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Bunyaviruses are Dependent on K_{2p} Channels to Infect Cells

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All viruses must infect cells in order to multiply and cause disease. Once inside cells, viruses hijack normal cellular processes to create an environment that favours their own survival, typically at the expense of the host. Through understanding how viruses do this, we can design new strategies to prevent viruses from multiplying, and thus stop their ability to cause disease. The *Bunyaviridae* are the largest family of negative stranded RNA viruses. These are a group of over 350 different viruses spread throughout the globe. They are predominantly transmitted to humans by insects and are capable of causing fatal disease in humans, often as a result of devastating hemorrhagic fevers. Bunyaviruses are a serious and worrying threat to human health because they have enormous capacity to mutate and evolve into new strains. In addition, the fact that bunyaviruses are mostly spread by biting insects that are highly mobile means they can rapidly move into new geographic loca-tions, causing widespread disease. Despite this huge threat, no preventative or therapeutic measures currently exist for any bunyavirus-mediated disease. Using model viruses within the *Bunyaviridae* family, predominantly Bunyam-wera virus (BUNV), we previously identified cellular potassium (K^+)
channels as an essential host cell factor required for bunyavirus infection. Using a rationale panel of Kþ channel modulators, we identified the two-pore Kþ (K2P) channel family as those required by BUNV. We have identified four possible K2P channels we believe are facilitating a cellular process required during BUNV infection. Using genetic silencing we plan to determine the specific channel(s) involved and from this, identify a new drug target for the development of novel anti-bunyavirus therapies.