Cancer cells that form DFT1 tumors overproduce transmembrane enzymes known as ERBB receptors.

When stimulated by specific proteins—likely EGF and NRG1, which are also overproduced in DFT1 cells—ERBB receptors induce production of a signaling protein and transcription factor called Stat3 and drive its activation.

In the nucleus, Stat3 drives the production of genes such as MMP2 that are known to trigger metastasis in humans.

Stat3 also binds to and inhibits another transcription factor, Stat1, which normally drives the expression of genes necessary for the generation of MHC class I molecules.

MHC class I molecules normally interact with receptors on cytotoxic T cells to discriminate self from foreign cells. By downregulating the production of MHC proteins, DFT1 cells are able to evade detection by the animals' immune system.

Under normal circumstances, cells lacking MHC markers would be detected by natural killer cells (NK), but for reasons that are unclear, devil NK cells don't react to DFT1.