

Correction: Nucleotide modifications in messenger RNA and their role in development and disease

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Biochemical Society Transactions (2016) 44; 1385–1393, DOI: 10.1042/BST20160110

The authors have become aware of an error in their paper that they would like to correct. The final paragraph in the section on Pseudouridine reads:

“A role of snoRNA-mediated pseudouridylation has been found in serotonin receptor 5-HT_{2c}R alternative splicing regulation contributing to the disease of the Prader–Willi syndrome. These patients do not express the brain-specific C/D box snoRNA HBII52 due to an imprinting defect, indicating that this complex involved in pseudouridylation has additional snoRNA-guided roles beyond modifying mRNA [31,32].”

The authors amend this to:

“A second class of snoRNAs, C/D box snoRNAs (SNORDs), can mediate 2'-O-methylation of the ribose, but can also act in a methylation-independent manner. About half of the SNORDs direct 2'-O-methylation in rRNA, while the rest has no sequence complementary to rRNA. Loss of predominantly brain-expressed SNORD116 and SNORD109a by deletion or as a result of imprinting defects contributes to Prader–Willi syndrome. A role for a methylation-independent function of SNORD115 (HBII52) has been shown in the serotonin receptor 5-HT_{2c}R, where binding affects splice-site selection resulting in alternative splicing defects [31, 32].”

Correction published:
12 June 2019