FRONTEO
～COMBAT COVID-19～
⑥探索結果（２）　新しい抗ウイルス剤の可能性（NAT10）

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Kibit & concept Encoder

MAY. 2020

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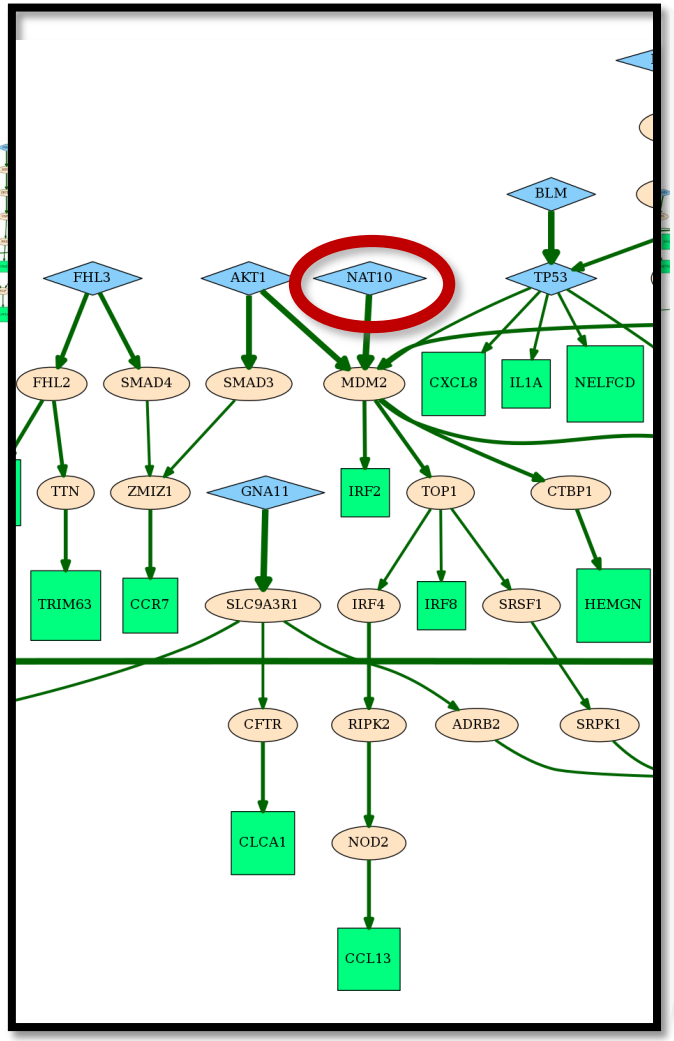
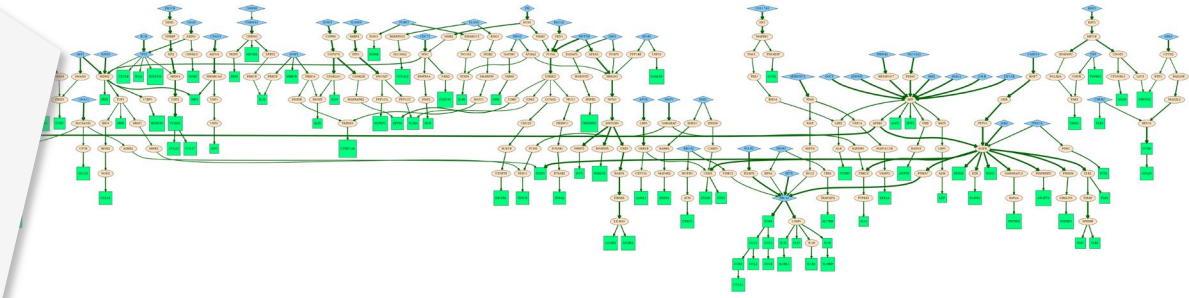
既存薬のターゲットとなっていない分子NAT10





RNAの安定化にかかわる分子で、AIにより原因性と判別されている。

NAT10:

The protein encoded by this gene is an RNA cytidine acetyltransferase involved in histone acetylation, tRNA acetylation, the biosynthesis of 18S rRNA, and the enhancement of nuclear architecture and chromatin organization. [provided by RefSeq, Oct 2016]

NAT10に関する文献

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Acetylation of cytidine residues boosts HIV-1 gene expression by increasing viral RNA stability

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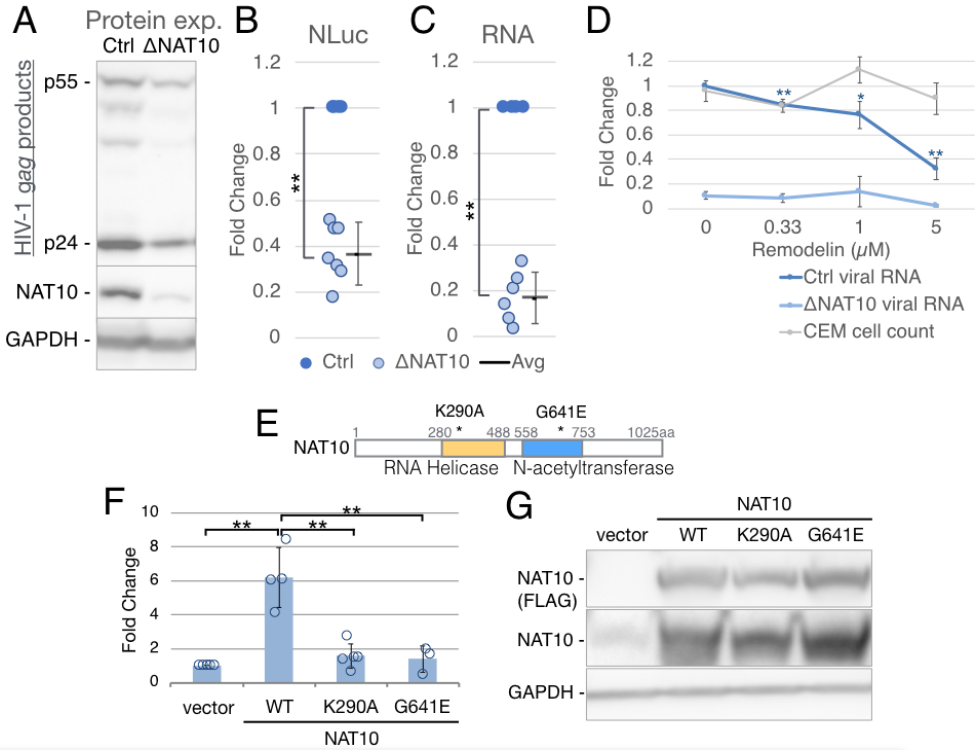
NAT10（シチジンをアセチル化する酵素）に関する文献。
NAT10によってもたらされたRNA安定性がHIVの遺伝子の発現を促進することが報告されている。

NAT10の減少がウイルスの増殖を抑える可能性

replication of human immunodeficiency virus 1 (HIV-1) and several other viruses[3-11]. Recently, acetylation of the N^4 position of cytidine (ac4C) was reported to boost cellular mRNA function by increasing mRNA translation and stability[12]. We therefore hypothesized that ac4C and N-acetyltransferase 10 (NAT10), the cellular enzyme that adds ac4C to RNAs, might also have been subverted by HIV-1 to increase viral gene expression. We now confirm that HIV-1 transcripts are indeed modified by addition of ac4C at multiple discreet sites and demonstrate that silent mutagenesis of a subset of these ac4C addition sites inhibits HIV-1 gene expression in *cis*. Moreover, reduced expression of NAT10, and the concomitant decrease in the level of ac4C on viral RNAs, inhibits HIV-1 replication by reducing HIV-1 RNA stability. Interestingly Remodelin, a previously reported inhibitor of NAT10 function[13,14], also inhibits HIV-1 replication without affecting cell viability, thus raising the possibility that the addition of ac4C to viral mRNAs might emerge as a novel cellular target for antiviral drug development.



NAT10の発現の減少は、同時にウイルスのRNAにおけるac4Cの減少を伴い、それによってウイルスのRNAが不安定になり、ウイルスのRNAの増殖が抑えられる。

NAT10阻害剤が作られれば、ウイルスの増殖を抑制できる可能性がある

この資料は下記により作成されました。

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