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涵蓋既深且廣的跨學科線上資源,主題分類包括15大類,104小類,類別如下:

- Agriculture, Aquaculture & Food Science 農業、漁業及食品科學
- Architecture & Planning 建築及規畫
- Art & Applied Arts 藝術及應用藝術
- Business, Economics, Finance & Accounting 商業、金融及會計
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- Earth & Environment 地球及環境科學
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John Wiley & Sons, Inc. 今日公告,根據2012年Thomson Reuters®公布的Journal Citation Reports,其旗下引文索引的期刊比例仍在持續攀升,目前達1,192(約77%)份,已高於2011年JCR的1,156。Wiley已躍升為50類別期刊佔比最高的佼佼者。

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- Wiley 目前有 1,192 份期刊擁有影響係數
- 25 份Wiley 期刊位居類別排名之首,並橫跨31個類別
- 264 份Wiley 期刊名列各類前十大排名,且涵蓋 341 個類別
- 59.4% 的 Wiley 期刊影響係數增加
- 在232 個主題中,Wiley 期刊包含218個主題
- Wiley 期刊在 50 類別中的佔比最高
- 《CA A Cancer Journal for Clinicians(臨床醫師癌症期刊)》連續獲得JCR 的最高影響係數

化學、物理科學及工程領域的成果卓越:

今年有四份刊物名列所屬類別之首,分別是《Computer-Aided Civil》以及《Infrastructure Engineering》(4.460,同時位居三大類別之冠)。代表IUCR發行之《Acta Crystallographica Section D》的影響係數增加至14.103,已躍居二大類別之首,而《International Journal of Energy Research》(1.987),還有《WIREs Computational Molecular Science》(5.738) 亦首度獲得影響係數,並以黑馬之姿衝上JCR數學及計算生物學類頂端。

其他表現亮眼而首度獲得影響係數的期刊,還有《Advanced Energy Materials》(10.043)、《Greenhouse Gases: Science and Technology》(2.679 / 與SCI聯合出版)、《International Journal of Applied Glass Science》(1.548 / 代表美國陶瓷協會發行),以及《WIRES Data Mining & Knowledge Discovery》(1.422),這些都是Wiley在出版上致力追求創新的卓越成果。

德國化學學會根據 Wiley-VCH 出版之《Angewandte Chemie》期刊的影響係數,也提高至13.734,並已穩坐發表基礎研究與審查著作之多學科化學期刊的龍頭寶座。由Wiley-VCH及ChemPubSoc Europe共同出版,《ChemSusChem, Angewandte》之姊妹期刊的最新影響係數,也有7.475,增加了43%,表現十分亮眼。

生命、地球及環境科學的卓越表現:

Wiley生命、地球及環境科學出版計畫中有85種期刊排名所屬類別前十名,總計101個排名前十大。八種期刊排名位居類別之首,包含影響係數為17.949,穩居生態學龍頭的《Ecology Letters》。

六種新期刊首度獲得影響係數,包括4.186的《WIREs RNA》、3.446的《Biotechnology Journal》以及係數1.184並於最近開放存取的期刊《Ecology and Evolution》。

《Annals of the New York Academy of Sciences》(4.375)、從10.960增加至13.231《FEMS Microbiology Reviews)微生物學文獻》、《Human Brain Mapping》(6.878),以及代表美國地球物理聯盟(AGU)出版,係數13.906,在地球化學與地球物理類別雙雙奪冠的《Reviews of Geophysics》,均有令人激賞的卓越成績。

於最近轉成開放存取模式的《Microbial Biotechnology》,第二次獲得之影響係數已從2.534增加至3.214,在微生物學類別的排名也前進了14名,到達38/116,而在生物技術與應用微生物學類別的排名也上升21名,達到41/159。

衛生科學的卓越成績 (I):

今年Wiley有329種衛生科學期刊進入JCR,其中有8種期刊名列第一,總計有62種期刊名列主題類別的前十名,還有3種期刊獲得第一次檢索以及首度取得影響係數。

第一名的期刊包含《CA – A Cancer Journal for Clinicians》(153.459 / 腫瘤學)、《American Journal of Transplantation》(6.192 / 移植)、《Addiction Biology》(5.914 / 物質濫用)、《Addiction》(4.577 / 物質濫用 - 社會科學)、《Periodontology 2000(4.012 / 牙醫、口腔外科及醫療)、《International Journal of Andrology》(3.565 / 男科學)、《Medical Education》(3.546 / 教育、科學學科),以及《BIRTH: Issues in Perinatal Care》(2.926 / 護理,涵蓋科學與社會JCR)。

Wiley 目前有三種期刊在物質濫用科學類別中排名前三,分別是《Addiction Biology》、《Addiction》以及《Alcoholism: Clinical and Experimental Research》。

衛生科學的卓越成績(Ⅱ):

《British Journal of Pharmacology》期刊也獲得5.067的影響係數 - 成為在藥理學與製藥類別中首屈一指的一般藥理學研究期刊。

代表美國風濕病學院出版的兩種期刊:《Arthritis & Rheumatism》以及《Arthritis Care & Research》,仍穩居風濕病學類別的前十名。

《Cochrane Database of Systematic Reviews》在一般與內科醫學類別的151種期刊中,名列第11位。《CDSR》被引用的總次數,從29,593增加至2011年的34,230,這是《CDSR》第六次榮獲該類別最高引用次數的殊榮。



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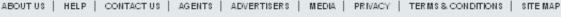
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Anytime a user enters a term in one of the Wiley Online Library search boxes, the system performs automatic stemming of the term(s) eliminating the need for users to manually type several common variations of the search term. The chart below summarizes the common expansions the system will automatically find for English language terms. In addition, for our German language journals and books, German specific stemming rules are applied.

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1. BASIC STEMMING

Search CLEAR (word root)

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- . Searching any of these variants (e.g., CLEARED) will also find all term variants.

CLEAR Word root CLEARS word root + s (plural) word root + ed (past tense) CLEARED CLEARING word root + ing (gerund) word root + er (comparative adjective) CLEARER CLEAREST

2. COMMON BRITISH vs. AMERICAN ENGLISH SPELLING VARIANTS Search TUMOR Search CENTER

. This search will find all of the following variants.

word root + est (superlative adjective)

. Searching any of these variants (e.g., TUMOUR) will also find all term variants.

TUMOR American English (singular) CENTER TUMORS CENTERS American English (plural) TUMOUR CENTRE British (singular) TUMOURS CENTRES British (plural)

3. NON-STANDARD PLURAL VARIANTS Search MOUSE

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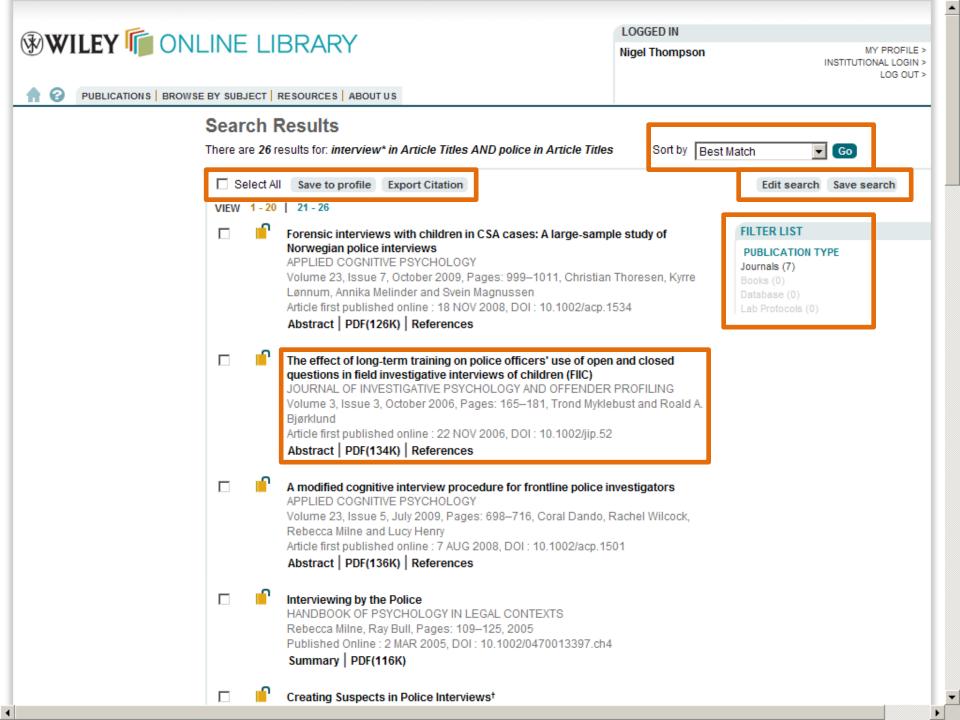
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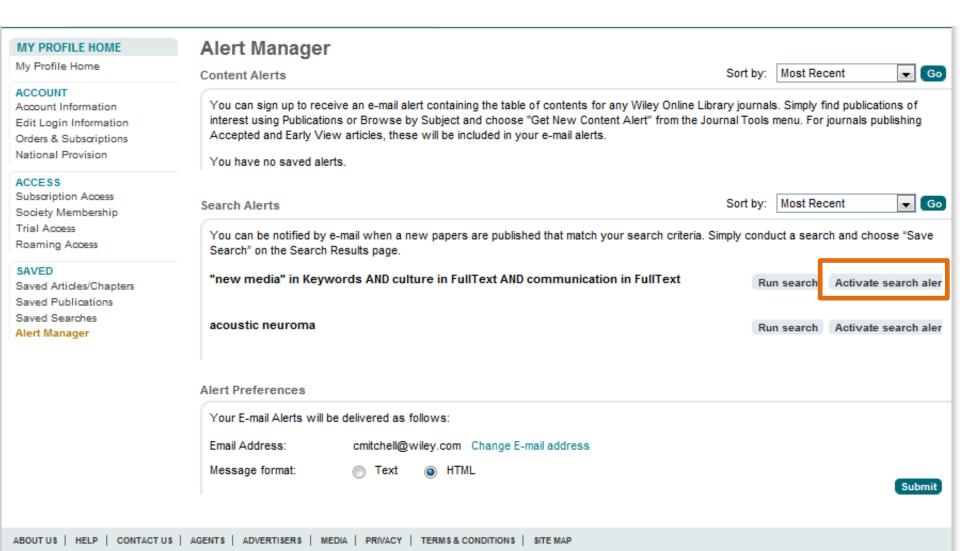


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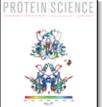
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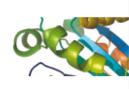
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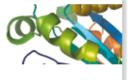
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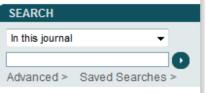
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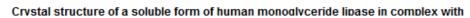
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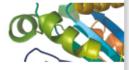
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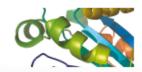








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The following Virtual Issues are available:

- Protein Folding: Short Question Long Answer (July, 2010)
- Learning about proteins that live in membranes (November, 2009)
- Celebrating the Structure of Myoglobin and its Impact on the Science of <u>Proteins</u> (May, 2009)

ISSUE 3, JULY 2010: PROTEIN FOLDING: SHORT QUESTION - LONG ANSWER

Arguably, more articles in *Protein Science* deal with the folding of proteins than any other subject. Solving the protein folding problem also remains as one of the major challenges in biology. This Virtual Issue combines two groups of articles from Protein Science that deal with this subject. The first selections have already established themselves as "citation classics". The second group of selections includes articles published within the past three years that can be characterized as "up-and-coming citation classics". Together, these contributions revisit established highlights and provide pointers to future developments.

Introduction to the Virtual issue

Brian W. Matthews

Principles of protein folding-A perspective from simple exact models

Ken A. Dill, Sarina Bromberg, Kaizhi Yue, Hue Sun Chan, Klaus M. Ftebig, David P. Yee

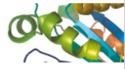
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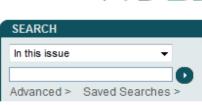






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Domain swapping in the kinase superfamily: OSR1 joins the mixt

Ying Li, Arthur G. Palmer III

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Volume 18, Issue 4, pages 678-681, April 2009

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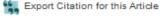
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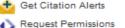
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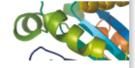
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Domain swapping in the kinase superfamily: OSR1 joins the mix†

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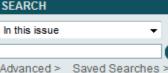
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Protein kinases constitute a large family of proteins that participate in the majority of cell signaling pathways. 1 Kinases act as mediators of a variety of biological processes in both normal physiology and pathogenesis, through specific interactions with upstream and downstream signaling molecules. Most kinases are multidomain proteins consisting of a catalytic kinase domain and several regulatory domains. The structures of kinase domains are conserved and have a two-lobed architecture consisting of a predominantly β -sheet Nterminal lobe and a predominantly α-helical C-terminal lobe.2 In contrast, the regulatory domains of different kinases often have distinct overall folds and local structural motifs required for maintaining pathway specificity. Structure determinations of protein kinases have provided more detailed descriptions of the regulation of kinase activity at the molecular level; however, the mechanisms of critical events, such as autophosphorylation, are still not fully understood.

Some recently determined structures of kinase domains, including Ste20-like kinase (SLK; PDB 2JFL, 2J51) and lymphocyte-originated kinase (LOK; PDB 2J7T) of the Ste20 family and death-associated kinase 3 (DAPK3, PDB 2J90) and checkpoint kinase 2 (CHK2, PDB 2CN5) of the CaMK family, suggest that domain swapping serves as a possible mechanism for trans autophosphorylation between two identical protein kinases.3, 4 In a domain-swapped dimer, one structural element of a molecule is replaced by the identical element from the partner molecule. Domain swapping is a general mechanism for forming protein oligomers and an efficient one from the evolutionary



Domain swapping in the kinase superfamily: OSR1 joins the mix

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Protein kinases constitute a large family of proteins that participate in the majority of cell signaling pathways. 1 Kinases act as mediators of a variety of biological processes in both normal physiology and pathogenesis, through specific interactions with upstream and downstream signaling molecules. Most kinases are multidomain proteins consisting of a catalytic kinase domain and several regulatory domains. The structures of kinase domains are conserved and have a two-lobed architecture consisting of a predominantly β-sheet N-terminal lobe and a predominantly α-helical C-terminal lobe. 2 In contrast, the regulatory domains of different kinases often have distinct overall folds and local structural motifs required for maintaining pathway specificity. Structure determinations of protein kinases have provided more detailed descriptions of the regulation of kinase activity at the molecular level; however, the mechanisms of critical events, such as autophosphorylation, are still not fully understood.

Some recently determined structures of kinase domains, including Ste20-like kinase (SLK; PDB 2JFL, 2J51) and lymphocyte-originated kinase (LOK; PDB 2J7T) of the Ste20 family and death-associated kinase 3 (DAPK3, PDB 2J90) and checkpoint kinase 2 (CHK2, PDB 2CN5) of the CaMK family, suggest that domain swapping serves as a possible mechanism for trans autophosphorylation between two identical protein kinases.3, 4 In a domain-swapped dimer, one structural element of a molecule is replaced by the identical element from the partner molecule. Domain swapping is a general mechanism for forming protein oligomers and an efficient one from the evolutionary point of view, because the interactions between monomers in the domain-swapped interface are native-like and new recognition sites need not be evolved. In the February, 2009 issue of Protein Science, Lee et al.5 reported the X-ray crystal structure of the kinase domain of oxidative stress responsive 1 (OSR1), which represents another example of a domain-swapped protein kinase. A similar report by Villa et al.6 appeared in the December 2008 issue of Proteins: Structure, Function and Bioinformatics, The structural coordinates have been deposited in the Protein Data Bank (PDB) as 3DAK and 2VWI, respectively. OSR1 is a Ser/Thr protein kinase belonging to the Ste20 family. It is one of the two human homologues of the putative Drosophila mitogen-activated protein kinase kinase kinase kinase (MAP4K) Fray,7 and a component of the recently identified WNK-OSR1/SPAK pathway, which is responsible for cell volume control and ion homeostasis in mammals and is activated by osmotic stress.8 In the pathway, with-no-lysine kinases (WNKs) activate OSR1, which in turn activates the Na +/K+/2Cl - cotransporters through direct phosphorylation. 9, 10 Mutations in WNKs lead to Gordon's syndrome, an autosomal dominant form of hypertension. 11 Full-length OSR1 contains an C-terminal extension, residues 291–527; within this sequence, the unique PF1 region, residues 291–344, is required for kinase activity, although the basis for this requirement is unknown. The structures of the OSR1 kinase domains reported by Lee et al.5 and Villa et al.6 used constructs that encompassed residues 1–295 and 1–303, respectively, and neither contains a complete PF1 region.

Both structural studies show that the OSR1 kinase domain forms a domain-swapped dimer (Fig. 1). The dimer interface is formed by exchange of the P+1 loop, located in the C-terminus of the activation segment, and the following helix αΕF (Fig. 2). The interface is stabilized by a salt bridge between Glu196 in helix αΕF of one monomer and Arg279, located between helices αl and αJ, in the other monomer, as well as van der Waals contacts between several hydrophobic residues from helix αΕF of one monomer and the hydrophobic pocket located between helices αG and αF of the partner monomer. The activation segment, which commonly is 20–30 residues in length and contains one or several phosphorylation sites, is a critical structural element for regulation of kinase activity. 12 Electron density is not clearly defined in either study for the phosphorylation site, Thr185, or the C-terminal part of the activation loop. The partially disordered activation segment suggests that the OSR1 kinase domain structures represent an inactive conformation, because a correctly positioned activation segment is required for ATP binding.



Figure 1. The kinase domain of oxidative stress responsive 1 (OSR1) forms a domain-swapped dimer. Each monomer binds one molecule of Mg-AMP-

mammals and is activated by osmotic stress.8 In the pathway, with-no-lysine kinases (WNKs) activate OSR1, which in turn activates the Na+/K+/2Cl⁻ cotransporters through direct phosphorylation.9, 10 Mutations in WNKs lead to Gordon's syndrome, an autosomal dominant form of hypertension.11 Full-length OSR1 contains an C-terminal extension, residues 291–527; within this sequence, the unique PF1 region, residues 291–344, is required for kinase activity, although the basis for this requirement is unknown. The structures of the OSR1

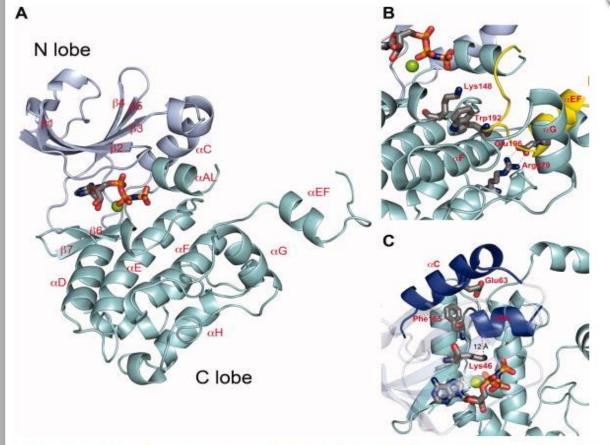


Figure 2. Structure of the OSR1 kinase domain monomer. (A) The first monomer from the coordinate file PDB 3DAK is shown. OSR1 has the typical bilobal kinase architecture consisting of a largely β -sheet N-terminal lobe (blue) and a helical C-terminal domain (aquamarine). (B) The domain-swapped interface between monomers contains a salt bridge between Arg279 of one monomer and Glu196 of the partner molecule, a cation- π interaction between Lys148 of one monomer and Trp192 of the partner molecule, and hydrophobic interactions between residues in α F and α G of one monomer with residues in α EF of the partner. (C) The structure shows characteristics of an inactive kinase, including absence of an ion pair between Lys46 and Glu63. An interactive view is available in the electronic version of the article, which also depicts superpositions with other kinase structures..Interactive View

ner interface is formed by Fig. 2). The interface is es al and aJ, in the other monomer and the hydrophobic ily is 20–30 residues in length tivity. 12 Electron density is not p. The partially disordered ecause a correctly positioned

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This outward movement has been observed in the structure of catalytically active cyclin-dependent kinase 2 (Cdk2) in complex with a phosphatase. 13 Solution NMR and kinetic data on dimeric p21-activated kinase 2 (PAK2) combined with the crystal structure of the highly homologous p21-activated kinase 1 (PAK1, PDB 1YHV) in its active state suggest that dimerization, which allows positioning of the active site of the partner melecular is assential for the transcruter hosphare phosphare in the active site of the partner melecular is assential for the transcruter hosphare phosphare in the active site of the partner melecular is assential for the transcruter hosphare phosphare in the active site of the partner melecular is assential for the transcruter has been observed in the structure of catalytically active cyclin-dependent kinase 2 (Cdk2) in complex with a phosphare product of the highly homologous p21-activated kinase 2 (PAK2) combined with the crystal structure of the highly homologous p21-activated kinase 3 (PAK1, PDB 1YHV) in its active state suggest that dimerization, which allows positioning of the

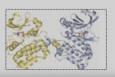
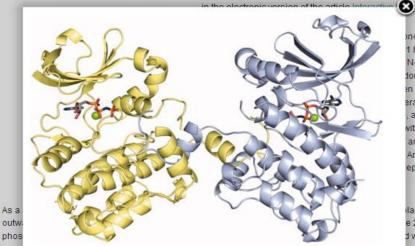


Figure 1. The kinase domain of oxidative stress responsive 1 (OSR1) forms a domain-swapped dimer. Each monomer binds one molecule of Mg-AMP-PNP. The two monomers from the coordinate file PDB 3DAK are depicted in blue and yellow, respectively; the AMP-PNP molecules are shown as CPK-colored bonds; and the Mg 2+ ions are shown as green spheres. An interactive view is available



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Lee et al. did not identify any key differences in the amino acid sequences between homologous domain-swapped and non-swapped kinases. Swapped domains often do not share similarity in sizes and sequences, based on the currently available high-resolution structures. 19 Owing to the relatively small differences in free energy of monomers and oligomers, many domain-swapped oligomers show low-affinity and are only observed in crystals. 20 Indeed, OSR1 kinase domain is monomeric in solution. 5, 6 In contrast, the previously identified domain-swapped kinase domains of SLK, LOK, and DAPK3 form stable dimers in solution. 4 Biochemical data for these kinases strongly support the involvement of dimers in the autophosphrylation reaction. However, the functional relevance of dimerization cannot be judged solely from the affinity in aqueous solution and coimmunoprecipitation experiments indicate that both the kinase domain alone and the full-length OSR1 are capable of forming oligomers in cells. 10

Intrinsic conformational plasticity is critical for structural transitions between active and inactive states of protein kinases, and biophysical evidence suggests that conformational dynamics may be coupled to catalysis.21-24 Results from NMR spectroscopy and molecular dynamics simulations have linked conformational flexibility to domain swapping in other protein molecules 25, 26 Therefore, domain swapping may be a consequence of the intrinsic conformational flexibility of kinase domains. The impact of domain swapping on the autophosphorylation reaction, if indeed they are coupled, remains to be fully elucidated, and additional structural and biochemical data are required to clarify the functional role

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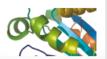
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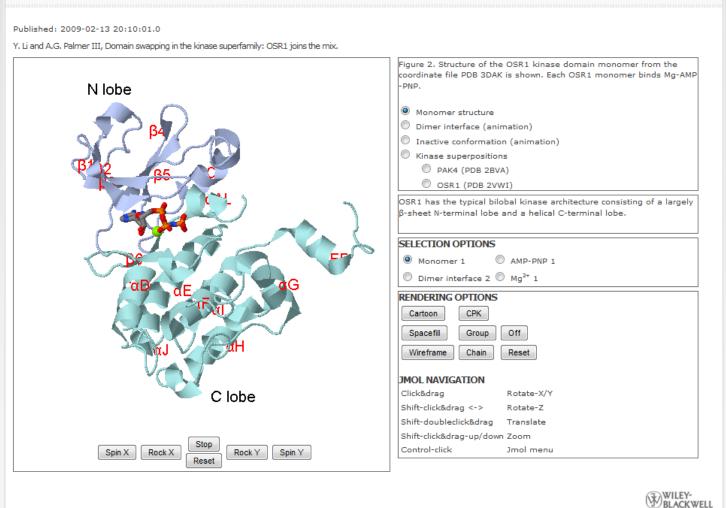




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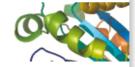
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Yu-Ran Na1,2, Chiwook Park1,2,1

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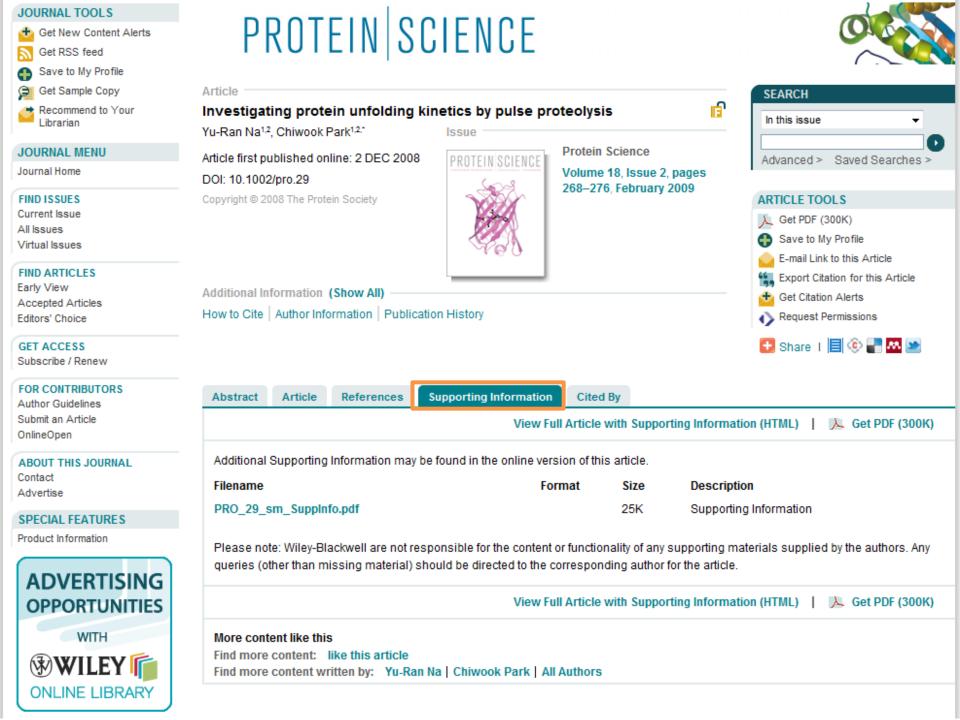
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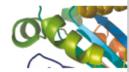
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