SPEAKER: Herceptin a drug that is currently used for women that have breast cancer, women that have tumors that express the proto-oncogene HER2. Too. And they have to not only over-express HER2, but they have to be-- to have the gene amplified. Herceptin works very well when it's combined with chemotherapy. And right now, also is used with a combination with other drugs, including pertuzumab. These drugs are successful, but the only problem is that patients do have resistance after a while when they are treated with Herceptin.

The study that we currently are performing in the laboratory-- and currently was published in the *Journal of the National Cancer Institute*-- demonstrates that a region in the HER2 protein is involved in the dimerization, or in the partnership between the HER2 two receptor with other receptors of the HER family. The HER family is basically four types of the HER receptors that are HER1, HER2, HER3, and HER4. And they are dimerized, either by a receptor in a ligand induction of receptor dimerization, or it can be dimerized by over-expression of the receptor.

So we identified this particular region that, when it's mutated, basically, it prevents not only the homodimerization of the receptors, but also the heterodimerization of the receptors. So we anticipate that the use of a drug that targets that particular region will be much more useful than any drug that we are currently using in the clinic. And we demonstrated that by using different breast cancer cells, as well as non-transformed breast epithelial cells.

This could have possibilities not only in breast cancer tumors that over-expressed the receptors and have amplification of the gene, but it could have a possibility in patients that their tumors have not only over-expression of HER2, but they also would have expression of HER2 in combination of expression of other HER2 receptors-- as well as other carcinomas, such as ovarian cancer, prostate cancer, and for example, gastric cancer.

We are currently conducting animal studies with these particular HER2 mutants in which we are determining whether the cells that contain these particular mutants are able to develop tumors in immunodeficient mice. If these tumors cannot develop tumors in mice, we are confirming what we are-- what we just published in the *Journal of the National Cancer Institute.*

If that's the case, that will definitely determine that this particular functional domain is crucial for the development of a drug that will target this particular domain. We believe that there is definitely hope, because this is the first time that anybody has identified any region that blocks homodimerization and heterodimerization as well, which will simplify the treatment of the cancer, rather than combining two, three, or four drugs together. This will be a one-stop shop, if you will.

And no, women will not at this point go to the doctor and say, "Please give me this drug." The drug does not exist. This is a promising area of future research. What excites us is that this is the first time that has been done. And by serendipity, if you wish, we got to this point. And we hope that this will pan out when we develop the drug.