The lariat-derived circRNA from ATXN2L: an outstanding circRNA in pigs

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In contrast to exon-derived circular RNAs (exonic circRNAs), which are widely studied, much less in known on intron-derived circRNAs: when intron lariats escape degradation due to debranching failure they can become circRNA precursors. When characterizing the intronic circRNA landscape of porcine and bovine testis, liver and muscle, only a circRNA derived from a lariat of the *ATXN2L gene* was identified in both species. This intronic *ATXN2L* circRNA was always among the strongest contributors of intronic circRNAs in testes. In pubertal porcine testes, we observed a single circRNA with a uniquely highly abundant expression pattern: this was again the intronic *ATXN2L* circRNA. Its dominant position goes far beyond intronic circRNAs, because it is most abundant across all circRNAs in pubertal testes (7 datasets). No analogous dominating position of an exonic or intronic circRNA was found neither in the adult porcine testis, nor in pubertal bovine testis.

When compiling data sets from different origins to conduct this study, we noticed that RNA preparation and sequencing protocols have a significant impact on circRNA recovery. Moreover, the comparison of porcine transcriptome of pubertal and adult testes generated with divergent protocols (at least for ribosomal sequence removal) suggested age-dependent differential expression for a large proportion of small-non-coding RNAs, so we suspect that additional non-biological effects (ribodepletion) might bias the recovery of small transcripts. This intronic *ATXN2L* circRNA is very small (118 nucl. in pigs) and we do not rule out that it may be partially lost by some experimental protocols.

Due to its particularly high expression level at a biologically sensitive time in testis development, we strongly assume that intronic *ATXN2L* circRNA has a specific function, at least in the pubertal pig testis. In this tissue, we did not obtain supporting evidence for an impact of this circular transcript on the transcription of the *ATXN2L* gene itself. Our data suggest that we may be witnessing the emergence of a new non-coding gene in pigs.

Annie Robic[1], Thomas Faraut[1], Chloé Cerutti[1], Julie Demars[1] and Christa Kühn[2,3]

[1] GenPhySE, Université de Toulouse, INRAE, ENVT, 31326 Castanet Tolosan, France
[2] Institute Genome Biology, Research Institute for Farm Animal Biology (FBN), Dummerstorf, Germany
[3] Faculty of Agricultural and Environmental Sciences, University of Rostock, Rostock, Germany