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Protective Antioxidant Effect of Argan Oil Compared with Olive, Colza and Cactus Seed Oils against Lipopolysaccharide-Induced Oxidative Stress in the Mouse Heart

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ABSTRACT: Injection of lipopolysaccharide (LPS) reproduces several features of Gram-negative sepsis in mice, among them an oxidative imbalance documented in liver and brain but rarely in cardiac tissue. We compared virgin argan oil (AO) from *Argania spinosa* (L.) Skeels with olive (OO), colza (CO) and cactus seed (CSO) oils for protection of the mouse heart against that challenge. The Swiss OF1 mouse model was given a diet in which 6% of the fats came from a single oil for 28 consecutive days. This diet was followed by injection of LPS extracted from *Escherichia coli* O111:B4 strain. Activities of enzymes super oxide dismutase (SOD) and catalase (CAT), levels of reduced glutathione (GSH) as well as malondialdehyde (MDA) were assessed in myocardial homogenates which were expressed in relation to total protein concentration (n = 5 for each group).. The heart responded unlike the organs described previously. LPS on its own left SOD and CAT activities and the GSH pool close to control values, and no macroscopic lesion was seen. Olive oil was the exception among the single-oil groups: SOD activity reached 95 ± 22 against 43 ± 14 U/mg protein in controls ($p = 0.0012$). Under LPS challenge, argan oil pretreatment raised cardiac GSH to about 2210 $\mu\text{mol/mg}$ protein against 860 in the LPS group ($p < 0.008$), while the three other oils lowered it below both LPS and control levels. Differences in CAT activity and in MDA content did not reach significance ($p > 0.99$). Argan oil therefore preserves the reduced glutathione pool of the challenged heart, and all four oils limited lipid peroxidation, but the cardiac response to LPS is milder than the hepatic and cerebral responses reported so far.

1. INTRODUCTION

Vegetable oils differ less in their energy content than in what they carry alongside it: fatty acids in varying proportions, and a minor fraction of sterols, tocopherols and phenolic compounds whose metabolic effects have been studied for three decades [1,2]. Argan oil, pressed from the almonds of *Argania spinosa* (L.) Skeels, is unusual on both counts. Oleic and linoleic acids occur in comparable amounts, and the unsaponifiable fraction contains schottenol and spinasterol, two sterols almost absent from other edible oils, together with γ -tocopherol and phenolic acids [2,3].

Research on the oil has followed two lines. One concerns lipid metabolism and cardiovascular risk: consumption improves paraoxonase activity and antioxidant status in healthy men [4], a phenolic extract inhibits low-density lipoprotein oxidation and promotes cholesterol efflux from macrophages [5], and supplementation lowers plasma lipids in high-fat-fed rats [6]. The other concerns antioxidant capacity as such, which is what motivated the present work [3].

A change in the equilibrium between reactive oxygen species and the processes that eliminate them is referred to as oxidative stress [7]. According to research, SOD, CAT, and GPx are enzymes used as cell protective agents while superoxide dismutase, vitamin E, and carotenoids are substances used for protective functions within the cells. Gram-negative bacteria stimulate the release of cytokines through the action of LPS which consequently causes the generation of ROS as well as damage in the organs.

Where argan oil has been tested against LPS, the target has been the liver or the brain [9,10]. The heart is less well covered, although oxidative stress contributes to most forms of cardiac and vascular disease [11]. We measured SOD, CAT, GSH and MDA in the hearts of mice fed argan, olive, colza or cactus seed oil and then challenged with LPS.

2. RESULTS

2.1. Composition of the studied oils

Fatty acid compositions of the four oils are presented in Table 1. The first oil which is argan oil contains oleic acid at a range of 43-49% and linoleic acid at between 29 and 36%. Olive oil and rapeseed oil ranked first in oleic acid content at 82% and 63% respectively while in prickly pear oil, linoleic acid (53.5%) is greater than oleic acid (18.3%), along with palmitic acid reaching a level of 20.1%. Tocopherol profiles diverge as sharply (Table 2). γ -Tocopherol accounts for roughly 90% of the tocopherols of argan oil, which also has the highest total content at 752 mg/kg; olive oil is dominated by the α form and colza oil by the δ form. Ferulic acid makes up most of the phenolic fraction of argan oil, 3147 of 3263 μ g/kg (Table 3).

Table 1. Fatty acid composition of the four studied oils, expressed as a percentage of the total triglyceride fraction.

Oil	Palmitic	Stearic	Oleic	Linoleic	Linolenic
<i>Argan oil</i>	11.5–15	4–7	43–49	29–36	0.1
<i>Olive oil</i>	7.5–15.6	1.4–3.4	60.9–82.1	4.5–16.1	0.4–1.2
<i>Colza oil</i>	4.5	1.6	63	19	9
<i>Cactus seed oil</i>	20.1	2.72	18.3	53.5	—

Values are percentages of total fatty acids. Adapted from El-Mostafa et al. and El Kharrassi et al. [12,3].

Table 2. Tocopherol distribution of the four oils (percentage of total tocopherols) and total tocopherol content.

Form	Argan oil	Colza oil	Cactus seed oil	Olive oil
<i>α-tocopherol</i>	5.86	24.03	56	88.01
<i>β-tocopherol</i>	0.12	—	12	7.57
<i>δ-tocopherol</i>	4.04	69.01	33	—
<i>γ-tocopherol</i>	89.98	6.96	5	4.42
<i>Total (mg/ kg)</i>	752.20	100	106	115.80

Adapted from El Kharrassi et al., Essadek et al. and El-Mostafa et al. [3,10,12].

Table 3. Phenolic composition of argan oil ($\mu\text{g/kg}$ of oil).

Phenolic compound	$\mu\text{g/kg}$ of oil
<i>Vanillic acid</i>	67
<i>Syringic acid</i>	37
<i>Ferulic acid</i>	3147
<i>Tyrosol</i>	12
<i>Total</i>	3263

Adapted from El Kebbaj [13].

2.2. Experimental design and protein assay

The activities of the enzymes and concentrations of the markers were standardized to total protein as determined by the Lowry technique. The standard curves were linear throughout the working range: $y = 0.0077x + 0.0048$, $R^2 = 0.944$ for BSA (Figure 1), and $y = 0.0015x + 0.0047$, $R^2 = 0.977$ for GSH (Figure 2).

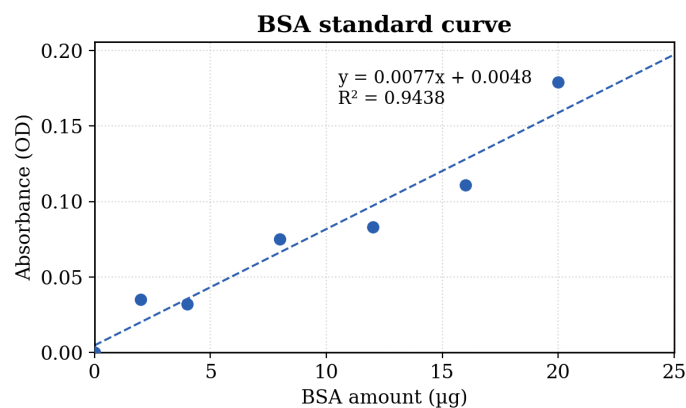


Figure 1. Bovine serum albumin (BSA) standard curve used for protein quantification by the Lowry method. Optical density (OD) at 750 nm as a function of BSA amount.

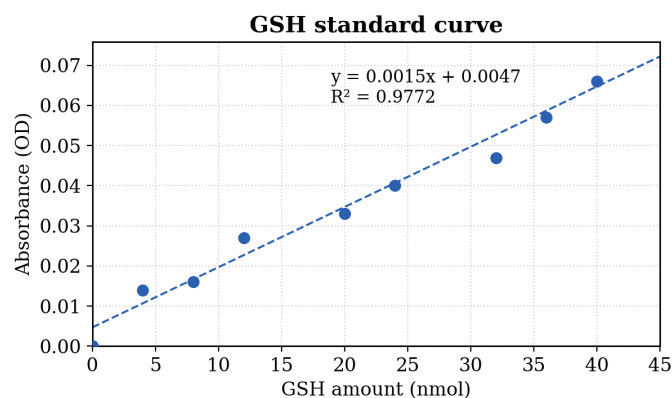


Figure 2. Reduced glutathione (GSH) standard curve. Optical density at 412 nm as a function of GSH amount.

2.3. Superoxide dismutase activity

Olive oil was the only single oil to change SOD activity appreciably (Figure 3). It more than doubled the control value, 95 ± 22 against 43 ± 14 U/mg protein ($p = 0.0012$), while argan, colza and cactus seed oils left activity within the control range. LPS by itself had no effect (52 ± 20 U/mg protein). The co-treatments then split in two directions. Argan and cactus seed oils lowered SOD activity relative to LPS alone, to 35 ± 15 and 29 ± 8 U/mg protein; olive oil raised it to 64 ± 25 ; the colza combination was indistinguishable from LPS.

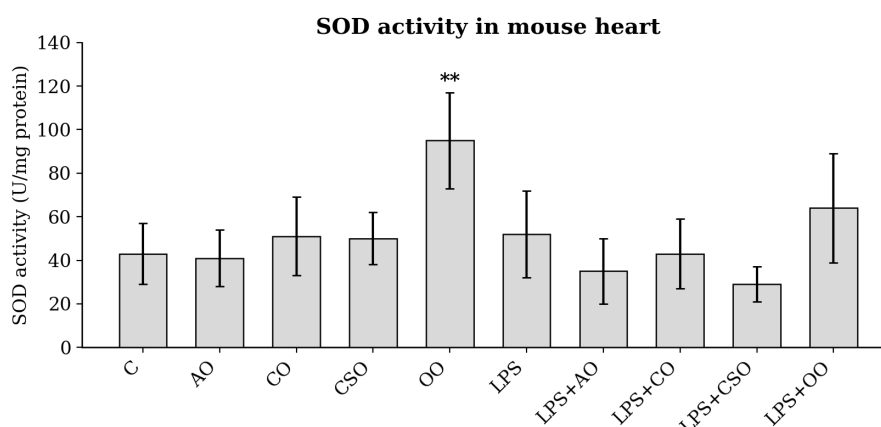


Figure 3. Superoxide dismutase (SOD) activity in the mouse heart, normalized to the control. Values are mean $\pm$ SEM of $n = 5$ mice per group. ** $p < 0.01$ versus control (two-way ANOVA with Tukey post-hoc test).

2.4. Catalase activity

There was no significant difference in catalase activity for any of the groups examined in this study ($p > 0.99$; Figure 4), with the colza oil group reaching the highest value of 12.2 ± 4.8 U/mg protein and the argan oil group reaching the lowest at 4.4 ± 2.3 , while the controls achieved an average of 7.3 ± 5.0 . The activity of catalase was not elevated by LPS treatment. Overall, in terms of co-treatment, both the argan and colza oils seemed to have increased effect, whereas the rest of the oils showed decreasing effect.

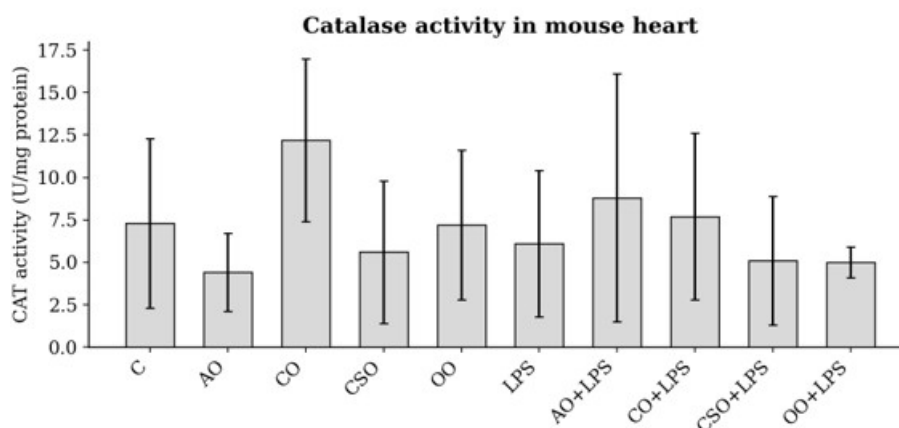


Figure 4. Catalase (CAT) activity in the mouse heart, normalized to the control. Values are mean $\pm$ SEM of $n = 5$ mice per group. Differences between groups were not significant ($p > 0.99$; two-way ANOVA).

2.5. Reduced glutathione (GSH)

The glutathione pool reacted to the presence of the oils. Argan, colza and olive oils lowered the cardiac GSH below the control value of 790 ± 380 μ mol/mg protein while the cactus seed oil had no effect. The LPS treatment did not decrease the GSH

levels either - the concentrations of 860 ± 280 $\mu\text{mol}/\text{mg}$ protein were very close to the control values. The effect of argan oil on the GSH levels was unique as it reached a value of 2210 ± 1150 $\mu\text{mol}/\text{mg}$ protein, which is around 2 and a half times greater than the LPS value. The colza, cactus seed and olive oils given prior to the LPS challenge resulted in the decrease of the GSH concentration to values of 475, 180 and 385 $\mu\text{mol}/\text{mg}$ protein, respectively, which were lower than both the LPS and control values.

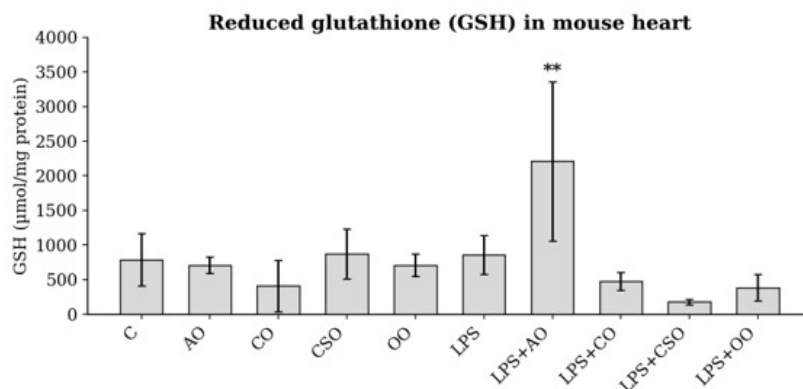


Figure 5. Reduced glutathione (GSH) content in the mouse heart, normalized to the control. Values are mean $\pm$ SEM of $n = 5$ mice per group. ** $p < 0.01$ for LPS + AO versus LPS (two-way ANOVA with Tukey post-hoc test).

2.6. Lipid peroxidation (MDA)

Each oil given alone raised malondialdehyde above the control value of 17 ± 6 mmol/mg protein, colza and cactus seed oils most of all (77 ± 20 and 66 ± 44 ; Figure 6). LPS raised it to 31 ± 8 . All four combinations returned MDA to between 16 and 22 mmol/mg protein, below both the corresponding oil-alone group and the LPS group. None of these differences was significant ($p > 0.99$), which the size of the standard errors makes unsurprising.

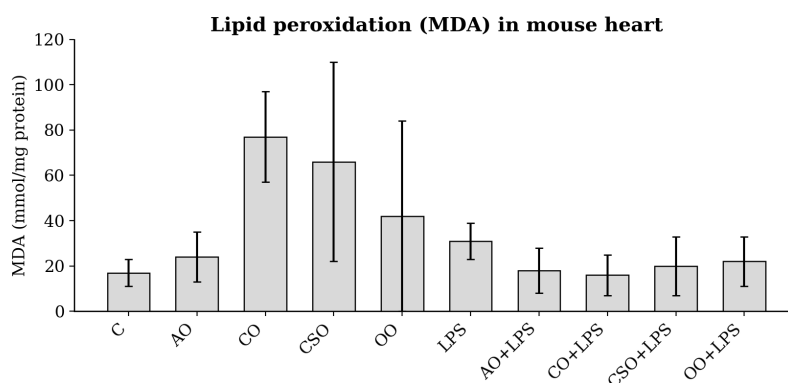


Figure 6. Malondialdehyde (MDA) content in the mouse heart, normalized to the control. Values are mean $\pm$ SEM of $n = 5$ mice per group. Differences between groups were not significant ($p > 0.99$; two-way ANOVA).

Table 4. Cardiac antioxidant enzyme activities and oxidative stress markers across the ten experimental groups (mean $\pm$ SEM, $n = 5$).

Group	SOD (U/mg)	CAT (U/mg)	GSH ($\mu\text{mol}/\text{mg}$)	MDA (mmol/mg)
C	43 ± 14	7.3 ± 5.0	790 ± 380	17 ± 6
AO	41 ± 13	4.4 ± 2.3	710 ± 120	24 ± 11
CO	51 ± 18	12.2 ± 4.8	410 ± 370	77 ± 20

Group	SOD (U/mg)	CAT (U/mg)	GSH (μ mol/mg)	MDA (mmol/mg)
CSO	50 $\pm$ 12	5.6 $\pm$ 4.2	875 $\pm$ 360	66 $\pm$ 44
OO	95 $\pm$ 22	7.2 $\pm$ 4.4	710 $\pm$ 160	42 $\pm$ 42
LPS	52 $\pm$ 20	6.1 $\pm$ 4.3	860 $\pm$ 280	31 $\pm$ 8
LPS + AO	35 $\pm$ 15	8.8 $\pm$ 7.3	2210 $\pm$ 1150	18 $\pm$ 10
LPS + CO	43 $\pm$ 16	7.7 $\pm$ 4.9	475 $\pm$ 130	16 $\pm$ 9
LPS + CSO	29 $\pm$ 8	5.1 $\pm$ 3.8	180 $\pm$ 40	20 $\pm$ 13
LPS + OO	64 $\pm$ 25	5.0 $\pm$ 0.9	385 $\pm$ 190	22 $\pm$ 11

Values were obtained by pixel-level digitization of the original experimental figures (mean $\pm$ SEM, $n = 5$), cross-checked with two independent measurement methods, and are expressed relative to the control. They are close estimates of the plotted data and should be replaced by the exact GraphPad Prism values before submission. AO, argan oil; OO, olive oil; CO, colza oil; CSO, cactus seed oil; SOD, superoxide dismutase; CAT, catalase; GSH, reduced glutathione; MDA, malondialdehyde.

3. DISCUSSION

The heart did not behave as the liver and the brain do under the same challenge. In those organs LPS raises antioxidant enzyme activities and MDA and disturbs the glutathione pool [9,10]; here it left SOD, CAT and GSH near control values and produced no visible lesion. Cardiac tissue holds a large constitutive antioxidant reserve under Nrf2 control [8], and at the dose and interval used the challenge may have stayed within what that reserve absorbs.

Catalase illustrates the point. El Kamouni et al. found the enzyme induced in the liver after LPS and further modulated by argan and olive oils [9]; in the heart, no group differed from any other. Polyphenols and tocopherols supplied by the diet may consume H_2O_2 before catalase is called upon [3,16], although nothing in the present data separates that reading from a simple absence of oxidative pressure.

SOD produced the one significant effect among the single oils, and it came from olive oil rather than argan oil. El Kamouni et al. reported the same direction in cardiac tissue [9], whereas the liver and brain data of Essadek et al. run the other way [10]. Under LPS, argan and cactus seed oils lowered SOD activity, as in both earlier studies, and olive oil again departed from the pattern. Treatment duration differs between these experiments, which is the first variable to examine before reading anything mechanistic into the discrepancy.

Glutathione carried the clearest signal. Argan oil was the only pretreatment to raise the cardiac GSH pool under LPS, and it did so by a wide margin. Essadek et al. observed the opposite in liver and brain, where most oil combinations depleted GSH, colza excepted [10], and El Kamouni et al. found cardiac GSH low in every group but the olive oil combination [9]. Why argan oil should protect the pool specifically is not established by these measurements. Its composition offers candidates, γ -tocopherol at 90% of the tocopherol fraction and ferulic acid at 3147 μ g/kg, yet the experiment was not designed to separate them. The variance in this group is also the largest in the study, and the mean rests on five animals.

Lipid peroxidation moved in the expected direction without statistical support. Every combination lowered MDA relative to the corresponding oil alone, which fits the tocopherol and polyphenol content of the four oils [3], and the same protective pattern appears in liver and brain [10]. El Kamouni et al. reported cardiac MDA rising instead under oil and LPS together [9].

Several limitations bear on how far these results can be taken. Only two comparisons reached significance, and five animals per group leave little power to detect the rest; the trends described above should be read as trends and nothing more. GPx was not assayed in the heart. The LPS dose and the interval before sacrifice were fixed rather than varied, so the absence of a cardiac response cannot be separated from an insufficient challenge. Gene expression was not measured, which leaves the Nrf2 interpretation untested. Finally, the values reported here were recovered from the original figures by digitization rather than read from the raw records, and should be replaced by the primary data before submission.

4. MATERIALS AND METHODS

4.1. Oils

Virgin argan oil came from the rural women's cooperative EFAS (Aghraisse village, Ait Baha province, Agadir, Morocco). Olive oil was obtained in the Aklim region (Berkane, north-eastern Morocco), colza oil from a commercial source, and cactus seed oil from *Opuntia ficus-indica*.

4.2. Animals

Swiss OF1 mice (IFFA CREDO), bred at the Pasteur Institute laboratory in Tit Mellil, Casablanca, were used, with a mean body weight of 18 ± 3 g. Handling followed the standards of the host institution.

4.3. Experimental design

Animals were assigned to ten groups of five. Four groups received a diet enriched with 6% of a single oil (argan, olive, colza or cactus seed) for 28 days. Challenged groups were given an intravenous injection of LPS from *Escherichia coli* O111:B4 (1 mg/mL), either alone or after the oil-enriched diet, and control animals received physiological saline. Mice were weighed before and after sacrifice and the heart was collected. Treatment and sacrifice preceded the analytical work reported here.

4.4. Preparation of cardiac homogenates

Each heart was cut into four pieces, each weighed on a precision balance to set the buffer volume. Homogenates were prepared at 4% (4000 mg per 100 mL) in 50 mM phosphate buffer, sonicated (Qsonica Misonix Q700, 700 W; three cycles of 20 s) and centrifuged at 3000 g for 10 min at 4 °C. Supernatants were collected and stored at -24 °C.

4.5. Protein quantification

Total protein was determined by the Lowry method against a bovine serum albumin standard curve prepared in 50 mM phosphate buffer, with optical density read at 750 nm on a Tecan spectrophotometer. Each enzymatic activity and marker value was normalized to the protein content of the corresponding sample.

4.6. Antioxidant enzyme activities

SOD activity was measured by the Paoletti method, which follows the riboflavin-driven reduction of nitroblue tetrazolium (NBT) and its inhibition by the enzyme, with absorbance read at 560 nm after 10 min of illumination. CAT activity was derived from the decomposition of hydrogen peroxide by the residual KMnO_4 method, with absorbance at 240 nm.

4.7. Oxidative stress markers

GSH was assayed with 5,5'-dithiobis-(2-nitrobenzoic acid) (DTNB). Homogenate was precipitated with 5% trichloroacetic acid and centrifuged at 12 000 g for 10 min at 4 °C, and the supernatant was reacted with DTNB in phosphate buffer, absorbance being read at 412 nm against a GSH standard curve. Lipid peroxidation was estimated from MDA by the thiobarbituric acid reaction [18]: homogenate was mixed with trichloroacetic acid and thiobarbituric acid, heated at 100 °C for 15 min, cooled, extracted with n-butanol and centrifuged at 3000 g for 15 min, absorbance of the supernatant being read at 532 nm.

4.8. Statistical analysis

Data are given as mean $\pm$ SEM. Groups were compared by two-way ANOVA followed by a Tukey post-hoc test when the null hypothesis was rejected, a p-value of 0.05 or less being taken as significant (** $p \leq 0.01$, * $p \leq 0.05$). Analyses were run in GraphPad Prism 8.

5. CONCLUSIONS

In the mouse heart, a single LPS injection did not disturb SOD or CAT activity, the glutathione pool or lipid peroxidation to a degree these assays could resolve, and left no visible lesion, in contrast with what has been described for liver and brain. Against that quiet background argan oil still produced an effect: it was the only oil to raise cardiac GSH under challenge, and all four oils lowered MDA relative to the oil given alone. Whether the cardiac reserve simply absorbed the dose used here, or the heart responds to LPS on a different timescale, can be settled by varying dose and interval. Assaying GPx, enlarging the groups and following antioxidant and β -oxidation transcripts by Q-PCR would supply the mechanistic account these measurements cannot.

DECLARATIONS

Author contributions: O.J. carried out the experimental work and drafted the manuscript; I.A. and O.E.H. contributed to data collection and laboratory procedures; M.A.E.C. and Y.B. designed and supervised the study. All authors have read and agreed to the published version of the manuscript.

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Data availability statement: The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of interest: The authors declare no conflict of interest.

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