

mGem: Horses for courses in mapping bacterial small RNA interaction networks

Simon L. Dove,¹ Sahar Melamed²

AUTHOR AFFILIATIONS See affiliation list on p. 5.

ABSTRACT Over the past few decades, it has become increasingly clear that small RNAs (sRNAs) play important regulatory roles in bacteria. These RNA species often work in concert with RNA chaperones such as Hfq that facilitate their base-pairing with target mRNAs. The mapping of sRNA interaction networks through the identification of the RNA species that sRNAs base-pair with can therefore provide critical insights into the potential regulatory roles sRNAs play. Indeed, sRNA interaction networks can be complex, with some bacteria producing more than a hundred different sRNAs, each capable of targeting anywhere from a single mRNA to several hundred different mRNAs. Here, we highlight two high-throughput approaches, RNA interaction by ligation and sequencing and intracellular RNA interaction by ligation and sequencing, that enable transcriptome-wide identification of sRNA interaction partners. Both methods capture RNA–RNA interactions *in vivo* and exploit chaperone-associated RNA–RNA ligation to capture native sRNA–target duplexes, providing powerful and scalable strategies for determining bacterial sRNA interaction networks.

KEYWORDS RIL-seq, iRIL-seq, sRNA, Hfq, ProQ, RNA–RNA interactions, post-transcriptional regulation

SMALL RNAs WORK IN CONCERT WITH RNA CHAPERONES AND PAIR WITH TARGET TRANSCRIPTS

Small RNAs (sRNAs) are bacterial RNA species that are anywhere between 50 and 500 nucleotides in length. Many sRNAs employ a short sequence (as few as 6 nucleotides), known as the seed sequence, to base-pair with target transcripts (1, 2). This base-pairing is often incomplete and, in gram-negative bacteria, is frequently mediated by the RNA chaperone Hfq (3). Hfq can bind simultaneously to both an sRNA and its mRNA target, resulting in activation or repression of translation, modulation of transcript stability, or both (1). For any given sRNA, of which there may be more than a hundred encoded by the bacterium (4), there can be as few as one, or as many as several hundred target transcripts (5). Post-transcriptional control by sRNAs can therefore have far-reaching effects.

HOW DO YOU IDENTIFY THE TARGETS OF sRNAs?

Although RNA-sequencing approaches have revolutionized the identification of sRNAs in a plethora of bacteria (2), relatively little is known about the regulatory roles these sRNAs play because it is not known which RNA species they pair with. Moreover, because of the limited and incomplete base-pairing that occurs between the sRNA and its target, computationally predicting targets for sRNAs is challenging. Here, we focus on approaches for the global identification of targets of sRNAs that function by base-pairing with other RNA species. Our intention is not to provide an exhaustive review of all the elegant approaches that are available for doing this, but instead to highlight two related genome-wide approaches, one of which has seen widespread adoption across the

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Address correspondence to Simon L. Dove, simon.dove@childrens.harvard.edu, or Sahar Melamed, sahar.melamed@mail.huji.ac.il.

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field, and another that shows particular promise. These approaches essentially involve trapping paired RNA species on an RNA chaperone to identify all chaperone-bound sRNAs together with their target RNAs at once.

RIL-seq: LINKING sRNAs TO TARGET RNAs

RIL-seq stands for RNA Interaction by ligation and sequencing and is conceptually similar to the ligation-based approaches developed for identifying the targets of miRNAs in eukaryotes (6, 7). RIL-seq was first used to identify RNA–RNA interactions occurring on Hfq in *Escherichia coli*, with a focus on those involving sRNAs and their mRNA targets (8). In this approach, cells expressing an epitope-tagged version of Hfq are first irradiated with UV to cross-link sRNAs together with their paired targets on individual molecules of Hfq (Fig. 1). Next, cells are lysed, and Hfq, together with any associated RNA, is immunoprecipitated using an antibody against the tagged Hfq. The RNAs bound to Hfq are then trimmed with a ribonuclease which facilitates subsequent ligation by generating RNAs with monophosphates at their 5′ ends. Paired RNA species trapped on Hfq are then linked together *in vitro* with RNA ligase (Fig. 1) (8). The resulting chimeric RNA species often correspond to an sRNA ligated to its mRNA target and are identified by high-throughput sequencing. A dedicated bioinformatic pipeline is then used to identify chimeric RNA molecules by detecting sequencing fragments whose ends map to two distinct, noncontiguous genomic loci, indicative of RNA–RNA ligation events. Following alignment, candidate chimeras are subjected to stringent filtering and threshold criteria, such as minimum read support and enrichment over random ligation, to eliminate mapping artifacts and low-confidence events, thereby retaining only statistically significant and biologically meaningful RNA–RNA interactions (8, 9). Importantly, the UV cross-linking step in RIL-seq helps preserve Hfq–RNA complexes present *in vivo*, limiting RNA exchange after cell lysis and improving the fidelity of captured RNA–RNA interaction pairs (8).

The first RIL-seq study enabled the identification of targets for over a hundred Hfq-bound sRNAs in cells of *E. coli* grown under a variety of conditions (8). This study recapitulated most of the RNA–RNA interactome in *E. coli* that was known at that time and revealed many new sRNAs and targets. Indeed, RIL-seq, together with the related approach CLASH (10), provides a snapshot of all the detectable interactions involving Hfq-bound sRNAs at any point in time. The information obtained not only provides important insights into the potential regulatory roles of sRNAs but also points to where the base-pairing between an sRNA and its target occurs on the target transcript (5, 11). This can help prioritize follow-up studies to determine the extent to which a given sRNA exerts a regulatory effect on a particular target. RIL-seq can also provide clues as to the possible regulatory mechanisms sRNAs employ. Although many sRNAs pair with their target transcripts close to or overlapping the Shine-Dalgarno sequence, RIL-seq data revealed that sRNAs can pair with an mRNA in the body of the transcript (i.e., internal to coding sequence (CDS)). This latter observation has led to the discovery that certain sRNAs can exert regulatory effects on protein activity by pairing deep within the CDS of an mRNA and potentially influencing the rate of translation (12).

Although we highlight the utility of RIL-seq in identifying mRNA species targeted by sRNAs, the approach also provides information on interactions between any RNA species that are occurring on a particular RNA chaperone such as Hfq, including both sRNA–sRNA interactions (see, for example, references 13, 14) and mRNA–mRNA interactions (15). In relation to the former, these sponging interactions between sRNAs on Hfq can modulate the availability or abundance of a given sRNA, thus modulating its activity, serving as an additional layer of regulation (16).

Since its initial use, RIL-seq has not only expanded our understanding of Hfq-bound sRNAs in *E. coli* under diverse conditions (17, 18), but has also been used successfully to identify the targets of Hfq-bound sRNAs in a variety of important environmental and pathogenic bacteria including enteropathogenic *E. coli* (19), *Salmonella enterica* (14), *Vibrio cholerae* (13), *Clostridium difficile* (20), *Pseudomonas aeruginosa* (21), *Klebsiella*

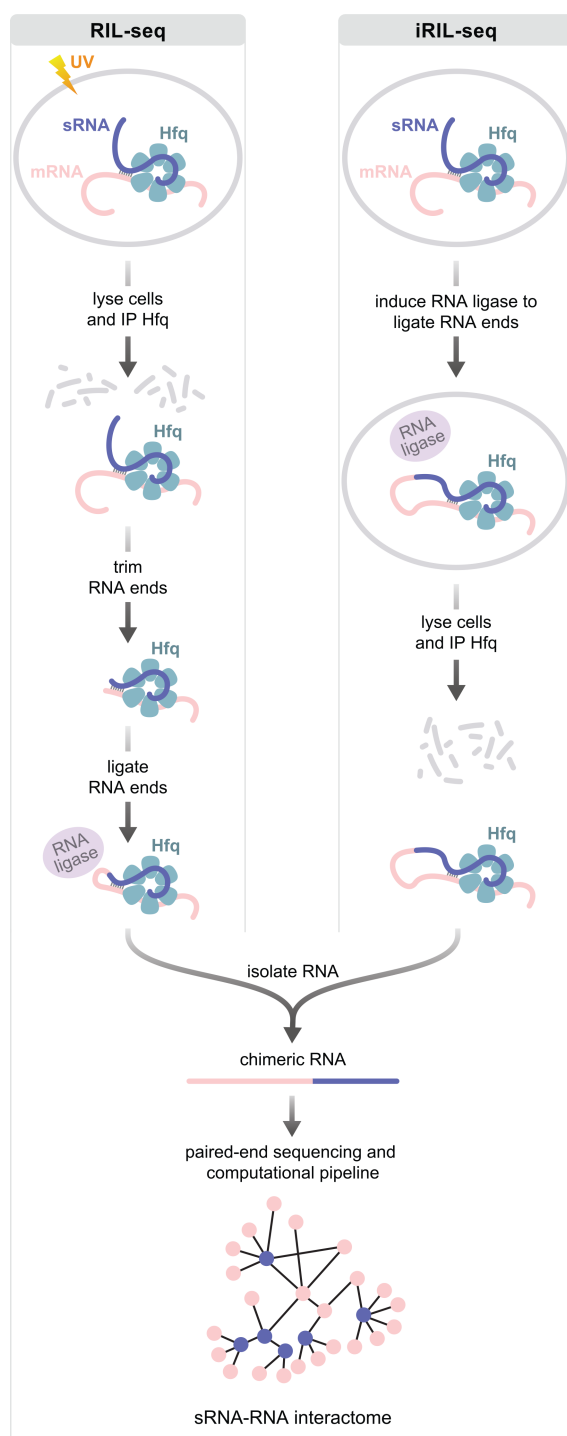


FIG 1 Schematic of RIL-seq (left) and iRIL-seq (right) approaches. Note that although mRNA species are depicted as paired with sRNAs on Hfq, other paired RNA species can be captured on Hfq using these approaches.

pneumoniae (22, 23), *Caulobacter crescentus* (24), *Bordetella pertussis* (25), and *Acinetobacter baumannii* (26). Many of these studies involve bacteria in which little was known about sRNA-mediated regulation beforehand and provide important resources to those interested in whether a gene or subset of genes in a particular organism might be subject to control at the post-transcriptional level by one or more sRNAs. Key specific insights gained from many of these studies are reviewed elsewhere (5). Recently, RIL-seq

has been applied to identify sRNA interaction networks occurring on Hfq in bacterial cells during prophage induction or phage infection, revealing roles played not only by sRNAs of the bacterial host, but also those of the phage, during these interactions (15, 27, 28).

Besides Hfq, RIL-seq can also be applied to the identification of RNA–RNA interactions occurring on another prominent RNA chaperone, ProQ, as was demonstrated in both *E. coli* and *V. cholerae* (29, 30). These studies led to the appreciation that ProQ can not only promote base-pairing between sRNAs and target mRNAs but also facilitate the pairing between different sRNA species (31). Applying RIL-seq to RNA-binding proteins other than Hfq or ProQ may reveal previously unrecognized roles for RNA-binding proteins as well as for sRNAs in bacterial regulation.

FROM RIL-seq TO iRIL-seq: EXPANDING THE RNA–RNA INTERACTION TOOLKIT

The second genome-wide approach we highlight is referred to as intracellular RIL-seq (iRIL-seq) (32). This approach involves expressing an RNA ligase inside cells to ligate base-paired RNA species together (Fig. 1). In this regard, it resembles the GRIL-seq approach developed originally for identifying the targets of individual sRNA species (33) and requires a 5′ monophosphate, typically present on processed transcripts, on either the sRNA or its target to allow ligation. Following induction of the RNA ligase, cells are harvested, then lysed, and Hfq is immunoprecipitated to enable the identification of those chimeric transcripts bound to Hfq by high-throughput sequencing (Fig. 1) (32). Thus, unlike RIL-seq, iRIL-seq does not employ a UV cross-linking step, nor does it involve trimming of RNA species with ribonuclease following the IP of Hfq. On the one hand, with fewer sample processing steps, iRIL-seq may offer certain advantages over the standard RIL-seq approach. On the other hand, ectopic expression of the RNA ligase in iRIL-seq can interfere with cell growth and introduce stress (32–35), which could, in principle, influence which sRNAs are produced by the cell under a particular growth condition. Additionally, because RNA ligation in iRIL-seq occurs *in vivo*, the method presumably preferentially captures RNAs that are processed *in vivo*, resulting in a subset of RNA–RNA interactions within the broader regulatory network. Recent analyses in *E. coli* suggest that RIL-seq and iRIL-seq indeed produce comparable, albeit not identical results (34). All in all, RIL-seq and iRIL-seq can be viewed as complementary approaches, with RIL-seq enabling more comprehensive interactome mapping, while iRIL-seq provides a simpler, more streamlined experimental protocol that has been suggested might capture more transient interactions between sRNAs and their targets (34). It is worth emphasizing that both methods use the same RIL-seq computational pipeline (9, 32). To date, iRIL-seq has been used to identify RNA–RNA interactions occurring on Hfq (32, 34, 35), and it will be interesting to investigate how well it can capture interactions on other RNA chaperones, such as ProQ or ProQ-related proteins that harbor a FinO RNA-binding domain.

FUTURE CHALLENGES

Both RIL-seq and iRIL-seq have provided significant insights into the regulatory roles of sRNAs in a variety of pathogenic and environmental bacteria (5, 35) grown as monocultures *in vitro*. How sRNA interaction networks are shaped in polymicrobial communities, where interspecies interactions and altered physiological states may substantially remodel RNA interactomes, is an open question. On a related note, how RNA–RNA interaction networks are shaped throughout growth in more complex and structured environments, such as those of the host, remains largely unexplored. The complementary features of RIL-seq and iRIL-seq may help address this gap, with RIL-seq suited for probing dynamic network changes over time and iRIL-seq potentially capturing interactions determined by a specific biogeographical location, provided RNA ligase induction can be linked to occupancy of a specific niche. Nonetheless, studying RNA interactomes in such complex settings presents significant challenges, including technical constraints associated with the use of UV irradiation or inducible systems. For example, in iRIL-seq, limited inducer accessibility under certain conditions may affect

efficient expression of the RNA ligase. Despite these technical challenges, recent studies demonstrate that mapping sRNA interaction networks under host-associated environments is feasible using both of the approaches outlined here (36, 37).

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AUTHOR AFFILIATIONS

¹Division of Infectious Diseases, Boston Children's Hospital, Harvard Medical School, Boston, Massachusetts, USA

²Department of Microbiology and Molecular Genetics, Institute for Medical Research Israel-Canada, Faculty of Medicine, The Hebrew University of Jerusalem, Jerusalem, Israel

AUTHOR ORCIDS

Simon L. Dove  <http://orcid.org/0000-0002-3758-608X>

Sahar Melamed  <http://orcid.org/0000-0003-1360-1297>

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AUTHOR CONTRIBUTIONS

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REFERENCES

- Papenfort K, Melamed S. 2023. Small RNAs, large networks: posttranscriptional regulons in gram-negative bacteria. *Annu Rev Microbiol* 77:23–43. <https://doi.org/10.1146/annurev-micro-041320-025836>
- Hör J, Matera G, Vogel J, Gottesman S, Storz G. 2020. Trans-acting small RNAs and their effects on gene expression in *Escherichia coli* and *Salmonella enterica*. *EcoSal Plus* 9:ESP-0030-2019. <https://doi.org/10.1128/ecosalplus.esp-0030-2019>
- Updegrove TB, Zhang A, Storz G. 2016. Hfq: the flexible RNA matchmaker. *Curr Opin Microbiol* 30:133–138. <https://doi.org/10.1016/j.mib.2016.02.003>
- Adams PP, Storz G. 2020. Prevalence of small base-pairing RNAs derived from diverse genomic loci. *Biochim Biophys Acta Gene Regul Mech* 1863:194524. <https://doi.org/10.1016/j.bbagr.2020.194524>
- Silverman A, Melamed S. 2025. Biological insights from RNA-RNA interactomes in bacteria, as revealed by RIL-seq. *Methods Mol Biol* 2866:189–206. https://doi.org/10.1007/978-1-0716-4192-7_11
- Grosswendt S, Filipchuk A, Manzano M, Klironomos F, Schilling M, Herzog M, Gottwein E, Rajewsky N. 2014. Unambiguous identification of miRNA:target site interactions by different types of ligation reactions. *Mol Cell* 54:1042–1054. <https://doi.org/10.1016/j.molcel.2014.03.049>
- Helwak A, Kudla G, Dudnakova T, Tollervey D. 2013. Mapping the human miRNA interactome by CLASH reveals frequent noncanonical binding. *Cell* 153:654–665. <https://doi.org/10.1016/j.cell.2013.03.043>
- Melamed S, Peer A, Faigenbaum-Romm R, Gatt YE, Reiss N, Bar A, Altuvia Y, Argaman L, Margalit H. 2016. Global mapping of small RNA-target interactions in bacteria. *Mol Cell* 63:884–897. <https://doi.org/10.1016/j.molcel.2016.07.026>
- Melamed S, Faigenbaum-Romm R, Peer A, Reiss N, Shechter O, Bar A, Altuvia Y, Argaman L, Margalit H. 2018. Mapping the small RNA interactome in bacteria using RIL-seq. *Nat Protoc* 13:1–33. <https://doi.org/10.1038/nprot.2017.115>
- Iosub IA, van Nues RW, McKellar SW, Nieken KJ, Marchioreto M, Sy B, Tree JJ, Viero G, Granneman S. 2020. Hfq CLASH uncovers sRNA-target interaction networks linked to nutrient availability adaptation. *eLife* 9:e54655. <https://doi.org/10.7554/eLife.54655>

11. Siemers M, Lippegaus A, Papenfort K. 2024. ChimericFragments: computation, analysis and visualization of global RNA networks. *NAR Genom Bioinform* 6:lqae035. <https://doi.org/10.1093/nargab/lqae035>
12. Thongdee N, Alaniz MM, Samatova E, Zhong A, Esnault C, Zhang H, Dale RK, Rodnina MV, Storz G. 2025. Modulation of protein activity by small RNA base pairing internal to coding sequences. *Mol Cell* 85:1824–1837. <https://doi.org/10.1016/j.molcel.2025.03.014>
13. Huber M, Lippegaus A, Melamed S, Siemers M, Wucher BR, Hoyos M, Nadell C, Storz G, Papenfort K. 2022. An RNA sponge controls quorum sensing dynamics and biofilm formation in *Vibrio cholerae*. *Nat Commun* 13:7585. <https://doi.org/10.1038/s41467-022-35261-x>
14. Matera G, Altuvia Y, Gerovac M, El Mouali Y, Margalit H, Vogel J. 2022. Global RNA interactome of *Salmonella* discovers a 5' UTR sponge for the MicF small RNA that connects membrane permeability to transport capacity. *Mol Cell* 82:629–644. <https://doi.org/10.1016/j.molcel.2021.12.030>
15. Silverman A, Nashef R, Wasserman R, Noy T, Born S, Yao T, Geng Y, Rotbard H, Levkowitz A, Kaufman Y, Golding I, Melamed S. 2025. Phage-encoded small RNA hijacks host replication machinery to support the phage lytic cycle. *Mol Cell* 85:4678–4697. <https://doi.org/10.1016/j.molcel.2025.11.019>
16. Vogt LN, Fröhlich KS. 2025. Impact of small RNA sponges on regulatory RNA networks in bacteria. *Annu Rev Genet* 59:69–89. <https://doi.org/10.1146/annurev-genet-013125-091919>
17. Faigenbaum-Romm R, Reich A, Gatt YE, Barsheset M, Argaman L, Margalit H. 2020. Hierarchy in Hfq chaperon occupancy of small RNA targets plays a major role in their regulation. *Cell Rep* 30:3127–3138. <https://doi.org/10.1016/j.celrep.2020.02.016>
18. McQuail J, Matera G, Gräfenhan T, Bischler T, Haberkant P, Stein F, Vogel J, Wigneshweraraj S. 2024. Global Hfq-mediated RNA interactome of nitrogen starved *Escherichia coli* uncovers a conserved post-transcriptional regulatory axis required for optimal growth recovery. *Nucleic Acids Res* 52:2323–2339. <https://doi.org/10.1093/nar/gkad1211>
19. Pearl Mizrahi S, Elbaz N, Argaman L, Altuvia Y, Katsowich N, Socol Y, Bar A, Rosenshine I, Margalit H. 2021. The impact of Hfq-mediated sRNA-mRNA interactome on the virulence of enteropathogenic *Escherichia coli*. *Sci Adv* 7:eabi8228. <https://doi.org/10.1126/sciadv.abi8228>
20. Fuchs M, Lamm-Schmidt V, Lenče T, Sulzer J, Bublit A, Wackenreuter J, Gerovac M, Strowig T, Faber F. 2023. A network of small RNAs regulates sporulation initiation in *Clostridioides difficile*. *EMBO J* 42:e112858. <https://doi.org/10.15252/embj.2022112858>
21. Gebhardt MJ, Farland EA, Basu P, Macareno K, Melamed S, Dove SL. 2023. Hfq-licensed RNA-RNA interactome in *Pseudomonas aeruginosa* reveals a keystone sRNA. *Proc Natl Acad Sci USA* 120:e2218407120. <https://doi.org/10.1073/pnas.2218407120>
22. Goh KJ, Altuvia Y, Argaman L, Raz Y, Bar A, Lithgow T, Margalit H, Gan YH. 2024. RIL-seq reveals extensive involvement of small RNAs in virulence and capsule regulation in hypervirulent *Klebsiella pneumoniae*. *Nucleic Acids Res* 52:9119–9138. <https://doi.org/10.1093/nar/gkac440>
23. Ruhland E, Siemers M, Gerst R, Späth F, Vogt LN, Figge MT, Papenfort K, Fröhlich KS. 2024. The global RNA-RNA interactome of *Klebsiella pneumoniae* unveils a small RNA regulator of cell division. *Proc Natl Acad Sci USA* 121:e2317322121. <https://doi.org/10.1073/pnas.2317322121>
24. Vogt LN, Gomariz MV, Siemers M, Papenfort K, Fröhlich KS. 2025. An RNA sponge directs the transition from feast to famine in *Caulobacter crescentus*. *Nat Commun* 16:9478. <https://doi.org/10.1038/s41467-025-65274-1>
25. Kumar D, Beles M, Saha A, Procházková I, Dienstbier A, Držmíšek J, Čapek J, Čurnová I, Hot D, Petráčková D, Večerek B. 2025. Global analysis of the Hfq-mediated RNA interactome discovers a MicA homolog that affects the cytotoxicity, biofilm formation, and resistance to complement of *Bordetella pertussis*. *Nucleic Acids Res* 53:gkaf614. <https://doi.org/10.1093/nar/gkaf614>
26. Intorcia V, Daum MN, Cheng B, Dove SL, Gebhardt MJ. 2026. Hfq orchestrates a robust RNA-RNA interaction network in *Acinetobacter baumannii*. *mBio* 17:e03231–25. <https://doi.org/10.1128/mbio.03231-25>
27. Lippegaus A, Haycocks JRJ, O'Driscoll E, Sprenger M, Thriene K, Jung E-M, Siemers M, Krautwurst S, Grainger DC, Papenfort K. 2025. A 3'UTR-derived small RNA modulates the life-cycle of the cholera toxin-encoding filamentous phage, CTXφ. *bioRxiv*. <https://doi.org/10.64898/2025.12.02.691856>
28. Sprenger M, Siemers M, Krautwurst S, Papenfort K. 2024. Small RNAs direct attack and defense mechanisms in a quorum sensing phage and its host. *Cell Host Microbe* 32:727–738. <https://doi.org/10.1016/j.chom.2024.03.010>
29. Ghandour R, Devlitsarov D, Popp P, Melamed S, Huber M, Siemers M, Krüger T, Kniemeyer O, Klingl A, Brakhage AA, Erhardt M, Papenfort K. 2025. ProQ-associated small RNAs control motility in *Vibrio cholerae*. *Nucleic Acids Res* 53:gkac1283. <https://doi.org/10.1093/nar/gkac1283>
30. Melamed S, Adams PP, Zhang A, Zhang H, Storz G. 2020. RNA-RNA interactomes of ProQ and Hfq reveal overlapping and competing roles. *Mol Cell* 77:411–425. <https://doi.org/10.1016/j.molcel.2019.10.022>
31. Melamed S. 2020. New sequencing methodologies reveal interplay between multiple RNA-binding proteins and their RNAs. *Curr Genet* 66:713–717. <https://doi.org/10.1007/s00294-020-01066-y>
32. Liu F, Chen Z, Zhang S, Wu K, Bei C, Wang C, Chao Y. 2023. *In vivo* RNA interactome profiling reveals 3'UTR-processed small RNA targeting a central regulatory hub. *Nat Commun* 14:8106. <https://doi.org/10.1038/s41467-023-43632-1>
33. Han K, Tjaden B, Lory S. 2016. GRIL-seq provides a method for identifying direct targets of bacterial small regulatory RNA by *in vivo* proximity ligation. *Nat Microbiol* 2:16239. <https://doi.org/10.1038/nmicrobiol.2016.239>
34. Luo X, Zhang A, Esnault C, De Mets F, Adams PP, Dale RK, Storz G, Gottesman S. 2025. RNA-RNA interactome approaches provide *in vivo* evidence for a critical role of the Hfq rim face in sRNA-mRNA pairing. *Nucleic Acids Res* 53:gkaf1313. <https://doi.org/10.1093/nar/gkaf1313>
35. Wu K, Lin X, Lu Y, Dong R, Jiang H, Svensson SL, Zheng J, Shen N, Camilli A, Chao Y. 2024. RNA interactome of hypervirulent *Klebsiella pneumoniae* reveals a small RNA inhibitor of capsular mucoviscosity and virulence. *Nat Commun* 15:6946. <https://doi.org/10.1038/s41467-024-51213-z>
36. Kanda T, Liu F, Kooshapour H, Reichardt S, Wang M, Icyishaka P, Obana N, Westermann AJ, Chao Y, Miyakoshi M. 2025. 3'UTR-derived small RNA couples acid resistance to metabolic reprogramming of *Salmonella* within macrophages. *Nucleic Acids Res* 53:gkaf1371. <https://doi.org/10.1093/nar/gkaf1371>
37. Kooshapour H, Matera G, Venturini E, Metka L, Bischler T, Vogel J, Westermann AJ. 2025. Intramacrophage RIL-seq uncovers an RNA antagonist of the *Salmonella* virulence-associated small RNA PinT. *Nucleic Acids Res* 53:gkaf1364. <https://doi.org/10.1093/nar/gkaf1364>