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Triazenes as inhibitors of HIV-1 and HCoV-OC43: A structure-activity relationship study

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ABSTRACT

Triazenes, or amino-substituted diazenes, are organic compounds containing three contiguous nitrogen atoms, that have potent biological activities. We previously demonstrated that triazenes, particularly those substituted with a phenyl or 3-pyridyl ring at the 1-position and a 2-pyridyl ring at the 3-position, exhibit anti-DENV properties. Here, we evaluated the antiviral activity against a betacoronavirus (HCoV-OC43) and a lentivirus (HIV-1). 1-(4-trifluoromethylphenyl)-2-imidazole-1-yldiazene (**21**) exhibited broad-spectrum activity (EC_{50} = 6.6–6.8 μM) but was cytotoxic to THP-1 cells. Pyridyl triazenes (**14, 15**) were the most potent against HCoV-OC43, while 1-(4-methoxyphenyl)-2-morpholin-4-yldiazene (**6**) and 1-(4-methoxyphenyl)3-(-6-methylpyridin-2-yl)triazene (**10**) inhibited HIV-1 the most. Structure–activity relationship analysis, supported by molecular docking, indicated that *para*-methoxy groups favored interactions with viral enzyme binding pockets, enhancing antiviral potency, while *meta* and *para*-trifluoromethyl groups were associated with reduced activity and increased cytotoxicity. These findings support the further development of triazenes as antiviral scaffolds.

1. Triazenes as antiviral compounds

Triazenes, or amino-substituted diazenes, are a class of organic compounds characterized by one double-bonded and two single-bonded nitrogen atoms. This unique chemical structure contributes to the versatility of their functions, including their use as anti-cancer drugs, primarily through their ability to alkylate DNA [1,2]. Dacarbazine and temozolomide are two FDA-approved antineoplastic examples of triazenes used in chemotherapy. Their success in cancer treatment has spurred further investigation into their biological activity in other areas of medical research. For instance, diminazene is an antiparasitic drug used in animals to prevent infections caused by various protozoa [3].

In vitro and *in cellulo,* triazenes have demonstrated antibacterial [4–7], antimalarial [8], and antifungal activities [9,10]. Triazenes' antibacterial properties have been associated with their ability to chelate metal ions in the microorganism's cell wall, impeding its synthesis [11]. The anti-cancer, antifungal, and antiparasitic properties were linked, instead, to their ability to form reactive methyldiazonium cations, capable of alkylating nucleic acids and proteins [12,13]. Despite the possible implications of triazenes' ability to block DNA synthesis and interact with RNA, their role in combating viral infections remains underexplored.

Recently, we demonstrated that derivatives, including phenyl,

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List of abbreviations

DENV	Dengue virus
HCoV-OC43	Human coronavirus OC43
HIV-1	Human immunodeficiency virus type 1
RdRp	RNA-dependent RNA polymerase
RT	Reverse transcriptase
Pro	Protease
M^{Pro}	Main protease
NS2B/NS3	Nonstructural proteins 2B and 3 (DENV protease)
CC_{50}	concentration leading to 50 % of cell death
EC_{50}	concentration leading to 50 % reduction of virus-infected cells
SAR	Structure–activity relationship
THP-1	Tohoku Hospital Pediatrics-1, human monocytic leukemia cell line
HCT-8	Human colon tumor (adenocarcinoma) cell line
Huh7	Human hepatocellular carcinoma cell line
NN inhibitor	Non-nucleoside inhibitor
CF_3	Trifluoromethyl group
OCH_3	Methoxy group
Br	Bromine
Cl	Chlorine
kcal/mol	Kilocalories per mole

phenyl-pyridine, imidazolo, and pyridyl, could impede cell infection with the dengue flavivirus [14]. Here, we explore the *in silico* and *in cellulo* effects of triazenes on infection by representative viruses from the betacoronavirus and lentivirus families, which continue to pose significant threats to global public health.

2. Structural classification of included triazenes

This study includes an analysis of the synthesized compounds described by Ref. [14], as well as novel derivatives 1-(4-methoxyphenyl)-2-morpholin-4-yldiazene (**6**), 1-(3-pyridyl)-3,3-diethyltriazene (**22**), 1-(3-pyridyl)-3-phenylethyltriazene (**26**), 1-(8-quinolinyl)-3,3-diethyltriazene (**27**), and 1-(3-pyridyl)-2-indolin-1-yldiazene (**29**) (Table S1 and S2 and Supplementary Material and methods, Fig. S1–5). Triazenes were synthesized in aqueous media (**1**–**21**) (Table S1) or organic solvents (**22**–**30**) (Table S2). The compounds were grouped into four clusters (I–IV) to facilitate comparison based on structural similarities, specifically variations in the R groups attached to the triazene core (Fig. 1). Structures incorporating an imidazole group (**20**, **21**), morpholine (**28**), indole (**29**), and quinoline (**27**) were named by considering these triazenes as amino-substituted diazenes.

Group I, 1-phenyl- and quinoline or morpholine-containing triazenes, is comprised of 1-phenyl-3,3-diethyltriazene **(1), 1-(4-methoxyphenyl)-3,3-diethyltriazene (2), 1-(4-trifluoromethylphenyl)- 3,3-diethyltriazene (3), 1-(4-trifluoromethylphenyl)-3-propargyltriazene (4), 1-(4-trifluoromethylphenyl)-2-(8-aminoquinolin-5-yl)diazene (5), and, (6), characterized by a phenyl group attached to the triazene backbone** (Fig. 1)**. (27**) has a quinoline group attached to the triazene backbone instead.

Group II, 1-phenyl-3-pyridyl triazenes, includes 1-phenyl-3-pyridin-2-yltriazene (**7**), 1-(*o*-tolyl)-3-pyridin-2-yltriazene (**8**), and other derivatives with a phenyl group at position one and a pyridyl group at the third nitrogen atom of the triazene backbone, such as 1-(4-methoxyphenyl)-3-pyridin-2-yltriazene (**9**), 1-(4-methoxyphenyl)3-(-6-methylpyridin-2-yl)triazene (**10**), 1-(4-bromophenyl)-3-pyridin-2-yltriazene (**11**), 1-(4-chlorophenyl)-3-pyridin-2-yltriazene (**12**), 1-(4-trifluoromethylphenyl)-3-pyridin-2-yltriazene (**13**), 1-(3-bromophenyl3-pyridin-2-yltriazene (**14**), 1-(3-chlorophenyl)- 3-pyridin-2-yltriazene (**15**), 1-(3-trifluoromethylphenyl)- 3-pyridin-2-yltriazene (**16**), 1-phenyl-3-pyridin-4-yltriazene (**17**), 1-(4-methoxyphenyl)-3-pyridin-4-yltriazene (**18**), and 1-(4-trifluoromethylphenyl)-2-pyridin-4-aminodiazene (**19**).

Group III, 1-phenyl-3-imidazole triazenes, includes two compounds, 1-(4-methoxyphenyl)-2-imidazole-1-yldiazene **(20) and 1-(4-trifluoromethylphenyl)-2-imidazole-1-yldiazene (21**), both featuring a phenyl group attached to first nitrogen of triazene backbone (position 7) and an imidazole encompassing the third nitrogen atom of the triazene backbone.

Group IV, pyridine-containing triazenes, comprises **(22)**, 1-(3-pyridyl)- 3-methyl-3-isopropyltriazene (**23**), 1-(3-pyridyl)-3-methyl-3-(2-methoxy-ethyl)triazene **(24)**, 1-(3-pyridyl)-3-methyl-3-phenyltriazene **(25)**, 1-(3-pyridyl)-3-phenylethyltriazene (**26**), 1-(3-pyridyl)-2-morpholin-4-yldiazene (**28**), 1-(3-pyridyl)-2-indolin-1-yldiazene (**29**), 1-(3-pyridyl)-2-tetrahydroquinolin-1-yldiazene (**30**), all featuring a *meta*-substituted pyridine attached to the first nitrogen of the triazene backbone, with various functional groups attached to the third nitrogen atom of the triazene backbone.

3. Antiviral and cytotoxic activity

Compounds were tested for antiviral and cytotoxic activity at concentrations ranging from 0.4 to 121.5 μM. Group I compounds did not display significant anti-DENV activity (Table S3, [14]); however, (**2**) showed toxicity towards hepatocarcinoma Huh7 cells, while (**6**) and (**5**) were cytotoxic to adenocarcinoma HCT-8 cells (CC_{50} = 12.8–12.9 μM; Table 1, Fig. 2A, Fig. S6A). (**2**) was the only one to display a weak but specific inhibition of HCoV-OC43 infection levels (EC_{50} = 20.6 μM), while (**2**), (**6**), and (**27**) blocked VSV-G-pseudotyped HIV-1 vector replication at noncytotoxic concentrations.

(**20**) and (**21**), from Group III, were previously shown to exhibit anti-flaviviral activity [14]. Here, (**20**) inhibited HCoV-OC43 with EC_{50} = 3.1 μM and CC_{50} = 71 μM (Fig. 2A–Table 1). (**21**) reduced HCoV-OC43 and HIV-1 infection levels with similar EC_{50} (6.6 and 6.8 μM, respectively), but the compound was toxic to THP-1 cells (CC_{50} = 19.7 μM) (Fig. S6A).

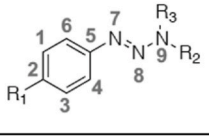
Among Group II (triazene scaffolds with phenyl and pyridine rings) molecules, (**7**), (**9**), (**10**), (**11**), (**14**), (**15**), and (**18**) were shown to dampen $DENV_{GFP}$ infection (Table S3, [14]), and many could also inhibit HCoV-OC43 (Fig. 2B, and Table 1). The most potent compounds against the betacoronavirus were (**14**) and (**15**) (EC_{50} = 0.8 μM and 1.2 μM, respectively), followed by (**7**) and (**8**) (EC_{50} = 3.8 μM and 3.9 μM), and by (**10**), (**12**) and (**17**) (EC_{50} = 8.3, 7.1, and 7.8 μM, respectively), while (**9**), (**11**) and (**18**) displayed inhibition at higher concentration (EC_{50} > 25 μM) (Table 1). In the case of HIV-1, (**10**) and (**18**) were the most potent (EC_{50} = 5 μM). Overall, compounds that inhibited the coronavirus also inhibited the flavivirus, while fewer were able to reduce HIV-1 infection levels. Most derivatives were cytotoxic to hepatocarcinoma cells (Huh-7, Table S3), but they did not display toxicity towards adenocarcinoma (HCT-8) and leukemic myeloid cells (THP-1), except for compound (**7**), with CC_{50} = 21 μM towards HCT-8 (Fig. S6B).

In group IV, pyridine triazene derivatives (**25**) and (**30**) were previously shown to display anti-flaviviral activity [14]. Only (**26**) consistently inhibited HCoV-OC43 infection (EC_{50} = 3.8 μM), while (**22**) and (**26**) displayed weak to moderate lentiviral inhibition (EC_{50} = 15.9 and 34.3 μM, respectively; Table S4, Fig. 2C, Fig. S6C).

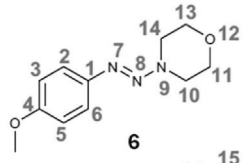
4. Structure-activity relationship (SAR), insights from functional groups and molecular docking

To identify SARs among diverse triazene derivatives, we analyzed antiviral activity data in conjunction with molecular docking to key viral enzymes (Table S4–6, Fig. S8). Among group I compounds, only (**2**) and (**6**) exhibited specific anti-HIV-1 activity. They both featured a *para*-methoxy (-OCH_3) substituent on the phenyl ring. This electron-donating

Group I



	R_1	R_2	R_3
1	H	Et	Et
2	MeO	Et	Et
3	CF_3	Et	Et
4	CF_3	H	Propyn-3-yle
5	CF_3	H	Quinolin-8-yle



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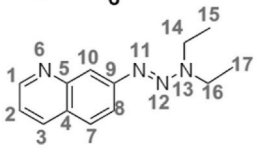

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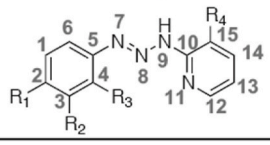
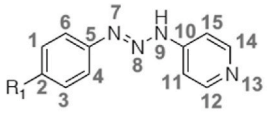
Group II

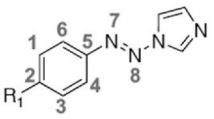


	R_1	R_2	R_3	R_4
7	H	H	H	H
8	H	H	Me	H
9	MeO	H	H	H
10	MeO	H	H	Me
11	Br	H	H	H
12	Cl	H	H	H
13	CF_3	H	H	H
14	H	Br	H	H
15	H	Cl	H	H
16	H	CF_3	H	H



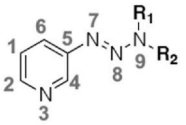
	R_1
17	H
18	MeO
19	CF_3

Group III

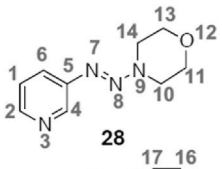


	R_1
20	MeO
21	CF_3

Group IV



	R_1	R_2
22	Et	Et
23	Me	i-Pr
24	Me	methoxymethyl
25	Me	phenyl
26	Me	Ethyl benzene



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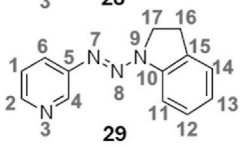

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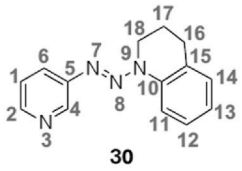

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Fig. 1. Triazene structures groups based on their substitution patterns. The main chain of Group I is a phenyl group attached to the triazene backbone's first nitrogen (position 7). In Group II, a phenyl group is attached at position 7 and a pyridine group is present at the triazene backbone's third nitrogen (position 9). In Group III, a phenyl group is attached to position 7 and an imidazole group to position 9 of the triazene backbone. In Group IV, the main chain features a *meta*-pyridine at position 7 of the triazene backbone, while different R groups are present in position 9.

Table 1
Antiviral activity of triazene derivatives.

		HCoV-OC43			HIV		
		EC_{50} (μM)	CC_{50} (μM)	SI	EC_{50} (μM)	CC_{50} (μM)	SI
	2	20.6 ± 0.5	>100	>4	13.9 ± 4.9	n.d.	>7
	5	18.2 ± 0.3	12.9	<1	>100	>100	n.a.
	6	26.4 ± 0.5	12.8 ± 0.1	<1	8.1 ± 5.1	92.3 ± 10.9	11
	27	>100	>100	n.a.	22.6 ± 14.2	90.8 ± 13.0	4
Group II	**7**	3.8 ± 0.5	21.0 ± 5.7	6	>100	>100	n.a.
	8	3.9 ± 0.8	>100	>25	>100	>100	n.a.
	9	25.4 ± 4.7	>100	>3	55.6 ± 1.1	>100	>1
	10	8.3 ± 0.4	>100	>12	5.1 ± 2.2	>100	>19
	11	39.1 ± 0.4	>100	>2	>100	>100	n.a.
	12	7.1 ± 0.3	>100	>14	>100	>100	n.a.
	14	0.8 ± 0.4	>100	>125	>100	>100	n.a.
	15	1.2 ± 1.2	>100	>83	>100	>100	n.a.
	17	7.8 ± 1.9	>100	>12	>100	>100	n.a.
	18	30.1 ± 1.4	>100	>3	5.2 ± 3.0	>100	>19
Group III	**20**	3.1 ± 0.1	70.7 ± 11.0	23	n.t.	n.t.	n.a.
	21	6.6 ± 2.0	>100	>15	6.8 ± 1.9	19.7 ± 15.5	>2
Group IV	**22**	>100	>100	n.a.	15.9 ± 5.8	>100	>6
	25	>100	>100	n.a.	64.9 ± 2.1	>100	>1
	26	3.8 ± 0.8	>100	>26	34.3 ± 3.7	>100	>2
	28	>100	>100	n.a.	55.0 ± 10.1	>100	>1
	29	>100	>100	n.a.	29.5 ± 0.9	>100	n.a.
	30	>100	>100	n.a.	5.7 ± 0.4	>100	n.a.

>100 means that activity was not detected at the highest concentration tested, n.t. not tested, n.a. not applicable. The EC_{50} and CC_{50} values specific to OC43 and HIV-1 of compounds (**1**), (**3**), (**4**), (**13**), (**16**), (**19**), (**23**), and (**24**) were all greater than 100 μM; therefore, they were excluded from the table.

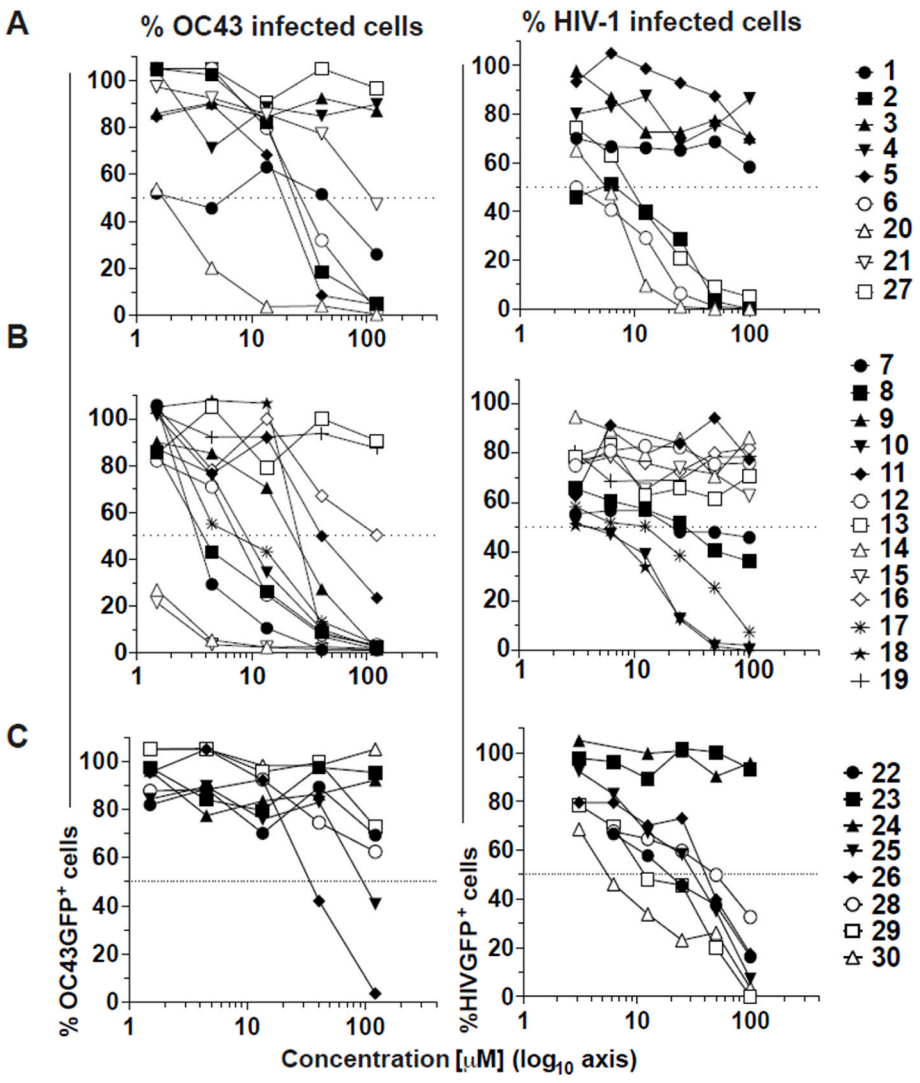

Fig. 2. Antiviral properties of triazene derivatives. HCT-8 cells and THP-1 cells were treated with Group I and III (**A**), Group II (**B**), and Group IV (**C**) triazenes. Cells were either infected with OC43 or with HIV-1. Infection levels (% GFP-positive cells) were determined by flow cytometry.

group could promote target binding, possibly through hydrogen bonding with active site residues, as observed with Asp29 and Asp30 in the HIV-1 protease active site (Table S6), or enhance compound solubility. By contrast, the presence of trifluoromethyl (CF_3), a powerful electron-withdrawing group, at the same position reduced antiviral activity in (**3**), (**4**), and (**5**), possibly due to unfavorable electronic or steric effects. The quinoline scaffold of compound (**27**) also conferred anti-HIV-1 activity, consistent with known quinoline-based antivirals [15].

Within group III, the imidazole of 1-phenyl-3-imidazolium triazenes (**20**) and (**21**) displayed potent anti-HCoV-OC43 activity. Docking results suggested that the imidazole participated in salt-bridge and hydrogen-bond interactions with key substrate recognition site residues in HCoV-OC43 protease (Fig. S8), consistent with the prediction of its involvement in compounds inhibiting SARS-CoV-2 M^{Pro} in recent computational studies [16]. The imidazole group was also predicted to form favorable interactions with catalytic Asp residues involved in RNA template positioning and Mg^{2+} coordination in the DENV and HCoV-OC43 RdRp active sites (Fig. S7 and S8, Table S4 and S5) [17,18]. Our results suggest that this group could confer broad antiviral activity. Replacing the O-CH_3 (**20**) with CF_3 in (**21**) at the *para* position of the phenyl ring maintained (HCoV-OC43) or increased antiviral activity and conferred toxicity to THP-1 leukemic cells. CF_3 has been shown to enhance the lipophilicity of molecules, thereby facilitating their ability to cross cell membranes and access intracellular targets, which may lead to increased anti-cancer effects [19].

By contrast, in the case of 1-phenyl-3-pyridyl triazenes (group II), inactive compounds, *i.e.* (**13**), (**16**), and (**19**), all contained a CF_3 group at the *meta* or *para* position of the phenyl ring, suggesting that it could cause steric hindrance or alter electrostatic interactions at target sites [20]. Triazenes with *para*-OCH_3 groups, (**10**) and (**18**) with anti-HIV-1 activity docked within the non-nucleoside (NN) inhibitor (I) binding pocket of RT, engaging in hydrophobic interactions with key residues Tyr188 and Trp229, like (**21**), and known NNRTIs efavirenz and rilpivirine (Table S6, Fig. S8C) [21,22]. (**14**) and (**15**), which bear *meta*--position halogens, exhibited the most potent anti-coronaviral activity in cell-based assays. Blind docking placed these compounds near the RdRp active site, where halogen groups enhanced hydrophobic packing and helped orient the triazene scaffold toward catalytic residues involved in *de novo* RNA initiation [23,24] (Fig. S8, and Table S4–5). Similar to most triazenes, the docking scores and binding modes (involvement of interaction with catalytic residues) were more specific towards the RdRps or the RT than towards the viral proteases. (**11**) and (**12**), which had these halogen groups at the *para* position, exhibited less activity, confirming that the position-specific electronic or steric effects of the substituents on the benzene ring affected the antiviral activity [25]. In group IV, very few displayed antiviral properties, suggesting that the

pyridine ring with a bulky hydrophobic tail in (**23**) and (**24**) may have hindered target enzyme binding.

5. Conclusion

This study highlights the antiviral potential of triazene derivatives against HCoV-OC43 and HIV-1. Several compounds, notably those with *para*-methoxy and *meta*-halogen substitutions, showed specific antiviral effects at non-cytotoxic concentrations. In contrast, trifluoromethyl groups were associated with increased cytotoxicity and reduced efficacy in most scaffolds. Docking analyses hinted that viral polymerases could be the molecular targets of triazenes, but this remains to be characterized in future studies. This study positions triazenes as promising scaffolds for the development of antivirals with potential broad-spectrum applications.

CRediT authorship contribution statement

Natacha Mérindol: Writing – review & editing, Writing – original draft, Visualization, Supervision, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Seyedeh Mahsa Hashemian:** Writing – review & editing, Validation, Investigation, Formal analysis, Data curation. **Seynabou Sokhna:** Writing – review & editing, Investigation, Formal analysis, Data curation, Conceptualization. **Marie-Pierre Girard:** Writing – review & editing, Investigation, Formal analysis. **Marc Presset:** Writing – review & editing, Investigation, Formal analysis. **Insa Seck:** Writing – review & editing, Formal analysis. **Lalla Aïcha Ba:** Writing – review & editing, Formal analysis. **Seydou Ka:** Writing – review & editing, Formal analysis. **Samba Fama Ndoye:** Writing – review & editing, Formal analysis. **Issa Samb:** Writing – review & editing, Formal analysis. **Erwan Le Gall:** Writing – review & editing, Validation, Resources, Conceptualization. **Lionel Berthoux:** Writing – review & editing, Validation, Resources. **Matar Seck:** Writing – review & editing, Validation, Resources, Project administration, Conceptualization. **Isabel Desgagné-Penix:** Writing – review & editing, Visualization, Validation, Supervision, Resources, Project administration, Funding acquisition, Conceptualization.

Declaration of generative AI and AI-assisted technologies in the writing process

Statement: During the preparation of this work, the authors used OpenAI ChatGPT to correct syntactic and grammatical errors. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Isabel Desgagne-Penix reports financial support was provided by Canada Research Chairs Program. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejmcr.2025.100288.

Data availability

Data will be made available on request.

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