MedChemComm

CONCISE ARTICLE

View Article Online
View Journal | View Issue

Cite this: *Med. Chem. Commun.*, 2014, **5**, 1693

Synthesis of a novel series of 2,3,4-trisubstituted oxazolidines designed by isosteric replacement or rigidification of the structure and cytotoxic evaluation†

Saulo F. Andrade,[ac] Claudia S. Teixeira,[a] Jonas P. Ramos,[b] Marcela S. Lopes,[a] Rodrigo M. Pádua,[a] Mônica C. Oliveira,[a] Elaine M. Souza-Fagundes[b] and Ricardo J. Alves*[a]

Received 24th March 2014
Accepted 21st July 2014

DOI: 10.1039/c4md00136b

www.rsc.org/medchemcomm

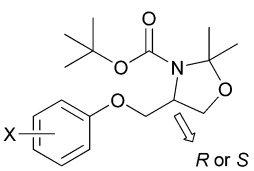
We have previously reported on a study of the structure–activity relationship in a series of 2,3,4-substituted oxazolidines recently discovered by our group varying the substituent at the ring or stereochemistry of the oxazolidine ring. We discovered the cytotoxic and pro-apoptotic potential of compounds **1** and **2** with good selectivity against cancer cell lines. In the present study we describe the synthesis and cytotoxic evaluation against cancer cell lines (HL60, JURKAT, MDA-MB-231 and LNCaP) of a series of oxazolidines designed by isosteric replacement or rigidification of the oxymethylene spacer of compounds **1** and **2**. Alkenes **3** and **4** retained the activity against MDA-MB-231 cells and they were more active on HL60, JURKAT and LNCaP cells. Considering LNCaP cells, *E*-isomer **4** was at least 7 times and about 3 times more potent than lead **1** and *Z*-isomer **3**, respectively. Compound **4** exerted significant activity against LNCaP with IC_{50} in the low micromolar range (11 μM) without affecting VERO cells and PBMC proliferation (IC_{50} > 100 μM) indicating its low toxicity to normal cells.

Introduction

Cancer is the leading cause of death worldwide. In addition, the number of new cases is increasing mainly due to population growth and aging.[1–3] Breast and prostate cancer are the most common types that affect women and men, respectively.[4] As successful cancer treatment remains a challenging goal, research into novel, selective and less toxic chemotherapeutic agents is gathering pace.[5–8] There is a great need to develop alternative and more effective therapies to improve both life expectancy and quality of a patient's life.[9–11]

As a part of an ongoing project aimed at the development of new anticancer compounds we have previously reported a study of the structure–activity relationship in a series of 2,3,4-trisubstituted oxazolidines recently discovered by our group (Fig. 1).[12,13] In this study, we prepared 25 compounds to evaluate the importance of the ring substituent and stereochemistry of oxazolidine in the antiproliferative activity against cancer cell lines.

It was observed that the hydrophobic and electron withdrawing $COOCH_3$ or NO_2 group is important for the activity of this class of compounds. The unsubstituted compounds (X = H) were inactive against cancer cell lines. The presence of hydrophilic and electron withdrawing COOH or hydrophobic and electron donor OMe results in poor activity. Regarding the substituent position, a substituent at the 3 or 4-position is important for the activity. All *ortho*-substituted compounds were inactive. The *S* isomers were generally more active than their enantiomers. In some cases, *S* isomers were 10 times more potent. Finally, significant activity difference between *S* isomers



X = NO_2, $COOCH_3$, OMe, COOH, H

Fig. 1 Structures of 2,3,4-trisubstituted oxazolidines with antiproliferative activity.

[a]*Departamento de Produtos Farmacêuticos, Faculdade de Farmácia, Universidade Federal de Minas Gerais (UFMG), Av. Antônio Carlos, 6627, Belo Horizonte, MG 31270-901, Brazil. E-mail: ricardodylan@farmacia.ufmg.br; Fax: +55 31 3409 6935; Tel: +55 31 3409 6955*

[b]*Departamento de Fisiologia e Biofísica, Instituto de Ciências Biológicas, Universidade Federal de Minas Gerais (UFMG), Brazil*

[c]*Departamento de Produção de Matéria-Prima, Faculdade de Farmácia, Universidade Federal do Rio Grande do Sul (UFRGS), Av. Ipiranga, 2752, Porto Alegre, RS 90610-000, Brazil*

† Electronic supplementary information (ESI) available: Chemical and biological procedures and characterisation data. See DOI: 10.1039/c4md00136b

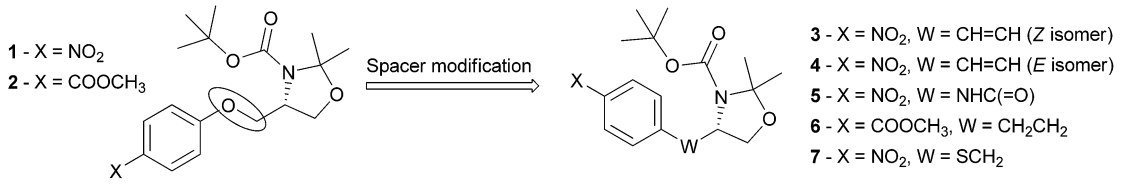


Fig. 2 Design of a new series of oxazolidines by modification of the oxymethylene spacer.

bearing $COOCH_3$ and NO_2 at the 3 or 4-position was not observed. However, *para*-substituted compounds appear to be more selective than *meta* against cancer cells. With this in mind, we decided to carry out further study to evaluate the importance of the oxymethylene spacer between benzene and oxazolidine rings of compounds **1** or **2** in order to obtain more potent and selective compounds (Fig. 2). This series was planned by rigidification of the structure (compounds **3–5**) or isosteric replacement (compounds **6** and **7**). In most cases NO_2 was chosen as the X group due to its intrinsic stability and synthetic viability. In the case of compound **6**, the chosen synthetic route demanded the $COOCH_3$ group.

The preliminary mechanism of action evaluation showed that compounds **1** and **2** were able to induce DNA fragmentation at 50 μM in HL60 cells.[13] In the case of compound **1**, about 90% of cells had fragmented DNA while compound **2** led to DNA fragmentation in about 40% of cells. This indicated that compound **1** has pro-apoptotic potential. Although the molecular target was not identified, it is an important finding because *apoptosis* is one of the most important pathways used to discover new anticancer drugs.[14,15] Despite different mechanisms of action, several important currently marketed anticancer drugs are able to trigger *apoptosis* in cancer cells (*i.e.* cisplatin and doxorubicin).[16,17] Thus, it is expected that compounds that modulate this pathway are promising *hit* compounds for the development of new anticancer drugs. So, we intended to evaluate in this study the cytotoxicity of this new series of oxazolidines against cancer cell lines (HL60, JURKAT, MDA-MB-231 and LNCaP) and the pro-apoptotic potential of the most potent compounds.

Results and discussion

The strategy for the synthesis of olefins **3** and **4** was based on the retrosynthetic analysis shown in Fig. 3. The disconnection of the double bond into two fragments offers two possibilities. In strategy 1, the new phosphonium salt **8** and 4-nitrobenzaldehyde **9** are the potential precursors, while in strategy 2 the disconnection furnishes the known Garner's aldehyde **10** and 4-nitrobenzyl phosphonium salt **11**. Taking into account that generally substituted benzaldehydes are cheap commercially available compounds and substituted benzyl phosphonium salts are expensive or not available, it seemed reasonable to adopt strategy 1. Thus, we aimed to prepare the novel compound **8**.

Initially, the key intermediate alcohol **15** was prepared as previously reported (Fig. 4).[13] In brief, commercially available D-serine **12** was protected with *tert*-butoxycarbonyl and carboxylic acid was converted into methyl ester by treatment with methyl iodide and potassium carbonate to give **13** in 78% overall yield.[18,19] Next, acetonide formation of **13** was carried out using 2,2-dimethoxypropane (DMP) and $BF_3 \cdot OEt_2$ to afford acetonide **14** in 77% yield.[20] Lastly, the methyl ester of acetonide **14** was reduced to alcohol **15** using $NaBH_4$ in 85% yield.[21]

With **15** in hand, we proceeded to the synthesis of compound **8**. Treatment of alcohol **15** with imidazole, iodine and PPh_3 gave **16** in 61% yield.[22] Unfortunately, this modest yield was obtained only using 200 mg of the starting material. When we scaled up to 400 mg, the yield decreases to about 45%. This could be explained by the instability of compound **16**. It was observed that storage of **16** at room temperature led to the formation of

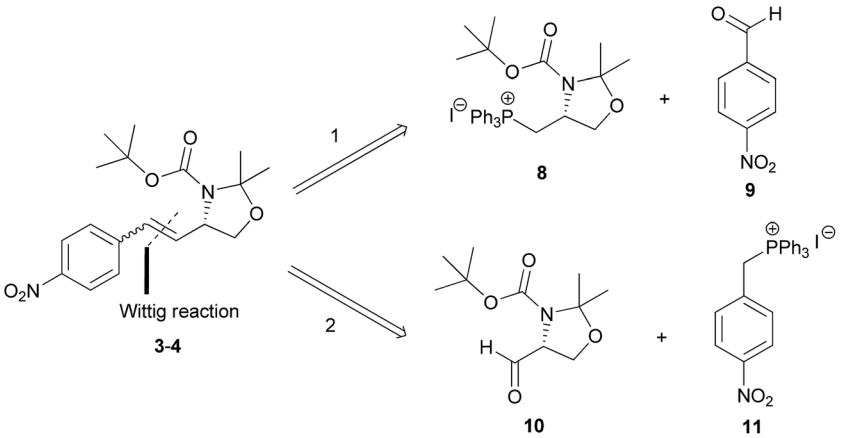


Fig. 3 Disconnection of the double bond.

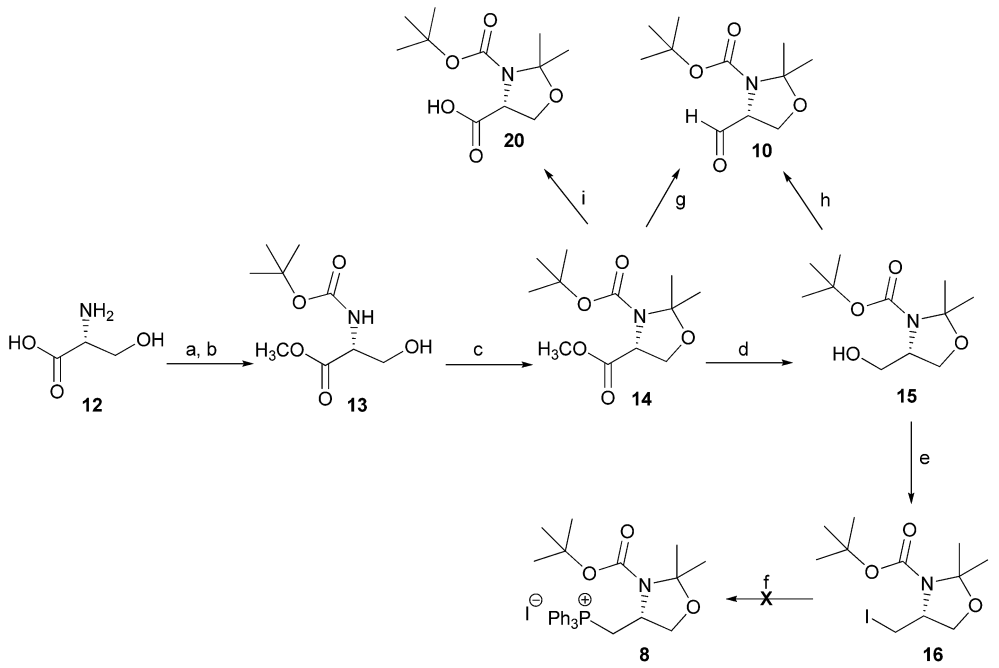


Fig. 4 Preparation of key intermediates. Reagents and conditions: (a) Boc_2O, NaOH, *t*-BuOH–H_2O (1 : 1); (b) CH_3I, K_2CO_3, DMF; (c) DMP, $BF_3{\cdot}OEt_2$, acetone; (d) $NaBH_4$, THF–MeOH (7 : 3), 0 °C → reflux; (e) imidazole, I_2, PPh_3, toluene; (f) PPh_3, toluene, 90 °C; (g) DIBAL, toluene, −78 °C; (h) DMSO, $(COCl)_2$, Et_3N, CH_2Cl_2, −78 °C → 0 °C; (i) LiOH, acetone–H_2O (7 : 3).

degradation products. Next, compound **16** was reacted with PPh_3 in toluene at 90 °C.[23] Usually, phosphonium salts precipitate during the reaction. In this case, precipitate formation was not observed. Indeed, TLC on silica gel revealed a large amount of starting material after 24 h and formation of at least 3 polar byproducts. It is possible that steric hindrance at electrophilic carbon of **16** (CH_2I) results in poor reactivity. Thus, we decided to abandon our initial synthetic strategy and proceed to the second strategy.

Our initial focus was on the synthesis of known Garner's aldehyde **10**. Treatment of **14** with DIBAL under classical Garner conditions gave **10** in only 40% yield.[19] We recovered 40% of the starting material using this method. Besides, a small amount of alcohol **15** (8%) was obtained. Unfortunately, aldehyde **10** and ester **14** had similar R_f values using many solvent mixtures. Thus, it was difficult to purify the desirable product **10**. We tried to increase the yield by using excess DIBAL and adding it very slowly but in all the cases we obtained similar yields. In order to prepare **10** in good yield, a second method was carried out. Alcohol **15** was oxidized under Swern conditions to give **10** in virtually quantitative yield.[24] Compound **10** was obtained in good purity using this method without using column chromatography. Next, we prepared benzyl phosphonium salt **11** (Fig. 5).

Benzaldehyde **17** was reduced to benzyl alcohol **18** using $NaBH_4$ in 88% yield.[21] Next, benzyl alcohol **18** was converted into iodide **19** by treatment with imidazole, iodine and PPh_3 in 78% yield.[25]

Treatment of iodide **19** with PPh_3 in toluene gave phosphonium salt **11** in 90% yield.[26] With **10** and **11** in hand, we were finally in conditions to prepare olefins **3** and **4**. Treatment of phosphonium salt **11** with BuLi followed by addition of Garner's aldehyde **10** gave olefins **3** and **4** in 70% yield as an isomer mixture which were separated by preparative TLC (*Z*/*E* 6 : 4).[27] Some controversy about the coupling constants (*J*) in ^1H NMR

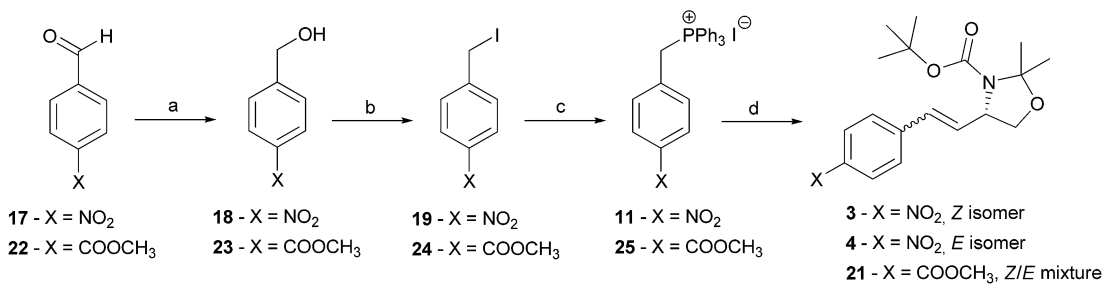


Fig. 5 Preparation of olefins **3**, **4** and **21**. Reagents and conditions: (a) $NaBH_4$, THF–MeOH (7 : 3), −15 °C → r.t.; (b) imidazole, I_2, PPh_3, toluene; (c) PPh_3, toluene; (d) I-BuLi 1.6 M in hexanes, THF; II-**10** in THF.

spectra was found in previously published studies. Unsubstituted *E*-olefins (X = H) with the *R* configuration was previously reported in the literature. Pellicciari's group reported that $J_{CH=CH}$ was 15.8 Hz for this compound while Raghavan's group found 12.2 Hz.[27,28] In our case, $J_{CH=CH}$ values for *E*- and *Z*-olefin (**4** and **3**, X = NO_2, *S* isomer) were 16.0 and 11.6 Hz, respectively (Fig. 6). These values are similar to the ones reported by Pellicciari's group.

In order to prepare amide analogue **5**, initially methyl ester of acetonide **14** was hydrolyzed under alkaline conditions to give **20** in 95% yield (Fig. 4).[29] An initial attempt to couple **20** with 4-nitroaniline to give **5** was carried out using NHS and EDC.[30] Unfortunately, this mild and useful method did not lead to product formation. We believe that delocalization of the nonbonding electron pair in 4-nitroaniline results in low nucleophilicity. Thus, we employed a better electrophile to accomplish this reaction. Carboxylic acid of **20** was activated with benzyl chloroformate and triethylamine followed by addition of 4-nitroaniline to give **5** in 57% yield (Fig. 7).[31]

Compound **6** was synthesized from *Z*/*E* olefin mixture **21**. Initially, this mixture was prepared from phosphonium salt **25** and Garner's aldehyde **10** under Wittig conditions in 68% yield (Fig. 5). Isomer mixture **21** was resistant to reduction with H_2 and catalytic Pd–C at room temperature and 1 atm. After testing different parameters including Pd–C ratio, temperature and pressure, we found that this reduction could be carried out using a 1 : 1 Pd–C (10% w/w) substrate at 55 °C and 55 atm to give **6** in 50% yield (Fig. 7). Finally, we prepared sulfur isostere **7** (Fig. 7). Treatment of alcohol **15** with 4-nitrothiophenol under Mitsunobu conditions provided sulfur isostere in 31% yield.[32]

The antiproliferative activity of compounds **1**–**7** was assessed on four human cancer cells lines, namely, HL60 promyelocytic leukemia, JURKAT T cell leukemia, MDA-MB-231 breast carcinoma cells and LNCaP prostate adenocarcinoma cells. Cytotoxic effects on normal cells were evaluated using VERO African green monkey kidney cells and PBMC peripheral blood mononuclear cells. The results are summarized in Table 1 and expressed as the concentration of drug inhibiting cell growth by 50% (IC_{50}).

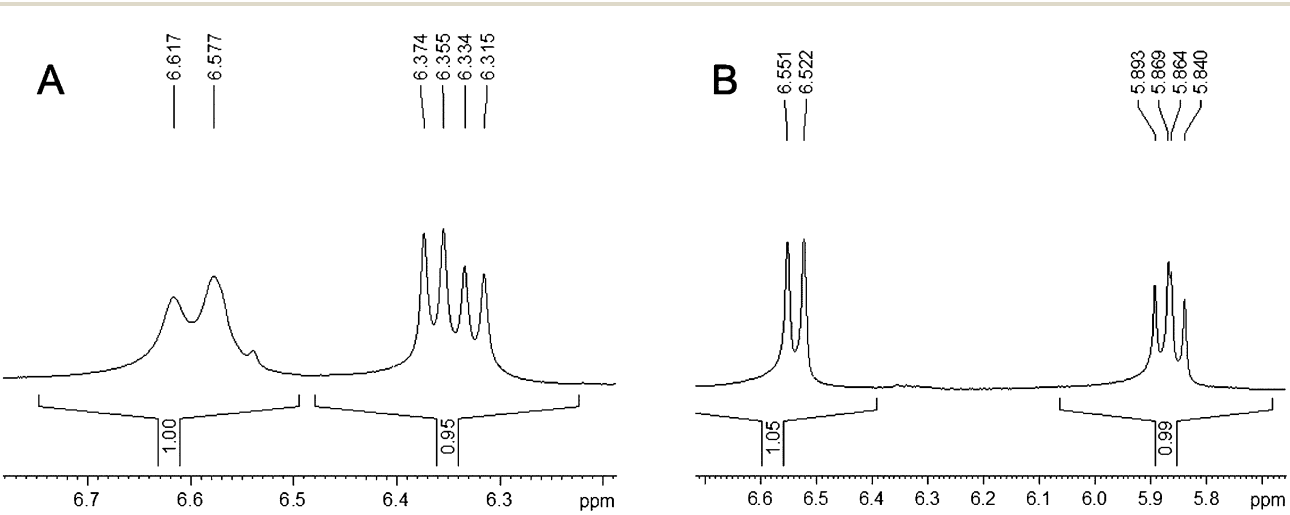


Fig. 6 Expansion of the ^1H NMR spectrum (400 MHz, $CDCl_3$, 45 °C) of olefins (**3** and **4**). Olefin protons (CH=CH) are shown. (A) *E*-Olefin spectrum. (B) *Z*-Olefin spectrum.

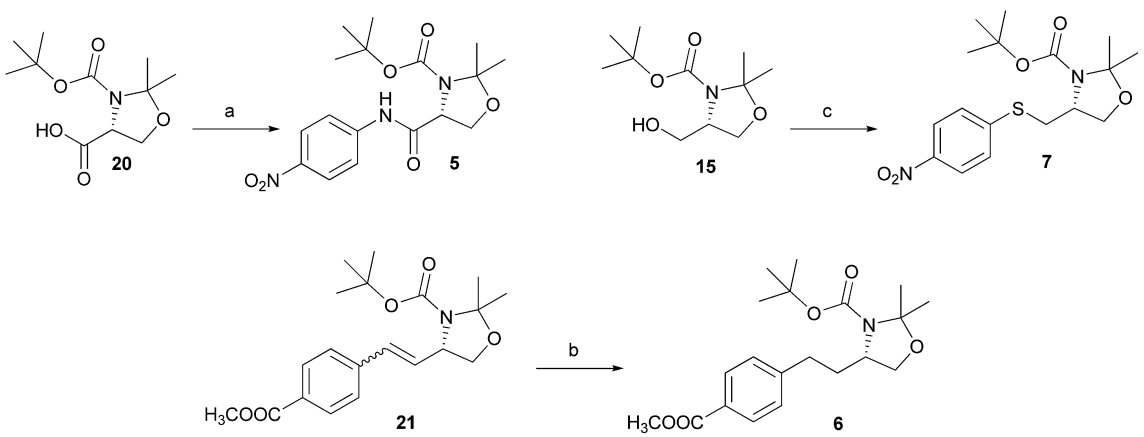


Fig. 7 Preparation of compounds **5**, **6** and **7**. Reagents and conditions: (a) I – benzyl chloroformate, Et_3N, CH_2Cl_2, −15 °C; II – 4-nitroaniline, −15 °C → r.t.; (b) H_2, Pd–C, THF, 55 °C, 55 atm; (c) 4-nitrothiophenol, PPh_3, DIAD, toluene, 80 °C.

Table 1 Antiproliferative activity of compounds **1–7** against cancer (HL60, JURKAT, MDA-MB-231 and LNCaP) and normal cells (VERO and PBMC)

	IC_{50}[a] (μM)					
Compound	HL60	JURKAT	MDA-MB-231	LNCaP	VERO	PBMC
1	28 ± 2	>50	37 ± 2	>80	50 ± 7	>100
2	32 ± 11	48 ± 4	25 ± 6	>80	>100	85 ± 33
3	18 ± 3	42 ± 9	32 ± 12	27 ± 1	>100	>100
4	23 ± 1	46 ± 8	32 ± 5	11 ± 4	>100	>100
5	>50	>50	>80	>80	ND[b]	ND[b]
6	20 ± 6	>50	>80	37 ± 5	ND[b]	ND[b]
7	>50	>50	>80	>80	ND[b]	ND[b]

[a] Each dataset represents mean ± SD of at least three independent experiments. In PBMC was carried out 8 independent experiments. [b] Not determined.

Olefins **3** and **4** were equally potent than lead **1** against MDA-MB231 cells. However, **3** and **4** were more active than **1** against HL60, JURKAT and LNCaP cells. Considering the LNCaP cell line (which is a cell line derived from a lymph node metastasis of a prostate adenocarcinoma), compounds **3** and **4** were the most active (IC_{50} = 27 and 11 μM) in this series. It is worth noting that lead compounds were inactive against this line (IC_{50} > 80 μM) and *E*-olefin **4** was at least 7 times more potent than lead **1**. Besides, prostate is the most common cancer in men, and thus there is a great need to develop new alternatives for this cancer type. In view of our previous results on the importance of the aromatic ring for the potency of this class, it was expected that alkenes *E* and *Z* would have different activities against cancer cells since the ring is positioned in opposite sides in these isomers.[13] However, this effect was noted only on LNCaP cells. In this case, rigidification of the structure using a double bond spacer enhances the activity. The *E*-isomer was about 3 times more potent than the *Z*-isomer. Other interesting fact is the great selectivity for cancer cells. Compound **4** showed good selectivity for these cancer cell lines with a selectivity index of >9 for LNCaP cells. Furthermore, **4** possesses drug-like physicochemical properties (MW = 348, 7 H-acceptor, 0 H-donors, 5 freely rotable bonds, $c\log P$ = 4.6). The conformationally restricted amide **5** was inactive against all cancer cells. Probably, amide **5** adopts an unfavorable conformation to bind with the molecular target.

Comparing compound **6** with lead **2**, it was observed that **6** is less active against JURKAT and MDA-MB-231 cells and equally potent against HL60 cells. However, compound **6** showed a relevant activity against the LNCaP cell line (IC_{50} = 37 μM) while lead **2** was inactive against this cell line (IC_{50} > 80 μM). Thus, isosteric replacement of –O– with $–CH_2–$ seems to be favorable on LNCaP cells. It appears that oxygen is not involved in the H-bond and methylene fairly changes the electron density of the

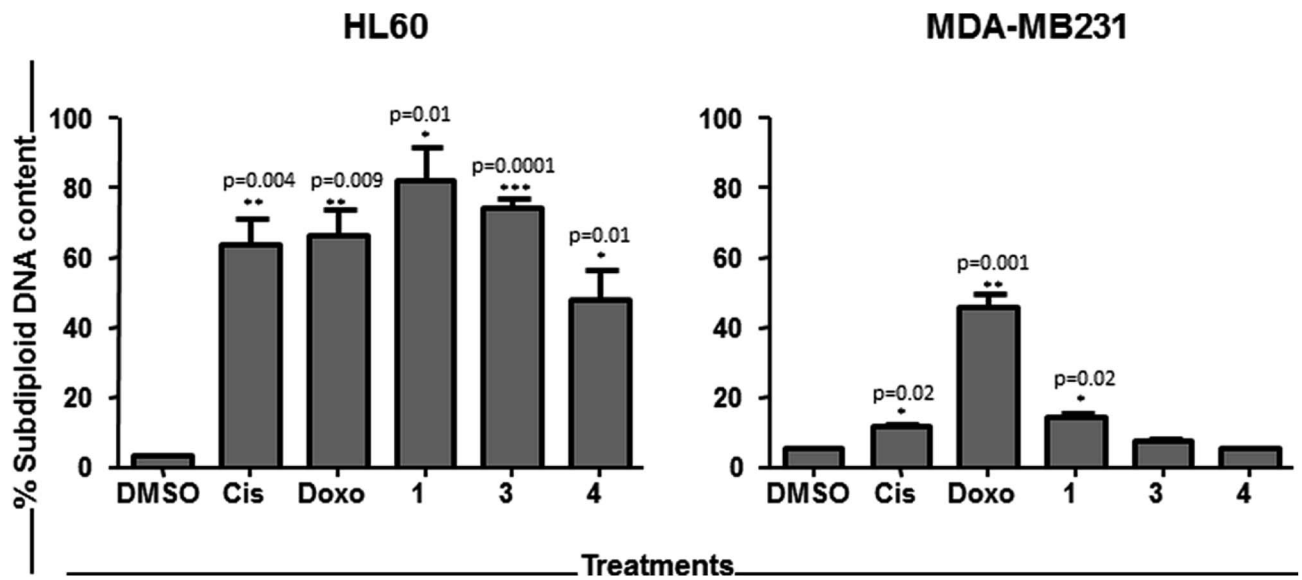


Fig. 8 Effects of compounds **1**, **3** and **4** on the DNA content of HL60 and MDA-MB231 cells. The DNA content was assessed by staining with propidium iodide and flow cytometric analysis. Cisplatin (cis) and doxorubicin (doxo), as positive controls are also shown. Representative data with mean ± SD from two independent experiments performed in duplicate.

ring. This could partially explain the good results obtained with alkenes **3** and **4** on LNCaP cells. Finally, isosteric replacement with sulfur was not tolerated. Compound **7** was inactive against these four cell lines. The larger size of sulfur as compared to oxygen or oxidation at sulfur may be responsible for the inactivity of compound **7**. This steric effect is in accordance with previous observations that *ortho*-substituted compounds are inactive.

Compounds **1**, **3** and **4** were incubated at 50 μM with HL60 or MDA-MB231 cells. After 24 h, DNA fragmentation was evaluated (Fig. 8). Significant increases in DNA fragmentation were detected after treatment with all compounds at 50 μM in HL60 cells. However, corresponding increases were not observed in MDA-MB231 cells. Considering HL60 cells, compound **1** induced more DNA fragmentation in comparison with **3** and **4**. Thus, the $-OCH_2-$ spacer is favorable to enhance the pro-apoptotic potential. In comparing isomers **3** and **4**, isomer *Z* **3** had more pro-apoptotic potential than *E* **4** in this case. It is possible that these compounds are involved in the same pathway for HL60 cells. Nevertheless, the apoptotic pathway does not appear to be important in MDA-MB231 cells. Thus, apparently this class modulates more than one pathway.

Conclusions

In summary, we described herein the synthesis and cytotoxic evaluation of a series of analogues of chiral oxazolidine **1** and **2** designed by isosteric replacement or rigidification of the oxymethylene spacer. Introduction of a double bond was well tolerated in almost all cases. Alkene *E* **4** had a relevant activity against LNCaP with an IC_{50} value of 11 μM without affecting Vero or PBMC cell proliferation. It was about 3 times more active than the *Z*-isomer on this cancer cell line. Besides, compound **4** has drug-like physicochemical properties. Thus, compound **4** has potential for further development as an anticancer agent. Rigidification using amide or isosteric replacement did not enhance the activity of this class and will not be considered for further modifications.

Notes and references

1 A. Sehgal, *Curr. Med. Chem.*, 2003, **10**, 749–755.
2 G. B. Rocque and J. F. Cleary, *Nat. Rev. Clin. Oncol.*, 2013, **10**, 80–89.
3 S. Fortin, L. Wei, E. Moreau, J. Lacroix, M.-F. Côté, E. Petitclerc, L. P. Kotra and R. C. Gaudreault, *Eur. J. Med. Chem.*, 2011, **46**, 5327–5342.
4 J. D. Merriman, M. Dodd, K. Lee, S. M. Paul, B. A. Cooper, B. E. Aouizerat, P. S. Swift, W. Wara, L. Dunn and C. Miaskowski, *Canc. Nurs.*, 2011, **34**, 345–353.
5 S. Fletcher, E. P. Keaney, C. G. Cummings, M. A. Blaskovich, M. A. Hast, M. P. Glenn, S.-Y. Chang, C. J. Bucher, R. J. Floyd, W. P. Katt, M. H. Gelb, W. C. van Voorhis, L. S. Beese, S. M. Sebti and A. D. Hamilton, *J. Med. Chem.*, 2010, **53**, 6867–6888.
6 G. A. Tunbridge, J. Oram and L. Caggiano, *MedChemComm*, 2013, **4**, 1452–1456.
7 X. Zheng, F. F. Zhao, Y. M. Liu, X. Yao, Z. T. Zheng, X. Luo and D. F. Liao, *Med. Chem.*, 2010, **6**, 6–8.
8 E. Leung, J. M. Hung, D. Barker and J. Reynisson, *MedChemComm*, 2014, **5**, 99–106.
9 J. K. Buolamwini, *Curr. Opin. Chem. Biol.*, 1999, **3**, 500–509.
10 V. A. Kulchitsky, V. I. Potkin, Y. S. Zubenko, A. N. Chernov, M. V. Talabaev, Y. E. Demidchik, S. K. Petkevich, V. V. Kazbanov, T. A. Gurinovich, M. O. Roeva, D. G. Grigoriev, A. V. Kletskov and V. N. Kalunov, *Med. Chem.*, 2012, **8**, 22–32.
11 N. Kumar, V. P. Raj, B. S. Jayshree, S. S. Kar, A. Anandam, S. Thomas, P. Jain, A. Rai and C. M. Rao, *Chem. Biol. Drug Des.*, 2012, **80**, 291–299.
12 M. C. X. Pinto, D. F. Dias, H. L. del Puerto, A. S. Martins, A. T. Carvalho, O. A. Martins-Filho, B. Badet, P. Durand, R. J. Alves and E. M. Souza-Fagundes, *Life Sci.*, 2011, **89**, 786–794.
13 S. F. Andrade, E. F. S. Campos, C. S. Teixeira, C. C. Bandeira, S. N. Lavorato, N. C. Romeiro, C. M. Bertollo, M. C. Oliveira, E. M. Souza-Fagundes and R. J. Alves, *Med. Chem.*, 2013, in press.
14 S. Nagata, *Exp. Cell Res.*, 2000, **256**, 12–18.
15 F. W. Fesik, *Nat. Rev. Cancer*, 2005, **5**, 876–885.
16 S. Tanida, T. Mizoshita, K. Ozeki, H. Tsukamoto, T. Kamiya, H. Kataoka, D. Sakamuro and T. Joh, *Int. J. Surg. Oncol.*, 2012, **2012**, 1–8.
17 H. Mizutani, S. Tada-Oikawa, Y. Hiraku, M. Kojima and S. Kawanishi, *Life Sci.*, 2005, **76**, 1439–1453.
18 D. Yoo, J. S. Oh, D.-W. Lee and Y. G. Kim, *J. Org. Chem.*, 2003, **68**, 2979–2982.
19 P. Garner and J. M. Park, *Org. Synth.*, 1992, **70**, 18–24.
20 F. W. Foss Jr, A. H. Snyder, M. D. Davis, M. Rouse, M. D. Okusa, K. R. Lynch and T. L. Macdonald, *Bioorg. Med. Chem.*, 2007, **15**, 663–677.
21 B. Duthion, T.-X. Metro, D. G. Pardo and J. Cossy, *Tetrahedron*, 2009, **65**, 6696–6706.
22 A. Ollivier, M. Goubert, A. Tursun, I. Canet and M.-E. Sinibaldi, *ARKIVOC*, 2010, **9**, 108–126.
23 B. D. Palmer, A. M. Thompson, R. J. Booth, E. M. Dobrusin, A. J. Kraker, H. H. Lee, E. A. Lunney, L. H. Mitchell, D. F. Ortwine, J. B. Smaill, L. M. Swan and W. A. Denny, *J. Med. Chem.*, 2006, **49**, 4896–4911.
24 A. Dondoni and D. Perrone, *Org. Synth. Coll.*, 2004, **10**, 320–326.
25 T. B. Lanni, K. L. Greene, C. N. Kolz, K. S. Para, M. Visnick, J. L. Mobley, D. T. Dudley, T. J. Baginski and M. B. Liimatta, *Bioorg. Med. Chem. Lett.*, 2007, **17**, 756–760.
26 S. Miljanic, L. Frkanec, Z. Meic and M. Zinic, *Eur. J. Org. Chem.*, 2006, **5**, 1323–1334.
27 S. Raghavan, A. Rajender, S. C. Joseph, M. A. Rasheed and K. R. Kumar, *Tetrahedron: Asymmetry*, 2004, **15**, 365–379.
28 R. Pellicciari, M. Marinozzi, G. Costantino, B. Natalini, F. Moroni and D. P. Giampietro, *J. Med. Chem.*, 1999, **42**, 2716–2720.

29 D. J. Chambers, G. R. Evans and A. J. Fairbanks, *Tetrahedron: Asymmetry*, 2005, **16**, 45–55.
30 S. F. Andrade, R. C. Figueiredo, J. D. Souza-Filho and R. J. Alves, *J. Braz. Chem. Soc.*, 2012, **23**, 1062–1069.
31 M. Falorni, S. Conti, G. Giacomolli, S. Cossu and F. Soccolini, *Tetrahedron: Asymmetry*, 1995, **6**, 287–294.
32 D. F. Dias, C. Roux, P. Durand, B. Iorga, M. A. B. Denisot, B. Badet and R. J. Alves, *J. Braz. Chem. Soc.*, 2010, **21**, 680–685.