ABSTRACT
Based on these data, Btk:ER is thought to regulate downstream signaling pathways in B cells primarily via PLC2 (i.e., regulatory protein coupled with nuclear protein chainase inhibitor PLSI 2), and thus it is not known whether activated BukR/STK copy-pasteurs interact with activatededBtky receptor expressed as a "conditional system", but this condition will probably allow for the dissection of Btons' (and other) role in various biological processes in many cell types.

INTRODUCTION
The B cell lymphocytes (BCL) are the most common type of lymphocyte in the human body, and they play a key role in the host's immune response. BCLs have been shown to be able to express multiple kinases such as tyrosine kinase (TRK) and phosphatidylinositol 3-kinase (PI3K), which are involved in a wide range of biological functions. However, their role in the host's immune response is poorly understood; the functional roles of BCLs in the immune response have been largely ignored.

Our aim is to develop a conditional form of tyrosine kinase (TYK) that can activate multiple downstream signaling pathways via PLC Gamma 2 in B Btk mutations, which are responsible for the human disease X-linked agammaglobulinemia (reviewed in reference), cause a severe blockage in the pro-B cell transition and result in no mature B cells. It is hypothesized that a two-way process involving PI 3-kinase and Src family PTK Lyn activates Btk upon cross-linking of the BCR. This enzyme causes Bkg to become phosphorylated upon binding to the PH domain of Bz3. The significance of Btk in BCR-induced phospholipase (PLC2) phosphatalysis, calcium mobilization, ERK, and NF-B activation and JNK-mediated signaling in deficient B cells has been established through biochemical analyses. However, it is not clear whether all downstream signals mediated by BTK are PLC2-dependent or if Button alone can perform these functions. By combining the full-length Btk protein with the hormone-binding domain of the estrogen receptor (Btkg:ER), we have produced a conditional variant of BTK. We demonstrate that Bukhariya kinase inhibitor BKT-1, KAL1, PLC-2, and PKC-K2 activation alone can activate various downstream signaling pathways in B cells, including calcium mobilization, ERK and JNK MAPK, as well as cellular death by mice.

CONCLUSION
Remarkable conclusions The Btk mutations observed in XLA patients and the xid mouse indicate that Bfk is essential for B cell development, but the important pathways activated by Bbk remain unresolved. In our paper, we demonstrate that a conditional form of Bcty can activate multiple signaling pathways downstream of the antigen receptor, including PLC2 phosphorylation, calcium mobilization (MS) activation or ERK and JNK activATION, and that all Bcky-mediated responses are dependent on BKC genes.