BEILSTEIN JOURNAL OF ORGANIC CHEMISTRY

Synthesis of electrophile-tethered $preQ_1$ analogs for covalent attachment to $preQ_1$ RNA

Laurin Flemmich and Ronald Micura*

Full Research Paper

Open Access

Address:
Institute of Organic Chemistry, Center for Molecular Biosciences Innsbruck (CMBI), Innrain 80-82, 6020 Innsbruck, Austria

Email:
Ronald Micura* - ronald.micura@uibk.ac.at

* Corresponding author

Keywords:
deazapurines; heterocycles; pyrrolopyrimidines; queuosine; riboswitches; ribozymes; RNA alkylation; RNA labelling

Beilstein J. Org. Chem. **2025,** *21,* 483–489.
https://doi.org/10.3762/bjoc.21.35

Received: 06 December 2024
Accepted: 17 February 2025
Published: 04 March 2025

Associate Editor: K. N. Allen

© 2025 Flemmich and Micura; licensee Beilstein-Institut.
License and terms: see end of document.

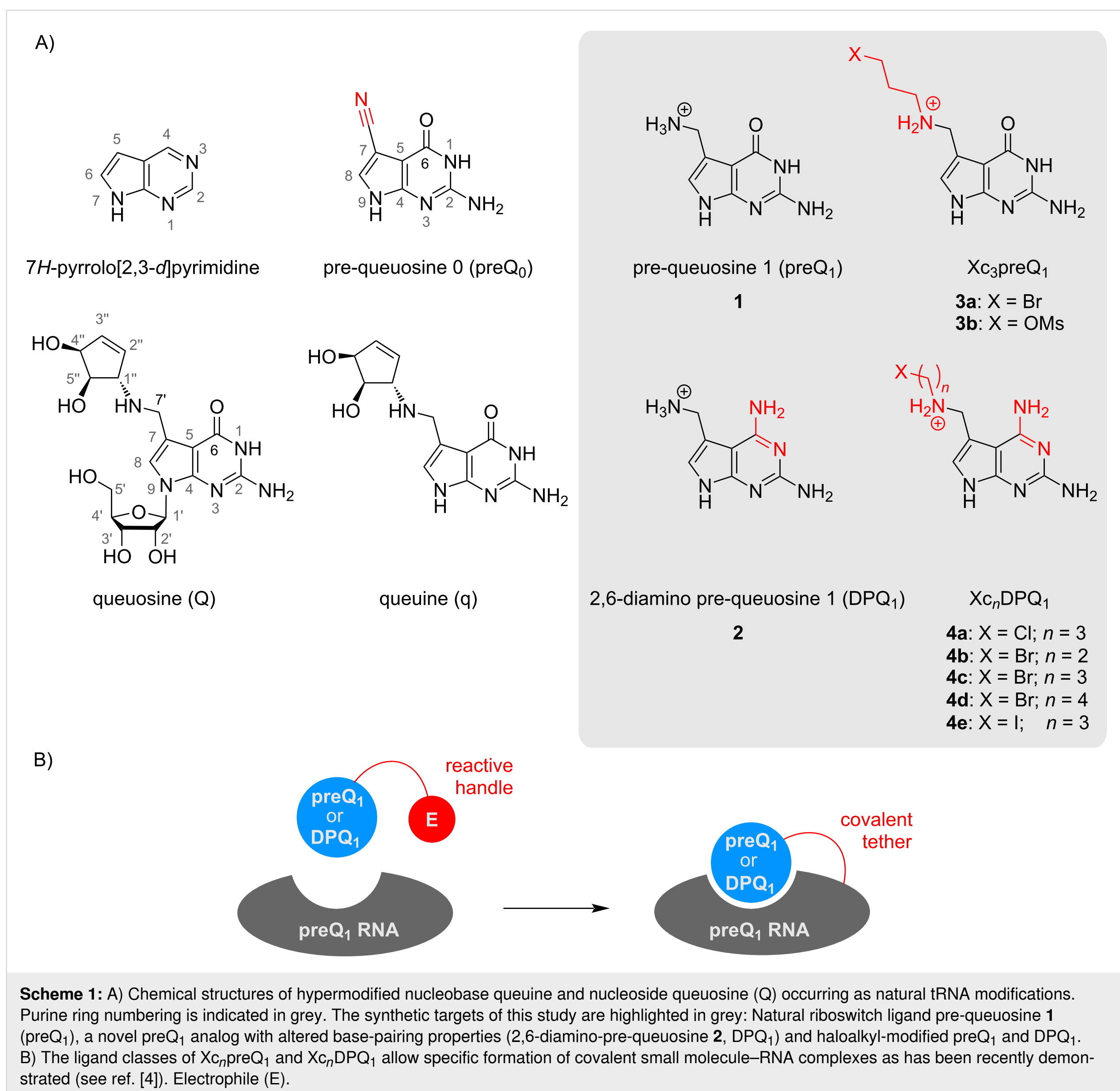
Abstract

The $preQ_1$ cIass-I riboswitch aptamer can utilize 7-aminomethyl-7-deazaguanine ($preQ_1$) ligands that are equipped with an electrophilic handle for the covalent attachment of the ligand to the RNA. The simplicity of the underlying design of irreversibly bound ligand–RNA complexes has provided a new impetus in the fields of covalent RNA labeling and RNA drugging. Here, we present short and robust synthetic routes for such reactive $preQ_1$ and (2,6-diamino-7-aminomethyl-7-deazapurine) DPQ_1 ligands. The readily accessible key intermediates of $preQ_0$ and DPQ_0 (both bearing a nitrile moiety instead of the aminomethyl group) were reduced to the corresponding 7-formyl-7-deazapurine counterparts. These readily undergo reductive amination to form the hydroxyalkyl handles, which were further converted to the haloalkyl or mesyloxyalkyl-modified target compounds. In addition, we report hydrogenation conditions for $preQ_0$ and DPQ_0 that allow for cleaner and faster access to $preQ_1$ compared to existing routes and provide the novel compound DPQ_1.

Introduction

Pre-queuosine 1 ($preQ_1$) is a biosynthetic precursor of the hypermodified nucleoside queuosine (Q) that is found in the wobble position of bacterial as well as eukaryotic aspartyl-, asparaginyl-, histidyl- and tyrosyl-tRNA isoacceptors bearing the $G_{34}U_{35}N_{36}$ anticodon motif [1]. Like other tRNA anticodon modifications, queuosine has been shown to increase translational fidelity and efficiency [2]. Structurally, queuosine and $preQ_1$ (compound **1**, Scheme 1A) belong to the 7-deazapurine family, which contain a pyrrolo[2,3-*d*]pyrimidine core. A rich pool of natural 7-deazapurine products has been identified, often (apparently) sharing a common biosynthetic pathway. Their functions are diverse; while some have been identified as having antifungal or antibiotic properties, others expand the chemical diversity and thus the functional sophistication of ribonucleic acids, as in the case of Q [3].

In most bacteria, Q biosynthesis is tightly regulated by riboswitches, which are highly structured RNA elements locat-



Scheme 1: A) Chemical structures of hypermodified nucleobase queuine and nucleoside queuosine (Q) occurring as natural tRNA modifications. Purine ring numbering is indicated in grey. The synthetic targets of this study are highlighted in grey: Natural riboswitch ligand pre-queuosine **1** ($preQ_1$), a novel $preQ_1$ analog with altered base-pairing properties (2,6-diamino-pre-queuosine **2**, DPQ_1) and haloalkyl-modified $preQ_1$ and DPQ_1. B) The ligand classes of $Xc_n preQ_1$ and $Xc_n DPQ_1$ allow specific formation of covalent small molecule–RNA complexes as has been recently demonstrated (see ref. [4]). Electrophile (E).

ed mostly in the 5'-leader of messenger RNA. $PreQ_1$ riboswitches sense the cellular concentration of $preQ_1$ and regulate the expression of downstream located genes associated with the biosynthesis or transport of Q in a feedback-like manner. Binding of $PreQ_1$ to the mRNA causes the riboswitch to commit an altered folding pathway, which affects the transcription or translation of the mRNA and results in altered transcript and/or protein levels [5].

In recent years, there has been a growing interest in the modification of $preQ_1$. $preQ_1$ derivatives, $preQ_1$ analogs and mimics of $preQ_1$ have greatly expanded our understanding of $preQ_1$-binding biomolecules, such as riboswitches [6-8] or the queuosine biosynthetic enzyme machinery [9-11]. Recently, even the self-methylation activity of a $preQ_1$ riboswitch has been discovered with a methylated $preQ_1$ derivative acting as a ribozyme cofactor [12]. Moreover, these analogs have found utility in several biotechnological applications, including the identification of queuosinylation sites in cellular RNA [13], RNA and DNA labeling [14,15], and mRNA photocaging [16]. The latter applications rely on the promiscuity of the tRNA-modifying enzyme *tRNA-guanine transglycosylase* (TGT), which can incorporate functionalized $preQ_1$ congeners into oligonucleotide strands at specific recognition sites.

In addition, the potential of modified $preQ_1$ for protein enzyme-independent RNA labeling has also been demonstrated [12,17]. In a recent study [4], sequence-specific RNA–small molecule

crosslinking (Scheme 1B) was achieved in vitro and in living cells using rationally designed electrophile-tethered derivatives of $preQ_1$ (**1**) and its Watson–Crick diamino-faced counterpart DPQ_1 (**2**, Scheme 1A). These ligands (compound classes **3** and **4**, Scheme 1A) were tailored to target a conserved guanine nucleobase within a natural $preQ_1$-binding mRNA domain, namely the $preQ_1$ class-I riboswitch ($preQ_1$-I) from *Thermoanaerobacter tengcongensis*. By rigorously analyzing the high-resolution structures available for this ligand–RNA complex, the approach exploits the natural, sequence-inherent reactivity hotspots of RNA and thus avoids the use of highly electrophilic warheads otherwise typically employed in RNA-small molecule crosslinking [18-21]. Instead, primary alkyl halides (or mesylates, Scheme 1, in particular compounds **3a**, **3b** and **4c**) were found to be potent yet mild alkylators that minimize off-target reactivity, while providing reasonably fast labeling kinetics and up to quantitative conversion under quasi-physiological conditions [4].

Obviously, the rapid dissemination and widespread acceptance of such labeling methods depend on fast and simple access to the small molecule probes. Here, we report efficient synthetic routes to haloalkyl- and mesylate-modified $preQ_1$ **3a** and **3b**, the corresponding variants with different Watson–Crick face of DPQ_1 (**4a–e**), as well as to the nucleobase precursors $preQ_1$ (**1**) and DPQ_1 (**2**), respectively.

Results and Discussion

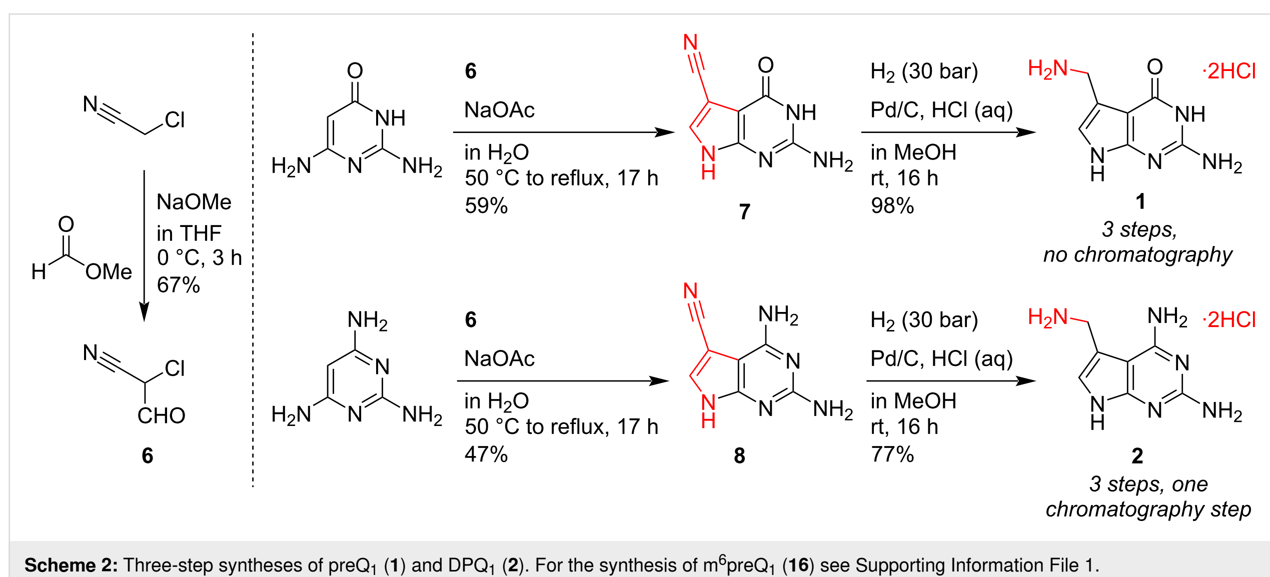
Synthesis of $preQ_1$ and DPQ_1

Several synthetic strategies towards $preQ_1$ and its derivatives have been reported [22-26]. Among these reports, various silylation and protection strategies have been employed to address the poor solubility of $preQ_1$ (and analogs) in organic solvents [9,22,23,25-29]. Herein, we report an optimized three-step protocol, free of protecting groups and time-consuming purification steps, that provides $preQ_1$ in 43% overall yield with a purity of >98% (Scheme 2). The approach is based on the cyclocondensation reaction between 2-chloro-3-cyanopropan-1-al (**6**) (itself obtained from chloroacetonitrile and methyl formate) and 2,6-diaminopyrimidin-4(*3H*)-one to afford $preQ_0$ (**7**), as originally reported by Townsend et al. [30]. The next step, namely the reduction of the nitrile moiety by hydrogenation is critical and notoriously difficult due to the low reactivity of this group in $preQ_0$ [26]. We solved this problem by applying strongly acidic protic conditions [31] together with a 7-fold increase in hydrogenation pressure (30 bar); this resulted in an almost quantitative conversion and pure $preQ_1$ (**1**) in the form of its dihydrochloride salt which was isolated after a simple filtration step.

Using the same approach, we were able to prepare the novel 2,6-diamino $preQ_1$ analog **2** (DPQ_1) by hydrogenation of **8** (DPQ_0 [32]) (Scheme 2). In this case, however, a final purification step (by reversed-phase chromatography) was required. Notably, using the hydrogenation conditions described here, we were also able to streamline our previously reported 7-step synthesis of O^6-methyl $preQ_1$ (**16**, m^6preQ_1) [28]. The direct reduction of the precursor O^6-methyl $preQ_0$ (**15**, m^6preQ_0) was possible, eliminating the need for the previously introduced protection/solubility concept, which shortened the synthetic route to only four steps (Supporting Information File 1).

Synthesis of $preQ_1$ and DPQ_1 derivatives with electrophilic handles

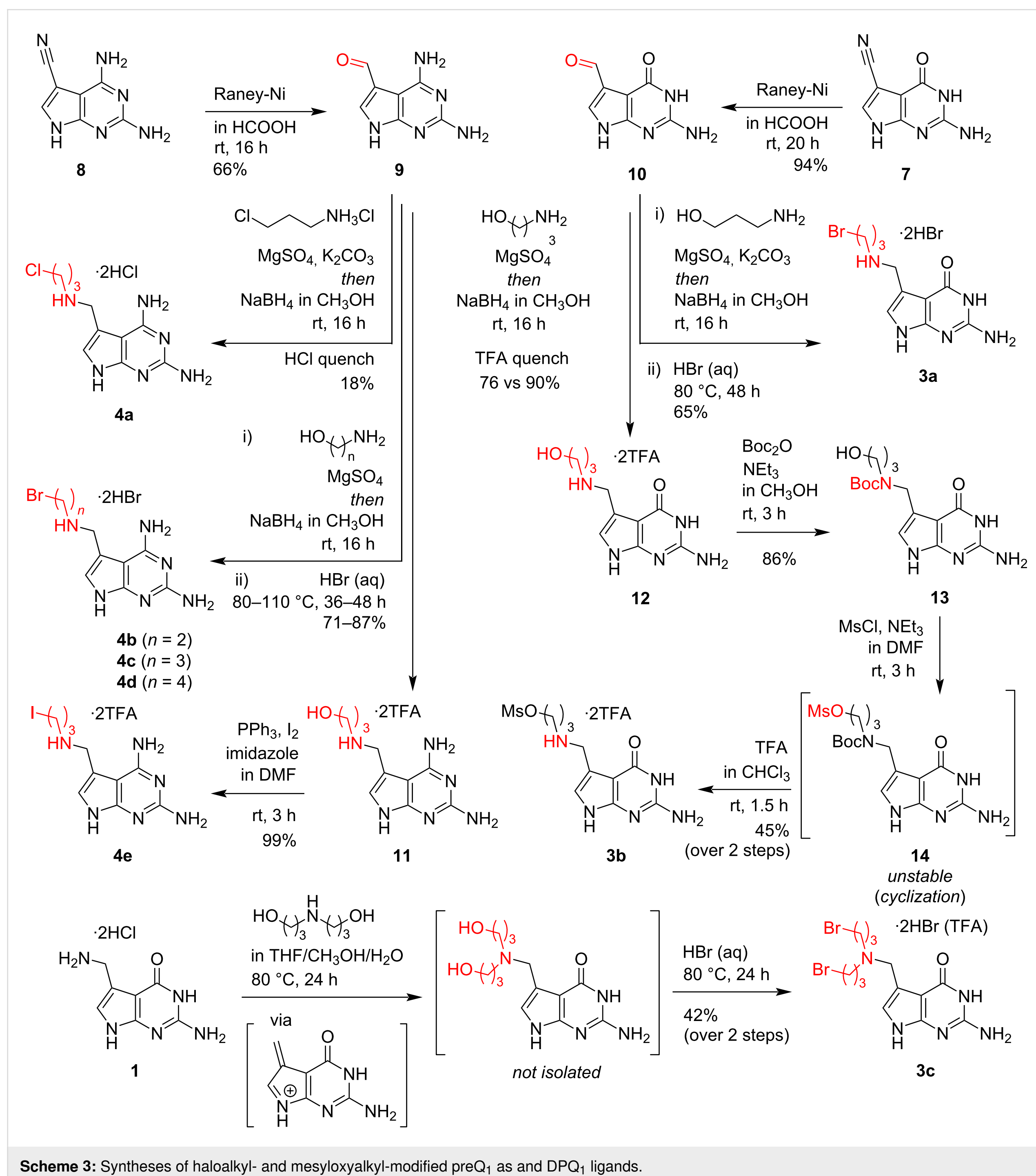
For the synthesis of haloalkyl- and mesyloxyalkyl-modified $preQ_1$ and DPQ_1 ligands **3a**,**b** and **4a–e** (for target structures see



Scheme 2: Three-step syntheses of $preQ_1$ (**1**) and DPQ_1 (**2**). For the synthesis of m^6preQ_1 (**16**) see Supporting Information File 1.

Scheme 1), a divergent synthetic route was sought that provided flexibility with respect to linker length and nature of the electrophile. We thus identified aldehydes **9** and **10** as suitable branching points, which were easily derivatized to their aminomethyl-modified preQ$_1$ analogs by reductive amination (Scheme 3). Their syntheses by Raney-Ni reduction of nitriles **7** and **8**, previously described by Gangjee and co-workers [33], proceeded cleanly in our hands.

In the case of compound **4a**, the chloropropyl moiety was directly installed by reductive amination of **9** with 3-chloropropylamine hydrochloride under basic conditions. A two-step reaction sequence, however, was necessary to generate derivatives **4b–e** and **3a**. First, the alkyl handles bearing a primary hydroxy group were introduced and then converted into the electrophile of choice. More specifically, to furnish compounds **4b–d**, precursor **9** was treated with the corresponding amino



Scheme 3: Syntheses of haloalkyl- and mesyloxyalkyl-modified preQ$_1$ as and DPQ$_1$ ligands.

alcohols in the presence of a desiccant. The imines formed were subjected to mild reduction with methanolic sodium borohydride. Upon purification by reversed-phase chromatography using aqueous hydrobromic acid as eluent (0.5% in H_2O), a considerable fraction of the alcohols was already converted to the desired bromides **4c**,**d**. Only in the case of **4b**, no deoxygenative bromination was observed, and the alcohol intermediate was isolated in pure form. In both cases, quantitative bromination was achieved by heating the compounds in concentrated aqueous hydrobromic acid, which after evaporation afforded the pure compounds **4b–d**.

To generate iodide **4e**, alcohol **11** was isolated and subjected to Appel conditions in DMF, using elemental iodine as the halogen source. Notably, we were not able to efficiently generate the corresponding bromides with the same strategy.

The $preQ_1$ derivative **3a** was synthesized in a 2-step reaction sequence analogous to the DPQ_1 derivative **4b**, while four steps were required to obtain the corresponding mesylate **3b**. Similar to **11**, compound **12** was isolated as its trifluoroacetate salt. Selective Boc protection of the aliphatic amine gave **13**, which was selectively *O*-mesylated to give compound **14**. Compound **14** was found to slowly undergo intramolecular cyclization by displacement of the mesyl group to give a six-membered cyclic carbamate, a reactivity that has been described earlier [34]. Thus, care was taken to quickly isolate compound **14** and use it immediately in the next step. Deblocking of the secondary amine by treatment with trifluoroacetic acid afforded **3b** in almost quantitative yield.

The bis(3-bromopropyl)-modified ligand **3c** was generated by heating $preQ_1$ together with bis(3-hydroxypropyl)amine. It is noteworthy that the amine exchange reaction is thought to proceed via a purine methide intermediate [11]. Subsequent treatment of the diol with aqueous hydrobromic acid provided **3c**.

Conclusion

We have developed a divergent synthesis of 7-aminomethyl-7-deazapurines ($preQ_1$ and DPQ_1) with various electrophilic handles extending the aminomethyl moiety. These derivatives are capable of covalent tethering to the $preQ_1$-I RNA aptamer. This aptamer occurs naturally in mRNA riboswitches in bacteria and is involved in ligand-dependent gene regulation. Therefore, this riboswitch (like others) has become an attractive target for drug design.

To date, most known RNA–small molecule binders interact in a non-covalent manner. The compounds presented here are part of our research program to tailor non-covalent RNA–small molecule ligands to their covalent counterparts. While “covalent drugs” have become a leading principle in medicinal chemistry in the “protein world” [35,36] – approximately 30% of all FDA-approved drugs form a covalent bond with their target protein – this concept is underexplored in the field of RNA drugging [37]. Recent studies suggest that the validation of RNA–small molecule interactions [38-40], drug efficacy or the identification of off-target effects of approved drugs on the transcriptome [41,42] could greatly benefit from covalency. We believe that these exciting new research directions will be furthered by the efficient synthetic routes to covalent RNA binders presented here.

Experimental

General procedure for reductive aminations (compounds 3a, 4b–d, 11, and 12). Aldehyde **9** or **10** (50.0 mg, 282 µmol) was suspended in methanol (1.3 mL). Anhydrous magnesium sulfate (340 mg, 2.82 mmol, 10 equiv) and the respective amino alcohol (2.82 mmol, 10 equiv) were added. The mixture was sonicated for 30 minutes and subsequently stirred at room temperature for 16 h. After cooling to 0 °C, sodium borohydride (96.1 mg, 2.54 mmol, 9 equiv) was added in portions over the course of 1 h. The mixture was stirred for additional 2.5 h at room temperature. Afterwards, the volatiles were removed under reduced pressure and the residue was taken up in dilute aqueous acid (for composition see individual experiments in Supporting Information File 1; compounds **3a**, **4b–d**, **11**, and **12**). If insolubles were present after pH 1–2 was reached, the suspension was filtered. Purification is described in Supporting Information File 1 for the individual compounds **3a**, **4b–d**, **11**, and **12**.

Supporting Information

Supporting Information File 1

Experimental part, HPLC analysis of preQ1 and NMR spectra.
[https://www.beilstein-journals.org/bjoc/content/supplementary/1860-5397-21-35-S1.pdf]

Acknowledgements

We thank Clemens Eichler (University of Innsbruck) for synthetic contributions.

Funding

This work was funded in part by the Austrian Science Fund FWF [P31691, F8011-B] and the Austrian Research Promotion Agency FFG [West Austrian Bio NMR 858017].

Author Contributions

Laurin Flemmich: conceptualization; investigation; validation; visualization; writing – original draft. Ronald Micura: conceptualization; funding acquisition; supervision; validation; visualization; writing – review & editing.

ORCID® iDs

Ronald Micura - https://orcid.org/0000-0003-2661-6105

Data Availability Statement

All data that supports the findings of this study is available in the published article and/or the supporting information of this article.

References

1. Zallot, R.; Yuan, Y.; De Crécy-Lagard, V. *Biomolecules* **2017,** *7,* 12. doi:10.3390/biom7010012
2. Iwata-Reuyl, D. *Bioorg. Chem.* **2003,** *31,* 24–43. doi:10.1016/s0045-2068(02)00513-8
3. McCarty, R. M.; Bandarian, V. *Bioorg. Chem.* **2012,** *43,* 15–25. doi:10.1016/j.bioorg.2012.01.001
4. Bereiter, R.; Flemmich, L.; Nykiel, K.; Heel, S.; Geley, S.; Hanisch, M.; Eichler, C.; Breuker, K.; Lusser, A.; Micura, R. *Nat. Chem. Biol.* **2025,** in press. doi:10.1038/s41589-024-01801-3
5. Serganov, A.; Nudler, E. *Cell* **2013,** *152,* 17–24. doi:10.1016/j.cell.2012.12.024
6. Roth, A.; Winkler, W. C.; Regulski, E. E.; Lee, B. W. K.; Lim, J.; Jona, I.; Barrick, J. E.; Ritwik, A.; Kim, J. N.; Welz, R.; Iwata-Reuyl, D.; Breaker, R. R. *Nat. Struct. Mol. Biol.* **2007,** *14,* 308–317. doi:10.1038/nsmb1224
7. Wu, M.-C.; Lowe, P. T.; Robinson, C. J.; Vincent, H. A.; Dixon, N.; Leigh, J.; Micklefield, J. *J. Am. Chem. Soc.* **2015,** *137,* 9015–9021. doi:10.1021/jacs.5b03405
8. Neuner, E.; Frener, M.; Lusser, A.; Micura, R. *RNA Biol.* **2018,** *15,* 1376–1383. doi:10.1080/15476286.2018.1534526
9. Wilding, B.; Winkler, M.; Petschacher, B.; Kratzer, R.; Egger, S.; Steinkellner, G.; Lyskowski, A.; Nidetzky, B.; Gruber, K.; Klempier, N. *Chem. – Eur. J.* **2013,** *19,* 7007–7012. doi:10.1002/chem.201300163
10. Fergus, C.; Al-qasem, M.; Cotter, M.; McDonnell, C. M.; Sorrentino, E.; Chevot, F.; Hokamp, K.; Senge, M. O.; Southern, J. M.; Connon, S. J.; Kelly, V. P. *Nucleic Acids Res.* **2021,** *49,* 4877–4890. doi:10.1093/nar/gkab289
11. Akimoto, H.; Nomura, H.; Yoshida, M.; Shindo-Okada, N.; Hoshi, A.; Nishimura, S. *J. Med. Chem.* **1986,** *29,* 1749–1753. doi:10.1021/jm00159a031
12. Flemmich, L.; Heel, S.; Moreno, S.; Breuker, K.; Micura, R. *Nat. Commun.* **2021,** *12,* 3877. doi:10.1038/s41467-021-24193-7
13. Bessler, L.; Kaur, N.; Vogt, L.-M.; Flemmich, L.; Siebenaller, C.; Winz, M.-L.; Tuorto, F.; Micura, R.; Ehrenhofer-Murray, A. E.; Helm, M. *Nucleic Acids Res.* **2022,** *50,* 10785–10800. doi:10.1093/nar/gkac822
14. Tota, E. M.; Devaraj, N. K. *J. Am. Chem. Soc.* **2023,** *145,* 8099–8106. doi:10.1021/jacs.3c00861
15. Alexander, S. C.; Busby, K. N.; Cole, C. M.; Zhou, C. Y.; Devaraj, N. K. *J. Am. Chem. Soc.* **2015,** *137,* 12756–12759. doi:10.1021/jacs.5b07286
16. Zhang, D.; Zhou, C. Y.; Busby, K. N.; Alexander, S. C.; Devaraj, N. K. *Angew. Chem., Int. Ed.* **2018,** *57,* 2822–2826. doi:10.1002/anie.201710917
17. Balaratnam, S.; Rhodes, C.; Bume, D. D.; Connelly, C.; Lai, C. C.; Kelley, J. A.; Yazdani, K.; Homan, P. J.; Incarnato, D.; Numata, T.; Schneekloth, J. S., Jr. *Nat. Commun.* **2021,** *12,* 5856. doi:10.1038/s41467-021-25973-x
18. Krochmal, D.; Shao, Y.; Li, N.-S.; DasGupta, S.; Shelke, S. A.; Koirala, D.; Piccirilli, J. A. *Nat. Chem. Biol.* **2022,** *18,* 376–384. doi:10.1038/s41589-021-00950-z
19. Ameta, S.; Jäschke, A. *Chem. Sci.* **2013,** *4,* 957–964. doi:10.1039/c2sc21588h
20. Wilson, C.; Szostak, J. W. *Nature* **1995,** *374,* 777–782. doi:10.1038/374777a0
21. McDonald, R. I.; Guilinger, J. P.; Mukherji, S.; Curtis, E. A.; Lee, W. I.; Liu, D. R. *Nat. Chem. Biol.* **2014,** *10,* 1049–1054. doi:10.1038/nchembio.1655
22. Kondo, T.; Ohgi, T.; Goto, T. *Chem. Lett.* **1983,** *12,* 419–422. doi:10.1246/cl.1983.419
23. Akimoto, H.; Imamiya, E.; Hitaka, T.; Nomura, H.; Nishimura, S. *J. Chem. Soc., Perkin Trans. 1* **1988,** 1637–1644. doi:10.1039/p19880001637
24. Gerber, H.-D.; Klebe, G. *Org. Biomol. Chem.* **2012,** *10,* 8660–8668. doi:10.1039/c2ob26387d
25. Chen, Y.-C.; Brooks, A. F.; Goodenough-Lashua, D. M.; Kittendorf, J. D.; Showalter, H. D.; Garcia, G. A. *Nucleic Acids Res.* **2011,** *39,* 2834–2844. doi:10.1093/nar/gkq1188
26. Klepper, F.; Polborn, K.; Carell, T. *Helv. Chim. Acta* **2005,** *88,* 2610–2616. doi:10.1002/hlca.200590201
27. Brooks, A. F.; Garcia, G. A.; Showalter, H. D. H. *Tetrahedron Lett.* **2010,** *51,* 4163–4165. doi:10.1016/j.tetlet.2010.06.008
28. Flemmich, L.; Moreno, S.; Micura, R. *Beilstein J. Org. Chem.* **2021,** *17,* 2295–2301. doi:10.3762/bjoc.17.147
29. Anthony, N. G.; Baiget, J.; Berretta, G.; Boyd, M.; Breen, D.; Edwards, J.; Gamble, C.; Gray, A. I.; Harvey, A. L.; Hatziieremia, S.; Ho, K. H.; Huggan, J. K.; Lang, S.; Llona-Minguez, S.; Luo, J. L.; McIntosh, K.; Paul, A.; Plevin, R. J.; Robertson, M. N.; Scott, R.; Suckling, C. J.; Sutcliffe, O. B.; Young, L. C.; Mackay, S. P. *J. Med. Chem.* **2017,** *60,* 7043–7066. doi:10.1021/acs.jmedchem.7b00484
30. Migawa, M. T.; Hinkley, J. M.; Hoops, G. C.; Townsend, L. B. *Synth. Commun.* **1996,** *26,* 3317–3322. doi:10.1080/00397919608004641
31. Dincer, S.; Cetin, K. T.; Onay-Besikci, A.; Ölgen, S. *J. Enzyme Inhib. Med. Chem.* **2013,** *28,* 1080–1087. doi:10.3109/14756366.2012.715288
32. Zhu, G.; Liu, Z.; Xu, Y.; Mao, Z. *Heterocycles* **2008,** *75,* 1631. doi:10.3987/com-08-11323
33. Gangjee, A.; Mavandadi, F.; Queener, S. F.; McGuire, J. J. *J. Med. Chem.* **1995,** *38,* 2158–2165. doi:10.1021/jm00012a016
34. Lee, J. W.; Lee, J. H.; Son, H. J.; Choi, Y. K.; Yoon, G. J.; Park, M. H. *Synth. Commun.* **1996,** *26,* 83–88. doi:10.1080/00397919608003865
35. Sutanto, F.; Konstantinidou, M.; Dömling, A. *RSC Med. Chem.* **2020,** *11,* 876–884. doi:10.1039/d0md00154f
36. Singh, J.; Petter, R. C.; Baillie, T. A.; Whitty, A. *Nat. Rev. Drug Discovery* **2011,** *10,* 307–317. doi:10.1038/nrd3410
37. Connelly, C. M.; Moon, M. H.; Schneekloth, J. S., Jr. *Cell Chem. Biol.* **2016,** *23,* 1077–1090. doi:10.1016/j.chembiol.2016.05.021
38. Guan, L.; Disney, M. D. *Angew. Chem., Int. Ed.* **2013,** *52,* 10010–10013. doi:10.1002/anie.201301639
39. Childs-Disney, J. L.; Yang, X.; Gibaut, Q. M. R.; Tong, Y.; Batey, R. T.; Disney, M. D. *Nat. Rev. Drug Discovery* **2022,** *21,* 736–762. doi:10.1038/s41573-022-00521-4

40. Velagapudi, S. P.; Li, Y.; Disney, M. D. *Bioorg. Med. Chem. Lett.* **2019,** *29,* 1532–1536. doi:10.1016/j.bmcl.2019.04.001
41. Fang, L.; Velema, W. A.; Lee, Y.; Xiao, L.; Mohsen, M. G.; Kietrys, A. M.; Kool, E. T. *Nat. Chem.* **2023,** *15,* 1374–1383. doi:10.1038/s41557-023-01309-8
42. Fullenkamp, C. R.; Schneekloth, J. S., Jr. *Nat. Chem.* **2023,** *15,* 1329–1331. doi:10.1038/s41557-023-01330-x

License and Terms

This is an open access article licensed under the terms of the Beilstein-Institut Open Access License Agreement (https://www.beilstein-journals.org/bjoc/terms), which is identical to the Creative Commons Attribution 4.0 International License (https://creativecommons.org/licenses/by/4.0). The reuse of material under this license requires that the author(s), source and license are credited. Third-party material in this article could be subject to other licenses (typically indicated in the credit line), and in this case, users are required to obtain permission from the license holder to reuse the material.

The definitive version of this article is the electronic one which can be found at:
https://doi.org/10.3762/bjoc.21.35