Supplementary material

Supplementary figure 1:

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An-LxrA     1  MSR-----------------------SLEGKFAIITGGSRGIGEAIAHNL
An-XhrA     1  M-------------------------SLKGKVAIVTGGARGIGAGIVRSL
Tr-LXR4     1  MAR-----------------------PYEGKLAIVTGASRGIGAAVARRL
Tr-LXR3     1  MTQMKNGAFPHDNAAVPNVERVLPLFSLKGRTAIVSGAGAGIGLAVAQAF

An-LxrA    28  ASKGCSLLLNYTSDSSRTRTESLCNTLSTTHKITCIPVQADLSDPAPAVN
An-XhrA    26  SEQGAKVAFNYVSSSSRKAADALIESLRQN-NNEATAVQADITDPN-APK
Tr-LXR4    28  AAKGSNVLITFTSDSSRDLTRGLVEELSSKHGVHVQSVQTDLAKASTAAP
Tr-LXR3    51  AEAGANVAIWYNSNKQA--VT-SAEDIAKTYGVKCKAYQVNVTSAE-AVD

An-LxrA    78  TIISAAKTHFTSPT----TNTLTIDILINNAGVSKDR-FLNDPSSGPIDP
An-XhrA    74  MIIQAALEAFQT---------DRIDILVNNAGAGDNR-PLEEV---TMDS
Tr-LXR4    78  IIVEAARTLFDSYSPPSGGKKFQVDILINNAGVSSNQ-FLNDPEKGAIDE
Tr-LXR3    97  KAITEIIKEFN----------GRLDVFVANSGITWTEGAFID---GSVES

An-LxrA   123  AYFNWHYTINVLAPLLLTQACAEYLPRKPA-------------HSGRIIN
An-XhrA   111  --YMRLMDVNVRAVIFMTQAILPYIP-----------------RGGRIIN
Tr-LXR4   127  AEFTRVYAINVLAPLLLTQAVAPHLPAD---------------RSGRIVN
Tr-LXR3   134  --ARNVMSVNVDGVMWCAKSAGAHFRRQKEEGTTIDGKPLDNFIAGSFIA

An-LxrA   160  ISSISSSLGF--TGQSVYGGTKAALEAMTRTWARELADV--ATVNAVNPG
An-XhrA   142  LSSISSRGGY--ATQSVYAASKAAVEGLTRVWATELGHKYGVTVNAANPG
Tr-LXR4   162  VSSVSASIGY--LGQSVYAGSKGALEVMTRTWARELAER--ATVNSVNPG
Tr-LXR3   182  TASMSGSIVNVPQLQAVYNSSKAAVIHFCKSLAVEWTGF--ARVNTVSPG

An-LxrA   206  PVVGDMYFATGEEFWKQMQGFQDNTPLSKLVDGEEAVEELL---SEEQKR
An-XhrA   190  PVDTDMYQAAGEVHLKRMEEQNK---------------------------
Tr-LXR4   208  PAWGDMYAEAGPTFWRRNQPYVDAAPLMA-YDGEEDVLRRAGGEADKFDR
Tr-LXR3   230  YIITEISNFVPP----------------------------------ETKT

An-LxrA   253  LIREKMGGRRPAFTREIAGVVGMLCTEDGAWCTGSVVCANGGLK--FT
An-XhrA   213  ---KVPAGQRCGTVQDIGDIVSFLAEERSRWVTGDVICANGGML--YI
Tr-LXR4   257  LVREQMGGRRPGFADEIAGTVDMLCTEESGWTTGSVVCANGGMRMSIA
Tr-LXR3   246  LWKDKIVMGREGRVGELKGAYLYLASDASSYTTGLDMIVDGGYS--LP
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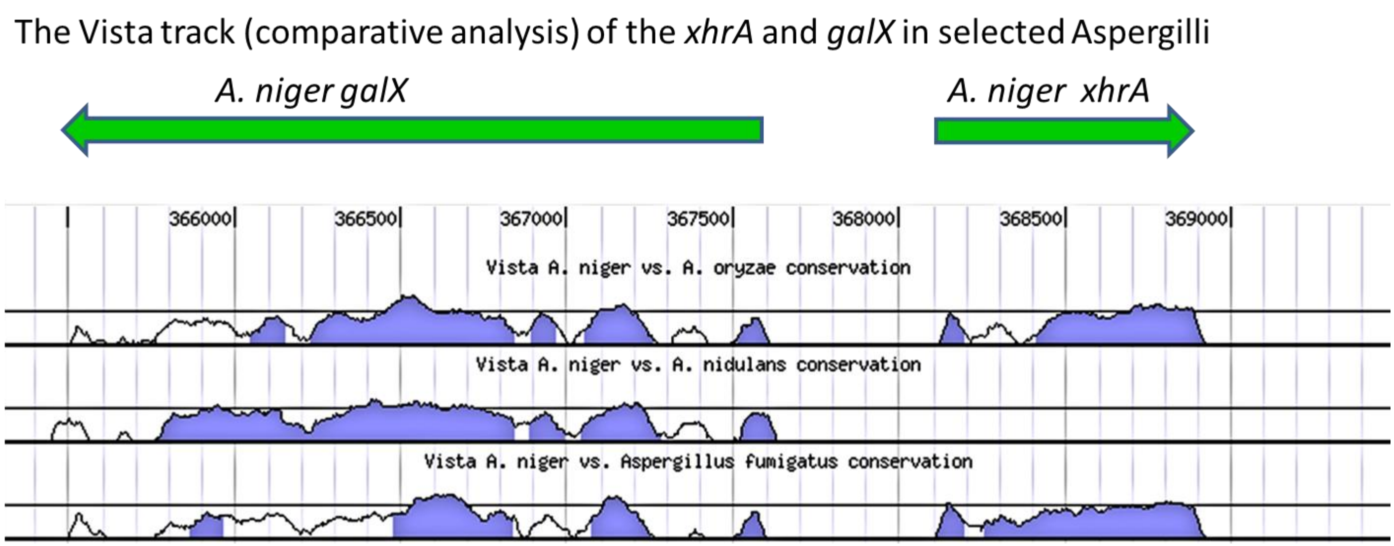
Legend figure S1:

Protein sequence alignment of LxrA, XhrA, LXR4, and LXR3. In *Aspergillus niger*, two close homologues, LxrA and XhrA, function in distinct catabolic pathways for L-arabinose and for D-galactose, respectively. In *Trichoderma reesei* (*Hypocrea jecorina*), the close homologue of these two proteins, LXR4, is responsible for L-xylo-3-hexulose conversion to D-sorbitol in the oxido-reductive D-galactose pathway; while the more distant homologue, LXR3, carries out the L-xylulose reduction in the L-arabinose catabolic pathway.

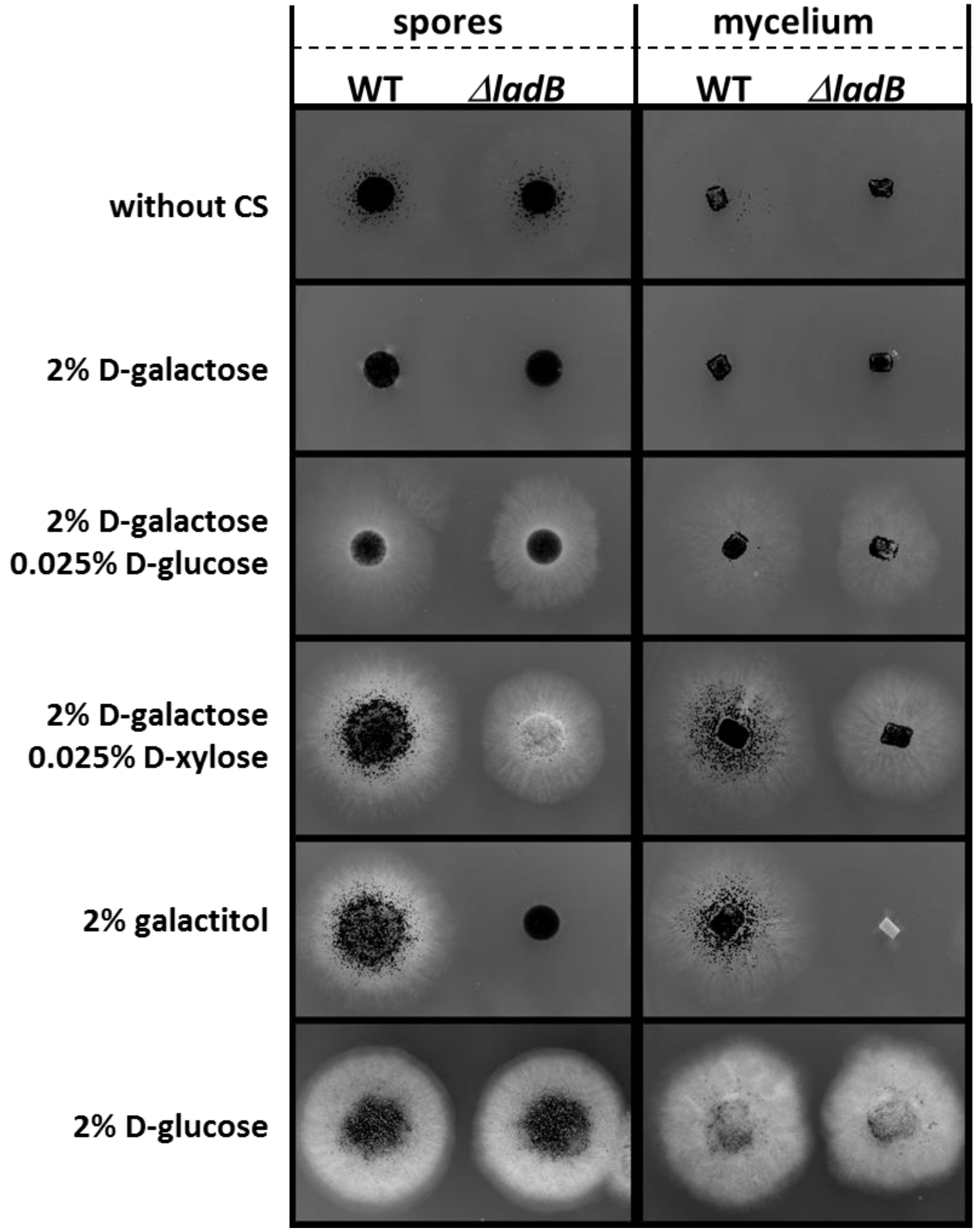
Supplementary figure 2:



Legend figure S2:

Comparison of the genomic regions covering the *galX* and *xhrA* genes in three *Aspergillus* species with *A. niger*. While all of the species contain the *galX* gene, only *A. oryzae* and *A. fumigatus*, but not *A. nidulans*, seem to have the *xhrA* gene (the figure is a modification of a vista visualization obtained from the JGI Aspergillus niger v3.0 web database - http://genome.jgi-psf.org/Aspni5/).

Supplementary figure 3:



Legend figure S3:

Growth of the *A. niger* strain ATCC 1015 (WT) and the ATCC 1015 with a deletion in *ladB* (*ΔladB*) on different carbon sources (CS). Comparison of the growth originated from either conidiospores (left panel) or pre-grown mycelium (right panel). The conidiospores were collected from cultures grown on PDA (potato-dextrose agar) plates and kept at -80°C. The mycelia were produced by inoculating the spores into YESG (2% yeast extract; 4% sucrose; 3% gelatin) medium and cultivating at 28°C for 18 hours. The mycelium was collected by filtration, washed with sterile water, and cut into identical pieces prior to the inoculation on the agar plates. The test plates contained YNB (yeast nitrogen base), 2% agar, and a carbon source as indicated in the figure. The cultivation was carried out at 28°C for 4 days in the case of spore-inoculation or 3 days in the case of mycelium-inoculation.

Here we summarise the observations supporting our claims:

- The mycelium pre-grown in the presence of sucrose and peptides is not able to grow after transfer to D-galactose.
- The addition of a small amount of D-xylose into the D-galactose-containing medium leads to considerable enhancement of growth of the wild type stain regardless of the form of inoculum. This is caused by induction of XyrA-dependent D-galactose reductase activity and not by the induction of the Leloir pathway (1).
- On the other hand, addition of a small amount D-glucose into the D-galactose-containing medium leads to only minimal growth probably caused by the D-glucose itself rather than by D-galactose. This is expected as the genes encoding the D-galactose oxido-reductive metabolism are repressed (Fig. 2, (1)). However, Fekete et al. showed that in their *A. niger* strain the genes encoding the Leloir pathway are expressed even in presence of D-glucose (2).
- The *A. niger* strain we used seemed to have no difficulty to sporulate in the presence of D-galactose when supplemented by a small amount of D-xylose (Fig. S3).

1. Mojzita, D., Koivistoinen, O. M., Maaheimo, H., Penttilä, M., Ruohonen, L., and Richard, P. (2012) *Fungal Genet. Biol.* **49**, 152-159
2. Fekete, E., de Vries, R. P., Seiboth, B., Vankuyk, P. A., Sándor, E., Fekete, E., Metz, B., Kubicek, C. P., and Karaffa, L. (2012) *FEMS Microbiol Lett.* **329**, 198-203