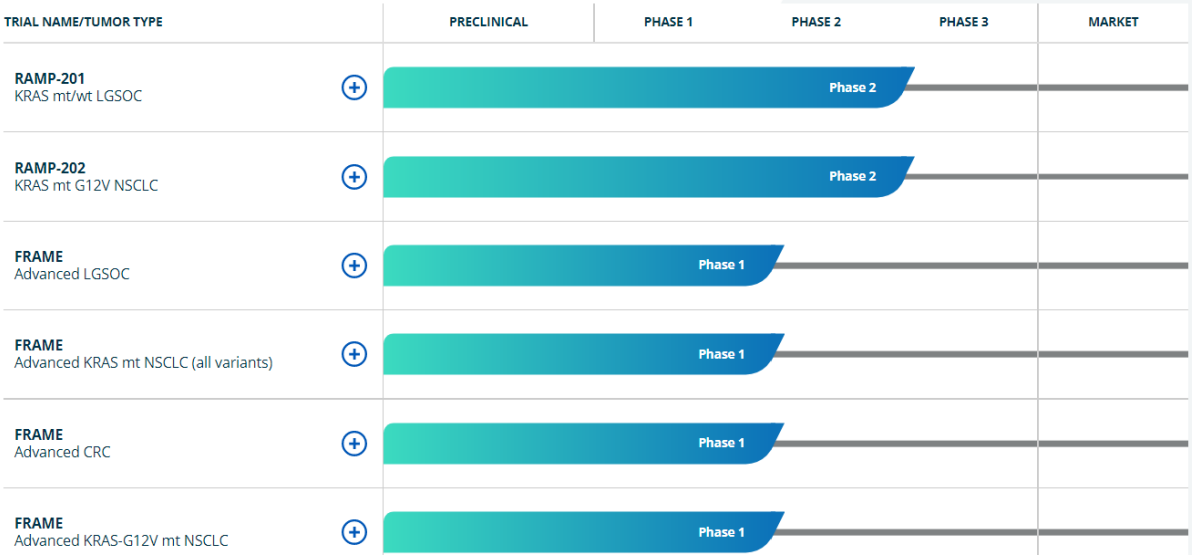
We're Recognizing Potential and Urgently Delivering on New Possibilities
VS-6766
Dual RAF/MEK Inhibitor
VS-6766 is a dual RAF/MEK inhibitor that offers more complete vertical blockade of the RAS pathway in a single molecule and is thought to be the only vertical blocker of the RAS pathway in clinical development.
Defactinib (VS-6063)
Selective FAK Inhibitor
10:45 PM
10/7/2021

Defactinib (VS-6063) is a best-in-class selective FAK inhibitor that has been studied as a monotherapy and in combination in patients with solid tumors.

VS-6766 + DEFACTINIB



FRAME Advanced pancreatic cancer	Phase 1
FRAME Advanced KRAS mt endometrioid cancer	Phase 1
Metastatic uveal melanoma	Phase 2

VS-6766 + OTHER COMBINATIONS

TRIAL NAME/TUMOR TYPE	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	MARKET
KRAS mt NSCLC	VS-6766 + everolimus (mTORi) Phase 2				

These studies are investigating treatments or outcomes that have not received approval from a health authority. We are working diligently to demonstrate the safety and efficacy of these products, but there is no guarantee that the outcome of these studies will result in approval. We invite you to review our expanded access policy.

Investigator Sponsored Trials (IST)

We believe in the power of collaboration to help us push forward with new and promising cancer treatments. Our Verastem Oncology Investigator Sponsored Trial (IST) program strives to advance medical and scientific knowledge for our product candidates and disease states of interest. We welcome partnerships with researchers and sponsoring institutions that build on this mission.

Investigator Sponsored Trial Form

For additional details regarding our IST program or to submit a research request, please download, complete and submit the form below via email to verastem-IST@verastem.com.

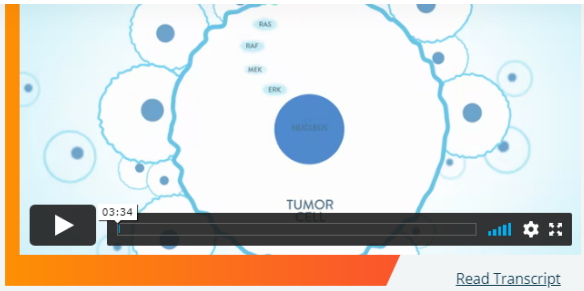
DOWNLOAD FORM

Research Collaborations

Verastem Oncology also accepts proposals for preclinical research collaborations. For more information, please reach out to us at research@verastem.com.

RAS Pathway | VS-6766 (RAF/MEK Inhibitor) | Defactinib (FAK Inhibitor) | VS-6766 as a Backbone of Therapy

RAS Pathway: Examining the Most Frequently Mutated Oncogene in Human Cancers



Read Transcript

Almost a third of all cancers in patients are associated with mutations of the RAS family of genes, which includes KRAS, NRAS, and HRAS. There are other oncogenes, including EGFR and BRAF, that also activate the RAS pathway—meaning an even higher percentage of cancers depend on this pathway for growth and survival. Patients with a RAS-mutated cancer tend to experience worse outcomes and a higher disease burden than those without RAS pathway mutations. Watch the video to see how customized combinations of VS-6766, a unique dual RAF/MEK inhibitor, including a combination with defactinib, a selective FAK inhibitor, have the potential to greatly expand the number of effective treatments for cancer patients who have limited options today.

Blocking the RAS Pathway Presents Challenges Due to Multiple Resistance Mechanisms

Cancer has a strong affinity for the RAS pathway and reacts to the blocking of any single target by either reactivating the RAS pathway elsewhere or activating parallel pathways to survive. For example, MEK inhibitors paradoxically induce MEK phosphorylation (pMEK) and RAS signaling by relieving ERK-dependent feedback inhibition of RAF. MEK inhibitors also cause compensatory activation of phosphorylated FAK (pFAK), and BRAF inhibition also induces compensatory activation of pFAK. Single-target therapies (eg, MEK inhibitors) are associated with resistance and may not be the best avenue to slowing tumor growth—and finding tolerable combination regimens with MEK inhibitors has been challenging. There has been only modest progress and a limited number of approved therapies. Novel therapies and combinations are urgently needed to deliver on the promise of better outcomes for patients.

VS-6766 (RAF/MEK Inhibitor) ►