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Synthesis of UDP-GalNAc analogues as probes for the study of polypeptide-α-GalNAc-transferases. Part 2

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Abstract—The synthesis of four UDP-GalNAc analogues (**1**–**4**) is described. The 3-, 4- and 6-deoxygenated analogues **1**–**3** were obtained by way of a divergent strategy starting from a 3,6-di-*O*-pivaloyl GlcNAc derivative as a common precursor. Analogue **4** bearing a *N*-trifluoroacetamido group was prepared from the trifluoromethylated oxazoline **24** as key intermediate. These compounds were designed to probe the substrate specificity of polypeptide-α-GalNAc transferases.

1. Introduction

As molecular markers at the surface of cells, glycan constituents of glycoproteins play a prominent role in numerous important biological processes such as cell–cell recognition or antigen–antibodies interactions. In particular, a direct correlation between the progression of cancer and the alteration of oligosaccharide structures N- or O-linked to proteins has been demonstrated.[1] While the comparatively more complex process of protein *N*-glycosylation has been extensively studied, little is known yet on the specific role of protein *O*-glycosylation and the factors triggering the glycosylation process.[2]

The first step of the biosynthesis of *O*-glycosylated proteins is catalysed by polypeptide-α-GalNAc-transferases (ppGalNAcTs),[3] which transfer a GalNAc unit from UDP-GalNAc to the hydroxyl group of a serine or a threonine residue with retention of configuration. Recently, we described the synthesis of three *O*-methylated UDP-GalNAc derivatives as probes for the study of the polypeptide-α-GalNAc-transferase T1.[4] Following biological evaluation, these analogues proved to be no donor substrates for the transferase and only weak competitive inhibitors. These results provided further evidence for the very high affinity and high selectivity of the sugar nucleotide binding site of the ppGalNAcT for UDP-GalNAc, as indicated also by affinity chromatography experiments.[5]

More recently, molecular modelling of the UDP-GalNAc binding site, based on structural information taken from the first X-ray crystallographic studies of retaining glycosyl transferases,[6] indicated that at least two of the OH groups of the sugar unit are involved in substrate binding and that the sugar nucleotide fits very tightly into a deep binding pocket, mostly through the GalNAc part.[7] In addition, the *N*-acetyl group may be involved in substrate recognition since UDP-Gal is not a substrate for the ppGalNAc transferase T1 although another member of this family has been shown to use UDP-Gal as a substrate albeit very poorly.[8] These findings prompted us to further explore the donor substrate specificity of ppGalNAc T1. Toward this goal, we synthesised the three deoxygenated analogues of UDP-GalNAc **1**–**3** as well as the *N*-trifluoroacetylated derivative **4** as probes of the sugar nucleotide–transferase interactions. Related studies revealed for example that the 2-, 4- and 6-deoxygenated analogues of UDP-Gal could serve as substrate for the β-(1→4)-galactosyltransferase,[9] and that the 3-, 4- (identical to **2**) and 6-deoxy derivatives of UDP-GlcNAc acted as donors in *N*-acetylglucosaminyl-transferase I mediated glycosylation reactions.[10] Last but not least, the *N*-trifluoroacetylated analogue of UDP-GlcNAc was shown to serve

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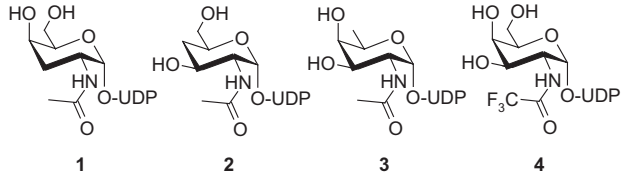
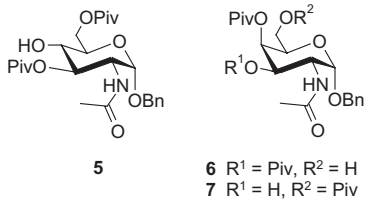


Figure 1. Analogues of UDP-GalNAc.

as a glycosyl donor for the 'core-2' GlcNAc transferase and for the GlcNAcT-V transferase (Fig. 1).[11]

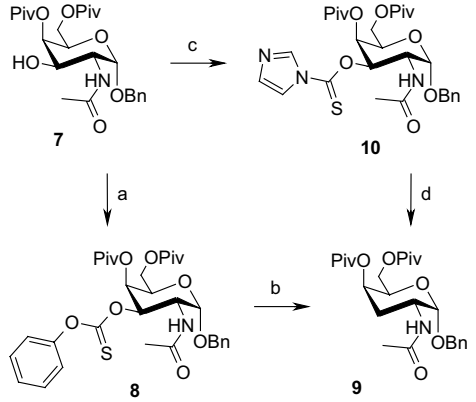


5 **6** R^1 = Piv, R^2 = H; **7** R^1 = H, R^2 = Piv

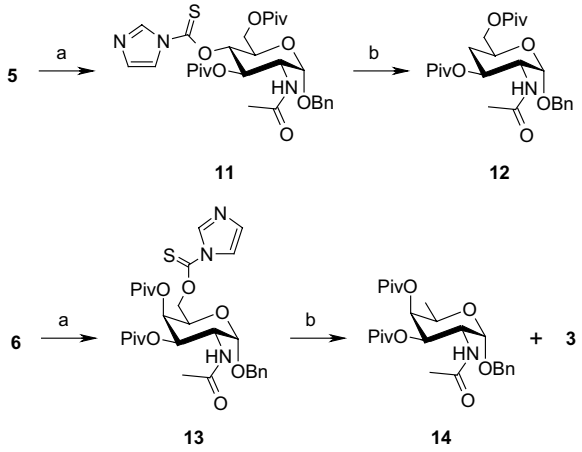
2. Synthesis of deoxygenated derivatives 1–3

Following the pathway previously developed for the synthesis of the *O*-methylated analogues,[4] deoxygenated analogues **1**–**3** were prepared by way of a divergent strategy starting from 3,6-di-*O*-pivaloyl GlcNAc derivative **5** as a common precursor. The latter compound as well as the two key intermediates **6** and **7** were readily obtained from GlcNAc.[12] Deoxygenation was achieved by the Barton–McCombie procedure using tributyltin hydride and 2,2′-azobis(2-methylpropionitrile).[13] The risk of pivaloyl migration under basic conditions prompted us to consider thionocarbamates and thionocarbonates as intermediates, since these groups can be introduced under essentially neutral conditions[14] and the imidazolylthiocarbonyl group has been used with success for the deoxygenation of galactosides.[15] Starting from **7** as model compound, we compared the two possible pathways (Scheme 1).

On one hand, compound **7** was reacted with phenyl chlorothionoformate in the presence of pyridine and



Scheme 1. (a) PhO-CS-Cl, DMAP, dichloromethane, pyridine, 4 h, 61%; (b) Bu_3SnH, AIBN, toluene, 90 °C, 3 h, 90%; (c) Im_2CS, toluene, 90 °C, 4 h, 96%; (d) Bu_3SnH, AIBN, toluene, 90 °C, 4 h, 83%.

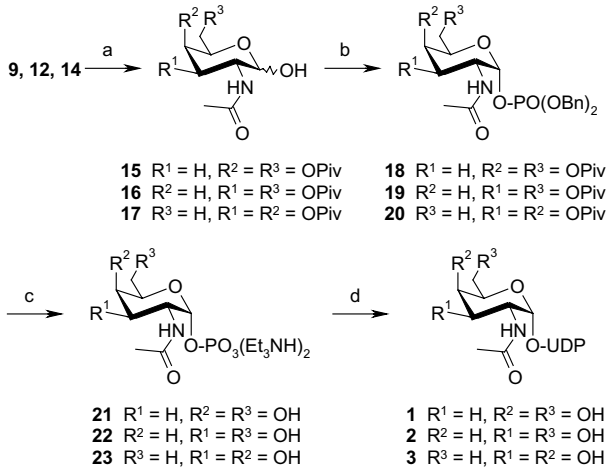


Scheme 2. (a) Im_2CS, toluene, 90 °C, 4 h, **11**: 97%, **13**: 94%; (b) Bu_3SnH, AIBN, toluene, 90%, 4 h, **12**: 86%, **14**: 30% (**6**: 50%).

N,*N*-dimethylaminopyridine to give the corresponding thionocarbonate **8** (61%), which in turn led to the desired deoxygenated compound **9** in 55% overall yield. On the other hand, treatment of **7** with 1,1′-thiocarbonyldiimidazole nicely furnished the parent thionocarbamate **10** (96%), which underwent the expected deoxygenation in 80% overall yield.

Hence we selected the latter conditions for further syntheses (Scheme 2). The 4-OH derivative **5** was successfully deoxygenated to give **12**[16] in 83% overall yield whereas the 6-deoxygenated compound **14** was obtained in a modest yield of 28% along with the starting alcohol **6** (50%) resulting from competitive hydrogen capture. Radical deoxygenation at primary positions is known to be difficult.[17] In some cases it has been possible to improve the yield by conducting the reaction at higher temperature and adding the hydride dropwise.[18] Unfortunately, in our hands the conversion never exceeded 30%; similar results were reported in the galactose series.[19]

Further elaboration to sugar-nucleotides was achieved by the strategy outlined below (Scheme 3). Deprotection of the anomeric position of benzyl glycosides **9**, **12** and **14** was accomplished under standard hydrogenolytic conditions in nearly quantitative yield. The resulting pyranoses **15**, **16** and **17** were phosphorylated using dibenzyl diethyl-phosphoramidite and 1*H*-tetrazole as catalyst.[20] Oxidation of the intermediate phosphites using hydrogen peroxide at −78 °C afforded the corresponding phosphotriesters **18**–**20** in yields ranging from 64 to 79%. The phosphorylation and anomeric configuration were unambiguously confirmed by ^1H and ^{31}P NMR data.[21] It should be noted that the di-*O*-acetylated analog of **19** was reported to be too labile for purification on silica gel.[10] All three phosphotriesters **18**-**20** however could be purified by flash chromatography without noticeable degradation. Debenzylation, followed by removal of the pivaloyl groups under Zemplen conditions, provided the parent sugar-monophosphates, which were immediately converted into their triethylammonium salts **21**, **22**[10] and **23** using a cationic

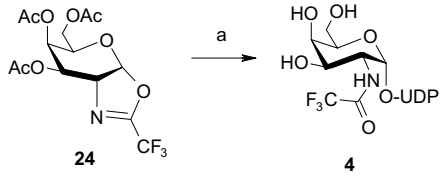


Scheme 3. (a) H_2, 10% Pd/C, EtOH, rt, 1 h, **15**: 97%, **16**: 99%, **17**: 99%. (b) (i) 1.5 equiv $(BnO)_2PNEt_2$, 1.5 equiv 1-*H*-tetrazole, THF, rt, 3 h; (ii) 3 equiv H_2O_2, −78 °C, 1 h, **18**: 79%, **19**: 64%, **20**: 70% (two steps). (c) (i) H_2, 10% Pd/C, EtOH, rt, 1 h; (ii) 2 equiv MeONa, MeOH, rt, overnight; (iii) Amberlite® IR-120 Et_3NH^+ form, **21**: 92%, **22**: 95%, **23**: 90% (three steps). (d) 2 equiv UMP-morpholidate, 3 equiv 1*H*-tetrazole, pyridine, rt, 4 days, **1**: 79%, **2**: 31%, **3**: 42%.

exchange resin, in yields ranging from 90% to 95% over three steps. Finally, UDP-GalNAc analogues **1**–**3** were obtained using improved Khorana conditions.[22] This coupling step, involving uridine 5′-monophospho-morpholidate as the activated UMP source and 1*H*-tetrazole provided the desired UDP-sugars in moderate to good yield (not optimised). Formation of the pyrophosphate was clearly established on the basis of ^{31}P NMR spectra showing two characteristic doublets.[23] Compound **2** was already reported as the 4-deoxy analog of UDP-GlcNAc and shown to be substrate for GlcNAc transferases.[10]

3. Synthesis of N-AcF_3 analogue

Analogue **4** bearing a 2-*N*-trifluoroacetamido group was prepared from the trifluoromethylated oxazoline **24** as key intermediate (Scheme 4), of which we have already reported the synthesis.[24,25] Subsequent phosphorylation was performed by opening **24** with dibenzyl phosphate, the phosphate was then deprotected by hydrogenolysis and selective *O*-deacetylation, was achieved by careful treatment with guanidine.[26] The triethylammonium salt



Scheme 4. (a) (i) 1.5 equiv HO–$PO(OBn)_2$, dichloroethane, rt, 2 h then reflux 10 h, 77%; (ii) 10% Pd/C, EtOH, rt, 1 h, 95%; (iii) 4 equiv guanidine, MeOH, rt, 2 h; (iv) Amberlite® IR-120 Et_3NH^+ form, 95% (2 steps); (v) 2 equiv UMP-morpholidate, 3 equiv 1-*H*-tetrazole, pyridine, rt, 4 days, 63%.

was obtained by ion exchange and finally, sugar nucleotide **4** was prepared by the UMP-morpholidate procedure (63%). Pyrophosphate bond formation was assigned on the basis of ^{31}P NMR data.[27]

4. Conclusion

The synthesis of the 3-, 4- and 6-deoxy derivative of UDP-GalNAc, and of its *N*-$COCF_3$ analog, has been achieved by short sequences. These analogues will be useful for the study of the substrate specificity of ppGalNAc transferases. Exploration of their activity as substrate or inhibitors is currently in progress and will be presented in due course.

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21. NMR data: **18** ^{1}H ($CDCl_3$) δ 1.15, 1.22 (2s, 2×9H, 2×3CH_3), 1.75 (s, 3H, CH_3CO), 1.74 (ddd, 1H, $J_{3a,4} = 2.5$ Hz, $J_{3a,2} = J_{3a,3eq} = 13.5$ Hz, H-3ax), 1.97 (ddd, 1H, $J_{3eq,2} = J_{3eq,4} = 3.0$ Hz, H-3eq), 3.96 (dd, 1H, $J_{6a,6b} = 11.0$ Hz, $J_{6a,5} = 7.0$ Hz, H-6a), 4.03 (dd, 1H, $J_{6b,5} = 7.0$ Hz, H-6b), 4.17 (ddd, 1H, $J_{5,4} = 2.0$ Hz, H-5), 4.40 (m, 1H, $J_{2,1} = 3.0$ Hz, $J_{2,NH} = 9.0$ Hz, H-2), 5.01 (m, 1H, H-4), 5.03–5.16 (m, 4H, 2×CH_2Ph), 5.65 (dd, 1H, $J_{1,P} = 5.5$ Hz, H-1), 5.72 (d, 1H, NH), 7.3–7.5 (m, 10H, 2×C_6CH_5); ^{13}C($CDCl_3$) δ 22.83 (CH_3CO), 26.93, 26.96 (2×3CH_3), 27.84 (C-3), 38.54, 38.99 (2×$C(CH_3)_3$), 43.35 (d, $J_{2,P} = 7.0$ Hz, C-2), 61.52 (C-6), 65.62, 68.30 (C-4,5), 69.74, 69.79 (2d, $J_{C,P} = 5.5$ Hz, 2×CH_2Ph), 96.38 (d, $J_{1,P} = 6.5$ Hz, C-1), 127.97–128.79 (10×CHPh), 135.19 (d, $J_{C,P} = 6.5$ Hz, Cq Ph), 135.37 (d, $J_{C,P} = 6.0$ Hz, Cq Ph), 169.62 (CH_3CO), 177.19, 177.63 (2×*t*Bu*C*O). **19** ^{1}H($CDCl_3$) δ 1.15, 1.19 (2s, 2×9H, 2×3CH_3), 1.70 (s, 3H, CH_3CO), 1.61–1.79 (m, 1H, H-4a), 1.93–2.05 (m, 1H, H-4b), 4.01 (d, 2H, 2H-6), 4.06–4.17 (m, 1H, H-5), 4.25 (dddd, 1H, $J_{2,1} = J_{2,P} = 3.0$ Hz, $J_{2,NH} = 9.5$ Hz, $J_{2,3} =$ 10.5 Hz, H-2), 4.98–5.16 (m, 5H, H-3, 2×CH_2Ph), 5.59 (d, 1H, NH), 5.70 (dd, 1H, $J_{1,P} = 5.5$ Hz, H-1), 7.2–7.5 (m, 10H, 2×C_6CH_5); ^{13}C ($CDCl_3$) δ 22.80 (CH_3CO), 26.92, 27.13 (2×3CH_3), 32.38 (C-4), 38.75, 38.80 (2×$C(CH_3)_3$), 52.00 (d, $J_{2,P} = 7.2$ Hz, C-2), 64.98 (C-6), 66.84, 67.99 (C-3,5), 69.71 (d, $J_{C,P} = 5.0$ Hz, CH_2Ph), 69.79 (d, $J_{C,P} = 5.5$ Hz, CH_2Ph), 97.85 (d, $J_{1,P} = 6.7$ Hz, C-1), 128.06–128.80 (10×CHPh), 135.34, 135.46 (2d, $J_{C,P} = 6.3$ Hz, 2×CqPh), 170.03 (CH_3CO), 178.05, 178.51 (2×*t*Bu*C*O). **20** ^{1}H ($CDCl_3$) δ 1.12 (d, 3H, 3H-6), 1.27, 1.30 (2s, 2×9H, 2×3CH_3), 1.70 (s, 3H, CH_3CO), 4.15 (q, 1H, $J_{5,6} = 7.0$ Hz, H-5), 4.63 (dddd, 1H, $J_{2,P} = 3.0$ Hz, $J_{2,1} = 3.5$ Hz, $J_{2,NH} = 10.0$ Hz, $J_{2,3} = 10.5$ Hz, H-2), 5.00–5.16 (m, 4H, 2×CH_2Ph), 5.19 (d, 1H, $J_{4,3} = 3.0$ Hz, H-4), 5.22 (dd, 1H, H-3), 5.58 (d, 1H, NH), 5.71 (dd, 1H, $J_{1,P} = 5.5$, H-1), 7.3–7.5 (m, 10H, 2×C_6H_5); ^{13}C ($CDCl_3$) δ 16.02 (C-6), 22.81 (C_3CO), 26.90, 27.26 (2×3CH_3), 38.82, 39.09 (2×$C(CH_3)_3$), 47.63 (d, $J_{2,P} = 7.2$ Hz, C-2), 66.22, 67.73, 70.25 (C-3–5), 69.72, 69.75 (2d, $J_{C,P} = 5.5$ Hz, 2×CH_2Ph), 97.98 (d, $J_{1,P} = 6.5$ Hz, C-1), 128.02–128.80 (10×CHPh), 135.27, 135.49 (2d, $J_{C,P} = 6.1$ Hz, 2×Cq Ph), 169.91 (CH_3CO), 177.47, 178.38 (2×*t*Bu*C*O).
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23. NMR data: **1** ^{31}P (D_2O) δ −10.68 (d, 1P, $J_{P,P} = 19.5$ Hz, P_α), −11.98 (d, 1P, P_β). **2** ^{31}P (D_2O) δ −10.74 (d, 1P, $J_{P,P} = 20.1$ Hz, P_α), −12.27 (d, 1P, P_β). **3** ^{31}P (D_2O) δ −10.71 (d, 1P, $J_{P,P} = 20.3$ Hz, P_α), −12.16 (d, 1P, P_β).
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25. NMR data: ^{1}H ($CDCl_3$) δ 2.07, 2.14 (2s, 3H and 6H, 3×CH_3CO), 4.14 (dd, 1H, $J_{6a,5} = 6.0$ Hz, $J_{6a,6b} = 11.5$ Hz, H-6a), 4.21 (dd, 1H, $J_{6b,5} = 6.0$ Hz, H-6b), 4.25 (dd, 1H, $J_{2,1} = 7.0$ Hz, $J_{2,3} = 7.5$ Hz, H-2), 4.31 (ddd, 1H, $J_{5,4} = 2.0$ Hz, H-5), 4.92 (dd, 1H, $J_{3,4} = 3.5$ Hz, H-3), 5.47 (ddd, 1H, H-4), 6.32 (d, 1H, H-1); ^{13}C ($CDCl_3$) δ 20.23, 20.37 (3×CH_3CO), 61.24 (C-6), 62.37 (C-2), 64.24, 69.93, 70.83 (C-3–5), 104.97 (C-1), 115.74 (q, $J_{C,F} = 274.5$ Hz, CF_3CO), 156.06 (q, $J_{C,F} = 41.0$ Hz, CF_3C), 168.54, 169.63, 170.17 (3×CH_3O).
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27. NMR data ^{31}P (D_2O): δ −10.76 (d, 1P, $J_{P,P} = 20.7$ Hz, P_α) −12.32 (d, 1P, P_β).