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Education

July 2009 - May 2017	**Ph. D.** in Plant Sciences, Dept. of Plant Sciences, School of Life Sciences, University of Hyderabad, Hyderabad, India. Guide: **Prof. Ragiba Makandar.**
July 2004 - July 2006	**M. Sc.** (Life Sciences), Dept. of Biochemistry and Molecular Biology, Pondicherry University, Pondicherry, India (CGPA- 7.23 /10)
July 2003 - July 2000	**B. Sc.** (Botany, Zoology, Chemistry), Deen Dayal Upadhyaya College, University of Delhi, India - (58.2 %)

Research Experience

July 2009 - May 2017	**Ph.D. Thesis**: "Characterization of powdery mildew causing *Erysiphe* pathogen on garden pea through molecular approaches." UGC-BSR-RFSMS Fellowship (2012-2015)
July 2008 - July 2009	**Project JRF**, at Indian Institute of Millets Research, Rajendra Nagar, Hyderabad. DBT project: "Genetic transformation of Indian sweet sorghum genotypes using *Bt* gene constructs and molecular analysis." The work involved introduction of *Bt* genes, *cry 1 Aa*, and *cry 1B* in Indian sweet sorghum genotypes, SSV-84 and RSSV-9 to induce resistance against spotted stem-borer, *Chilo partellus* using genetic transformation methods as *Agrobacterium*-mediated transformation, particle bombardment and *in planta* transformation. Also, contributed in an institute project on intra-specific and inter-specific hybridization of male sterile lines of sorghum with wild relatives and maize lines respectively.

Publications:

1. Barkha Ravi, Poonam Kanwar, Sibaji K. Sanyal, **Malathi Bheri,** Girdhar K. Pandey. VDACs: An outlook on biochemical regulation and function in animal and plant systems. **Frontiers in Physiology,** 2021; doi: 10.3389/fphys.2021.683920. (IF=4.566)

2. **Bheri M.**, Mahiwal S., Sanyal SK. and Pandey GK. Plant protein phosphatases: What are we learning about their mechanism of action? **The FEBS Journal**. 288(3) 756–785, 2021; doi: 10.1111/febs.15454. (IF=4.392)

3. **Bheri M.** and Pandey GK. Protein Tyrosine Phosphatases: Implications in regulation of stress responses in plants. In: Pandey G.K. (eds.) **Protein Phosphatases and Stress Management in Plants**. Springer, Cham. 2020; doi: 10.1007/978-3-030-48733-1_17.

4. Bhosle S., Nitinkumar M., **Bheri M.**, and Makandar, R. Detection of putative pathogenicity and virulence genes of *Erysiphe pisi* using genome-wide *in silico* search and their suppression by *er2* mediated resistance in garden pea. **Microbial Pathogenesis**. Vol. 136, 103680, 2019; doi: 10.1016/j.micpath.2019.103680. (IF=2.914)

5. **Bheri M.**, Pandey G. K. PP2A phosphatases take a giant leap in the post-genomics era. **Current Genomics**. Vol. 20, Issue 3, 154-171, 2019a; doi: 10.2174/1389202920666190517110605. (IF=2.630)

6. **Bheri M.**, Bhosle S. and Makandar, R. Shotgun proteomics provides an insight into pathogenesis-related proteins using anamorphic stage of the biotroph, *Erysiphe pisi* pathogen of garden pea. **Microbiological Research**. Vol. 222, 25-34, 2019; doi: 10.1016/j.micres.2019.02.006. (IF=3.970)

7. **Bheri M.**, Pandey G. K. Protein phosphatases meet reactive oxygen species in plant signaling networks. **Environmental and Experimental Botany**. Vol. 161, Pages 26-40, 2019b; doi: 10.1016/j.envexpbot.2018.10.032. (IF=4.027)

8. **Bheri M.**, Fareeda G., Makandar, R. Assessing host specialization of *Erysiphe pisi* on garden pea germplasm through genotypic and phenotypic characterization. **Euphytica**. Vol. 212, 1-14, 2016, doi: 10.1007/s10681-015-1511-3. (IF=1.614)

9. GenBank Submissions (KM189820-25, KM096758-60, KM189826-29, KM099155-KM099156), 2014; (KR028332- KR028334) 2017.

Technical Skills:

1. **Culture and maintenance of micro-organisms:** Fungal and bacterial culture maintenance in the form of glycerol stocks and stab cultures**.**
2. **Genetic transformation:** *Agrobacterium*-mediated transformation, particle bombardment and *in planta* method.
3. **Plant tissue culture:** Tissue culture using explants as immature embryos, immature infloresences and shoot apical meristems, methods as Gus and Basta assays.
4. **Molecular Biology:** Plasmid isolation, genomic DNA isolation, restriction digestion, PCR, ligation, heat shock transformation, electroporation, DNA gel elution, RAPD-

SCAR marker approach (random amplified polymorphic DNA- Sequence characterized amplified region), Southern blotting**.**

5. **Proteomics:** Protein isolation and proteome profiling using LC-MS/MS protocol, Protein over-expression analysis by SDS-PAGE.
6. **Morphological analysis:** Characterization of fungal specimens using Laser scanning Confocal Microscopy (LSCM) and Scanning Electron Microscopy (SEM).
7. **Plant pathology:** Detached and whole plant assays using fungal pathogens for pathogenicity assays.
8. **Computational Biology:** Data mining using ENTREZ, BLAST, etc., Sequence submission and retrieval systems, homology searches, pairwise alignment and multiple sequence alignment and phylogenetic software MEGA; STRING analysis.

Conferences/Symposia/Workshops attended:

1. Delivered oral presentation in Plant Sciences Colloquium – 2013 organized by Dept. of Plant Sciences, University of Hyderabad on 4 March, 2013.
2. Presented a poster titled "Characterization of *Erysiphe pisi* isolates and garden pea lines to identify a set of host differentials against powdery mildew disease" - **Malathi B.** and Ragiba M. in "64th Annual Meeting of Indian Phytopathological Society and National Symposium on Biology of Infection, Immunity and Disease Control in Pathogen-Plant Interactions", at UoH during Dec. 2-4, 2011.
3. Participated in Genomics training workshop (UoH-DBT-CREBB) for Quantitative PCR and Microarray from Nov. 8th-Nov. 12th, 2010 at UoH.
4. Presented poster on "Identification of genetic components involved in pathogenesis and avirulence of a fungal pathogen *Erysiphe pisi*, causing powdery mildew disease in pea" - **Malathi B.**, Santosh H., Shyam G., Priyanka Divya A. and Ragiba M. in Bioquest 2010, The 34th Annual Research Gala at School of Life Sciences, University of Hyderabad, Hyderabad on March 20, 2010.
5. Presented poster on "Identification of genetic components involved in pathogenesis and avirulence of a fungal pathogen *Erysiphe pisi*, causing powdery mildew disease in pea" - **Malathi B.**, Santosh H., Shyam G., Priyanka Divya A. and Ragiba M. in the National Biotechnology Symposium on "Genomics and Crop Improvement: Relevance and Reservations", jointly organized by Indian Council of Agricultural Research and Department of Biotechnology, Govt. of India at Institute of Biotechnology, Rajendra

Nagar, Hyderabad during Feb. 25-27, 2010.

6. Participated in 5th AOHUPO Congress, 14th ADNAT Convention and 1st PSI conference on "New Perspectives in Proteome Research" at Centre for Cellular and Molecular Biology (CCMB), Hyderabad during February 21-25, 2010.

7. Presented poster on "Identification of genetic components involved in pathogenesis and avirulence of a fungal pathogen *Erysiphe pisi*, causing powdery mildew disease in pea" - **Malathi B.**, Santosh H., Shyam G., Priyanka Divya A. and Ragiba M. in the XXXIII All India Cell biology Conference & International Workshop on Cell Cycle Regulation at University of Hyderabad, Hyderabad during Dec.10-13, 2009.
8. Participated in International conference on "Current trends in Biotechnology and its implications in Agriculture" held at Meerut, UP from Feb. 19-21, 2009.
9. Participated in national seminar on "Bioinformatics and Functional Genomics" sponsored by the Dept. of Biotechnology, Govt. of India, from Feb. 15-17, 2006 at Pondicherry University, Puducherry.
10. Participated in the 93rd Indian Science Congress (Jan. 3-7, 2006) held at Acharya N.G. Ranga Agricultural University, Hyderabad (Jan. 3-7, 2006).
11. Participated in the 75th Annual session (Dec.8-9, 2005) of The National Academy of Sciences, India held at Pondicherry University.
12. Participated in seminar on "Nanosciences and Nanobiotechnology" sponsored by the Dept. of Biotechnology, Dept. of Science and Technology, Govt. of India and Pondicherry University, from Dec. 5-6, 2005 at Pondicherry University, Puducherry.
13. Organization and Participation in National Science Day Seminar on 28 Feb. 2006, "Gene'sis 06" in the Dept. of Biochemistry and Molecular Biology, Pondicherry University.

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Shotgun proteomics provides an insight into pathogenesis-related proteins using anamorphic stage of the biotroph, *Erysiphe pisi* pathogen of garden pea

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ABSTRACT

E. pisi is an ascomycete member causing powdery mildew disease of garden pea. It is a biotrophic pathogen, requiring a living host for its survival. Our understanding of molecular mechanisms underlying its pathogenesis is limited. The identification of proteins expressed in the pathogen is required to gain an insight into the functional mechanisms of an obligate biotrophic fungal pathogen. In this study, the proteome of the anamorphic stage of *E. pisi* pathogen has been elucidated through the nano LC–MS/MS approach. A total of 328 distinct proteins were detected from *Erysiphe* isolates infecting the susceptible pea cultivar, Arkel. The proteome is available via ProteomeXchange with identifier PXD010238. The functional classification of protein accessions based on Gene Ontology revealed proteins related to signal transduction, secondary metabolite formation and stress which might be involved in virulence and pathogenesis. The functional validation carried through differential expression of genes encoding G-protein beta subunit, a Cyclophilin (Peptidyl prolyl cis-transisomerase) and ABC transporter in a time course study confirmed their putative role in pathogenesis between resistant and susceptible genotypes, JI2480 and Arkel. The garden pea-powdery mildew pathosystem is largely unexplored, therefore, the identified proteome provides a first-hand information and will form a basis to analyze mechanisms involving pathogen survival, pathogenesis and virulence.

1. Introduction

Garden pea (*Pisum sativum* L.) is an important edible legume crop cultivated worldwide. The crop is consumed as vegetable and pulse due to its high nutritive value and is also used as fodder for cattle. *Erysiphe pisi* is an obligate biotrophic fungal pathogen which causes the destructive powdery mildew disease in garden pea worldwide. The disease appears as white specks on infected pea leaves which gradually infect the whole plant resulting in loss of nutrients to the fungus, disruption of photosynthetic process and premature death of leaves and other infected plant tissues. This devastative disease affects pea yield and its productivity potential leading to 25–50% yield losses globally (Bhardwaj and Sharma, 1984; Munjal et al., 1963; Warkentin et al., 1996). In India, a loss of 20–30% in pod number and 25% reduction in pod weight has been reported as a result of *E. pisi* attack (Smith et al., 1988). Nisar et al. (2006) have reported crop losses ranging from 25% to 86%.

Although powdery mildews are most commonly occurring Ascomycete plant pathogenic fungi, studies in these pathogens are limited due to their obligate biotrophic nature rendering difficulty in pursuing *ex-situ* or *in-vitro* studies. Studies have been carried to unravel the genetic and molecular nature of interactions between powdery mildews and their hosts, mostly on powdery mildew fungi infecting barley, wheat and *Arabidopsis thaliana*, the model plant (Bindschedler et al., 2011, 2009; Hacquard et al., 2013; Spanu, 2014; Spanu et al., 2010; Wicker et al., 2013). Though proteome based studies have been reported in the fungus, *Blumeria graminis*, another powdery mildew causing member of the Poaceae family, the pathogen is phylogenetically distinct from *E. pisi*, as it forms a separate clade from other powdery mildew fungi (Braun, 1987, 1981; Cook et al., 1997; Saenz and Taylor, 1999; Sperr, 1973; Zeller, 1995).

The whole genome sequencing studies of three of the powdery mildew fungi, *B. graminis* f. sp. *hordei* (*Bgh*), *Erysiphe pisi* and *Golovinomyces orontii* deciphered sequence sizes of ~120 Mb, ~151 and ~160 Mb respectively (Spanu et al., 2010). The genome of *E. necator* was also estimated to be 126 ± 18 Mb by Jones et al. (2014). The studies of fungal genomes reveal major gene losses despite their large genome size which accounts to be more than four times greater in comparison to the median of other ascomycete members. The *Blumeria* genome showed only 5854 curated genes and reduced number of protein-coding genes (~6500 annotated genes in *Bgh* and *Bgt*) in spite of their large genome size (120–160 Mbp) owing to genome expansion resulting from

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Assessing host specialization of *Erysiphe pisi* on garden pea germplasm through genotypic and phenotypic characterization

Malathi Bheri · Gemmalamadugu Fareeda · Ragiba Makandar

Received: 6 January 2015 / Accepted: 2 July 2015

Abstract *Erysiphe pisi, Erysiphe baeumleri and Erysiphe trifolii* cause powdery mildew disease in garden pea. So far, three genes *er1, er2* and *Er3* imparting resistance against *E. pisi* have been reported in pea. The *E. pisi* isolate used in this study, Ep01 (GenBank Accession No. KM096758) was identified using the internal transcribed spacer sequence of the ribosomal DNA region. Testing of garden pea germplasm lines and cultivars against *E. pisi* pathogen under green house conditions revealed varying levels of resistance. The resistant control lines JI2302, JI2480 and P660-4 with reported resistance genes *er1, er2* and *Er3* respectively showed significant levels of resistance to the *E. pisi* isolate, Ep01 while the susceptible control lines, Messire and Lincoln showed significant levels of susceptibility. Out of the forty six pea lines (including the control lines) tested, three lines were found to be highly resistant, three lines were resistant, eighteen lines were moderately resistant, twelve were moderately susceptible, six were susceptible and four lines were highly susceptible. The molecular screening using SCAR markers for *er1* gene, Sc-OPO-18_{1200}, Sc-OPE-16_{1600}, ScOPX 04_{880} and ScOPD 10_{650} did not lead to identification of *er1* gene due to lack of expected amplification with Sc-OPE-16_{1600} and Sc-OPO-18_{1200} while no polymorphism was observed between resistant and susceptible controls with ScOPX 04_{880} and ScOPD 10_{650}. The SCAR marker for *er2* gene, ScX17_1400 led to identification of twenty four lines that may be carrying *er2* gene while SCAR markers for *Er3* gene, $SCW4_{637}$ and $SCAB1_{874}$, helped identify four lines as potential sources of *Er3* gene.

Keywords Garden pea · Powdery mildew disease · *Erysiphe pisi* · SCAR markers · Percent disease index · *er1, er2* and *Er3* resistance genes · Ep01 (GenBank Accession No. KM096758)

Introduction

Garden pea (*Pisum sativum* L.) is one of the most important edible legume crops cultivated worldwide. The crop is a rich source of nutrients and is consumed as either a vegetable or a pulse and also used as fodder for cattle. The pea seeds with an estimated total protein content of 22 %, serves as a rich source of protein and fibre. Among the pathogens that infect garden pea, the powdery mildew disease caused by the fungal pathogens belonging to *Erysiphe* sps., is a devastative disease affecting crop yield and its productivity potential. Losses due to the disease are severe causing

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Detection of putative pathogenicity and virulence genes of *Erysiphe pisi* using genome-wide *in-silico* search and their suppression by *er2* mediated resistance in garden pea

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ABSTRACT

The biotrophic fungus, *Erysiphe pisi* is the chief causal agent of powdery mildew disease of garden pea. A genome-wide search using *in-silico* approach was carried to detect putative pathogenicity and virulence genes of *E. pisi*, since information about these genes and their interaction with pea is limited. Nineteen putative pathogenicity gene sequences were detected through genome-wide pathogenicity gene-search and confirmed them to be conserved in *E. pisi* through genomic PCRs. Fifteen of these genes expressed through reverse transcriptase-polymerase chain reaction (RT-PCR) amplifying expected band size along with fungal and plant specific internal controls. Gene sequencing and annotation revealed them to be *Erysiphe*-specific. A time course study was carried to monitor expression of nine of these genes through real-time quantitative (qRT)-PCR in *Erysiphe*-challenged plants of powdery mildew resistant pea genotype, JI-2480 carrying *er2* gene and susceptible pea cultivar, Arkel. Expression of these genes was differentially and temporally regulated. They were found mostly related to signaling; cAMP-PKA (*cPKA*, *CRP* and *AC*) and MAPK (*MST7*) pathways along with *MFP*, *TRE* and *PEX* which are reported pathogenicity factors in other ascomycete members indicating that similar conserved pathways function in *E. pisi* also. These genes expressed at higher level at initial hours post inoculation (hpi) as early as 6 hpi in Arkel compared to JI-2480 implying them as pathogenicity factors. The elevated level of expression of *MFP*, *TRE*, *CRP* and *cPKA* gene sequences in *E. pisi*-challenged JI-2480 genotype at 12 hpi alone suggests these genes to possess a role in avirulence in JI-2480, conferring *er2* mediated resistance.

1. Introduction

The powdery mildew disease caused by *Erysiphe pisi* is a serious concern in garden pea (*Pisum sativum* L.). Previous studies to identify resistance to powdery mildew disease had revealed presence of two recessive genes *er1* and *er2* in *Pisum sativum* and one dominant gene *Er3* in *P. fulvum* and these genes are the reported sources that confer resistance to powdery mildew disease in pea [1–4]. Host resistance mechanism as well as the nature of pathogenicity and virulence of *E. pisi* necessitates investigation to combat powdery mildew disease [5]. The manipulation of the host machinery by the pathogen is demonstrated to be genetically regulated through previous research studies carried in other biotrophic fungal pathogens [6,7]. Previous studies attempted to make an insight into the mechanisms of pathogenesis of biotrophic fungal pathogens by characterizing powdery mildew pathogens of barley, *Blumeria graminis* [8] and the model plant *Arabidopsis thaliana* [9] caused by members of the genus, *Golovinomyces* sp. Research studies aiming at unraveling the molecular mechanisms of host-pathogen interaction between *E. pisi* and garden pea are limiting. Therefore, it necessitates studies to detect pathogenicity and virulence of *E. pisi* pathogen in garden pea.

Erysiphe pisi being an adapted pathogen of pea is recognized by the host through pathogen associated molecular patterns (PAMPs) [10]. Effectors are reported to target host immunity by suppressing defense responses and promoting pathogen growth in host tissues. Delivery of these effector proteins into susceptible host cells would enhance susceptibility mediated through effector triggered-susceptibility (ETS). On the contrary, effectors recognized by specific resistance genes trigger Effector triggered immunity (ETI) which manifests in a resistant genotype to confer resistance against adapted pathogens. Targeting pathogenicity and virulence factors would enable combating powdery mildew infection in garden pea. Therefore, the present study was aimed at identifying putative pathogenicity genes using *E. pisi*-pea interaction.

Erysiphe pisi genome sequence information available in the public

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REVIEW ARTICLE

Plant protein phosphatases: What do we know about their mechanism of action?

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Keywords
dephosphorylation; Dual-specificity phosphatases; metallo-dependent protein phosphatases; phosphoprotein phosphatases; plant signaling; PP2A-like phosphatases; protein kinases; protein phosphatases; Protein Tyrosine Phosphatases; Serine/Threonine-specific phosphatases

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Protein phosphorylation is a major reversible post-translational modification. Protein phosphatases function as 'critical regulators' in signaling networks through dephosphorylation of proteins, which have been phosphorylated by protein kinases. A large understanding of their working has been sourced from animal systems rather than the plant or the prokaryotic systems. The eukaryotic protein phosphatases include phosphoprotein phosphatases (PPP), metallo-dependent protein phosphatases (PPM), protein tyrosine (Tyr) phosphatases (PTP), and aspartate (Asp)-dependent phosphatases. The PPP and PPM families are serine(Ser)/threonine(Thr)-specific phosphatases (STPs), while PTP family is Tyr specific. Dual-specificity phosphatases (DsPTPs/DSPs) dephosphorylate Ser, Thr, and Tyr residues. PTPs lack sequence homology with STPs, indicating a difference in catalytic mechanisms, while the PPP and PPM families share a similar structural fold indicating a common catalytic mechanism. The catalytic cysteine (Cys) residue in the conserved HCX_5R active site motif of the PTPs acts as a nucleophile during hydrolysis. The PPP members require metal ions, which coordinate the phosphate group of the substrate, followed by a nucleophilic attack by a water molecule and hydrolysis. The variable holoenzyme assembly of protein phosphatase(s) and the overlap with other post-translational modifications like acetylation and ubiquitination add to their complexity. Though their functional characterization is extensively reported in plants, the mechanistic nature of their action is still being explored by researchers. In this review, we exclusively overview the plant protein phosphatases with an emphasis on their mechanistic action as well as structural characteristics.

Protein phosphatases—What do they do?

Protein phosphorylation is a major reversible post-translational modification, in which protein kinases (PKs) transfer the γ-phosphoryl group of donor ATP to the acceptor protein side chains, while protein phosphatases (PPs) dephosphorylate the phosphoproteins. The human protein kinome and

Abbreviations
ALPH, ApaH-like phosphatases; BR, Brassinosteroid; DsPTPs/DSPs, Dual-specificity phosphatases; HEAT, huntingtin, elongation factor 3, A subunit of PP2A and TOR; MKP, MAPK (Mitogen-activated protein kinase) phosphatases; PKs, protein kinases; PME-1, protein phosphatase methylesterase 1; PPKL, protein phosphatases with Kelch-like domains; PPM, metallo-dependent protein phosphatases; PPP, phosphoprotein phosphatases; PPs, protein phosphatases; PTP, protein Tyrosine (Tyr) phosphatases; PYL, Pyrabactin Resistance1-Like; RLPH, Rhizobiales/Rhodobacterales/Rhodospirillaceae-like phosphatases; SLP, *Shewanella*-like phosphatases; SnRK2, SUCROSE NONFERMENTING1-related protein kinase 2; STPs, Serine (Ser)/Threonine (Thr)-specific phosphatases; TOPP, type-one protein phosphatase.

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Protein phosphatases meet reactive oxygen species in plant signaling networks☆

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Metallo-dependent protein phosphatases (PPM)
Protein tyrosine phosphatases (PTP)
Serine/threonine phosphatases
Dual specificity phosphatases (DSPs)
PP2A
PP2C
PTP
DsPTP
Stress

ABSTRACT

The cellular redox state is vital for the survival of all living systems. The dynamic nature of reactive oxygen species (ROS) are reflected in their functioning both as a damage causal and control agent. They are known primarily for causing oxidative damage and induction of programmed cell death (PCD) in case of pathogen attacks to prevent pathogen proliferation, while at low levels, they act as signal transducer(s) in different signaling pathways. Protein phosphatase(s) and kinase(s) co-ordinate the reversible post-translational modification of proteins, thereby, regulating a number of signaling pathways. In plant systems, protein kinases outnumber the protein phosphatases, which is countered by the diverse functional assemblage of the latter. Protein phosphatases are redox-regulated through reversible oxidation of critical Cysteine (Cys) residues leading to their activation and inactivation. Thus, their diversity and critical involvement in a number of cellular processes, which require redox homeostasis as a crucial element, brings them under the control of redox regulation. The observations made with regard to their involvement in photo-oxidative stress and responses involving ROS as signaling molecules, make a strong case for the potential of protein phosphatases in ROS signaling, with an emphasis on stress biology. In this review, we present the common ground on which ROS and protein phosphatases operate in view of their regulatory functions.

1. Introduction

Protein phosphorylation, an important reversible post-translational modification involved in the regulation of a number of critical cellular processes, occurs in a coordinated manner through two classes of enzymes, the kinases and the phosphatases. The kinases transfer the γ-phosphoryl group of donor ATP to the acceptor protein side chains, while the phosphatases dephosphorylate the phosphoproteins (Barford, 1996). At least two-thirds of human cellular proteins are phosphorylated with phosphorylation on Serine (86.4%), Threonine (11.8%) and Tyrosine (1.8%), respectively (Olsen et al., 2010, 2006). The eukaryotic protein phosphatases are classified as the phosphoprotein phosphatases (PPP), metallo-dependent protein phosphatases (PPM), protein tyrosine phosphatases (PTP), and Aspartate-dependent phosphatases (Kerk et al., 2008; Uhrig et al., 2013a). The PPP and PPM families are Ser/Thr-specific phosphatases (STPs) while PTP are Tyr specific. Dual-specificity phosphatases (DSP's) dephosphorylate all three phosphoresidues (Keyse, 1995; Stone and Dixon, 1994; Tonks and Neel, 1996). The PPP family includes PP1, PP2A, PP2B (Calcineurin, found in fungi and animal systems only), distantly related PP4-7 with unknown functions while the PPM family includes PP2C phosphatases and other Mg^{2+} or Mn^{2+}-dependent protein phosphatases (Kerk et al., 2008). The classical PPP family in eukaryotes also includes *Shewanella*-like (SLP) phosphatases, Rhizobiales-like (RLPH) phosphatases and ApaH-like (ALPH) phosphatases that are highly similar to PPP-like protein phosphatases with a prokaryotic origin (Andreeva and Kutuzov, 2004; Uhrig et al., 2013b; Uhrig and Moorhead, 2011). The molecular evolution of these bacterial-like PPP classes identified in eukaryotes involve ancient mitochondrial/archaeal origin and lateral gene transfer. SLP phosphatases are absent in red alga, cyanobacteria, amoebozoa, animalia and archaea, but found in plants, mosses, and green algae (Uhrig and Moorhead, 2011). Homologs of eukaryotic protein phosphatases, PTPs, low molecular weight PTP (LMWPTP), PPPs and PPMs are present in archaea as well as bacteria and function as translation factor(s), small ribosome-associated GTPase, phosphotransferase system, stress responses, phosphoprotein anti-anti-sigma factor, sigma B regulator, negative effector of development, purine biosynthesis, transcriptional regulator and histone-like protein(s) (Pereira et al., 2011). The human proteome encodes up to 255 phosphatases (Sacco et al., 2012), having implications in cancers, auto-immune disorders and inherited genetic

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