¹H, ¹⁵N, and ¹³C Resonance Assignments and Secondary Structure of the SWIRM Domain of Human BAF155, a Chromatin Remodeling Complex Component

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Mammalian SWI/SNF complexes are evolutionary conserved, ATP-dependent chromatin remodeling units. BAF155 in the SWI/SNF complex contains several highly conserved domains, including SANT, SWIRM, and leucine zipper domains. The biological roles of the SWIRM domain remain unclear; however, both structural and biochemical analyses of this domain have suggested that it could mediate protein-protein or protein-DNA interactions during the chromatin remodeling process. The human BAF155 SWIRM domain was cloned into the *Escherichia coli* expression vector pMAL-c2X and purified using affinity chromatography for structural analysis. We report the backbone ¹H, ¹⁵N, and ¹³C resonance assignments and secondary structure of this domain using nuclear magnetic resonance (NMR) spectroscopy and the TALOS+ program. The secondary structure consists of five α -helices that form a typical histone fold for DNA interactions. Our data suggest that the BAF155 SWIRM domain interacts with nucleosome DNA ($K_d = 0.47 \mu\text{M}$).

INTRODUCTION

The mammalian SWI/SNF complexes are evolutionary conserved, ATP-dependent chromatin remodeling components that use the energy of ATP hydrolysis to rearrange and remodel chromatin structure. These complexes play roles in chromatin remodeling and controlling diverse related processes, including transcriptional regulation, cell differentiation, cell death, and tumorigenesis (Clapier and Cairns, 2009; Kim et al., 2012; Roberts and Orkin, 2004). The mammalian SWI/SNF complexes consist of invariant core protein components and variable protein components, and the core components are BRG1 or hBRM, SNF5/INI1/BAF47, BAF155/SRG3, and BAF170.

BAF155 (SWI/SNF complex subunit SARCC1), a human homolog of yeast Swi3, is a core component of the SWI/SNF

complex. BAF155 is necessary for various cellular processes associated with transcriptional regulation, development, differentiation, and proliferation (Phelan et al., 1999). A previous report has shown that the expression level of SRG3 is down regulated after positive selection of developing thymocytes, a process that is critical in determining glucocorticoid sensitivity in T cells (Jeon et al., 1997). In addition, the T-cell receptor and Notch signaling inhibit glucocorticoid-induced apoptosis of developing thymocytes by down regulating expression of the murine homolog SRG3 (Choi et al., 2001; Jeon et al., 1997; Ko, 2004). However, the function of BAF155 as a component of the SWI/SNF complex remains clear. BAF155, which consists of 1,105 amino acids, has 33–47% sequence identity with Swi3 proteins (Sohn et al., 2007). BAF155 contains several highly conserved domains such as the SANT, SWIRM, and leucine zipper domains (Aasland et al., 1996). Each domain of the BAF155 homologs is important to the interaction with other components of the SWI/SNF complex. A recent report showed that the SWIRM domain of BAF155 directly interacts with another core component of the SWI/SNF complexes and plays a critical role in stabilization of the SWI/SNF complexes (Sohn et al., 2007).

The SWIRM domain is a globular module with 85–95 residues and was named after the proteins SWI3, RSC8, and MOIRA, in which the domain was first recognized by computational analysis of the eukaryotic genome (Crosby et al., 1999). The SWIRM domain is present in many different chromatin interactive or modifying proteins, including Swi3/SMARCC1/SMARCC2, Rsc8, histone demethylase1 (LSD1), and transcriptional adaptor ADA2 of the SAGA histone acetyl transferase complexes (Barlev et al., 2003; Forneris et al., 2005). The functional roles of the SWIRM domain remain unknown, but both structural and biochemical analyses of the SWIRM domain have suggested that the SWIRM domain could mediate protein-protein or protein-DNA interactions during the chromatin remodeling process. For example, the SWIRM domain of the

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ADA2a and SMARCC2 binds to double-stranded nucleosomal DNA, and the Swi3 SWIRM domain interacts with DNA. The SWIRM domain of LSD1 does not interact with DNA; however, it could mediate protein-protein interactions (Yoneyama et al., 2007). Here, we report resonance assignments and secondary structures of the BAF155 SWIRM domain using nuclear magnetic resonance (NMR) spectroscopy (Lee et al., 2010). We have found that the BAF155 SWIRM domain interacts with nucleosomal DNA and the important residues for the protein-DNA interaction have been identified from structural and biochemical data.

MATERIALS AND METHODS

Cloning, expression, and purification of the BAF155 SWIRM domain

cDNA fragments encoding the SWIRM domain of human BAF155 (amino acid sequence from Ile446 to Met543) were amplified by polymerase chain reaction (PCR). Amplified cDNA fragments were subcloned into the modified expression vector pMAL-c2X (Novagen Inc.) harboring an N-terminal hexa-histidine [(His)₆] affinity tag, maltose binding protein (MBP), and Tobacco Etch Virus (TEV) protease cleavage sites, which were transformed into *Escherichia coli* (Strain BL21 DE3). BAF155 SWIRM domain-expressing cells were grown in M9 minimal media at 37°C until an optical density value of 0.6 was reached, at which time 1.0 mM concentration of IPTG was added for induction, and the cells were further cultured for 12 h at 25°C (Jeong et al., 2013). Cells were harvested by centrifugation and stored frozen at -70°C. Uniformly ¹⁵N- and ¹³C/¹⁵N-labeled proteins were incubated in minimal medium with ¹⁵NH₄Cl, U-¹³C₆-glucose or both as the sole nitrogen and carbon sources in M9 minimal media. Harvested cells were lysed by sonication in lysis buffer [25 mM sodium phosphate, 300 mM NaCl, 5 mM 2-mercaptoethanol, and protease inhibitor cocktail (Sigma), pH 6.8]. The (His)₆-tagged proteins were purified by immobilized metal affinity chromatography on a Ni-NTA column (Amersham Pharmacia) and cleaved using TEV protease for 15 h at 25°C. Further purification was performed by size-exclusion column chromatography using a HiLoad Superdex 75 column. Size-exclusion chromatography with four reference proteins [albumin (66 kDa), carbonic anhydrase (29 kDa), cytochrome C (12.4 kDa) and aprotinin (6.5 kDa)] was used to determine the molecular weight of the BAF155 SWIRM domain using the following equation: $\log Y = -1.2177.0 X + 3.2038$, where X = elution fraction. Purified protein was concentrated to 1.0 mM using an Amicon Ultra centrifugal filter device (Millipore). The buffer solution for gel filtration chromatography consisted of 10 mM HEPES, 100 mM NaCl, 10 mM dithiothreitol (DTT), and 0.01% NaN₃ at pH 6.8.

NMR spectroscopy and resonance assignments

All NMR experiments were recorded at 25°C on a Bruker DRX500 spectrometer equipped with a z-shielded gradient triple-resonance cryoprobe (Lee et al., 2010). ¹H chemical shifts were referenced to the methyl proton signals of 2,2-dimethyl-2-silapentane-5-sulfonic acid at ppm. Both ¹³C and ¹⁵N chemical shifts were calculated indirectly from the ¹H spectrometer frequency. Three-dimensional (3D) experiments using HNCACB, CBCA(CO)NH, HNCA, and HCCH-TOCSY for sequential resonance assignments have been performed on a uniformly ¹⁵N/¹³C-labeled protein (Lee et al., 2010). For distance constraints, ¹⁵N-edited 3D nuclear Overhauser effect spectroscopy (NOESY; τ_m = 150 ms) and ¹³C-edited 3D NOESY-heteronuclear single-

quantum correlation spectroscopy (HSQC; τ_m = 120 ms) experiments were performed. All NMR data were processed and analyzed using XWIN-NMR (Bruker Instruments, Karlsruhe, Germany), NMRpipe/NMRDraw (Bio-sym/Molecular simulation, Inc., USA) and the PINE-SPARKY program (Lee et al., 2009). Dihedral angle restraints were derived from ¹H_α, ¹³C_α, ¹³C_β, ¹³CO chemical shifts using the TALOS+ program (Shen et al., 2009).

NMR titration experiments

All NMR titration experiments were recorded at 25°C on a Bruker DRX500 spectrometer equipped with a z-shielded gradient triple-resonance cryoprobe. Titration experiments were performed by recording a series of two-dimensional (2D) [¹H-¹⁵N] HSQC using the ¹⁵N-labeled BAF155 SWIRM domain (0.2 mM) in the presence of different concentrations of DNA ranging from 0 to 0.2 mM. Protein and DNA solutions were prepared in 10 mM HEPES buffer at pH 6.8 containing 100 mM NaCl, 10 mM DTT, and 0.01% NaN₃. Double-stranded DNA of 5'-AAATTGGCAACTGCCAAA-3' was used for NMR titration (Ko et al., 2010). All NMR data were processed using NMRpipe/NMRDraw software (Delaglio et al., 1995; Wuthrich, 1990). To analyze chemical shift changes upon DNA binding, chemical shift differences were calculated using the following equation: $\Delta\delta_{ppm} = \{(\Delta\delta H^N)^2 + (\Delta\delta N^H)^2\}^{1/2}$.

Fluorescence experiments

Binding constants between nucleosomal linker DNA and the BAF155 SWIRM domain were measured on a model RF-5301PC spectro-fluorophotometer (Shimadzu, Japan). BAF155 SWIRM protein in 10 mM HEPES, 100 mM NaCl, 10 mM DTT, and 0.01% NaN₃ (pH 6.8) was titrated to a molar ratio of 1:1 (SWIRM:DNA) using a 2 ml thermostat cuvette. Samples were excited at 290 nm, and emission spectra were recorded for light scattering effects from 280 to 450 nm (Nissler et al., 2004). We calculated the K_d value using the equation $\log (F_0 - F / F) = \log (1 / K_d) + n \log [\text{ligand}]$, where F_0 and F represent fluorescence intensity of the protein at 335 nm in the absence and presence of DNA, respectively. The number n represents the ligand binding site of the protein (Barik et al., 2005).

RESULTS AND DISCUSSION

Expression and purification of BAF155 SWIRM

Biologically functional domains of BAF155 consist of the Chromo domain, SWIRM domain, SANT domain, Glu-rich region, and Pro-rich region (Fig. 1A). The Chromo domain binds to methylated histones. The SANT domain is a novel motif found in several eukaryotic transcriptional regulatory proteins that was identified based on its homology to the DNA binding domain of c-myc. The SWIRM domain of BAF155 corresponding residues from 447 to 543 was successfully cloned into the pMAL-c2x expression vector using the TEV protease enzyme site (ENLYFQG; Fig. 1B). The purified protein has been characterized at a purity of > 95% by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (Fig. 2A). Data from size-exclusion chromatography show that the molecular weight of the BAF155 SWIRM domain was determined to be a monomer (~11.4 kDa) in solution (Fig. 2B). The final concentration of the NMR sample was about 1 mM in 90% H₂O/10% D₂O solution at pH 6.8. A one-dimensional ¹H NMR spectrum of BAF155 SWIRM was used to check the sample condition. A number of high-field, shifted, methyl proton resonances from 0.5 to 1.0 ppm and dispersion of the amide proton resonances (7.5-9.5 ppm) strongly suggest that the BAF155 SWIRM domain has a stable folded structure

Fig. 1. Domain structure and vector map of the BAF155 SWIRM domain. (A) BAF155 consists of a Chromo domain, SWIRM domain, SANT domain, Glu-rich region, and Pro-rich region. (B) Schematic diagram of the recombinant plasmid construct of the BAF155 SWIRM domain. The vector consists of an MBP tag, a (His)₆ tag, a TEV cleavage site (ENLYFQG), and a BAF155 SWIRM domain.

Fig. 2. Purification profile of the BAF155 SWIRM domain. (A) Sodium dodecyl sulfate-polyacrylamide gel electrophoresis shows the protein purity and expression yield of the BAF155 SWIRM domain. The molecular weight of the BAF155 SWIRM domain was measured using a protein size marker. Lanes 1 and 2 are the whole cell lysate before and after induction, respectively. Lanes 3 and 4 are the supernatant and pellet of the cell lysate, respectively. Lanes 5 to 7 are fractions from Ni-NTA column elution [flow through, washing, fusion protein (55.4 kDa)]. Lane 8 shows the protein band after TEV cleavage (each band is the MBP tag(*1), TEV(*2), target protein). Lane 9 is a final target protein (11.4 kDa) after reverse column work. (B) The elution profile of the size-exclusion chromatography (Superdex 75). Standard marker proteins were loaded and analyzed using origin 7.01 (origin Lab.). BAF155 SWIRM was eluted from 84 to 96 ml. The highest elution fraction (89.9 ml) indicated the molecular weight of the BAF155 SWIRM domain. The molecular weight of BAF155 SWIRM was calculated using the following equation: $\log Y = -1.2177 X + 6.263$ ($R^2 = 0.9942$, X = elution fraction).

under our experimental condition.

NMR resonance assignments

The 2D [^1H - ^{15}N]-HSQC spectrum demonstrated that most of the backbone NH resonances are well dispersed over the spectrum (Fig. 3A); however, many resonances were found in the narrow range from 7 to 9 ppm in the ^1H dimension, implying that the BAF155 SWIRM domain is mostly α -helical in structure. The sequential resonance assignments of the BAF155 SWIRM domain were performed using a combination of HNCACB,

CBCA(CO)NH, and HNCA data. Side chain assignments were made from 3D HCCH-TOCSY, ^{15}N -edited NOESY, and ^{13}C -edited NOESY data. Sequential assignments were completed, as shown in the HNCACB strip plot in helix 3 from residue E482 to T494 of the BAF155 SWIRM domain (Fig. 3B). The ^{15}N -edited NOESY spectrum was used to confirm all sequential resonance assignments. A summary of the backbone assignments was deposited in the Biological Magnetic Resonance Bank (<http://www.bmrb.wisc.edu>) under the accession number 19204.

Fig. 3. Backbone and side-chain assignments of the BAF155 SWIRM domain. (A) ^1H - ^{15}N heteronuclear single quantum correlation (HSQC) spectrum of uniformly $^{13}\text{C}/^{15}\text{N}$ labeled BAF155 SWIRM domain. Each peak corresponds to an amide N and H of an amino acid residue. The spectrum was collected at pH 6.8 and 298 K on a Bruker DRX 500 MHz NMR spectrometer. (B) The HNCACB strip plot shows an example of the sequential assignment from E482 to T494. Each strip plot corresponds to the ^{15}N plane of the indicated residues. Each strip shows resonance for the C_α and the C_β of each $i, i+1$ correlation to intrasidues ^{15}N and NH chemical shifts for helix 3 (residues 482-494) of the BAF155 SWIRM domain. (C) Strip plots taken from 3D ^{15}N -correlated NOESY collected on a Bruker 500 MHz. Each strip is positioned at the amide ^{15}N frequency in ω_1 , and proton frequencies in ω_2 and ω_3 of the residue indicated at the top by one-letter amino acid code and the sequence number. The strip plot shows sequential NOE connectivity as an arrow in a solid box, and $d_{\alpha\text{N}}$ ($i, i+3$) connectivity as a dotted box with a dotted line in the helix 3 region.

Secondary structures of BAF155 SWIRM

Data from the 3D ^{15}N -correlated $[^1\text{H}-^1\text{H}]$ -NOESY spectrum (Fig. 3C) shows NOEs of d_{NN} , $d_{\alpha\text{N}}$ together with sequential NOE connectivity for α -helical regions. Support for the prevalence of the helical structures was further obtained from the observation of strong $d_{\alpha\text{N}}$ ($i, i+3$) NOEs (Bartels and Wüthrich, 1994). A number of NOEs such as $d_{\alpha\text{N}}$ ($i, i+3$), $d_{\alpha\text{N}}$ ($i, i+4$) were observed to support that the BAF155 SWIRM domain consists of five α -helices (Val462-Ala467, Pro468-Phe472, Pro481-Leu497, Ser505-Asn511, and Val516-Trp527). The secondary structures of the BAF155 SWIRM domain were confirmed by TALOS+ data based on $\Delta\text{C}_\alpha - \Delta\text{C}_\beta$ Chemical shift values. Figure 4 shows the dihedral Ψ and ϕ angles together with the secondary structures. The BAF155 SWIRM domain consists of five α -helices and loops: α_1 (Val462-Ala467), α_2 (Pro468-Phe472), α_3 (Pro481-Leu497), α_4 (Ser505-Asn511), and α_5 (Val516-Trp527) (Fig. 4). Helices 1, 2, and 4 are relatively short, whereas helices 3 and 5 are long with a length between five and four turns. The N-terminal (Ile 447-His461) and C-terminal (Gly527-Met543) regions have long flexible tails. The three loops connecting helices are L1 (Asn473-Thr480), L2 (Asn498-Thr504), and L3 (Leu512-Asp515). NMR data revealed that the BAF155 SWIRM domain consists of five helices, which form a typical histone fold and HTH motif for protein-DNA interactions (Fig. 4).

Comparison of the BAF155 SWIRM domain with SWIRM family proteins

The SWIRM domain is evolutionally conserved in several chromatin remodeling complexes, and it is divided into three subfamilies: Swi3 of ATP-dependent chromatin remodeling complexes, LSD1 of histone demethylase, and the ADA2 transcriptional adaptor (Yoneyama et al., 2007) (Fig. 5A). Multiple sequence alignment data based on secondary structure have suggested that the BAF155 SWIRM domain belongs to the Swi3 subtype among SWIRM family proteins. The SWIRM domain of BAF155 has 36-37% of sequence homology with those of other SWIRM families (Swi3, LSD1, Ada2) (Fig. 5B), and all SWIRM domains have a helical structure. It has been suggested that the BAF155 SWIRM domain might interact with DNA sequences, such as TATA box DNA and nucleosomal linker DNA (Da et al., 2006; Gui and Dean, 2003; Qian et al., 2005). Here, we showed that the BAF155 SWIRM domain interacts with nucleosomal linker DNA. Because the precise roles of SWIRM domains are still undefined, our report will provide a functional clue regarding the BAF155 SWIRM domain during the chromatin remodeling process (Jung et al., 2012; Reisman et al., 2009; Wu et al., 2009).

DNA binding properties of the BAF155 SWIRM domain

We have shown that the double-stranded DNA of the nucleo-

Fig. 4. TALOS and chemical shift index (CSI) values for secondary structure analysis. Secondary structures of the BAF155 SWIRM domain were predicted from CSI values. PHI (Φ) and PSI (Ψ) values indicate backbone dihedral angles. Backbone dihedral angles (phi, psi) of the BAF155 SWIRM domain were calculated using TALOS+. Secondary structure (SS) values suggest α -helices from TALOS+. Predicted secondary structures of the BAF155 SWIRM domain are drawn on the bottom with the sequence.

Fig. 5. Sequence comparison between the BAF155 SWIRM domain and SWIRM family proteins. (A) Domain structure of SWIRM family proteins (BAF155, SWI3, Ada2a, and LSD1). Blue and red colors indicate the SWIRM and SANT domains, respectively. (B) Multiple sequence alignment of SWIRM domains of BAF155, SWI3, LSD1, and Ada2a. Sequence alignment was performed using T-coffee software and further rendered by Esprit software.

Fig. 6. Molecular interaction between the BAF155 SWIRM domain and DNA. (A) Superimposition of 2D ^1H - ^{15}N HSQC spectra of free protein (red) and protein/DNA complex (cyan). The molar ratio of protein to DNA is 1:1. (B) Backbone amide chemical shift perturbations of the BAF155 SWIRM domain upon DNA binding. The average chemical shift changes were calculated using the following formula: $\Delta\delta_{\text{total}} = \{(\Delta\delta^{\text{H}})^2 + (\Delta\delta^{\text{N}})^2\}^{1/2}$. (C) Fluorescence spectra with a fitting plot of BAF155 SWIRM in the presence of difference mixing ratios of double-stranded DNA [1:0 (black), 1:0.2 (blue), 1:0.4 (red), 1:0.6 (cyan), 1:0.8 (orange), 1:1 (green)] at pH 6.8.

somal linker region binds to the BAF155 SWIRM domain (Da et al., 2006; Qian et al., 2005; Yoneyama et al., 2007). The DNA binding mode of the BAF155 SWIRM domain has been confirmed by NMR titration experiments (Fig. 6A). The DNA binding site of the BAF155 SWIRM domain was identified by collecting ^1H - ^{15}N HSQC spectra upon nucleosomal linker DNA binding. Residues that were perturbed upon DNA binding were detected in $\alpha2$ and $\alpha5$, suggesting that DNA interacts with residues F471 and F472 in the $\alpha2$ and V516, C517, M520, R521, A524, and W529 in $\alpha5$ (Fig. 6B). The binding affinity K_d between the BAF155 SWIRM domain and DNA was determined to be 4.67×10^{-7} M using fluorescence-quenching experiments (Fig. 6C). Taken together, these results propose that the BAF155 SWIRM domain binds to nucleosomal DNA, and this domain might play a functional role as a remodeler during the chromatin remodeling process. Recent report suggested that the SWIRM domain might interact with other core proteins in chromatin remodeling complexes (Qian et al., 2005), and the interaction study is currently in progress.

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REFERENCES

- Aasland, R., Stewart, A.F., and Gibson, T. (1996). The SANT domain: a putative DNA-binding domain in the SWI-SNF and ADA complexes, the transcriptional co-repressor N-CoR and TFIIIB. *Trends Biochem. Sci.* 21, 87-88.
- Barik, A., Mishra, B., Shen, L., Mohan, H., Kadam, R.M., Dutta, S., Zhang, H.Y., and Priyadarsini, K.I. (2005). Evaluation of a new copper(II)-curcumin complex as superoxide dismutase mimic and its free radical reactions. *Free Radic. Biol. Med.* 39, 811-822.
- Barlev, N.A., Emelyanov, A.V., Castagnino, P., Zegerman, P., Bannister, A.J., Sepulveda, M.A., Robert, F., Tora, L., Kouzarides, T., Birshtein, B.K., et al. (2003). A novel human Ada2 homologue functions with Gcn5 or Brg1 to coactivate transcription. *Mol. Cell. Biol.* 23, 6944-6957.
- Choi, Y.I., Jeon, S.H., Jang, J., Han, S., Kim, J.K., Chung, H., Lee, H.W., Chung, H.Y., Park, S.D., and Seong, R.H. (2001). Notch1 confers a resistance to glucocorticoid-induced apoptosis on developing thymocytes by down-regulating SRG3 expression. *Proc. Natl. Acad. Sci. USA* 98, 10267-10272.
- Clapier, C.R., and Cairns, B.R. (2009). The biology of chromatin

- remodeling complexes. *Annu. Rev. Biochem.* 78, 273-304.
- Crosby, M.A., Miller, C., Alon, T., Watson, K.L., Verrizer, C.P., Goldman-Levi, R., and Zak, N.B. (1999). The trithorax group gene *moira* encodes a brahma-associated putative chromatin-remodeling factor in *Drosophila melanogaster*. *Mol. Cell. Biol.* 19, 1159-1170.
- Da, G., Lenkart, J., Zhao, K., Shiekhatair, R., Cairns, B.R., and Marmorstein, R. (2006). Structure and function of the SWIRM domain, a conserved protein module found in chromatin regulatory complexes. *Proc. Natl. Acad. Sci. USA* 103, 2057-2062.
- Delaglio, F., Grzesiek, S., Vuister, G.W., Zhu, G., Pfeifer, J., and Bax, A. (1995). NMRPipe: a multidimensional spectral processing system based on UNIX pipes. *J. Biomol. NMR.* 6, 277-293.
- Fornieri, F., Binda, C., Vanoni, M., Mattevi, A., and Battaglioli, E. (2005). Histone demethylation catalysed by LSD1 is a flavin-dependent oxidative process. *FEBS Lett.* 579, 2203-2207.
- Gui, C.Y., and Dean, A. (2003). A major role for the TATA box in recruitment of chromatin modifying complexes to a globin gene promoter. *Proc. Natl. Acad. Sci. USA* 100, 7009-7014.
- Jeon, S.H., Kang, M.G., Kim, Y.H., Jin, Y.H., Lee, C.J., Chung, H.Y., Kwon, H., Park, S.D., and Seong, R.H. (1997). A new mouse gene, SRG3, related to the SWI3 of *Saccharomyces cerevisiae*, is required for apoptosis induced by glucocorticoids in a thymoma cell line. *J. Exp. Med.* 185, 1827-1836.
- Jeong, K.W., Ko, H., Lee, S.A., Hong, E., Ko, S., Cho, H.S., Lee, W., and Kim, Y. (2013). Backbone dynamics of an atypical orphan response regulator protein, *Helicobacter pylori* 1043. *Mol. Cells* 35, 158-165.
- Jung, I., Sohn, D.H., Choi, J., Kim, J.M., Jeon, S., Seol, J.H., and Seong, R.H. (2012). SRG3/mBAF155 stabilizes the SWI/SNF-like BAF complex by blocking CHFR mediated ubiquitination and degradation of its major components. *Biochem. Biophys. Res. Commun.* 418, 512-517.
- Kim, B., Kang, S., and Kim, S.J. (2012). Differential promoter methylation and histone modification contribute to the brain specific expression of the mouse Mbu-1 gene. *Mol. Cells* 34, 433-437.
- Ko, M. (2004). T cell receptor signaling inhibits glucocorticoid-induced apoptosis by repressing the SRG3 expression via Ras activation. *J. Biol. Chem.* 279, 21903-21915.
- Ko, S., Kang, G.B., Song, S.M., Lee, J.G., Shin, D.Y., Yun, J.H., Sheng, Y., Cheong, C., Jeon, Y.H., Jung, Y.K., et al. (2010). Structural basis of E2-25K/UBB+1 interaction leading to proteasome inhibition and neurotoxicity. *J. Biol. Chem.* 285, 36070-36080.
- Lee, W., Westler, W.M., Bahrami, A., Eghbalnia, H.R., and Markley, J.L. (2009). PINE-SPARKY: graphical interface for evaluating automated probabilistic peak assignments in protein NMR spectroscopy. *Bioinformatics* 25, 2085-2087.
- Lee, C.J., Yun, J.H., Lim, S.K., and Lee, W. (2010). Solution structures and molecular interactions of selective melanocortin receptor antagonists. *Mol. Cells* 30, 551-556.
- Nissler, L., Gebhardt, R., and Berger, S. (2004). Flavonoid binding to a multi-drug-resistance transporter protein: an STD-NMR study. *Anal. Bioanal. Chem.* 379, 1045-1049.
- Phelan, M.L., Sif, S., Narlikar, G.J., and Kingston, R.E. (1999). Reconstitution of a core chromatin remodeling complex from SWI/SNF subunits. *Mol. Cell* 3, 247-253.
- Qian, C., Zhang, Q., Li, S., Zeng, L., Walsh, M.J., and Zhou, M.M. (2005). Structure and chromosomal DNA binding of the SWIRM domain. *Nat. Struct. Mol. Biol.* 12, 1078-1085.
- Reisman, D., Glaros, S., and Thompson, E.A. (2009). The SWI/SNF complex and cancer. *Oncogene* 28, 1653-1668.
- Roberts, C.W., and Orkin, S.H. (2004). The SWI/SNF complex--chromatin and cancer. *Nat. Rev. Cancer* 4, 133-142.
- Shen, Y., Delaglio, F., Comilescu, G., and Bax, A. (2009). TALOS+: a hybrid method for predicting protein backbone torsion angles from NMR chemical shifts. *J. Biomol. NMR.* 44, 213-223.
- Sohn, D.H., Lee, K.Y., Lee, C., Oh, J., Chung, H., Jeon, S.H., and Seong, R.H. (2007). SRG3 interacts directly with the major components of the SWI/SNF chromatin remodeling complex and protects them from proteasomal degradation. *J. Biol. Chem.* 282, 10614-10624.
- Wu, J.I., Lessard, J., and Crabtree, G.R. (2009). Understanding the words of chromatin regulation. *Cell* 136, 200-206.
- Wuthrich, K. (1990). Protein structure determination in solution by NMR spectroscopy. *J. Biol. Chem.* 265, 22059-22062.
- Yoneyama, M., Tochio, N., Umehara, T., Koshiba, S., Inoue, M., Yabuki, T., Aoki, M., Seki, E., Matsuda, T., Watanabe, S., et al. (2007). Structural and functional differences of SWIRM domain subtypes. *J. Mol. Biol.* 369, 222-238.