MARY CROWLEY
CANCER RESEARCH CENTERS
HOPE LIVES HERE.

MELANOMA

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Melanoma: Where's Our Menelaus?

Most melanomas are black skin lesions due to pigment production and, although most of them (approximately 84%) are localized with a high probability of cure, the remaining ones have a short survival time and, until recently, have defied effective treatment. There have been two recent treatment breakthroughs: Yervoy™, which modulates the immune system, produces a 10% response rate and almost doubles the average survival rate (to 10 months) and Vemurafenib, a therapy inactivating the specific gene mutation occurring in 60% of melanomas, which has a high response rate but also a high recurrence rate. Why such problems? Melanoma is like Proteus, the wily shape shifter in Homer's Odyssey who could change from one shape to another. When one dominant mutant gene that controls growth signals is blocked (by Vemurafenib), melanoma cells can open up new detours and bypasses allowing for resumption of growth. Likewise, following immunotherapy, the cancer cells can produce immune inhibiting proteins, a tough defensive line. At Mary Crowley, we are devising ways to circumvent these shape shifting capabilities. Many of our targeted agents are directed at both the known abnormal growth signal pathway and the known and presumed detours and bypasses to try to block resistance from emerging. We are also investigating innovative immune approaches including the FANG™ vaccine, which incorporates a mechanism to block the cancer's ability to make immune suppressive proteins, allowing us to break through its defensive line. Basically, we are looking for a Menelaus, the Greek warrior who finally caught and controlled the slippery Proteus.

Neil Senzer, MD, Executive Scientific Director at Mary Crowley

Menelaus Goes To The "Street"

Advanced melanoma is still a deadly disease with limited therapeutic options. However with recent advances focused on targeted immune and molecular technology, melanoma survival has improved. So, to follow-up in the unique way of Dr. Senzer involving Greek mythology, we have discovered a weakness that Proteus is unaware of! Utilization of Vemurafenib then YervoyTM provide an additive 1:2 punch of signal control (V600 mutation-directed) followed by targeted immune attack (Treg inhibition) WITHOUT the crippling effects of chemotherapy-induced immune suppression. This approach works and passes FDA's assessment. To temporarily get away from Greek mythology (Sorry Neil) and go to the streets of America, patients with melanoma progressing after Vemurafenib/ Yervoy (or earlier if V600 "-") have an opportunity to participate in several other (potentially additive) melanoma experimental studies at Mary Crowley, (see below).

Study 11-26 - CAVATAK™ uses coxsackievirus, an oncolytic virus similar to Oncovex™. The virus is designed to grow within the tumor (and not in healthy cells), break up and shrink the tumor, and reactivate the immune system to systemically fight cancer.

In this effort, Mary Crowley is going to the street and adding "brass knuckles" to the boxing gloves of Menelaus (our physicians) in his effort to control Proteus (cancer).

"Think about research for your advanced melanoma patient"
- John Nemunaitis, MD, Executive Medical Director at Mary Crowley