Chapter 5
Current Understanding of the TCTP Interactome

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Abstract Evolutionarily conserved and pleiotropic, the translationally controlled tumor protein (TCTP) is a housekeeping protein present in eukaryotic organisms. It plays an important role in regulating many fundamental processes, such as cell proliferation, cell death, immune responses, and apoptosis. As a result of the pioneer work by Adam Telerman and Robert Amson, the critical role of TCTP in tumor reversion was revealed. Moreover, TCTP has emerged as a regulator of cell fate determination and a promising therapeutic target for cancers. The multifaceted action of TCTP depends on its ability to interact with different proteins. Through this interaction network, TCTP regulates diverse physiological and pathological processes in a context-dependent manner. Complete mapping of the entire sets of TCTP protein interactions (interactome) is essential to understand its various cellular functions and to lay the foundation for the rational design of TCTP-based therapeutic approaches. So far, the global profiling of the interacting partners of TCTP has rarely been performed, but many interactions have been identified in small-scale studies in a specific biological system. This chapter, based on information from protein interaction databases and the literature, illustrates current knowledge of the TCTP interactome.

5.1 Introduction

Translationally controlled tumor protein (TCTP), also termed as tumor protein translationally controlled 1 (TPT1), histamine releasing factor (HRF), p23, or fortilin, was initially discovered in the tumor cells in mice by researchers working on translationally regulated genes (Yenofsky et al. 1983; Macdonald et al. 1995). It

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was subsequently found that TCTP is an evolutionarily conserved protein that shares a high degree of homology with the protein from plants to mammals (Acunzo et al. 2014; Amson et al. 2013; Bommer and Thiele 2004). Numerous studies have shown that TCTP plays indispensable roles in various physiological processes, including cell proliferation (Gu et al. 2014; Hsu et al. 2007), cell death (Chen et al. 2014; Lucibello et al. 2011; Susini et al. 2008), cell cycle (Burgess et al. 2008; Johnson et al. 2008), the cytoskeleton (Jeon et al. 2016; Jaglarz et al. 2012; Bazile et al. 2009), protein synthesis (Chen et al. 2013a; Rho et al. 2011), immune responses (Tsai et al. 2014; Kaarbo et al. 2013), malignant transformation (Huang et al. 2015), and nuclear reprogramming (Amson et al. 2013; Roque et al. 2016; Wu et al. 2012; Cheng et al. 2012; Sirois et al. 2011). Telerman and colleagues demonstrated that TCTP plays an important role in tumor reversion, which is defined as the process by which cancer cells lose their malignant phenotype (Tuynder et al. 2002, 2004). Our previous study also revealed that downregulation of TCTP in multiple myeloma cells can lead to tumor reversion (Ge et al. 2011). Importantly, increasing evidences suggest that TCTP is a promising therapeutic target for cancer prevention and intervention (Acunzo et al. 2014; Lucibello et al. 2015; Baylot et al. 2012).

The role of TCTP in many cellular functions is the result of its dynamic interactions with numerous cellular proteins. It is well known that tumorigenesis is the consequence of multiple genetic and epigenetic events that induce cell proliferation and the progression of tumor growth. TCTP was implicated in diverse cellular functions due to interactions with other proteins related to tumorigenesis. Therefore, identification and the characterization of the TCTP interacting proteins on a large scale is important for the understanding of its regulatory mechanisms and revealing its functions in tumorigenesis.

5.2 Global Interactome Profiling Methods

Individual proteins perform their functions through interactions with other proteins and these interactions are crucial for all cellular processes. The knowledge about the entire set of protein interactions (interactome) is essential for our understanding of both the function of individual proteins and the functional organization of the whole cell (Lage 2014). Many experimental high-throughput (HTP) approaches have been developed to determine the protein interactomes in various organisms on a large scale. Through their integration with other "omics" data, interactome datasets have provided valuable information to uncover the functional cellular protein networks and the origin of many diseases. In this chapter, we discuss the emerging and established techniques currently employed to identify the interactome with a particular focus on yeast two-hybrid screens (Y2H) and mass spectrometry (MS)-based approaches. Excellent in-depth reviews on HTP approaches are already available (Mehta and Trinkle-Mulcahy 2016; Lievens et al. 2010).

The yeast two-hybrid (Y2H) assay has been used for over 25 years and remains the most popular choice for researchers investigating interactomes (Rajagopala 2015). This assay is a genetic complementation technique where the proteins to be tested for interaction (referred to as "bait" and "prey") are fused to the DNA-binding domain and the activation domain of the transcription factor (Bruckner et al. 2009). The proteins are co-expressed in a yeast strain reconstituting transcription factor activity, which drives the expression of a reporter gene (Lentze and Auerbach 2008). Large-scale Y2H strategies have been applied to map the human interactome and to generate protein interactome in a number of model organisms (Rajagopala 2015; Zhang et al. 2010; Yu et al. 2008; Li et al. 2004). There are many different variations of the Y2H assay developed such as the recruitment of the bait and prey to the cytosol, plasma membrane, and endoplasmic reticulum or using multiple baits (Koegl and Uetz 2007; Stellberger et al. 2010).

MS-based proteomics has become a widely used technology to identify protein–protein interactions (PPIs) during the past decade (Smits and Vermeulen 2016). The workflows of the commonly used MS-based interaction proteomics are based on affinity-purification MS (AP-MS) of the protein of interest using specific antibodies. The application of quantitative proteomics such as quantitative immunoprecipitation combined with knockdown (QUICK) for protein enrichments from crude lysates to discriminate bona fide interactors from background proteins has proved to be particularly useful (Ge et al. 2010; Selbach and Mann 2006; Zheng et al. 2012; Chen et al. 2013b). Recently, many different MS-based global interactome profiling approaches have been developed, such as proximity-ligation technology based on engineered ascorbate peroxidase (APEX) labeling and global interactome profiling based on the co-behavior of proteins in biochemical fractionations (Havugimana et al. 2012; Kristensen et al. 2012) or perturbation experiments (Christoforou et al. 2016).

An alternative method that has proved invaluable in protein interactome research is protein arrays, which are miniaturized parallel assay systems that contain small amounts of purified proteins in a high-density format (Phizicky et al. 2003). They allow the simultaneous determination of a variety of analytes from small amounts of samples in a single experiment (Tao et al. 2007; Tao and Zhu 2006). This technique has undergone considerable developments since it was first introduced (Chen et al. 2013b; Yang et al. 2016). This has become one of the most powerful multiplexed detection platforms, which can be used for identification of protein interactome, antibody classification, and protein functional analysis.

5.3 The Current Knowledge of the TCTP Interactome

The results of TCTP interactome analysis imply that TCTP interacting proteins belong to a range of functional groups (Li et al. 2016), including nucleic acid-binding proteins, cytoskeletal proteins, chaperones, enzyme modulators, and transferases, which is consistent with the multifunctional nature of TCTP.

5.3.1 Chaperone Proteins

Chaperone proteins from the highly conserved HSP70 family were identified to be interacting partners of TCTP, and HSPA9 was subsequently confirmed as a TCTP-binding partner (Li et al. 2016). HSP70 family members have key regulatory roles in a variety of cellular stress responses (Murphy 2013; Daugaard et al. 2007). The study of over-expression of HSPA9 in tumor cells demonstrated its function in protecting cells from oxidative damage (Liu et al. 2005; Orsini et al. 2004). Interestingly, TCTP is upregulated during oxidative stress and has been implicated as an antioxidant protein (Lucibello et al. 2011; Oikawa et al. 2002; Rupec et al. 1998). Thus, it is likely that the HSPA9–TCTP complex can function together in resistance to intracellular oxidative stress. The complex of TCTP and HSP70 family may also be involved in anti-apoptotic processes (Fig. 5.1a), which is similar to the HSP27–TCTP complex (Baylot et al. 2012).

5.3.2 Nucleic Acid-Binding Proteins

A large number of nucleic acid-binding proteins are the regulatory proteins of transcription and translation. Among them, XRCC6 (Ku70) has been demonstrated to play a critical role in genomic stability maintenance by binding to TCTP and XRCC5 (Ku80) (Zhang et al. 2012; Wang et al. 2015; Gullo et al. 2006). When DNA double-strand breaks (DSB) occur, TCTP accumulates at the damage sites, co-localizing with XRCC6 and XRCC5 (Ku80) and forms complexes for DSB repairs (Fig. 5.1b). However, the levels of XRCC5 and XRCC6 in nuclei are reduced in the absence of TCTP (Zhang et al. 2012), suggesting that TCTP can act as a chaperone. Moreover, Gurdon et al. demonstrated that TCTP directly binds to the promoter region of *oct4* and acts as a transcription factor for this gene (Koziol et al. 2007). They further showed that TCTP also indirectly activates *nanog* transcription by binding to a distant site from its promoter (Koziol et al. 2007). Together, the interactome analysis further confirmed that TCTP is a critical transcription and translation regulator and may fulfill its functions by binding to other proteins.

5.3.3 Cytoskeletal Proteins

The cytoskeleton is a highly dynamic system comprising of different groups of structural proteins including tubulin, actin, and intermediate filaments to form polymers and associated proteins with diverse regulatory functions (Petrasek and Schwarzerova 2009). TCTP has been reported to be associated with cytoskeleton proteins and many related cellular processes (Bazile et al. 2009; Gachet et al. 1999;

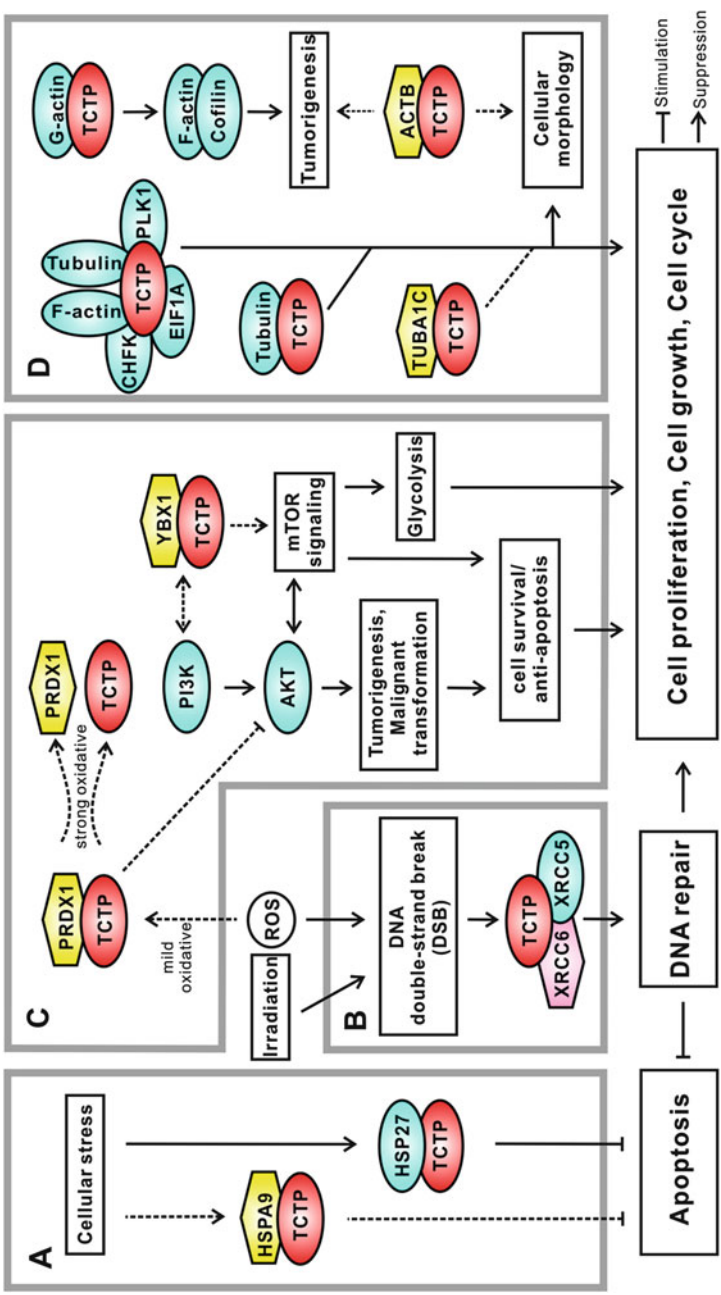

Fig. 5.1 Proposed model depicting the molecular mechanism of the validated TCTP-binding proteins. (**a**) When exposed to cellular stress, HSPA9–TCTP may form complexes to resist apoptosis, which is similar to HSP27–TCTP complex. (**b**) When exposed to irradiation or ROS, which may result in DSB, the TCTP–XRCC6–XRCC5 complexes will form and exert their function in DSB sensing and repairing. (**c**) The TCTP–PRDX1 complex may prevent Akt-driven transformation by similar mechanism as TCTP–PTEN in a mild oxidative stress. However, under high doses of oxidative stress, TCTP may also dissociate from oxidized PRDX1 and inactivated. The TCTP–YBX1 complexes are associated with PI3K–Akt signaling pathway which regulates tumorigenesis, malignant transformation, and mTOR signaling. The complexes may also activate mTOR signaling directly and further regulate cell proliferation, cell growth, and cell cycle. (**d**) The ACTB–TCTP and TUBA1C–TCTP complexes may probably have similar functions as the interaction of TCTP between F-actin, G-actin, and tubulin and exert their function in cell morphology, tumorigenesis, cell proliferation, cell growth, and cell cycle. *Red*: TCTP protein. *Yellow*: the novel TCTP-binding proteins; *Pink*: TCTP-binding proteins that identified previously. *Blue*: molecules contribute to those functional pathways. The *solid line* indicates the reported regulation relationships, while the *dash line* indicates the conjectural regulation relationships

Tsarova et al. 2010). For example, TCTP is involved in regulating cell shape, probably via complex interactions with both F-actin and the microtubule cytoskeleton (Bazile et al. 2009). TCTP also associates with microtubules during specific phases of the cell cycle by binding to tubulin (Gachet et al. 1999). TCTP can release the binding of cofilin to G-actin and transfer the active cofilin to F-actin, increasing the cofilin-activity cycle in invasive tumor cells (Tsarova et al. 2010). The proteomics analysis reveals 15 TCTP interacting cytoskeleton proteins and sheds new light on the role of TCTP in cytoskeleton-related functions like cell morphology, tumorigenesis, cell proliferation, cell growth, and cell cycle (Fig. 5.1d).

5.3.4 Other Functions

PRDX1 and YBX1 were also validated to be TCTP-binding partners. PRDX1 was the first antioxidant protein reported to protect other proteins from inactivation through interaction (Neumann et al. 2009). When exposed to mild oxidative stress, PRDX1 is upregulated and binds to phosphatase and tensin homolog (PTEN) to protect it from oxidation-induced inactivation (Neumann et al. 2009). However, under high doses of oxidative stress, PTEN irreversibly dissociates from oxidized PRDX1 and becomes inactivated, resulting in hyperactivation of Akt signaling (Neumann et al. 2009; Stambolic et al. 1998, 2000; Backman et al. 2004). Notably, TCTP upregulation has been detected in surviving cells after oxidative stress (Lucibello et al. 2011). Conversely, when exposed to a strong oxidative stress, cancer cells caused a downregulation of TCTP, followed by cell death (Lucibello et al. 2011). Therefore, the binding of TCTP and PRDX1 may also be involved in antioxidant pathways, and the TCTP–PRDX1 complex may prevent Akt-driven transformation by a similar mechanism as PTEN under mild oxidative stress (Fig. 5.1c).

YBX1 has been implicated in numerous cellular processes similar to TCTP. The pleiotropic functions of YBX1 and TCTP indicate that the TCTP–YBX1 complex may be involved in vital signaling pathways. YBX1 is closely related to the PI3K/Akt/mTOR signaling pathway (Dazert and Hall 2011). It transcriptionally activates the expression of *PIK3CA* in basal-like breast cancer cells (Astanehe et al. 2009). Serine phosphorylation of the YBX1 102 residue relies on Akt kinase activity (Sutherland et al. 2005; Basaki et al. 2007; Sinnberg et al. 2012). The inhibition of the PI3K pathway can also reduce the expression of YBX1 (Sinnberg et al. 2012). Through experiments of YBX1 silencing, Lee et al. confirmed that the reduction of YBX1 resulted in decreasing of mTOR protein levels (Lee et al. 2008). Interestingly, the translation of *TCTP* mRNA is regulated by PI3-K/Akt/mTOR signaling, and a positive feedback loop between TCTP and mTOR contributes to tumor formation (Bommer et al. 2015; Kobayashi et al. 2014). By interacting with each other, TCTP and YBX1 may work cooperatively in the PI3K/Akt/mTOR pathway, which regulates tumorigenesis, malignant transformation, and mTOR signaling. The complexes may also be directly related to mTOR signaling and

may further influence the glycolysis pathway to regulate cell proliferation, cell growth, and cell cycle (Fig. 5.1c).

5.4 Concluding Remarks

As described above, the TCTP interactome is complex, multifunctional, and has many missing pieces. There are still many unexplained functions that have not yet been attributed to a specific TCTP interactor. Much work is still required to understand the TCTP interactome and its physiological significance. Large-scale approaches, such as next generation Y2H, tandem-affinity purification coupled to MS, and protein arrays might lead to the identification of the new interactors of TCTP and the entire TCTP interactome. The identification of bona fide TCTP interactors can reveal novel functional properties of TCTP. A complete identification of the TCTP interactome will give us a better understanding of the role of TCTP, its mechanism of action, and its associations with the interacting proteins to affect diverse biological and pathological processes. It is critical to identify the key hubs and nodes in the TCTP interaction networks as well as to obtain a detailed molecular characterization of the TCTP interactome. This knowledge will facilitate the development of agents to perturb (or mimic) these interactions. As an example of the potential of this approach, the identification of the interaction between TCTP and YBX1 gives us an idea to find the therapeutic small molecule compound (inhibitor) which can specifically block the interaction (Li et al. 2016). Therefore, elucidating the mechanisms of action of TCTP interactome should provide substantial benefits for the discovery of novel drug targets and biomarkers of disease.

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