

Transcript Details

This is a transcript of an educational program. Details about the program and additional media formats for the program are accessible by visiting: <https://reachmd.com/programs/practical-dermatology/understanding-btk-inhibition-in-chronic-spontaneous-urticaria/84983/>

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Understanding BTK Inhibition in Chronic Spontaneous Urticaria

Dr. Neal Bhatia: Hi. I'm Dr. Neal Bhatia. I'm chief medical editor of Practical Dermatology, and this is another installment of Updates in Urticaria. And there's a lot of good movement in urticaria nowadays, especially with the discovery and the application of therapies directed towards the Bruton tyrosine kinase pathway, and what does BTK, what does it mean, where does it fit, et cetera. We've heard a lot about tyrosine kinases.

We've heard a lot about different enzymatic pathways, especially in atopic dermatitis, psoriasis, and elsewhere, including lupus down the road. But in urticaria, from what we're understanding, Bruton tyrosine kinase has a very significant role signaling through a lot of different pathways, as well as with a lot of important components that we're familiar with. You can start with Fc epsilon receptor. You can look at 4 and 13. We can look at some of the other signaling of mast cells, as well as some of the other histaminergic pathways. All of them seem to actually come back to that central role of where Bruton tyrosine kinase fits in the mast cell. And as opposed to some of the others that are more on the cell membrane with the receptor, BTK is actually centrally placed in the cytoplasm, and all, again, pathways have to signal through that.

That being said, what we're trying to figure out is the activation of BTK, where does it fit in mast cells and basophils, and how does that impact not just histamine degranulation, but also the process of all of the vasomotor and vaso-driven pathways that come from those urticarial pathways that we see in front of us. That being said, you look at how do we modulate between an autoallergic response and an autoimmune response, that again goes back to some of the types of receptors that are bound, as well as, again, some of the signaling that leads to BTK doing its purpose.

If you remember, any kind of kinase is there to phosphorylate. So phosphorylation means we're trying to get those processes in motion, trying to get those enzymatic pathways to talk to each other, and again, that's what releases histamine from the mast cells in the first place. If, again, you think about some of the other benefits of BTK inhibition, there's an impact on B-cell receptor signaling, as well as, again, some of the other parts of autoantibodies that work in chronic spontaneous urticaria. So many different paths leading from what BTK can do, what suppression of it can do, and where we're going to see that fit into other pathways.

Main take-homes for urticaria, again, is don't be fooled by just urticaria alone. Every urticaria has a flavor, chronic spontaneous urticaria specifically, but there's going to be some data for inducible urticaria down the road. Again, don't want to be fooled by mastocytosis, so make sure your patients are looking into any visceral effects, like any kind of GI effects, rheumatological effects, or anything else that's under the hood with them. Make sure with urticaria patients that they're aware now, that these oral treatments such as remibrutinib that have come aboard, these are very safe. They don't have a box warning, and they don't require any kind of EpiPen or anything else that we might have been used to from before.

So again, a lot of good data with the BTK pathways, a lot of understanding of potential applications outside of urticaria. But I think for as well as we're studying how we treat hives, we can get even more aggressive and even more safe with the use of some of the oral therapies that are in our path.