Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
A-to-I RNA editing is a fundamental biological process with 2 major forms, namely adenosine-to-inosine (A-to-I, recognized as A-to-G) and cytosine-to-uracil (C-to-U) deamination, mediated by ADAR and APOBEC enzyme families, respectively. A-to-I RNA editing has been shown to directly affect the genome/transcriptome of RNA viruses with significant repercussions for viral protein synthesis, proliferation and infectivity, while it also affects recognition of double-stranded RNAs by cytosolic receptors controlling the host innate immune response. Recent evidence suggests that RNA editing may be present in SARS-CoV-2 genome/transcriptome. The majority of mapped mutations in SARS-CoV-2 genome are A-to-G/U-to-C (opposite strand) and C-to-U/G-to-A (opposite strand) substitutions comprising potential ADAR-/APOBEC-mediated deamination events. A single nucleotide substitution can have dramatic effects on SARS-CoV-2 infectivity as shown by the D614G(A-to-G) substitution in the spike protein. Future studies utilizing serial sampling from patients with COVID-19 are warranted to delineate whether RNA editing affects viral replication and/or the host immune response to SARS-CoV-2.

1. Introduction

Currently, more than 170 known RNA base modifications expand the RNA alphabet from 4 to hundreds of individual nucleotides [1]. The most abundant RNA modification in humans is RNA editing, which comes in 2 main forms, namely adenosine-to-inosine (A-to-I) and cytosine-to-uracil (C-to-U) deamination, mediated by the ADAR and APOBEC family of enzymes, respectively [2-4]. Inosine (I) is in turn recognized as guanosine (G) by polymerases during RNA-dependent RNA replication (viral replication) and by ribosomes during translation [2,5,6]. A-to-I RNA editing is a widespread phenomenon in the human transcriptome, mainly located in the endogenous Alu retroelements, which locally form double-stranded RNA regions, a pre-requisite for the binding and catalytic deamination by ADARs [7,8]. A-to-I RNA editing has been shown to affect multiple facets of the RNA metabolism [2,5,9], while we and others have previously shown that ADAR1-induced RNA editing is enhanced under chronic inflammatory conditions leading to stabilization of proinflammatory transcripts, thus having a “fuel-on-fire” effect on the perpetuation of the inflammatory response [10,11]. More importantly, A-to-I RNA editing has been shown to directly affect the genome and transcriptome of RNA viruses with significant repercussions for viral protein synthesis, proliferation and infectivity [6,12]. Of interest, recent data suggest that RNA editing may also take place in the genome/transcriptome of SARS-CoV-2, the virus responsible for the ongoing COVID-19 pandemic.

2. Single nucleotide variants in SARS-CoV-2 genome: hints towards the involvement of host RNA editing machineries

As of February 2021, COVID-19 accounts for more than 2 million deaths worldwide. Despite the intensive efforts of the scientific and medical community, there is currently no available targeted therapy, while numerous vaccines are in the stage of clinical trials with only a few having reached the clinic. SARS-CoV-2 has an approximately 30

**Abbreviations:** eIF-2a, Eukaryotic translation initiation factor 2 subunit 1; IFN, interferon; IRF, interferon regulatory factor; ISG, interferon stimulated genes; MDA-5, melanoma differentiation-associated protein 5; PKR, protein kinase R; RIG-I, retinoic acid-inducible gene 1.

* Corresponding author.

E-mail address: dparask@med.uoa.gr (D. Paraskevis).
The observed frequency of SNVs does not follow the pattern of RNA-dependent RNA polymerase (RdRP) errors, as revealed by previous mechanistic studies removing the 3′-5′ exoribonuclease activity (“proofreading”) of coronavirus [21].

1) The observed frequency of SNVs does not follow the pattern of RNA-dependent RNA polymerase (RdRP) errors, as revealed by previous mechanistic studies removing the 3′-5′ exoribonuclease activity (“proofreading”) of coronavirus [21].

2) C-to-U substitutions observed in SARS-CoV-2 genome/transcriptome follow the APOBEC deamination motif [A/U][A/U] [3,22-24]. C residues surrounded by A/U both upstream (5′) and downstream (3′) were ~10-fold more likely to be substituted by U compared to C residues surrounded by either G or C [24].

3) A-to-G substitutions in SARS-CoV-2 genome show a depletion of G at ~1 position [22,25], and a slight G enrichment 1-base downstream [25], which is also observed in human ADAR1/2-induced A-to-I editing events [26].

4) The increased frequency of A-to-G base substitutions in association with increased type I interferon (IFN) response and ADAR1 expression in Calu-3 cells infected with SARS-CoV-2 supports the potential involvement of the interferon-inducible ADAR1p150 enzyme [25].

In a first report utilizing nanopore direct RNA sequencing, researchers detected at least 41 RNA modification sites on viral transcripts [15]. Of interest, modified viral RNAs had shorter poly(A) tails than unmodified RNAs, suggesting that RNA modifications may affect RNA stability and consequently viral protein synthesis [15]. While this initial report excluded the presence of A-to-I RNA editing events, later studies have detected multiple RNA editing sites in the SARS-CoV-2 transcriptome and genome [22,25]. Di Giorgio et al. used bronchoalveolar lavage fluid (BALF) samples from patients with COVID-19 to examine the presence of RNA editing events in SARS-CoV-2 transcriptome [22]. Using metagenomic sequencing they analysed 8 samples of appropriate sequencing depth and detected multiple base substitutions in SARS-CoV-2 transcriptome with A-to-G being the most prevalent. Of note, depletion of G bases in position ~1 of the A/G substitutions was evident in SARS-CoV-2 samples supporting the involvement of ADARs [22]. Similarly, C-to-U substitutions following a motif compatible with APOBEC editing were detected in the examined SARS-CoV-2 transcriptome.
| Virus                          | Main “editor” | Model of study | Editing sites | Treatment - ADAR overexpression / knockdown | Key findings                                                                 | Ref. |
|-------------------------------|---------------|----------------|---------------|---------------------------------------------|-------------------------------------------------------------------------------|------|
| HCV                           | ADAR1 p150    | In vitro Huh-7 cells | Radiolabeled AMP | IFNs treatment ADAR1-knockdown ADAR2 knockdown | Increased A-to-I editing of radiolabeled AMP. Inhibition of HCV replicon (B^7) synthesis. 5-41-fold increase of HCV replicons. | [46] |
| HDV                           | ADAR1 p150    | In vitro Huh-7, HEK293 cells | HDAg (A1012) (“Amber/W” site) | ADAR1-knockdown | No effect on HDV replicon. HDAg-L production Switch from replication to packaging | [28-30] |
|                               | ADAR1 p150    | In vitro Huh-7, HEK293 cells | HDAg (A1012) (“Amber/W” site) | ADAR1-knockdown | Inhibition of HDV-antigeneome editing. Reduced production of HDV virions. ADAR1p110 is primarily responsible for HDV antigenome editing during replication. | [47,48] |
| ADAR1/ADAR2                   | In vitro Huh-7 cells | HDAg (A1012) (“Amber/W” site) | IFNs treatment (ADAR1p150 up-regulation) ADAR1/ADAR2 overexpression | Increased editing of amber/W site. Hyper-editing at non-amber/W sites inhibited HDV RNA replication. Increased HDAg-L production. Inhibition of HDV replication. ADAR1p150 is mainly responsible for IFNs-induced HDV RNA editing. | [49-51] |
| HIV                           | ADAR1 p150    | In vitro COS-7, HEK293T cells | env | ADAR1 overexpression Catalytically-inactive ADAR1 overexpression ADAR1-knockdown | Upregulation of p24 Gag protein expression No effect on p24 Gag protein expression | [52] |
|                               | ADAR1 p150    | In vitro HEK293T, Jurkat T cells | 5’ UTR, rev, tat | ADAR1-knockdown ADAR1-overexpression | Downregulation of p24 Gag protein expression. Significantly increased release of HIV virions. Increased viral infectivity in primary human CD4^+ T cells. | [53] |
|                               | ADAR2 p150    | In vitro HEK293T, Jurkat T cells | 5’ UTR | ADAR2-knockdown ADAR2-overexpression | Significantly increased release of HIV virions. No effect on viral infectivity. | [54] |
|                               | ADAR1 p150    | Ex vivo PBMCs, primary CD4^+ T cells | n/a | ADAR1-knockdown ADAR1-overexpression ADAR1-knockdown | Impaired HIV protein synthesis and replication. Decreased HIV replication in Aicardi Goutières Syndrome (defective ADAR1) compared to healthy control-derived PBMCs. | [55] |
|                               | ADAR1 p150    | In vitro Jurkat T cells | rev, env | ADAR1-knockdown ADAR1-overexpression ADAR1-knockdown | Downregulation of viral protein synthesis and replication. Inhibition of HIV protein synthesis and viral infectivity. | [56] |
|                               | ADAR1 p150    | Ex vivo Macrophages, BALF cells | Envelope gp120 V3 | ADAR1-knockdown ADAR1-overexpression | No significant effect on viral protein synthesis or infectivity. | [40] |
|                               | ADAR1 p150    | In vitro HEK293T, A549 cells | Reporter plasmid | ADAR1-knockdown ADAR1-overexpression | No significant effect on viral protein synthesis or infectivity. | [31] |
|                               | SARS-CoV-2    | ADAR1 p150    | BALF samples | Adenovirus viral transcriptome / genome | Decreased viral protein expression and viral production. | [57] |
|                               | Measles Virus | ADAR1 p150    | In vitro Vero, HeLa cells | Defective Interfering (DI) RNAs | Several measles virus DI s had a large number of A-to-G substitutions, suggestive of ADAR1-mediated A-to-I editing. | [22] |
|                               | SARS-CoV-2    | ADAR1 p150    | Calu-3, Vero cells | viral genome / transcriptome | Several A-to-G / U-to-C mutations were observed in SARS-CoV-2 transcriptome (most common single nucleotide variants). Significantly fewer A-to-G/U-to-C substitutions were detected in the viral genome. No nonsense A-to-G/U-to-C substitutions were detected in SARS-CoV-2 genome or transcriptome, proposing a potential deleterious effect for SARS-CoV-2 replication. | [25] |

Abbreviations: HCV: hepatitis C virus; HDV: hepatitis D virus; HIV: human immunodeficiency virus; BALF: bronchoalveolar lavage fluid.

The Table includes the main results from selected publications on the role of ADAR1/2 in RNA viral infections and does not comprise an exhaustive literature review.
binding to ACE2 and consequently cellular entry and virulence is indeed
amino-acid substitution (D614G) in the spike protein affecting viral
dsRNA for degradation by specific endonucleases [41], remains to be
2. However, SARS-CoV-2 seems to avoid extensive A-to-I RNA editing as
ADAR1-induced RNA editing in the host immune response to SARS-CoV-
SARS-CoV-2 [37]. ADAR1, and specifically the cytoplasmic ADAR1p150
(Table 1 and Fig. 1). An excellent example of this comes from the hep-
factor for the fate of multiple RNA viruses including HIV-1, HCV, HDV,
Influenza A and Measles virus (Table 1). Host-dependent A-to-I RNA
editing of the viral genome or transcriptome can have either pro-viral or
anti-viral effects depending on the host-virus interaction (excellently
reviewed in [6]). A-to-I RNA editing in coding regions may affect protein
synthesis and consequently proliferation and infectivity of the virus
(Table 1 and Fig. 1). An excellent example of this comes from the hep-
atitis delta virus (HDV): HDV encodes two forms of the Hepatitis Delta
Antigen (HDAg) protein, namely a shorter form (p24/ HDAg-S) that is
essential for viral RNA replication, and a longer form extended by 19
amino acids (p27/ HDAg-L), which facilitates packaging of the viral
genome and viral particle assembly [6]. A-to-I editing of a stop codon
(UAG, “amber”) is necessary to turn it into tryptophan [UI(G), “W”]
thus enabling the production of HDAg-L. [28–30] (Table 1). Moreover,
ADAR1 may interact with viral proteins, such as Influenza A NS1,
through its RNA-binding domains with potential implications for type I
IFN pathway activation [31]. Viruses may take advantage of the host
RNA editing machinery to avoid recognition by innate immune re-
ceptors. More specifically, previous studies have shown that dsRNAs
containing multiple IU-pairs suppress the activation of the innate immune
receptors MD2A5 and RIG-I and subsequently IRF-3, thus inhibiting
the induction of the type I IFN pathway [32] (Fig. 1). Similarly, ADAR1
can directly interact with the antiviral PKR protein and prevent its
hyperactivation thus promoting viral replication [33–36].
Type I IFN seems to be the determining factor of host response to
SARS-CoV-2 [37]. ADARI, and specifically the cytoplasmic ADARlp150
isoform, is IFN-inducible [38], suggesting a potential involvement of
ADARI-induced RNA editing in the host immune response to SARS-Co-
V. However, SARS-CoV-2 seems to avoid extensive A-to-I RNA editing as
shown by the low levels (<1%) of A-to-I editing detected in the isolated
viral genomes/transcriptomes from patient cells [22,25], which is in line
with low type I IFN gene expression observed in SARS-CoV-2 infected
cells [39]. Whether exogenous IFN administration to COVID-19
patients could affect viral replication partly through induction of
multiple RNA editing events (hyper-editing) that can inhibit viral pro-
tein synthesis, as has been previously shown for HIV-1 [40], or mark
dsRNA for degradation by specific endonucleases [41], remains to be
proven by future studies.
Finally, the best-studied SARS-CoV-2 mutation to date leading to an
amino-acid substitution (D614G) in the spike protein affecting viral
binding to ACE2 and consequently cellular entry and virulence is indeed
an A-to-G substitution [16]. Whether this was originally an ADARI-
mediated RNA editing event cannot be proven, however it supports
the significant repercussions of single nucleotide substitutions in the
spike protein for SARS-CoV-2 infectivity [16,17] through various
mechanisms including ACE2 binding affinity [42], conformational
changes leading to an ACE2 binding-competent state [43] or higher
availability of spike protein in virions [44]. With CRISPR-Cas13 being
intensively investigated in the last few years [45], identification of
deleterious A-to-G substitutions in the SARS-CoV-2 genome through
computational modeling and validation of reduced proliferation/infect-
vity in vitro could unravel new, targeted therapeutic approaches.
Finally, future studies examining sequential samples from patients with
different disease course could shed more light on the up- or down-
regulation of RNA editing in patients and its association with viral
replication and host innate immune response during the early stages of
disease (type I IFN-mediated immunity) or during the hyper-
inflammatory syndrome observed later on.
In conclusion, the widespread nature of RNA editing and its estab-
lishment role in viral infection the inducible character of ADAR1 by
interferon and other proinflammatory cytokines, and the potential of
single base substitutions to significantly alter the infectivity of SARS-
CoV-2 along with the establishment of CRISPR-Cas13, make RNA editing
worth exploring in the COVID-19 pandemic.

Author contributions
NIV and KM performed literature search. NIV wrote the 1st draft of
the manuscript after input from all authors. All authors reviewed the
manuscript and approved the final version.

Declaration of Competing Interest
none.

References
[1] P. Boccaletto, M.A. Machnicka, E. Purta, P. Piątkowski, B. Baginski, T.K. Wierci,
V. de Crecy-Lagard, R. Ross, P.A. Lambach, A. Kotter, M. Helm, J.M. Bujnicki,
MODOMICS: a database of RNA modification pathways. 2017 update, Nucleic
Acids Res. 46 (2018) D303–D307, https://doi.org/10.1093/nar/gkx1030.
[2] K. Nishikura, A-to-I editing of coding and non-coding RNAs by ADARs, Nat.
Rev. Mol. Cell Biol. 17 (2016) 83–96, https://doi.org/10.1038/nrm.2015.4.
[3] T. Lerner, F.N. Papavasilious, R. Pecori, RNA editors, cofactors, and mRNA targets:
an overview of the C-to-U RNA editing machinery and its implication in human
disease, Genes 10 (2018), https://doi.org/10.3390/genes10010013.
[4] A. Gatsiou, K. Stellos, Dawn of epitranscriptomic medicine. Circ. Genomic Precis.
Med. 11 (2018), e001927, https://doi.org/10.1136/circgenm.118.001927.
[5] A. Gatsiou, N. Vlachogiannis, F.F. Lunella, M. Sachse, K. Stellos, Adenosine-to-
inosine RNA editing in health and disease. Antioxid. Redox Signal. 29 (2018)
846–863, https://doi.org/10.1089/ars.2017.7295.
[6] C.E. Samuel, Adenosine deaminases acting on RNA (ADARs) are both antiviral and
proviral, Virology. 411 (2011) 180–193, https://doi.org/10.1016/j.
virology.2010.12.004.
[7] A. Athanasiadis, A. Rich, S. Maas, Widespread A-to-I RNA editing of Alu-containing
mRNAs in the human transcriptome, PloS Biol. 2 (2004), e91, https://doi.
org/10.1371/journal.pbio.0020091.
[8] D.D.Y. Kim, Widespread RNA editing of embedded Alu elements in the human
transcriptome, Genome Res. 14 (2004) 1719–1725, https://doi.org/10.1101/
gr.285504.
[9] L.E. Dorn, S. Tual-Chalot, K. Stellos, F. Acconnero, RNA epigenetics and
vascular, J. Mol. Cell. Cardiol. 129 (2019) 272–280, https://doi.org/
10.1016/j.yycard.2019.03.010.
[10] K. Stellos, A. Gatsiou, K. Stamatepolouzos, L. Pericic Matic, D. John, F.F. Lunella,
N. Jae, O. Rouzbah, C. Amrhin, F. Sigala, R.A. Boon, B. Fürtig, Y. Manavski,
Y. Xu, S. Uchida, T. Keller, J.-N. Boekel, A. Franco-Cereceda, L. Maegdefessel,
W. Chen, H. Schwalbe, A. Bindreff, F. Eriksson, U. Hedin, A.M. Zehe, S.
Dimmel, Adenosine-to-inosine RNA editing controls cathespin S expression in
atherosclerosis by enabling HuR-mediated post-transcriptional regulation, Nat.
Med 22 (2016) 1140–1150, https://doi.org/10.1038/nm.4172.
[11] N.I. Vlachogiannis, A. Gatsiou, D.A. Silvestris, K. Stamatepolouzos, M.
G. Tektidasou, A. Gallo, P.P. Sifakis, K. Stellos, Increased adenosine-to-inosine
RNA editing in rheumatoid arthritis, J. Autoimmun. 106 (2020) 102329, https://
doi.org/10.1016/j.jaut.2019.102329.
A.G. Polson, H.L. Ley, B.L. Bass, J.L. Casey, RNA editing of hepatitis delta virus antigenome reveals numerous new sites, Nat. Genet. 45 (2013) 970–976, https://doi.org/10.1038/ng.2608.

F. Zhang, RNA editing with CRISPR-Cas13, Science. 358 (2017) 1019–1027, https://doi.org/10.1126/science.aam8499.

C.D. Lyssowski, C. Waidler, M. Kanter, J. Storm, J.E. Lemieux, J.B. Munro, A. Rafique, A. Barve, P.C. Sabeti, C.A. Kyratsous, N. Spaan, B.S. Razooky, H.-H. Hoffmann, E. Michailidis, L. Moens, J.E. Han, L. Lorenzo, S. Baric, SARS-CoV-2 spike protein variant, Cell 183 (2020), https://doi.org/10.1016/j.cell.2020.06.043, 812–827.e19.

A. Albrecht, B.R. tenOever, Imbalanced host response to SARS-CoV-2 drives COVID-19 severity, Infect. Genet. Evol. J. Mol. Epidemiol. Evol. Genet. Infect. Dis. 79 (2020) 6298–2015, https://doi.org/10.1016/j.coi.2021.09.015.

A. Bhoopathi, G. Ramesh, S. Assim, V. Senthil, S. Sivasubramanian, A. Sivakumar, N. Thirumalai, J. Jagadish, G. Shanthi, S. Jayaraman, S. Suresh, V. Gnanam, H. Prabakaran, S. Swaminathan, Impact of SARS-CoV-2 spike protein variant, Cell 183 (2020), https://doi.org/10.1016/j.cell.2020.06.043, 812-827.e19.

P. Zhang, Y. Hozumi, Y.-H. Zheng, C. Yin, G.-W. Wei, Host immune response to SARS-CoV-2 spike protein variant, Cell 183 (2020), https://doi.org/10.1016/j.cell.2020.06.043, 812-827.e19.

S. Baric, SARS-CoV-2 D614G variant exhibits efficient replication ex vivo and infection in vivo through a mechanism involving ADAR1 and ADAR2, J. Virol. 76 (2002) 6298–2005, https://doi.org/10.1128/JVI.79.10.6291–6298.2005.

S. Bhoopathi, G. Ramesh, S. Assim, V. Senthil, S. Sivasubramanian, A. Sivakumar, N. Thirumalai, J. Jagadish, G. Shanthi, S. Suresh, V. Gnanam, H. Prabakaran, S. Swaminathan, Impact of SARS-CoV-2 spike protein variant, Cell 183 (2020), https://doi.org/10.1016/j.cell.2020.06.043, 812–827.e19.

A. Albrecht, B.R. tenOever, Imbalanced host response to SARS-CoV-2 drives COVID-19 severity, Infect. Genet. Evol. J. Mol. Epidemiol. Evol. Genet. Infect. Dis. 79 (2020) 6298–2015, https://doi.org/10.1016/j.coi.2021.09.015.

A. Albrecht, B.R. tenOever, Imbalanced host response to SARS-CoV-2 drives COVID-19 severity, Infect. Genet. Evol. J. Mol. Epidemiol. Evol. Genet. Infect. Dis. 79 (2020) 6298–2015, https://doi.org/10.1016/j.coi.2021.09.015.

A. Albrecht, B.R. tenOever, Imbalanced host response to SARS-CoV-2 drives COVID-19 severity, Infect. Genet. Evol. J. Mol. Epidemiol. Evol. Genet. Infect. Dis. 79 (2020) 6298–2015, https://doi.org/10.1016/j.coi.2021.09.015.

A. Albrecht, B.R. tenOever, Imbalanced host response to SARS-CoV-2 drives COVID-19 severity, Infect. Genet. Evol. J. Mol. Epidemiol. Evol. Genet. Infect. Dis. 79 (2020) 6298–2015, https://doi.org/10.1016/j.coi.2021.09.015.

A. Albrecht, B.R. tenOever, Imbalanced host response to SARS-CoV-2 drives COVID-19 severity, Infect. Genet. Evol. J. Mol. Epidemiol. Evol. Genet. Infect. Dis. 79 (2020) 6298–2015, https://doi.org/10.1016/j.coi.2021.09.015.
editing in interferon-alpha-stimulated host cells, J. Viral Hepat. 13 (2006) 150-157, https://doi.org/10.1111/j.1365-2893.2005.00663.x.

[51] D. Hartwig, L. Schoeneich, J. Greeve, C. Schütte, I. Doen, H. Kirchner, H. Hennig, Interferon-α stimulation of liver cells enhances hepatitis delta virus RNA editing in early infection, J. Hepatol. 41 (2004) 667-672, https://doi.org/10.1016/j.jhep.2004.06.025.

[52] A. Phuphuakrat, R. Kraiwong, C. Boonarkart, D. Laubakritti, T.-H. Lee, P. Auewarakul, Double-stranded RNA adenosine deaminases enhance expression of human immunodeficiency virus type 1 proteins, J. Virol. 82 (2008) 10864-10872, https://doi.org/10.1128/JVI.00238-08.

[53] M. Doria, F. Neri, A. Gallo, M.G. Farace, A. Michienzi, Editing of HIV-1 RNA by the double-stranded RNA deaminase ADAR1 stimulates viral infection, Nucleic Acids Res. 37 (2009) 5848–5858, https://doi.org/10.1093/nar/gkp604.

[54] M. Doria, S. Tomasselli, F. Neri, S.A. Ciafre, M.G. Farace, A. Michienzi, A. Gallo, ADAR2 editing enzyme is a novel human immunodeficiency virus-1 proviral factor, J. Gen. Virol. 92 (2011) 1228-1232, https://doi.org/10.1099/vir.0.028043-0.

[55] E. Cuadrado, T. Boomman, J.L. van Hamme, M.H. Jansen, K.A. van Dort, A. Vanderver, G.L. Rice, Y.J. Crow, N.A. Koostra, T.W. Kuijpers, ADAR1 facilitates HIV-1 replication in primary CD4+ T cells, PLoS One 10 (2015), e0143613, https://doi.org/10.1371/journal.pone.0143613.

[56] N. Biswas, T. Wang, M. Ding, A. Tumne, Y. Chen, Q. Wang, P. Gupta, ADAR1 is a novel multi targeted anti-HIV-1 cellular protein, Virology. 422 (2012) 265-277, https://doi.org/10.1016/j.virol.2011.10.024.

[57] C.K. Pfaller, G.M. Mastorakos, W.E. Matchett, X. Ma, C.E. Samuel, R. Cattaneo, Measles virus defective interfering RNAs are generated frequently and early in the absence of C protein and can be destabilized by adenosine deaminase acting on RNA 1-like hypermutations, J. Virol. 89 (2015) 7735-7747, https://doi.org/10.1128/JVI.01017-15.