HETEROCYCLES, Vol. 101, No. 2, 2020, pp. 621 - 630. © 2020 The Japan Institute of Heterocyclic Chemistry
Received, 26th July, 2019, Accepted, 1st October, 2019, Published online, 16th October, 2019
DOI: 10.3987/COM-19-S(F)53

N-GLYCOSYLATION REACTION OF *THIO*-GLYCOSIDE USING HYPERVALENT IODINE(III) REAGENT

Koji Morimoto,[a,b] Kana Yanase,[a] Takumi Ikeda,[a] Chihiro Uchikawa,[a] Yasuyuki Kita,[b]* and Tetsuya Kajimoto[a,b]*

[a] College of Pharmaceutical Sciences, Ritsumeikan University, 1-1-1 Nojihigashi, Kusatsu, Shiga, 525-8577, JAPAN. [b] Research Organization of Science and Technology, Ritsumeikan University, 1-1-1 Nojihigashi, Kusatsu, Shiga, 525-8577, JAPAN. *Corresponding author. Tel.: +81-77-561-5829; fax: +81-77-561-5829; e-mail: kita@ph.ritsumei.ac.jp; (Y. Kita); kajimoto@fc.ritsumei.ac.jp (T. Kajimoto).

Abstract – We discovered that the hypervalent iodine(III) reagent could mediate the *N*-glycosylation reaction using the *thio*-glycoside donors with triazoles. By using this method, glycosyl triazole could be easily synthesized under mild reaction conditions.

In Celebration of Professor Kaoru Fuji on His 80th Birthday

INTRODUCTION

Glycoconjugates and glycosyl derivatives having carbon-nitrogen (C-N) bonds at the anomeric position play important roles in a number of biological events, such as fertilization, immune response, and bacterial and viral infections.[1] Actually, a number of biological functions of *N*-glycans in the events was identified in these two decades and many inhibitors against glycosidases and glycosyltransferases having the C-N bond were newly developed.[2] Especially, in the latter case, some *N*-glycans are now utilized as clinical medicines.[3] Based on, *N*-glycosyl mimics of glycosides have become attractive targets for organic synthesis; however, the *N*-glycosylation reactions are not well established compared to those for the *O*-glycosylation.[4] Thus, an effective methodology for the C-N bond formation is quite useful for the construction of *N*-glycan derivatives and the libraries of mimics of the *N*-linked glycopeptides. The former could be inhibitors against glycosidases and glycosyltransferases, and the latter would be excellent tools to investigate the functions of glycoenzymes, which constructs and cleaves glycosyl bonds of *N*-glycans in the biosynthetic pathways.

Among the *N*-glycans, glycosyl azoles are most attractive molecules because they showed potent inhibitory activities against glycosidase and glycosyltransferases, while they were employed as biologically-inactive linkers in the study of neoglycoconjugates.[5] Moreover, some glycosyl triazoles have been used as sugar donors in glycosylation reactions.[6] Various types of *N*-linked glycosyl 1,2,3-triazoles have also been reported. [7]

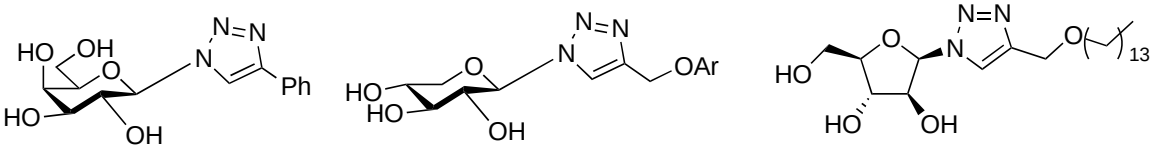
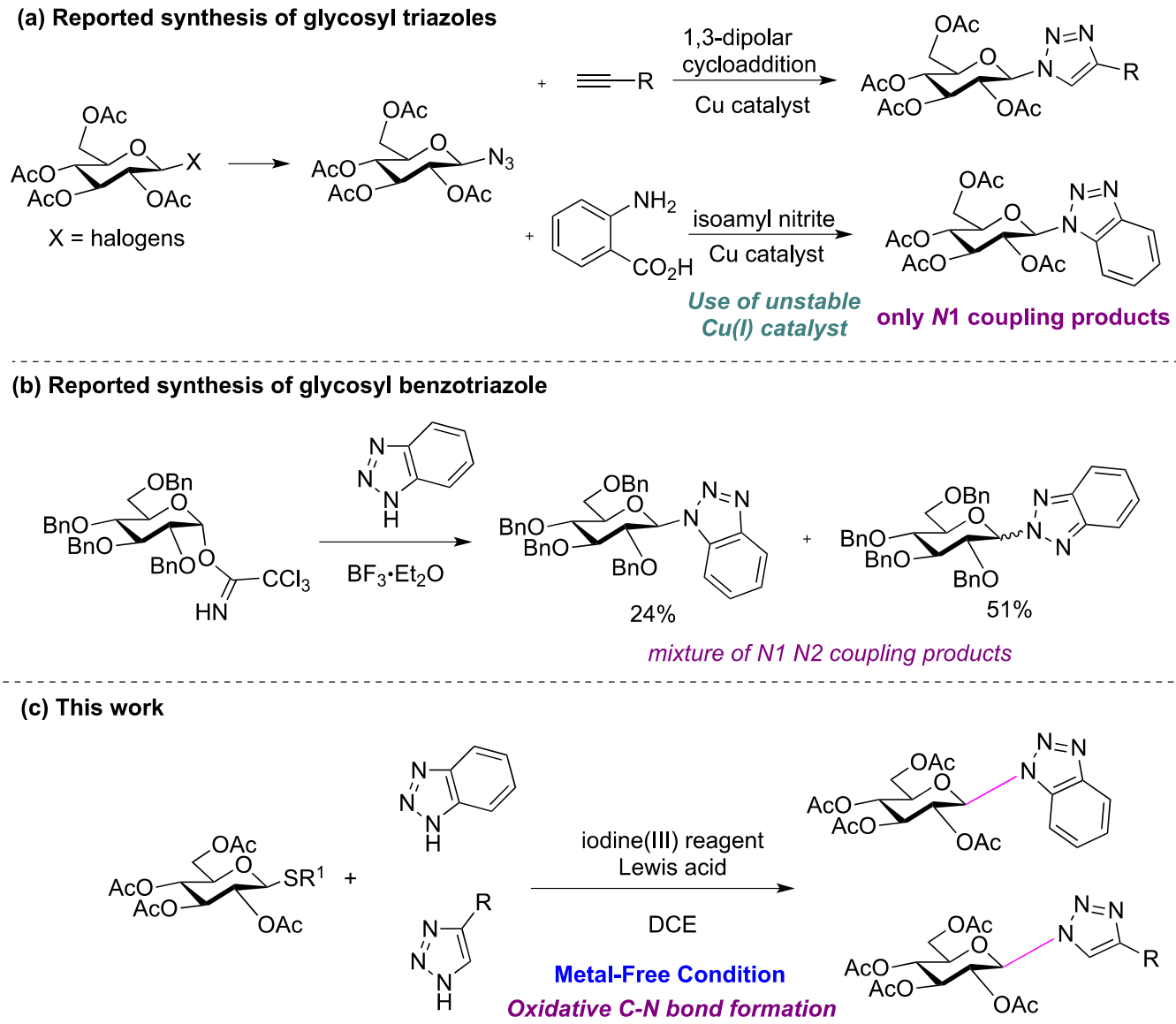


Figure 1. Utility of the glycosyl triazoles

Thus, various types of *N*-linked glycosyl 1,2,3-triazoles have already been reported, and most of them have been generally synthesized by the Huisgen [3 + 2] cycloaddition reaction, in which glycosyl azides and activated symmetrical alkynes reacted in the presence of the Cu(I) ion (Scheme 1a).[8] The CuI-catalyzed azide-terminal alkyne cycloaddition (CuAAC) reaction produced 1,4-disubstituted 1,2,3-triazoles with a high regioselectivity. Despite the usefulness of CuAAC, these reactions suffer from drawbacks, not only in a medicinal chemistry perspective but also often in the case of the materials sciences: (i) copper catalyst contamination of the product, and (ii) Cu(I) oxidation state instability, which requires an inert gas or the addition of an excess reducing agent.[9] Glycosyl benzotriazoles have been prepared by the glycosidation of 1,2,3-benzotriazole, resulting in mixtures of the 1- and 2-substituted regio-isomers (Scheme 1b).[10] To the best of our knowledge, glycosyl azoles are considered to be useful compounds, but their synthetic methods are limited to the Huisgen reaction. Therefore, the development of efficient methods for the glycosylation reaction of these glycosyl azoles is highly fascinating in organic synthesis.

The oxidation with the environmentally-benign hypervalent iodine(III) reagent has witnessed profound progress in organic chemistry.[11] Recently, we have been involved in the development of the hypervalent iodine(III)-mediated glycosylation leading to oligosaccharides.[12] During this glycosylation reaction, the hypervalent iodine reactant activated the sulfur atom of *thio*-glycoside and the glycosylation reaction efficiently proceeded. With this in mind, we envisioned that iodine(III) reagents could serve to promote the *N*-glycosylation to the corresponding glycosyl azoles **3**. In this communication, we report our preliminary investigation of the iodine(III)-mediated *N*-glycosylation of *thio*-glycosides to develop a convenient synthesis method of glycosyl azoles using the hypervalent iodine(III) reagent as an activator (Scheme 1c).



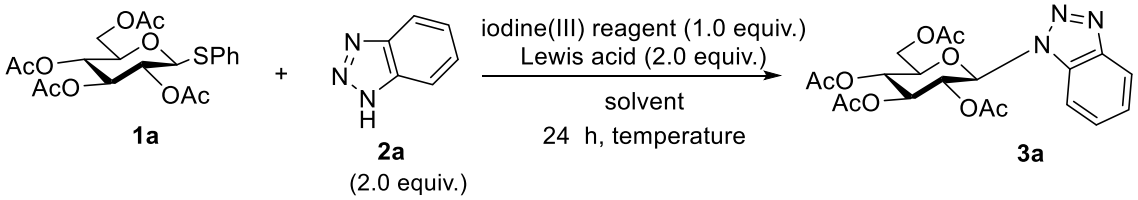
Scheme 1. Synthesis of glycosyl triazoles

RESULTS AND DISCUSSION

Initially, we examined the reaction conditions using *thio*-glycoside **1a** and 1,2,3-benzotriazole **2a** as model substrates (Table 1). Based on our previous research, our initial studies revealed that the combination of phenyliodine(III) bis(trifluoroacetate) (PIFA) with TfOH in CH_2Cl_2 could promote the glycosylation reaction at room temperature, affording the *N*1 coupling product **3a** in 46% yield. Meanwhile, we also tested some other solvents, such as acetonitrile and HFIP, but no better results were obtained (entries 2 and 3). Next, the use of TMSOTf instead of TfOH improved the yield up to 67% (entry 4). The treatment of the *thio*-glycoside with $BF_3 \cdot Et_2O$ showed the formation of **3a**, but the yields decreased to 31% (entry 5). To our delight, **1a** was easily converted to the desired product **3a** in 85%

yield using TMSOTf as an acid in dichloroethane (DCE) and at 80 ºC (entry 6). Switching to other iodine(III) reagents, i.e., phenyliodine(III) diacetate (PIDA) and PhI(OH)OTs, did not produce good results (entries 7 and 8). In all cases, a trace amount of the *N*2 coupling product was detected. When the reaction was performed with NBS used to activate the thioglycoside, 42% of the *N*1 coupling product was obtained and 22% of the *N*2 coupling product was also produced. In the case of NIS, only about 10% of the glycosylated product was obtained.

Table 1. Optimization of Reaction Conditions



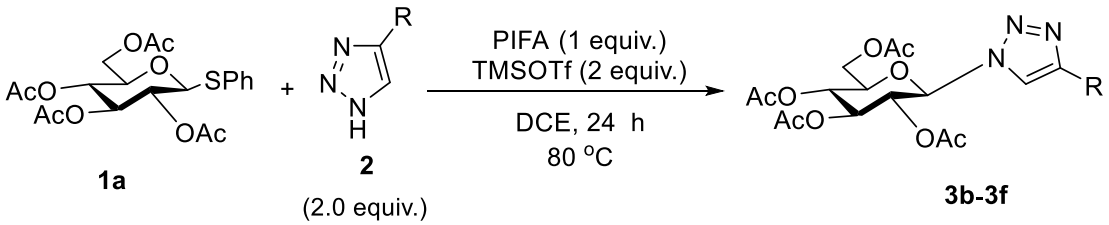
entry	iodine(III) reagent	acid	solvent (0.1 M)	temperature	yield (%)[a,b]
1	PIFA	TfOH	CH_2Cl_2	rt	46
2	//	//	MeCN	//	n.d.
3	//	//	$(CF_3)_2CHOH$ (HFIP)	//	n.d.
4	//	TMSOTf	CH_2Cl_2	//	67
5	//	$BF_3 \cdot Et_2O$	//	//	31
6	//	TMSOTf	$(CH_2)_2Cl_2$ (DCE)	80 ºC	85
7	PIDA	TMSOTf	//	//	46
8	PhI(OH)OTs	//	//	//	trace

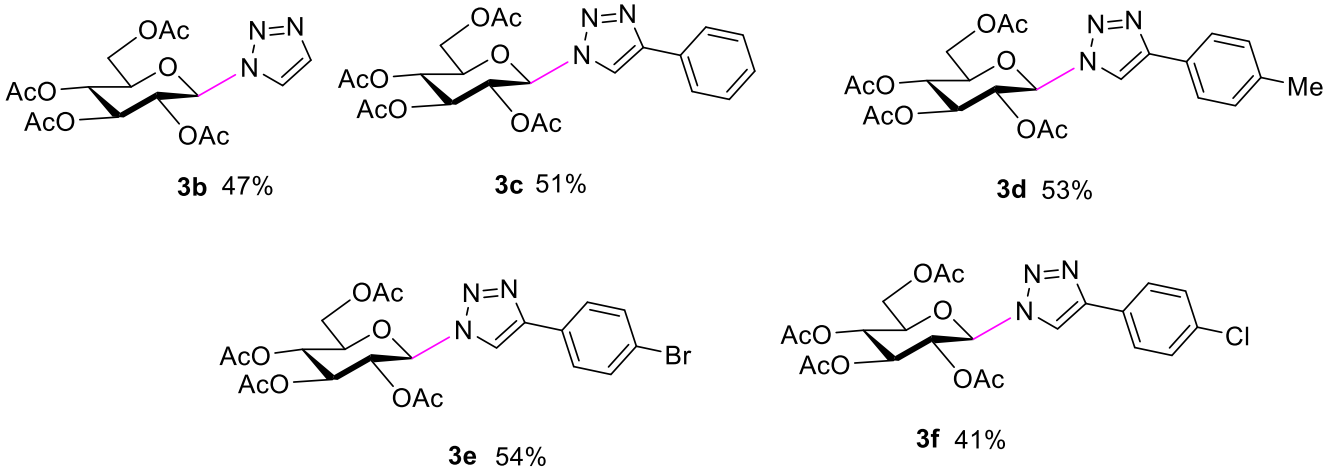
[a] Reaction conditions: under a nitrogen atmosphere, *thio*-glycoside **1a** (0.1 mmol), 1,2,3-benzotriazole **2a** (0.2 mmol), iodine(III) reagent (0.1 mmol), acid (0.2 mmol). [b] Isolated yield.

With these optimized condition in hand, we set out to explore the substrate scope of this glycosylation reaction. As shown in Table 2, the *thio*-glycoside **1a** with triazoles **2** gave the desired coupling products **3b**-**3f** in good yields. The 1,2,3-triazole **2b** smoothly occurred producing the glycosylation product **3b** in

satisfactory yield. Furthermore, 4-phenyl-1*H*-1,2,3-triazole **2c** afforded the corresponding products **3c** and **3d** in 51−53% yields. The halogen-substituted 4-aryl-1*H*-1,2,3-triazoles afforded the corresponding glycosyl azoles **3e** and **3f** in moderate yields.

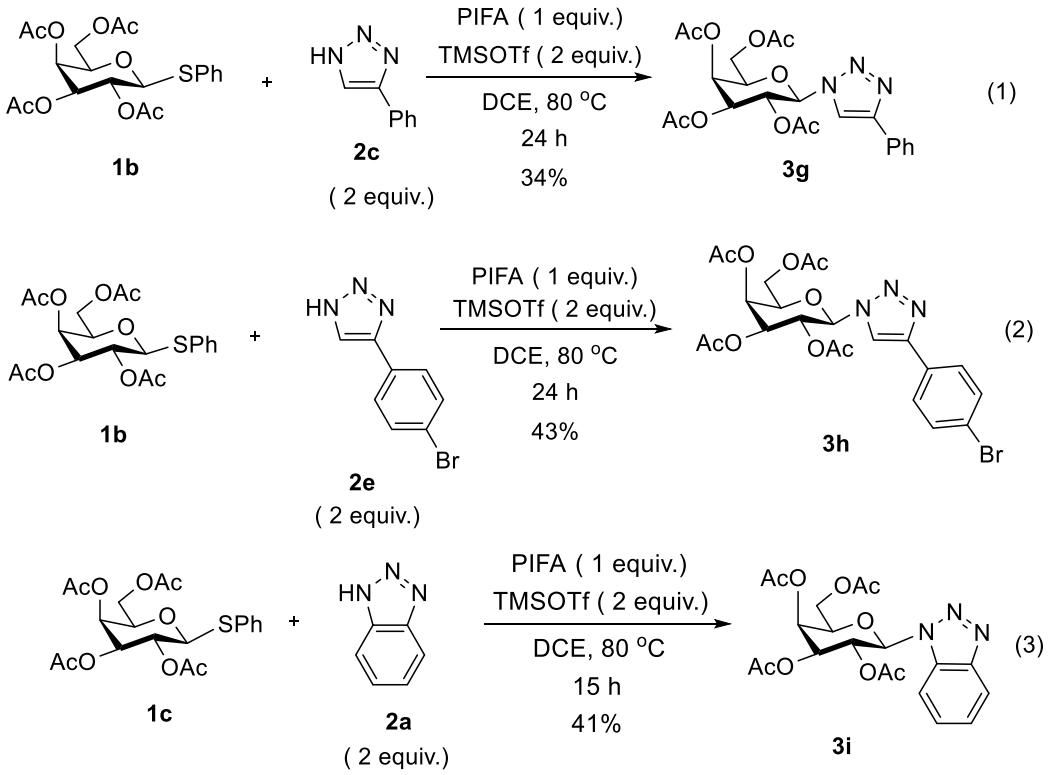
Table 2. Scope of substrates[a,b]





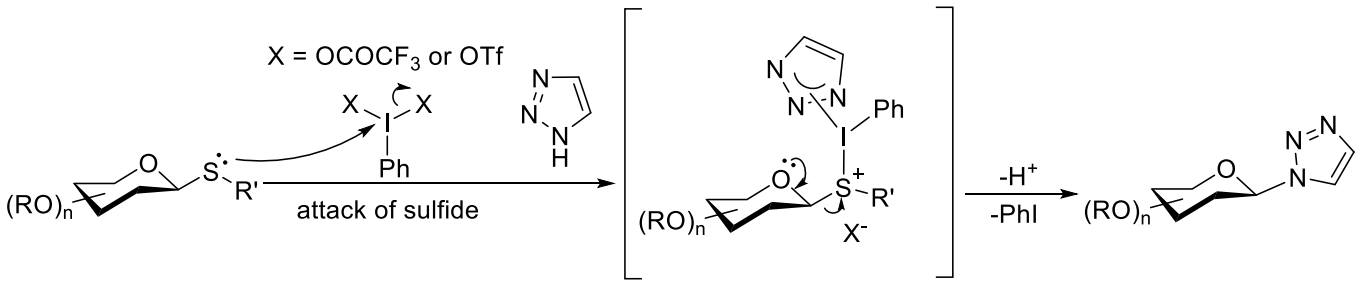
[a] Reaction conditions: under a nitrogen atmosphere, *thio*-glycoside **1a** (0.1 mmol), 1*H*-1,2,3-triazole **2** (0.2 mmol), PIFA (0.1 mmol), TMSOTf (0.2 mmol). [b] Isolated yield.

Encouraged by these results, we next examined the glycosylation reaction of the *thio*-glycosides derived from galactose **1b** with 4-phenyl-1*H*-1,2,3-triazole **2c**. (Scheme 2). For the acetyl-protected galactose **1b**, the reaction proceeded to produce the glycosylated product **3g** in 34% yield (eq. 1). The bromo-substituted 4-aryl-1*H*-1,2,3-triazole **2e** afforded the corresponding glycosyl azole **3h** in moderate yield (eq. 2). The treatment of 1,2,3-benzotriazole **2a** formed the desired product **3i** in 41% yield (eq. 3). A trace amount of the *N*2 coupling product was detected. In our reaction condition, *N*1 glycosylated product obtained in good yields, though the reason for this high regioselectivity is yet unclear.



Scheme 2. Glycosylation of galactose derivatives

Although the exact reaction mechanism is not clear, the proposed reaction mechanism is shown in Scheme 3. Initially, the sulfonium intermediate was formed by the reaction of the glycosyl donor with the hypervalent iodine(III) reagent. Next, it was assumed that ligand exchange occurred between iodine reagent and triazole. Subsequent elimination of iodobenzene and thiol followed by attack of *N*1 position of trizaole leads to the selective glycosylated product. This study on selectivity is ongoing now.



Scheme 3. Proposed reaction mechanism

In conclusion, we have developed a straightforward and efficient method for the synthesis of glycosyl azoles. In this reaction, when 1,2,3-benzotriazole was used, the *N*1 selective glycosylation reaction proceeded. This protocol offers a rapid approach to glycosyl azoles along with the achievement of a remarkable structural diversity. The process also features readily available starting materials, simple

operation and good scalability, and would be synthetically useful in organic synthesis.

EXPERIMENTAL

General Remarks; The ^{1}H-NMR and ^{13}C-NMR spectra were recorded by a JEOL JMN-400 spectrometer operating at 400 MHz in $CDCl_3$ at 25 ^{o}C with tetramethylsilane as the internal standard. Data are reported as follows: chemical shift in ppm (δ), integration, multiplicity (s = singlet, d = doublet, t = triplet, q = quartet, bs = broad singlet, m = multiplet), coupling constant (Hz). The mass spectra were obtained using a Shimadzu GCMS-QP 5000 instrument with ionization voltages of 70 eV. The high resolution mass spectra were performed by the Elemental Analysis Section of Osaka University. Column chromatography and TLC were carried out on Merck Silica gel 60 (230-400 mesh) and Merck Silica gel F254 plates (0.25 mm), respectively. The spots and bands were detected by UV irradiation (254, 365 nm).

Materials: $PhI(OCOCF_3)_2$ (PIFA), 1,2,3-triazole, 1,2,3-benzotriazole are commercially available and used as received. All other starting materials are commercially available. They were used without further purification.

General Procedure for the Glycosylation Reaction

To a stirred solution of phenyl 2,3,4,6-tetra-*O*-acetyl-1-*thio*-β-D-glucopyranoside **1a** and 1,2,3-benzotriazole **2a** (0.2 mmol, 2 equiv.) in DCE (2 mL, 0.05 M), TMSOTf (0.2 mmol, 2 equiv.) was added at room temperature. PIFA (0.1 mmol, 1 equiv) was subsequently added to the reaction mixture with stirring, then stirred for an additional 24 h at 80 ^{o}C, while the reaction was monitored by TLC. A saturated aqueous solution of sodium hydrogen carbonate was added to the mixture when the reaction was completed. The aqueous phase was extracted with CH_2Cl_2. The extract was dried over anhydrous Na_2SO_4, then evaporated to dryness. The crude residue was purified by column chromatography on silica gel (eluent: *n*-hexane/ CH_2Cl_2) to form the pure glycosylation product **3a** in 85% yield.

Characterization of the glycosylation products 3

1-(2,3,4,6-Tetra-*O*-acetyl-β-D-glucopyranosyl)-1*H*-benzo[*d*][1,2,3]triazole 3a[7]

^{1}H-NMR (400 MHz, $CDCl_3$): δ 1.77 (3H s), 2.04 (3H s), 2.09 (3H s), 2.12 (3H s), 4.10 (1H, ddd, *J* = 10.0, 4.8, 2.0 Hz, H5), 4.22 (1H, dd, *J* = 12.8, 2.4 Hz), 4.33 (1H, dd, *J* = 12.8, 4.8 Hz), 5.38 (1H, dd, *J* = 10.0, 9.4 Hz), 5.52 (1H, dd, *J* = 9.4, 9.4 Hz), 5.78 (1H, dd, *J* = 9.4, 9.4 Hz), 6.21 (1H, d, *J* = 9.6 Hz, H1), 7.43 (1H, dd, *J* = 8.0, 7.2 Hz), 7.57 (1H, dd, *J* = 8.0, 7.2 Hz), 7.73 (1H, d, *J* = 8.0 Hz), 8.08 (1H, d, *J* = 8.0 Hz). ^{13}C-NMR (100 MHz, $CDCl_3$): δ 20.1, 20.6, 20.6, 20.7, 61.6, 67.8, 69.2, 72.7, 75.0, 86.0, 101.6, 120.3, 124.8, 128.4, 131.7, 146.5, 168.6, 169.5, 170.15, 170.5 ppm.

1-(2,3,4,6-Tetra-*O*-acetyl-β-D-glucopyranosyl)-1*H*-1,2,3-triazole 3b[13]

^{1}H-NMR ($CDCl_3$, 400 MHz): δ 1.85 (3H s), 2.01 (3H s), 2.05 (3H s), 2.06 (3H s), 4.01 (1H, ddd, *J* = 10.0 Hz, H5), 4.14 (1H, dd, *J* = 2.0 Hz), 4.29 (1H, dd, *J* = 12.4, 4.8 Hz), 5.26-5.21 (1H, m), 5.47-5.39 (2H,

m), 5.93 (1H, d, *J* = 9.5 Hz, H1), 7.76 (1H, s), 7.83 (1H, s): ^{13}C-NMR ($CDCl_3$, 100 MHz): δ 20.3, 20.5, 20.6, 20.7, 61.6, 67.9, 70.4, 72.8, 75.2, 85.9, 122.1, 134.6, 169.1, 169.5, 170.0, 170.5 ppm.

1-(2,3,4,6-Tetra-*O*-acetyl-β-D-glucopyranosyl)-4-phenyl-1*H*-1,2,3-triazole 3c[14]

^{1}H-NMR ($CDCl_3$, 400 MHz): δ 1.88 (3H, s), 2.04 (3H, s), 2.07 (3H, s), 2.08 (3H, s), 4.06-4.03 (1H, m), 4.18-4.15 (1H, m), 4.35-4.31 (1H, m), 5.28 (1H, t, *J* = 9.6 Hz), 5.47-5.43 (1H, m), 5.53 (1H, t, *J* = 9.6 Hz), 5.95 (1H, d, *J* = 9.6 Hz, H1), 7.36 (1H, d, *J* = 7.4 Hz), 7.45-7.42 (2H, m), 7.84 (2H, d, *J* = 7.5 Hz), 8.02 (1H, s): ^{13}C-NMR ($CDCl_3$, 100 MHz): δ 20.2, 20.6, 20.6, 20.7, 61.6, 67.8, 70.2, 72.8, 75.2, 85.9, 117.8, 125.9, 128.6, 128.9, 129.9, 148.6, 169.1, 169.4, 170.0, 170.5 ppm.

4-*p*-Tolyl-1-(2,3,4,6-tetra-*O*-acetyl-β-D-glucopyranosyl)-1*H*-1,2,3-triazole 3d[15]

^{1}H-NMR ($CDCl_3$, 400 MHz): δ 1.85 (3H, s), 2.01 (3H, s), 2.06 (3H, s), 2.35 (3H, s), 3.99-4.03 (1H, m) 4.13 (1H, dd, *J* = 12.4, 1.6 Hz), 4.30 (1H, dd, *J* = 12.6, 5.0 Hz), 5.24 (1H, t, *J* = 9.6 Hz), 5.43 (1H, t, *J* = 9.6 Hz), 5.47 (1H, t, *J* = 9.6 Hz), 5.91 (1H, d, *J* = 8.0 Hz, H1) 7.21 (2H, d, *J* = 7.6 Hz,), 7.69 (2H, d, *J* = 8.0 Hz), 7.94 (1H, s); ^{13}C-NMR (100 MHz, $CDCl_3$): δ 20.1, 20.5, 20.6, 21.3, 61.6, 67.8, 70.2, 72.8, 75.1, 85.7, 117.3, 125.8, 127.1, 129.5, 138.4, 148.5, 168.9, 169.3, 169.9, 170.4 ppm.

1-(2,3,4,6-Tetra-*O*-acetyl-β-D-glucopyranosyl)-4-(4-bromophenyl)-1*H*-1,2,3-triazole 3e[16]

^{1}H-NMR ($CDCl_3$, 400 MHz): δ 1.85 (3H, s), 2.01 (3H, s), 2.06 (3H, s), 2.23 (3H, s), 4.03 (1H, ddd, *J* = 10.2, 4.8, 2.0 Hz), 4.16 (1H, dd, *J* = 12.8, 2.0 Hz), 4.33 (1H, dd, *J* = 12.7, 5.1 Hz), 5.26 (1H, dd, *J* = 10.0, 9.2 Hz), 5.44 (1H, dd, *J* = 9.6, 9.2 Hz), 5.50 (1H, dd, *J* = 9.6, 9.2 Hz), 5.92 (1H, d, *J* = 9.2 Hz, H1), 7.56 (2H, d, *J* = 8.4 Hz), 7.71 (2H, d, *J* = 8.4 Hz), 8.00 (s, 1H): ^{13}C-NMR (100 MHz, $CDCl_3$): δ 20.2, 20.5, 20.6, 20.7, 61.6, 67.7, 70.2, 72.7, 75.3, 85.9, 117.8, 122.5, 127.4, 128.9, 132.0, 147.4, 169.0, 169.3, 169.9, 170.4 ppm.

1-(2,3,4,6-Tetra-*O*-acetyl-β-D-glucopyranosyl)-4-(4-chlorophenyl)-1*H*-1,2,3-triazole 3f

^{1}H-NMR ($CDCl_3$, 400 MHz): δ 1.89 (3H, s), 2.02 (3H, s), 2.05 (3H, s), 2.21 (3H, s), 4.01-4.05 (1H, m), 4.16 (1H, d, *J* = 12.0 Hz), 4.33 (1H, dd, *J* = 12.8, 4.8 Hz), 5.27-5.30 (1H, m), 5.45-5.51 (2H, m), 5.92 (1H, d, *J* = 9.2 Hz, H1), 7.40 (2H, d, *J* = 8.4 Hz), 7.78 (2H, d, *J* = 8.4 Hz), 8.00 (s, 1H): ^{13}C-NMR (100 MHz, $CDCl_3$): δ 20.6, 20.9, 21.0, 21.1, 61.9, 68.1, 70.6, 73.0, 75.6, 86.3, 118.3, 127.6, 128.8, 129.5, 134.8, 147.9, 169.5, 169.8, 170.3, 171.0 ppm. HRMS (MALDI): calcd for $C_{22}H_{25}ClN_3O_9$ $[M+H]^+$ 510.1279, found 510.1282.

1-(2,3,4,6-Tetra-*O*-acetyl-β-D-galactopyranosyl)-4-phenyl-1*H*-1,2,3-triazole 3g[14]

^{1}H-NMR ($CDCl_3$, 400 MHz): δ 1.91 (3H, s), 2.02 (3H, s), 2.05 (3H, s), 2.24 (3H, s), 4.17-4.25 (3H, m), 5.27 (1H, dd, *J* = 9.0, 3.2 Hz, 1H), 5.58 (1H, d, *J* = 2.8 Hz), 5.65 (1H, t, *J* = 7.2 Hz), 5.92 (1H, d, *J* = 9.6 Hz, H1), 7.36-7.33 (1H, m), 7.45-7.42 (2H, m), 7.86 (2H, d, *J* = 7.2 Hz), 8.06 (1H, s): ^{13}C-NMR (100 MHz, $CDCl_3$): δ 20.1, 20.4, 20.5, 20.6, 61.1, 66.8, 67.7, 70.7, 73.9, 86.2, 117.7, 125.8, 128.4, 128.7, 129.8, 148.3, 169.0, 169.7, 169.9, 170.2 ppm.

1-(2,3,4,6-Tetra-*O*-acetyl-β-D-galactopyranosyl)-4-(4-bromophenyl)-1*H*-1,2,3-triazole 3h[17]

^{1}H-NMR ($CDCl_3$, 400 MHz): δ 1.91 (3H, s), 2.03 (3H, s), 2.06 (3H, s), 2.25 (3H, s), 4.17-4.25 (3H, m), 5.27 (1H, dd, *J* = 9.2, 3.2 Hz), 5.58 (1H, d, *J* = 3.0 Hz), 5.65 (1H, t, *J* = 7.4 Hz), 5.92 (1H, d, *J* = 9.6 Hz), 7.56 (2H, d, *J* = 8.8 Hz), 7.73 (2H, d, *J* = 8.8 Hz), 8.06 (s, 1H) ppm.

1-(2,3,4,6-Tetra-*O*-acetyl-β-D-galactopyranosyl)-1*H*-benzo[*d*][1,2,3]triazole 3i[7]

^{1}H-NMR ($CDCl_3$, 400 MHz): δ 1.75 (3H, s), 1.98 (3H, s), 2.00 (3H, s), 2.27 (3H, s), 4.15–4.26 (2H, m), 4.31 (1H, t, *J* = 6.4 Hz), 5.33 (1H, dd, *J* = 10.0, 3.2 Hz, H2), 5.60 (1H, d, *J* = 3.2 Hz), 5.85 (1H, t, *J* = 9.6 Hz), 6.17 (1H, d, *J* = 9.6 Hz), 7.38 (1H, t, *J* = 7.6 Hz), 7.53 (1H, t, *J* = 7.6 Hz), 7.66 (1H, d, *J* = 8.4 Hz), 8.03 (1H, d, *J* = 8.4 Hz): ^{13}C-NMR (100 MHz, $CDCl_3$): δ 20.3, 20.8, 20.9, 21.0, 61.5, 66.9, 67.3, 71.2, 73.9, 86.9, 111.1, 120.6, 125.0, 128.51, 132.0, 146.8, 169.0, 170.2, 170.3, 170.6 ppm.

ACKNOWLEDGEMENTS

This work was partially supported by Grants-in-Aid for Scientific Research (S) and Grants-in-Aid for Scientific Research (A) from the Japan Society for the Promotion of Science (JSPS), a Grant-in-Aid for Scientific Research on Innovative Areas “Advanced Molecular Transformation by Organocatalysts” from The Ministry of Education, Culture, Sports, Science and Technology (MEXT). K.M. acknowledges a Grant-in-Aid for Scientific Research (C) from JSPS.

REFERENCES

1. (a) M. E. Taylor and K. Drickamer, *Introduction to Glycobiology*, 2006; (b) N. W. Owens and F. Schweizer, in Comprehensive Biotechnology, Second Edition, 2011, vol. 1, pp. 122–138; (c) X. Yu, K. Baruah, C. N. Scanlan, and M. Crispin, in Antibody Fc: Linking Adaptive and Innate Immunity, Elsevier Inc., 2013, pp. 179–194; (d) T. B. Parsons, W. B. Struwe, J. Gault, K. Yamamoto, T. A. Taylor, R. Raj, K. Wals, S. Mohammed, C. V. Robinson, J. L. P. Benesch, and B. G. Davis, *Angew. Chem. Int. Ed.*, 2016, **55**, 2361; (e) S. V. Moradi, W. M. Hussein, P. Varamini, P. Simerska, and I. Toth, *Chem. Sci.*, 2016, **7**, 2492.
2. A. Kobata, *Proc. Japan Acad. Ser. B*, 2013, **89**, 97.
3. T. Kajimoto and M. Node, *Curr. Top. Med. Chem.*, 2009, **9**, 13.
4. (a) G. J. van der Heden van Noort, M. G. van der Horst, H. S. Overkleeft, G. A. van der Marel, and D. V. Filippov, *J. Am. Chem. Soc.*, 2010, **132**, 5236; (b) H. Tanaka, Y. Iwata, D. Takahashi, M. Adachi, and T. Takahashi, *J. Am. Chem. Soc.*, 2005, **127**, 1630; (c) K. Tanaka, T. Miyagawa, and K. Fukase, *Synlett*, 2009, 1571; (d) B. Wang, Y. Liu, R. Jiao, Y. Feng, Q. Li, C. Chen, L. Liu, G. He, and G. Chen, *J. Am. Chem. Soc.*, 2016, **138**, 3926; (e) Y. Kobayashi, Y. Nakatsuji, S. Li, S. Tsuzuki, and Y. Takemoto, *Angew. Chem. Int. Ed.*, 2018, **57**, 3646.

5. (a) L. V. Lee, M. L. Mitchell, S. J. Huang, V. V. Fokin, K. B. Sharpless, and C. H. Wong, *J. Am. Chem. Soc.*, 2003, **125**, 9588; (b) P. van der Peet, C. T. Gannon, I. Walker, Z. Dinev, M. Angelin, S. Tam, J. E. Ralton, M. J. McConville, and S. J. Williams, *ChemBioChem*, 2006, **7**, 1384; (c) N. J. Agard, J. M. Baskin, J. A. Prescher, A. Lo, and C. R. Bertozzi, *ACS Chem. Biol.*, 2006, **1**, 644; (d) N. J. Agard, J. A. Prescher, and C. R. Bertozzi, *J. Am. Chem. Soc.*, 2004, **126**, 15046.
6. (a) B. A. Salameh, H. Leffler, and U. J. Nilsson, *Bioorg. Med. Chem. Lett.*, 2005, **15**, 3344; (b) L. L. Rossi, and A. Basu, *Bioorg. Med. Chem. Lett.*, 2005, **15**, 3596.
7. J. A. Watt, A. C. T. Gannon, A. K. J. Loft, A. Z. Dinev, and S. J. Williams *Aust. J. Chem.*, 2008, **61**, 837.
8. (a) C. W. Tornøe, C. Christensen, and M. Meldal, *J. Org. Chem.*, 2002, **67**, 3057; (b) V. V. Rostovtsev, L. G. Green, V. V. Fokin, and K. B. Sharpless, *Angew. Chem. Int. Ed.*, 2002, **41**, 2596; (c) H. C. Kolb, M. G. Finn, and K. B. Sharpless, *Angew. Chem. Int. Ed.*, 2001, **40**, 2004.
9. A. Mandoli and Alessandro, *Molecules*, 2016, **21**, 1174.
10. R. R. Schmidt and J. Michel, *J. Carbohydr. Chem.*, 1985, **4**, 141.
11. For selected reviews and accounts, see: (a) Y. Kita, H. Tohma, and T. Yakura, *Trends Org. Chem.*, 1992, **3**, 113; (b) G. F. K. T. Wirth, M. Ochiai, V. V. Zhdankin, H. Tohma, and Y. Kita, *Hypervalent Iodine Chemistry*, Springer Berlin Heidelberg, Berlin, Heidelberg, 2003, vol. 224; (c) H. Tohma and Y. Kita, *Top. Curr. Chem.*, 2003, 209; (d) V. V. Zhdankin and P. J. Stang, *Chem. Rev.*, 2008, **108**, 5299; (e) T. Dohi and Y. Kita, *Chem. Commun.*, 2009, 2073; (f) Y. Kita and T. Dohi, *Chem. Rec.*, 2015, **15**, 886; (g) A. Yoshimura and V. V. Zhdankin, *Chem. Rev.*, 2016, **116**, 3328; (h) K. Morimoto, T. Dohi, and Y. Kita, *Synlett*, 2017, **28**, 1680.
12. (a) T. Kajimoto, K. Morimoto, R. Ogawa, T. Dohi, and Y. Kita, *Eur. J. Org. Chem.*, 2015, 2138; (b) T. Kajimoto, K. Morimoto, R. Ogawa, T. Dohi, and Y. Kita, *Chem. Pharm. Bull.*, 2016, **64**, 838; (c) K. Morimoto, K. Yanase, I. Odaka, Y. Kita, and T. Kajimoto, *Heterocycles*, 2019, **99**, 680.
13. K. Slámová, P. Marhol, K. Bezouška, L. Lindkvist, S. G. Hansen, V. Křen, and H. H. Jensen, *Bioorg. Med. Chem. Lett.*, 2010, **20**, 4263.
14. K. Kumari, A. S. Singh, K. K. Manar, C. L. Yadav, V. K. Tiwari, M. G. B. Drew, and N. Singh, *New J. Chem.*, 2019, **43**, 1166.
15. D. Nandi, A. Taher, R. U. Isalm, and K. Mallick, *Appl. Organomet. Chem.*, 2019, **33**, 1.
16. S. Tejera, R. L. Dorta, and J. T. Vázquez, *Tetrahedron: Asymmetry*, 2016, **27**, 896.
17. I. Carvalho, P. Andrade, V. L. Campo, P. M. M. Guedes, R. Sesti-Costa, J. S. Silva, S. Schenkman, S. Dedola, L. Hill, M. Rejzek, S. A. Nepogodiev, and R. A. Field, *Bioorg. Med. Chem.*, 2010, **18**, 2412.