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Review

Multiple faces of protein interacting with C kinase 1 (PICK1): Structure, function, and diseases

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ABSTRACT

Protein interacting with C-kinase 1 (PICK1) has received considerable attention because it is the only protein that contains both PSD-95/DlgA/ZO-1 (PDZ) domain and Bin-Amphiphysin-Rvs (BAR) domain. Through PDZ and BAR domains, PICK1 binds to a large number of membrane proteins and lipid molecules, and is thereby of multiple functions. PICK1 is widely expressed in various tissues, particularly abundant in the brain and testis. In the central nervous system (CNS), PICK1 interacts with numerous neurotransmitters receptors, transporters, ion channels, and enzymes, and controls their trafficking. The best characterized function of PICK1 is that it regulates trafficking of α-amino-3-hydroxy-5-methylisoxazole-4-propionic acid receptor (AMPAR) subunit GluA2 during long-term depression and long-term potentiation. Recent evidence shows that PICK1 participates in various diseases including neurobiological disorders, such as chronic pain, epilepsy, oxidative stress, stroke, Parkinson's disease, amyotrophic lateral sclerosis, schizophrenia, and non-neurological disorders, such as globozoospermia, breast cancer, and heart failure. In this review, we will summarize recent advances focusing on the structure and regulation of PICK1 and its functions in protein trafficking, neurological and non-neurological diseases.

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1. Structure, function, and regulation of PICK1

1.1. Structure

Protein interacting with C-kinase 1 (PICK1) was cloned as a protein kinase C-α (PKCα)-binding protein, which is expressed in multiple tissues and organs, mostly abundant in the brain and testis (Staudinger et al., 1995, 1997; Wang et al., 2003, 2007). The expression of PICK1 starts as early as embryonic 15 days, and continues to increase until two weeks after birth (Xia et al., 1999). The high evolutionary conservatism of PICK1 from non-vertebrate animals to mammals strongly suggests that it has important biological functions, though PICK1-knockout is not lethal in mammals (Gardner et al., 2005; Steinberg et al., 2006). PICK1 possesses two important domains, a PSD-95/DlgA/ZO-1 (PDZ) domain and a Bin-Amphiphysin-Rvs (BAR) domain, making it the only known protein containing these two domains. Through the PDZ domain, PICK1 interacts with a broad range of neurotransmitter receptors, transporters, and enzymes (Deken et al., 2001; Xu and Xia, 2006; Hanley, 2008). In general, BAR domain can drive the curvature of membrane (Peter et al., 2004), through which PICK1 may regulate synaptic localization and function of target proteins (Xu and Xia, 2006; Hanley, 2008). Moreover, PICK1 possesses an N-terminal acidic region (NAR), PDZ domain, BAR domain, and a C-terminal acidic amino acid region (CAR). NAR combines with Ca^{2+} to regulate the interaction between PICK1 and α-amino-3-hydroxy-5-methylisoxazole-4- propionic acid receptor (AMPAR) subunit GluA2 (Hanley and Henley, 2005). CAR can negatively regulate the lipid binding of BAR domain (Jin et al., 2006). PICK1 is dispersed in the cytosol of many cells and usually gathered around perinuclear region (Staudinger et al., 1995). In neurons, PICK1 is expressed at synapses, both presynaptically and postsynaptically (Haglerød et al., 2009).

A number of recent findings have promoted our understanding to the structure of PICK1. It has been shown that PICK1 interacts with Arp2/3 complex and acts on actin polymerization (Rocca et al., 2008; Nakamura et al., 2011; Murk et al., 2013; Rocca et al., 2013). A recent study showed that PICK1-Arp2/3 interaction may not be mediated by CAR (Madasu et al., 2015). Using X-ray scattering analysis, Karlsen et al. (2015) discovered that PICK1 inclines to form higher-order structures in a parallel mode of BAR–BAR oligomerization. In these structures, the positions of PDZ domains are highly flexible, which enables PICK1 to perform long-range and dynamic scaffolding of membrane-associated proteins (Karlsen et al., 2015; Cheng et al., 2009; Sheng and Sala, 2001). These findings help to explain how PDZ domain of PICK1 binds putative partners. PKCα, dopamine transporter, and acid-sensing ion channel ASIC1a have either canonical or dual binding mode with PICK1, suggesting that recognition domains in PICK1 are evolved to expand its repertoire of functional interactions (Erlendsson et al., 2014; Jin et al., 2010).

Recently, two studies reported the tertiary structure of PICK1 using small angle X-ray scattering (SAXS) (Madasu et al., 2015; Karlsen et al., 2015). Unexpectedly, these studies reached different structural and functional conclusions of PICK1, although similar SAXS analysis was used. The controversies were mainly derived from the approach for sample stabilization, which is particularly challenging for SAXS because PICK1 is indeed prone to aggregation, and other analytic methods (Boczkowska et al., 2015; Erlendsson et al., 2015). Future work is needed to clarify the structural model of PICK1 based on present results.

1.2. Functions

A wealth of information shows that PICK1 has multiple functions in the central nervous system (CNS), mainly relevant to the transport of its-associated proteins. The function of PICK1 in the activity-dependent modulation of synaptic transmission has been well studied (Alfonso et al., 2014; Hanley, 2008). PICK1 is involved in the endocytosis of membrane AMPARs during cerebellar long-term depression (LTD) (Anggono et al., 2013; Matsuda et al., 2000; Xia et al., 2000; Kim et al., 2001). PICK1 is also important for hippocampal long-term potentiation (LTP) as blockade of PDZ domain or PICK1 deficiency prevents LTP (Terashima et al., 2008). PICK1 regulates the trafficking of GluA2 during the expression of synaptic plasticity (Xia et al., 2000; Hanley and Henley, 2005; Steinberg et al., 2006; Terashima et al., 2008; Malinow and Malenka, 2002). Blocking PICK1 alters the subunit composition and trafficking of GluA2 (Anggono et al., 2011; Hirbec et al., 2003). However, the role of PICK1 in regulating AMPAR trafficking is controversial. Several studies indicate that PICK1 regulates the recycling of AMPARs by retaining them in the intracellular compartments (Lin and Huganir, 2007; Citri et al., 2010; Anggono et al., 2011; Madsen et al., 2012). Calcium is a critical regulator in PICK1-mediated retention of internalized AMPARs (Citri et al., 2010). PICK1 may reduce the reinsertion rate of AMPAR in a Rab11-dependent manner (Madsen et al., 2012).

Recent findings expand our knowledge about PICK1 functions in neurons, including miRNA activity, AMPAR trafficking, synaptogenesis, and vesicle biogenesis. MicroRNA-related protein argonaute associates with endosomal compartments at synapses. PICK1 binds to argonaute and inhibits its function in translational repression, providing a link between endosomal trafficking and local translational repression (Antoniou et al., 2014). Two studies provide novel mechanisms of AMPAR trafficking and synaptogenesis. First, PICK1 interacts with Ca^{2+}/calmodulin activated kinase II (CamKII) through BAR domain (Lu et al., 2014). Second, PICK1 recruits AMPARs to immature postsynaptic sites and promotes neurexin-induced synaptogenesis (Xu et al., 2014). PICK1 might also affect spinogenesis by regulating in actin polymerisation: PICK1 binds to Rho-family members Rac1 and Cdc42 and mediates the subcellular localization of Cdc42 (Rocca and Hanley, 2015). Moreover, PICK1 functions in vesicle biogenesis, as shown by fewer and reduced size of large dense core vesicles in PICK1 knockout mice (Pinheiro et al., 2014).

Interestingly, a series of studies revealed new roles of PICK1 in regulating D-serine release and neurodevelopment. Earlier studies demonstrated that PICK1 interacts with D-serine synthesizing enzyme, serine racemase (SR), and is associated with schizophrenia

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