Figure S1. Unique RRM3 features are conserved between *Saccharomyces cerevisiae* Hrb1/Gbp2 and *Aspergillus nidulans* snxA. Sequences were aligned using MUSCLE (https://www.ebi.ac.uk/Tools/msa/muscle/). Shown are the C-termini of snxA, Hrb1, and Gbp2. Green, RNA Recognition Motif 3; non-bolded red, conserved G – G – E/D – K/R motif that in budding yeast participates in forming a 'closed loop' to block RNA binding at RRM3; red and bolded with gray highlight, conserved VD or AD dipeptide. In Hrb1 the conserved Asp427 forms a stabilizing H-bond with Arg374. Yellow highlight, conserved YxYGG motif essential for association of GBP2 with THO2.
Figure S2. AN6228-gyfA deletion and reconstructed tIL:tIIR translocation allele. Maps of each construct showing strategy for diagnosis by PCR and Southern blot is shown on the left. PCR diagnosis is shown at right. PCR primers lying outside of the linear DNA replacement constructs were used in molecular diagnosis. A. AN6228 was deleted from TN02A25 (pyrG89 paba1; argB2; ΔnkUA::argB; riboB2) by one-step gene replacement with A. fumigatus pyrG. The primers should amplify an 8993 nt product from the parent AN6228+ strain, and a 6162 nt amplicon from the deleted locus. B. AN6228+ in TN02A25 was replaced by a 7489 nt linear amplicon harboring the tIL:tIIR gyfA fragment, containing the truncated gyfA gene (exons 1-3 and part of exon 4, gyfAΔ1061-1506) fused with 210 nt of snxA intron 1 (ChrI nt 841,834 fused with ChrII nt 3,223,165), and followed by the Aspergillus fumigatus riboB selectable marker for transformation. This gyfA truncation allele can express 1060 out of 1506 aa from AN6228, including the GYF domain in exon 4, plus an additional 17 aa encoded by intron I of snxA prior to the first in-frame stop codon after the chromosome I-II fusion, for a total of 1077 aa. Diagnostic PCR should amplify a 5984 nt product from the parent AN6228+ locus, and a 6504 nt amplicon from the truncated allele. C. Southern blot verification of the reconstructed AN6228 truncation allele. Genomic DNAs from two wild-type strains (SWJ 400 and SWJ 2973) and 16 riboB+ transformants (1-6, -9,-11, 13-20) were digested with EcoRV and subjected to chemilluminescent Southern blotting using a 2175 nt digoxigenin-labeled DNA probe (pink box in B). Transformants #1, 2, 13, and 17 were saved and used in subsequent experiments. They were named, respectively, tMLB 6742 (#1), tMLB 6743 (#2), tMLB 6744 (#13), and tMLB 6745 (#17).

A and B Key to lanes: λ, Lambda HindIII DNA size markers; Par, TN02A25 host strain; Δ, deletion strains; tr, strains harboring a replacement of AN6228 locus with the reconstructed AN6228 translocation.
**Figure S3.** C-terminal tagging of AN6228-*gyfA*+ with GFP. GFP-tagged *gyfA* constructs replaced the wild-type *gyfA*+ locus in tSWJ 4286 and tSWJ 4287. **A. Gene modification strategy.** Black brackets indicate the amplicons that were used to verify correct integration of the tags. **B. Trans-locus PCR** of the parent *gyfA*+ host and three *pyrG*+ transformants. **Key to lanes:** λ, Lambda HindIII DNA size markers; Par, tSWJ 4286; G, GFP-tagged transformants, G1 (tMLB 6665) and G14 (tMLB 6666).
Figure S4. AN8825-setB deletion. setB (AN8825) was deleted from SWJ 5586 (ya2; snxA2; argB2; pyroA4 ΔnkuA::argB; nicA2; riboB2) by one-step gene replacement with A. fumigatus riboB. **A. Deletion strategy.** Arrows indicate the positions of PCR primers that lie outside of the linear DNA used to delete setB. **B. Trans-locus PCR** of the parent setB+ strain and two ΔsetB::riboB transformants.

Key to lanes: λ, Lambda HindIII DNA size markers; Par, SWJ 5586, setB+ parent; Δ1 and Δ2, ΔsetB::riboB transformants.
Figure S5. **setB-G255V knock-in mutation.** To verify that the G255V mutation detected by whole-genome sequencing was causative for suppression of *snxA2*, the mutation was knocked into strains carrying *setB+ snxA+* (tSWJ 5347, tSWJ 5590), and *setB+ snxA2* (SWJ 5586), as described in the methods. **A. Gene replacement strategy.** Arrows indicate the positions of PCR primers that lie outside of the 7555 nt linear DNA used to knock in the mutation. Black shading, exons; gray shading, 5' UTR. **B. Trans-locus PCR** of two representative transformants. Sanger sequencing confirmed the knock-in mutations in each of two selected transformants from each host strain. 

**Key to lanes:** λ, Lambda HindIII DNA size markers; Par, SWJ 598 *setB*+ parent; Ki-1, Ki-2 *setB-G255V* knock-in transformants. Ki-1 host: tSWJ 5347; Ki-2 host: SWJ 5586.
Figure S6. **AN7033-Histone H3-K36L knock-in mutation.** To create an allele of Histone H3 that is non-methylatable on lysine-36, the mutation was knocked into strains carrying setB+ snxA+ (tSWJ 5347, tSWJ 5590) as described in the methods. **A. Gene replacement strategy.** Arrows indicate the positions of PCR primers that lie outside of the 7.607 kb linear DNA used to knock in the mutation. Black shading, exons; gray shading, 5' and 3' UTR. **B. Trans-locus PCR** of two representative transformants. Sanger sequencing confirmed the knock-in mutations in each of two selected transformants from each host strain. **Key to lanes:** λ, Lambda HindIII DNA size markers; Par, SWJ 598; Ki-1, Ki-2 Histone H3-K36L knock-in transformants. Ki-1 host: tSWJ 5347; Ki-2 host: SWJ 5590.
Figure S7. AN4865-nsrA deletion. nsrA (AN4865) was deleted from strains carrying nsrA+ snxA+ (SWJ 4285) and nsrA+ snxA2 (tSWJ 5978) by one-step gene replacement with A. fumigatus pyrG. A. Deletion strategy. Arrows indicate the positions of PCR primers that lie outside of the linear DNA used to delete nsrA. B. Trans-locus PCR of the parent snxA+ and snxA2 strains (Par) and 7 ΔnsrA::pyrG transformants. λ, Lambda HindIII DNA size markers.
Figure S8. Phenotypes of snxA2 suppressors. Two independently generated Class 1 and Class 2 suppressors were outcrossed to a wild-type strain, and F1 progeny containing snxA2 and each suppressor were recovered and tested for suppression of snxA2 cold-sensitivity at the restrictive temperature of 20°. Fresh conidia were toothpicked onto minimal media and grown for 3 days at 37° and for 11 days at 20°.

Strains: Wild-type, SWJ 424; snxA2, SWJ 5586; snxA2-sup26, SWJ 6012; snxA2-sup59, SWJ 6015; snxA2-sup22, SWJ 6010; snxA2-sup49, SWJ 6014.
Figure S9. Transcript-specific qPCR strategy and candidate transcription startsites (TSS) for the 9-exon snxA mRNA. The 5’ end of the wild-type snxA gene is shown, including 169 nt exon 1, 766 nt intron 1, 23 nt exon 2, 547 nt intron 2, and the alternative start codons for the 11-exon and 9-exon proteins. The differing N-terminal amino acid sequences are shown at the bottom of the figure. Gray box, 5’ UTR of the 11-exon transcript; light blue box, 42 nt sequence encoding the first 14 amino acids of the 9-exon allele, located in the 547 nt second intron of the 11-exon transcript. This 42 nt codegenic sequence lies upstream of and includes the 3’ splice junction of intron 2 of the 11-exon allele, and continues in-frame with exon 3. The red horizontal arrows indicate the transcript-specific 11-exon qPCR primer pair (c11F + c9-11R); blue arrow, 9-exon transcript-specific qPCR forward primer paired with c9-11R; black arrows, primer pair to detect total snxA mRNA. The red ‘x’ indicates the location of the snxA1/A2 translocation breakpoint in intron 1. Dark blue boxes mark the positions of candidate transcription startsites (TSSs) mapped by Sibthorp et al. (2013).
Cross: PAR REC Map distance (cM)

**tSWJ 6345 x tSWJ 6483:**

- Intervals:
  - pyrG – snxA: 185 7 3.6
  - snxA – trpA: 191 1 0.5
  - pyrG – trpA: 186 6 3.1

**SWJ 3403 x tSWJ 6507:**

- Intervals:
  - snxA – pyrG: 221 18 7.5
  - snxA – acuJ: 197 42 17.6
  - pyrG – acuJ: 210 29 12.1

**tSWJ 5978 x SWJ 6326:**

**tSWJ 5981 x SWJ 6325:**

- Intervals:
  - adA – snxA: 316 4 1.3
  - pyrG – adA: 310 10 3.1
  - pyrG – snxA: 306 14 4.4

  - pyrG – snxA total: 712 39 5.1

---

**Figure S10.** A reciprocal translocation detected via genetic linkage of Chromosome I markers with *snxA1/A2* cold-sensitivity. Cold-sensitivity associated with *snxA* mutants was previously mapped to AN3739 on Chromosome II, and a deletion of AN3739 (*snxA*)
phenocopied the cold-sensitivity of $snxA1/A2$ mutants (James et al., 2014). Subsequent discovery of strong linkage between $pyrG89$ on Chromosome I and cold-sensitivity suggested the possibility that $snxA1/A2$ mutants were caused by a ChrI – ChrII translocation. Further evidence was obtained from crosses using Chr I markers $adA55$ and $acuJ211$ and the ChrII marker $trpA69$, as shown above. **Abbreviations:** E, Exon; ATG$_1$, Start codon for 11-exon $snxA$ mRNA; ATG$_2$, Start codon for 9-exon $snxA$ mRNA; PAR, parental genotype; REC, recombinant genotype.
A

**MPD2_Spombe**

- **MNSEASVFSNM**---------------------**DWSQL**------------------------**AHSLHKKANN** 26
  
  *MPTLPSSFAASAAGNTQADSRDGTSSGFEWSTRMMNGATQTFRRPSVATNPSTHDRAT** 60

**AN6228_gyfA**

- **SSSTTGAFAQPLTSTKATAPNGVSTPT**------------------------54
  
  *SATPTGSAAGGASTHSTRGANGSD** 120

**MPD2_Spombe**

- **SSSHIDPSVS----AKLDSQRKLSSGKMNWD-------------------SL** 81
  
  *NH1ETPTANGAWGKRDHKDPFICPEVCDHGQFELPGVLVDMDKETTFSTSVNSPL* 180

**AN6228_gyfA**

- **AN6228_gyfA**
  
  *AN6228_gyfA** 360

**MPD2_Spombe**

- **SSSSSTTGAFPQLTSFTKPTATNGVDSPT**---------------------------------54
  
  *MPD2_Spombe** 137

**AN6228_gyfA**

- **SATTPTGSAGGAYST**
  
  **MPD2_Spombe** 120

**AN6228_gyfA**

- **HMSSTRNGASVDT**
  
  **MPD2_Spombe** 137

**AN6228_gyfA**

- **TRYSKEQFLDLYKARQRESQVLSKNVA**
  
  **MPD2_Spombe** 240

**AN6228_gyfA**

- **AN6228_gyfA**
  
  *AN6228_gyfA** 185

**MPD2_Spombe**

- **SSTNPVGLPGSTSPWPSASQNANFSHPHAGFANLGNTSSAAQPTTEKRPGFGSLRGS**
  
  **MPD2_Spombe** 360

**AN6228_gyfA**

- **AN6228_gyfA**
  
  **AN6228_gyfA** 360

**MPD2_Spombe**

- **PRSGSAALGGSQDVSTPSQVADQLGF**
  
  **MPD2_Spombe** 540

**AN6228_gyfA**

- **PRSGSAALGGSQDVSTPSQVADQLGF**
  
  **MPD2_Spombe** 540
MPD2_Spombe: RSDPFTGSPTSFAARPQNLFLNPNQVATMLOQRALQEQEQFDSTHGDTLDFQARRE 1020
AN6228_gyfA: RSDPFTGSPFAARPQNLFLNPNQVATMLOQRALQEQEQFDSTHGDTLDFQARRE 1020

AN6228_gyfA: RSDPFTGSPFAARPQNLFLNPNQVATMLOQRALQEQEQFDSTHGDTLDFQARRE 1020

MPD2_Spombe: SEETKQEKPSTKLKETVEKSLQGQSAPAASEKIPVTSGQSTXPSKLPFP 633
AN6228_gyfA: SEETKQEKPSTKLKETVEKSLQGQSAPAASEKIPVTSGQSTXPSKLPFP 633

MPD2_Spombe: PSLDETISREMSIASSEALPQVEKSNSDQPPVAIPSTSKTGPSWAK-VDVSTSMQAQEI 691
AN6228_gyfA: PSLDETISREMSIASSEALPQVEKSNSDQPPVAIPSTSKTGPSWAK-VDVSTSMQAQEI 691

MPD2_Spombe: SKLQQVVSMSGHS------PDFLAWCKSLKSFLKQGTEWNSAFKVVTSKKNKKRV 990
AN6228_gyfA: SKLQQVVSMSGHS------PDFLAWCKSLKSFLKQGTEWNSAFKVVTSKKNKKRV 990

B

GYF domain alignments:

AN6228 gyfA: RMRW YKDRPOQJNIQCPWTGLEMHDWFKAGFSPLDQIRKLED
Mpd2+ S. pombe: LLHLYKDPQNNVCOPPTGVDMHQWYRAGYFPLGLPIKLLLE
Smy2 S. cerevisiae: ESSWYIDTQGQIHPFTQMQMSQWYGFASTLQISRLG
GIGYF1 Hs: ARWKFYKDPQGEIQCPFPTQMAEWFQAGYFGSMLLVKRGCD
GIGYF2 Hs: MQKYKDPQGEIQCPFPTQMAEWFQAGYFGTMSLLVVRAD
CD2BP2 Hs: DVMVWKJENTGD-AELYGFPTSAQNOVSEGYPDGVYCRK
AN0328 (Lin1): RVMWYKREDGAGGYYCAGPGTNNESWKGACFYGEVF
Figure S11. Alignment of AN6228-gyfA with Schizosaccharomyces pombe Mpd2+.  
A. Sequences were aligned using MUSCLE. Aqua, GYF domain; gray, putative Cdk1-CyclinB kinase phosphorylation site on Mpd2+ (Swaffer et al., 2016); green, phosphosites mapped to gyfA: aa 399, 1130, 1132, 1158, 1160, 1317, and 1357 (Ramsubramaniam et al., 2014); yellow, potential other phosphosites (R-x-x-S-x-S and R-x-x-S/T-P); red, gyfA translocation breakpoint between Glu (E) and Asp (D) in snxA1/A2.  B. Alignments of GYF domains from Aspergillus nidulans, Schizosaccharomyces pombe, Saccharomyces cerevisiae, and human (Hs). Both A. nidulans GYF domain proteins are shown. Blue, canonical GYF residues conserved across all species and both subfamilies of GYF domain proteins. Yellow, invariant residues in the Smy2/Mpd2+/GIGYF/gyfA subfamily of GYF domain proteins that are not shared with the CD2BP2 GYF subfamily represented by CD2BP2 and AN0328/Lin1. Green, conserved Trp (W) residue in CD2BP2 subfamily that differs from invariant Asp (D) residue in Smy2/Mpd2+/GIGYF subfamily.
Figure S12. alcA::gyfA construct. A gyfA wild-type genomic clone was fused with the alcA alcohol dehydrogenase gene promoter. A wild-type gyfA allele was amplified from genomic DNA using forward and reverse primers that contained SnaBI and BglII restriction enzyme sites, respectively. This fragment was cloned into the Smal and BamHI sites of the plasmid pSDW194 (James et al. 2014), in front of the inducible-repressible alcA alcohol dehydrogenase gene promoter and followed by the selectable marker argB. The plasmid was transformed into gyfA+ (SWJ3629), and a strain carrying an argB integration was verified through Southern Blot. A.
Southern blot strategy for integration at the argB locus. B, BamHI. B. Southern blots were probed with a 3.3 kb argB fragment. Single-copy integration at the argB locus should replace the 9.4 kb wild-type BamHI band with two bands of 10.2 and 6.7 kb, respectively. In the Southern blot, Lane 1 contains a strain bearing a copy of alcA::gyfA integrated at argB, and containing one or two additional copies integrated ectopically. This alcA::gyfA transformant, designated tMLB 6801, was crossed with nimX1 and nimX2 and ΔgyfA strains to generate ΔgyfA alcA::gyfA nimX+ and ΔgyfA alcA::gyfA nimX1/X2 mutants. All double and triple mutants were verified through outcrosses to wild-type tester strains, and by complementation of ΔgyfA by overexpression of alcA::gyfA. C. gyfA+ overexpression partially rescues nimX1/X2 heat sensitivity. Cells were tested to determine if alcA-driven complementation of ΔgyfA would fully restore nimX1/X2 heat-sensitivity. Complementation of the ΔgyfA growth and suppressor phenotypes was assessed by comparing growth at 32° and 37° during alcA repression (rich media with 2% glucose) or alcA induction (minimal media containing 100 mM threonine). Strains were point-inoculated using conidia from fresh streaks. Glucose-containing plates were incubated for 3 days; threonine-containing plates were incubated for 4 days.
**Figure S13.** *setB* suppressor mutations are *snxA*-dependent. A *setB* deletion and *setB* point mutations can suppress the cold-sensitivity of hypomorphic *snxA*1/A2 mutations but cannot suppress the cold-sensitivity of a Δ*snxA* null mutation. Fresh conidiospores were point-inoculated on minimal media and grown for 3 days at 37° and 9 days at the restrictive temperature of 20°.

**Strains:** WT, SWJ 424; Δ*snxA*, tSWJ 4412; Δ*setB*, tSWJ 6407; Δ*setB* Δ*snxA*, tSWJ 6164; Δ*setB* snxA1, tSWJ 6247; Δ*setB* Δ*snxA*, tSWJ 6257; *setB*-G255V Δ*snxA*, tSWJ 6075; *setB*-G255W Δ*snxA*, tSWJ 6136.
A

Figure S14. The hypomorphic setB point-mutations setB-sup26-G255W and setB-sup59-G255V elevate snxA2 transcripts to wild-type level and snxA1 transcripts to 40-55% of wild-type level. Relative snxA mRNA expression was measured in randomly cycling vegetative mycelia using primers to detect the 9-exon snxA transcript in wild-type, snxA1/A2 mutants, and snxA1/A2/setB suppressor strains. A. snxA1-derived strains and suppressors. B. snxA2-derived strains and suppressors. Relative expression was normalized to actA using the ΔΔCt method using four replicates. Error bars, SEM. Strains: WT, PCS439; snxA1, SWJ 5594; snxA1 setB-sup26-G255W, SWJ 5995; snxA1 setB-sup59-G255V, SWJ 5998; snxA2, SWJ 5562; snxA2 setB-sup26-G255W, SWJ 6012; snxA2 setB-sup59-G255V, SWJ 6015.
Figure S15. The 9-exon transcript represents the majority of total snxA transcript in the setB histone H3K36 point mutants. Relative snxA mRNA expression was measured in randomly cycling vegetative mycelia using primers to detect total snxA mRNA or the alternative 11- and 9-exon snxA transcripts in snxA2, snxA2/nsrA39, snxA2/nsrA49, snxA2/nsrA39, snxA2/setB26, and snxA2/setB59 strains. Relative expression was normalized to actA using the ΔΔCt method using four replicates in each of two independent RNA preps. Levels of expression for each data set were normalized against snxA+ wild-type levels (strain PCS 439), which were set at an expression level of 1.0 (not shown). Error bars, SEM. Strains: snxA2, SWJ 6034; snxA2/nsrA39, SWJ 6013; snxA2/nsrA49, SWJ 6014; snxA2/setB26, SWJ 6012; snxA2/setB59, SWJ 6015.
Figure S16. \( \Delta \text{setB} \) is epistatic to \( \Delta \text{cclA} \) for rescue of \( \text{snxA1/A2} \) cold-sensitivity. Single-, double-, and triple-mutant strains derived from \( \text{snxA1} \) or \( \text{snxA2} \) with \( \Delta \text{setB} \) and/or \( \Delta \text{cclA} \) were tested for ability to rescue cold-sensitivity of the \( \text{snxA} \) mutants. Fresh conidiospores were point-inoculated on minimal media and grown for 3 days at 37\(^\circ\) and 7 days at the restrictive temperature of 22\(^\circ\). 

**Strains:** WT, SWJ 424; \( \text{snxA1} \), tSWJ 5594; \( \text{snxA2} \), tLC 5602; \( \Delta \text{setB} \), tSWJ 6250; \( \Delta \text{cclA} \), tSWJ 5617; \( \Delta \text{setB} \) \( \Delta \text{cclA} \), tSWJ 7116; \( \Delta \text{setB} \) \( \text{snxA1} \), tSWJ 6247; \( \Delta \text{setB} \) \( \text{snxA2} \), tSWJ 6164; \( \Delta \text{cclA} \) \( \text{snxA1} \), tSWJ 5635; \( \Delta \text{cclA} \) \( \text{snxA2} \), tSWJ 5637; \( \Delta \text{setB} \) \( \Delta \text{cclA} \) \( \text{snxA1} \), tSWJ 7115; \( \Delta \text{setB} \) \( \Delta \text{cclA} \) \( \text{snxA2} \), tSWJ 7114.
Begin 4865 Nsr1 5’UTR

735,041

rsrA_Anidulans    MSKTSSVKKGASAGKLADKALSKVDDAGVTQRQPEKAKSKQIAREIA
nsr1_Scerevisiae  MAKTTKVKNKKE---------------------------VKASKQAKEEK

nsrA_Anidulans    SKEKKPKSKKKEPTPSSSESESESESESESESESESESESESESESESESESESESESESEDKPKVKKEVKK
nsr1_Scerevisiae  AKAVSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSSS
**Figure S17.** Alignment of *A. nidulans nsrA* with *S. cerevisiae* ortholog *nsr1*. Sequences were aligned using MUSCLE (https://www.ebi.ac.uk/Tools/msa/muscle/). Shown is the *nsrA* 5’ UTR DNA sequence (chr III nt 735,041 to the START codon at 734,552) followed by protein alignment. Green highlight, RNA Recognition Motifs 1 and 2; Bold red, 8 RGG motifs in *A. nidulans* C-terminus; non-bolded red, 6 RGG motifs in *S. cerevisiae* C-terminus; gray highlight, *nsrA* 5’ UTR; yellow highlight, non-coding intron in 5’ UTR; aqua highlights, mutations in *nsrA* that suppress *snxA1/A2* cold-sensitivity; red highlight, N-terminal SEDE motif – candidate casein kinase II phosphorylation site.