A ~60-min brisk walk increases insulin-stimulated glucose disposal but has no effect on hepatic and adipose tissue insulin sensitivity in older women

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Wang X, Patterson BW, Smith GI, Kampelman J, Reeds DN, Sullivan SA, Mittendorfer B. A ~60-min brisk walk increases insulin-stimulated glucose disposal but has no effect on hepatic and adipose tissue insulin sensitivity in older women. J Appl Physiol 114: 1563–1568, 2013. First published March 14, 2013; doi:10.1152/japplphysiol.01364.2012—The purpose of this study was to determine whether brisk walking improves multiorgan (liver, muscle, adipose tissue) insulin sensitivity in older women. Ten nonobese older women (age: 66.7 ± 1.5 yr, mean ± SE) completed two 2-stage hyperinsulinemic-euglycemic clamp procedures [insulin infusion rate stage 1: 10 mU/m2 body surface area (BSA) per min; stage 2: 50 mU/m2 BSA per min] in conjunction with stable isotope-labeled glucose and palmitate tracer infusions: one in the morning after a single, ~1-h bout of brisk treadmill walking, the other after an equivalent period of rest in the late afternoon of the preceding day. We found that basal glucose rate of appearance (Ra) into plasma was not different after rest and after exercise (17.3 ± 0.8 and 17.1 ± 0.4 μmol/kg fat-free mass per min, respectively). The insulin-mediated decrease in glucose Ra during stage 1 of the clamp was also not different after rest and exercise (82.2% ± 3.4% and 77.7% ± 2.1%, respectively), but glucose rate of disappearance (Rd) during stage 2 of the clamp was significantly greater (P < 0.05) after exercise than rest (88.0 ± 5.9 and 78.4 ± 6.5 μmol/kg fat-free mass per min, respectively). There were no differences in palmitate Ra during basal conditions or insulin infusion after exercise and after rest. Therefore, we conclude that a single bout of brisk walking for ~1 h improves muscle insulin sensitivity but has no effect on liver and adipose tissue insulin sensitivity in older women.

insulin sensitivity; exercise; older adults

INSULIN RESISTANCE IS AN IMPORTANT risk factor for type 2 diabetes mellitus and cardiovascular disease (17, 34). Aerobic exercise has very potent insulin-sensitizing effects (4, 21) and regular physical activity is the cornerstone for maintaining insulin sensitivity and metabolic health. The beneficial effect of exercise on insulin sensitivity is transient and lasts for only a couple of days after an exercise session in young and middle-aged adults; thereafter it returns to baseline values even in highly trained athletes (2, 5, 10, 25, 29, 30, 32). Improved insulin sensitivity occurs in many tissues [muscle, liver, adipose tissue (9, 11, 19, 20, 27, 35)] after exercise but skeletal muscle insulin sensitivity is the most comprehensively studied feature and is mediated by a transient increase in insulin-mediated translocation of GLUT4 to the cell surface (18, 21, 23). The cellular signaling pathways mediating this response are not yet fully understood but lie downstream from and do not involve the classic, proximal insulin signaling cascade including IRS1, PI3K, and Akt (21, 23). On the other hand, it is well-established that GLUT4 translocation is directly related to the degree of glycogen breakdown (21, 23). Consequently, exercise intensity and volume modulate the magnitude of the improvement in insulin sensitivity in muscle (3, 11, 12, 28), and it has been suggested that a threshold for an exercise-induced insulin-sensitizing effect equivalent to approximately 1 h of moderate intensity exercise may exist in young and middle-aged adults (28). The American College of Sports Medicine and the United States Department of Health and Human Services recommend a minimum of 150 min (but ideally 300 min) of moderate-intensity exercise a week for adults (i.e., 30–60 min of exercise on ≥5 days a week) (14, 39). Whether this is adequate for older adults is not known. Although older adults retain the ability to improve insulin sensitivity through exercise (8, 11, 13, 22, 24, 27, 38), studies in older adults to date have focused solely on high-intensity and/or high-volume exercise (8, 11, 13, 15, 22, 24, 27, 36, 38), and some investigators report no improvement even after intense exercise training (15, 36).

The purpose of this study was to determine whether a single session of brisk walking improves insulin sensitivity in older adults. The two-stage hyperinsulinemic-euglycemic clamp procedure in conjunction with stable isotope-labeled glucose and palmitate tracer infusions was used to evaluate insulin sensitivity in the liver, muscle, and adipose tissue. A low-dose insulin infusion was used to evaluate the ability of insulin to suppress the endogenous rates of appearance (Ra) of glucose (an index of hepatic glucose production) and palmitate (an index of adipose tissue lipolytic rate) in plasma; both endogenous glucose production and adipose tissue lipolysis are exquisitely sensitive to insulin and are nearly completely suppressed at plasma insulin concentrations in the postprandial range (16, 33). A “high-dose” insulin infusion resulting in postprandial plasma insulin concentrations was used to evaluate the ability of insulin to stimulate glucose rate of disappearance (Rd) from plasma (an index of muscle glucose uptake) (7, 33).

MATERIALS AND METHODS

Subjects and Screening Evaluations

Ten healthy, nonobese older women (66.7 ± 1.5 yr, mean ± SE, age range 60–73 yr) participated in this study (Table 1), which was approved by the Institutional Review Board of Washington University School of Medicine in St. Louis, MO. Written informed consent was obtained from all subjects before participation in the study. None of the subjects smoked and all were weight stable (±1 kg) and sedentary (<20 min of exercise no more than 3 times/wk) for at least 3 mo before entering the study. All subjects completed a comprehensive screening evaluation. The data on demographics, medical history, medications, and physical activity are reported in Table 1. All subjects were postmenopausal, and the majority of the cohort experienced menopause prior to age 50 yr. Subjects were not using any hormone replacement therapy (HRT) at the time of this study. Individuals with chronic diseases were excluded (Table 1). No individual was taking any medications known to alter insulin sensitivity or circulating hormone concentrations (e.g., statin drugs, insulin sensitizers, or any other medications used for treatment of diabetes, cardiovascular disease, or atherosclerotic disease). A majority of the cohort was taking a multivitamin (70%), a vitamin D supplement (90%), and fish oil (90%). All other medications were reviewed and considered to have minimal effects on insulin sensitivity.

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Table 1. Subjects’ body composition, aerobic fitness, and basal (overnight fasted) metabolic variables at screening

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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<tbody>
<tr>
<td>Body weight, kg</td>
<td>68.2 ± 2.6</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>24.9 ± 0.9</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>41.8 ± 1.1</td>
</tr>
<tr>
<td>VO₂peak, ml·kg body wt⁻¹·min⁻¹</td>
<td>22.2 ± 1.1</td>
</tr>
<tr>
<td>Glucose, mg/dl</td>
<td>86.6 ± 1.4</td>
</tr>
<tr>
<td>Triglycerides, mg/dl</td>
<td>81 ± 12</td>
</tr>
<tr>
<td>Total cholesterol, mg/dl</td>
<td>118 ± 11</td>
</tr>
<tr>
<td>HDL-cholesterol, mg/dl</td>
<td>61 ± 6</td>
</tr>
<tr>
<td>LDL-cholesterol, mg/dl</td>
<td>111 ± 9</td>
</tr>
</tbody>
</table>

Data are means ± SE. VO₂peak, peak oxygen consumption; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Medical examination, including a detailed history and physical examination, a resting electrocardiogram, standard blood tests, and an oral glucose tolerance test. Blood tests included complete metabolic panel, complete blood count, blood lipids, and thyroid-stimulating hormone. Standard cut-offs that are used in our hospital and associated clinics for above/below normal values were used to include or exclude subjects. During the oral glucose tolerance test, a 75-g glucose drink (Azer Scientific, Morgantown, VA) was consumed within 3 min, and blood samples to measure plasma glucose concentration were obtained immediately before and 2 h after consumption of the drink; subjects were seated in a chair for the duration of the glucose tolerance test. Subjects with diabetes, impaired fasting glucose, or impaired glucose tolerance based on American Diabetes Association criteria (1) were excluded from the study. None of the subjects had evidence of illness, or were taking medications known to affect carbohydrate or lipid metabolism. Body composition was determined by using dual-energy X-ray absorptiometry (Hologic QDR 1000w; Hologic, Waltham, MA).

Peak oxygen consumption (VO₂peak) was evaluated during a graded exercise test on a treadmill. Heart rhythm and rate were continuously monitored (Marquette MAX-1, Sandy, UT), and expired air was analyzed by using a metabolic cart (ParvoMedics TrueOne 2400) and expired air was monitored (Marquette MAX-1, Sandy, UT), and expired air was analyzed by using a metabolic cart (ParvoMedics TrueOne 2400) to ensure the appropriate exercise intensity was achieved. The duration of exercise was variable and terminated when subjects achieved a total energy expenditure of 3.5 kcal/kg body wt, based on the volume of the oxygen consumed. We based the duration of the exercise bout on the total amount of energy expended rather than a fixed time period to minimize differences in energy and substrate use between subjects.

Hyperinsulinemic-euglycemic clamp procedure. At 0600, a catheter was inserted into a forearm vein for stable isotope-labeled tracer (purchased from Cambridge Isotope Laboratories, Andover, MA) infusions. A second catheter was inserted into a contralateral hand vein for blood sampling. The hand was heated to 55°C by using a thermostatically controlled box to obtain arterialized blood samples. At 0700, a bolus of [6,6-²H₂]glucose (18 µmol/kg), dissolved in 0.9% NaCl solution, was administered through the catheter in the forearm vein, followed by a constant infusion of [6,6-²H₂]glucose (0.22 µmol·kg⁻¹·min⁻¹) dissolved in 0.9% NaCl solution. At 0830, a constant infusion of [2,2-²H₂]palmitate (0.03 µmol·kg⁻¹·min⁻¹), dissolved in a 25% human albumin solution, was started. At 1000, a hyperinsulinemic-euglycemic clamp procedure was initiated. During stage 1 of the clamp (3.0–5.5 h from the start of the glucose tracer infusion), insulin was infused at 10 µU/m² body surface area (BSA) per minute (following a two-step priming dose of 40 µU/m² BSA per minute for 5 min and 20 µU/m² BSA per minute for 5 min, respectively). At 1230, stage 2 of the clamp procedure (5.5–9.0 h) was initiated by a two-step priming dose of insulin (200 µU/m² BSA per minute for 5 min and 100 µU/m² BSA per minute for 5 min), followed by constant infusion at 50 µU/m² BSA per minute. Euglycemia was maintained at a plasma glucose concentration of ~100 mg/dl by variable-rate infusion of 20% dextrose (enriched with [6,6-²H₂]glucose) (7). The [6,6-²H₂]glucose and [2,2-²H₂]palmitate infusion rates were reduced by 50% of basal infusion rates during the first stage of the clamp to account for the insulin-mediated suppression of glucose and palmitate Ra (7).

Sample Collection and Analysis

Blood samples were obtained before the tracer administration to determine plasma insulin, glucagon, glucose, and free fatty acid (FFA) concentrations, and background tracer-to-tracer ratio (TTR) of glucose and palmitate, and every 10 min during the last 30 min of the basal period and each stage of the clamp procedure to determine glucose and fatty acid kinetics and plasma glucose, glucagon, insulin and FFA concentrations. Blood samples to monitor blood glucose concentration were collected every 10 min throughout the insulin infusion. Blood samples were collected in chilled tubes containing heparin to determine plasma glucose and insulin concentrations. The remaining samples were collected in chilled tubes containing EDTA. Plasma was separated by centrifugation within 30 min of collection and stored at −80°C until final analyses were performed.

Plasma glucose concentration was determined by using an automated glucose analyzer (YSI 2300 STAT plus; Yellow Springs Instrument, Yellow Springs, OH). Plasma insulin concentration was measured by using an automated chemiluminescent immunoassay (IMMULITE, Siemens Healthcare Diagnostic, Los Angeles, CA). Plasma glucagon concentration was measured by using a radioimmunoassay (EMD Millipore, Billerica, MA). Plasma FFA concentrations were quantified by gas chromatography (Hewlett-Packard 5890-II,
Palo Alto, CA) after adding heptadecanoic acid to plasma as an internal standard (31). Plasma glucose and palmitate TTR were determined by using gas chromatography/mass spectrometry (MSD 5973 system with capillary column; Hewlett-Packard) (31).

Calculations

Isotopic steady states were achieved during the final 30 min of the basal period and stages 1 and 2 of the clamp procedure (Table 2), and Steele’s equation for steady-state conditions was used to calculate substrate kinetics (37). During isotopic and metabolic steady-state conditions, total glucose rate of appearance (Ra) in plasma equals total glucose rate of disappearance (Rd) from plasma; accordingly, glucose Rd during the basal period and stages 1 and 2 of the clamp procedure was calculated as the sum of unlabeled glucose Ra (calculated by dividing the glucose tracer infusion rate by the average plasma glucose TTR during the last 30 min of the basal period and stages 1 and 2 of the clamp procedure) and the glucose tracer infusion rate. Endogenous glucose Ra was calculated by subtracting the glucose infusion rate (labeled and unlabeled) from total glucose Rd. Palmitate Ra into plasma was calculated by dividing the palmitate tracer infusion rate by the average plasma palmitate TTR during the final 30 min of the basal and stage 1 of the clamp procedure.

Statistical Analysis

Data are reported as means ± SE. Paired Student’s t-test was used to compare basal plasma glucose, insulin, and FFA concentrations after exercise and rest. Repeated-measures analysis of variance with subject condition (exercise vs. rest) and clamp period (basal vs. stage 1 or 2 of the clamp) was used to evaluate the effect of exercise on plasma glucagon concentration and glucose and palmitate kinetics. A P value of ≤0.05 was considered statistically significant.

RESULTS

By design, all subjects performed the same amount of work in relationship to total body weight during the exercise session (i.e., 3.5 kcal/kg body wt), which lasted on average 54 ± 2 min. The absolute amount of work performed ranged from 193 to 275 kcal, and subjects burned 139 ± 17 kcal (~2.1 kcal/kg) as carbohydrates (i.e., ~35 g carbohydrates) and 92 ± 11 kcal (~1.4 kcal/kg) as fat (i.e., ~10 g lipids).

Table 2. Plasma glucose tracer-to-tracee ratios during basal conditions and stages 1 and 2 of the hyperinsulinemic-euglycemic clamp procedure

<table>
<thead>
<tr>
<th></th>
<th>Sample</th>
<th>Rest</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal</td>
<td>1</td>
<td>0.021 ± 0.0014</td>
<td>0.020 ± 0.0007</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.020 ± 0.0013</td>
<td>0.021 ± 0.0007</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.021 ± 0.0013</td>
<td>0.021 ± 0.0007</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.021 ± 0.0014</td>
<td>0.020 ± 0.0007</td>
</tr>
<tr>
<td>Stage 1</td>
<td>1</td>
<td>0.027 ± 0.0009</td>
<td>0.026 ± 0.0007</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.027 ± 0.0007</td>
<td>0.026 ± 0.0007</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.027 ± 0.0012</td>
<td>0.026 ± 0.0006</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.027 ± 0.0010</td>
<td>0.026 ± 0.0009</td>
</tr>
<tr>
<td>Stage 2</td>
<td>1</td>
<td>0.025 ± 0.0009</td>
<td>0.025 ± 0.0008</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.025 ± 0.0011</td>
<td>0.025 ± 0.0006</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.026 ± 0.0010</td>
<td>0.025 ± 0.0007</td>
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<tr>
<td></td>
<td>4</td>
<td>0.026 ± 0.0009</td>
<td>0.026 ± 0.0010</td>
</tr>
</tbody>
</table>

Data are means ± SE. Four blood samples each (1–4) were collected during the last 30 min of the basal period and each stage of the clamp procedure. See text for details.

Basal plasma glucose concentration after exercise was marginally, but statistically significantly, lower than after rest (87.6 ± 1.3 vs. 90.7 ± 1.6 mg/dl, P = 0.019). Basal plasma insulin (6.3 ± 1.5 vs. 7.2 ± 1.8 mU/l, P = 0.20), glucagon (58 ± 4 vs. 59 ± 3 pg/ml, P = 0.72), and FFA (0.53 ± 0.06 vs. 0.48 ± 0.05 mM, P = 0.40) concentrations were not significantly different after exercise and rest. There were no differences in basal glucose Ra (696 ± 22 vs. 707 ± 38 μmol/min, P = 0.74) or basal palmitate Ra (71.6 ± 6.4 vs. 68.0 ± 4.8 μmol/min, P = 0.53) after exercise and after rest (Figs. 1 and 2).

During the hyperinsulinemic-euglycemic clamp procedure, plasma insulin concentration increased to 19.8 ± 2.6 and 20.0 ± 2.4 mU/l (P = 0.84) during stage 1 and to 75.7 ± 5.3 and 76.7 ± 4.7 mU/l (P = 0.76) during stage 2 after exercise and rest, respectively. Plasma glucagon concentration decreased to 39 ± 1 and 39 ± 3 pg/ml during stage 1 and to 30 ± 1 and 34 ± 3 pg/ml during stage 2 after exercise and rest, respectively (significant effect of insulin infusion, P < 0.01 but no significant effect of trial or trial × insulin infusion interaction). Euglycemia was successfully maintained, and the average plasma glucose concentration was similar after exercise and rest (stage 1: 101.3 ± 0.9 vs. 99.7 ± 1.1 mg/dl, respectively; stage 2: 102.7 ± 1.4 vs. 102.3 ± 0.8 mg/dl, respectively).

Low-dose insulin infusion caused a significant decrease (by ~80%; P < 0.001) in glucose Ra, and the insulin-mediated suppression of glucose Ra was different after rest and exercise (Fig. 1). High-dose insulin infusion nearly completely suppressed endogenous glucose Ra (to 5 ± 3% and 6 ± 4% of basal values after exercise and rest, respectively) and increased glucose Rd by ~3-fold above basal values; the insulin-stimulated glucose Rd was ~20% greater after exercise than rest (2,864 ± 242 vs. 2,452 ± 266 μmol/min above basal values, respectively, P = 0.037) (Fig. 1). The same was true when glucose Rd was adjusted for small individual differences in plasma insulin concentrations during the clamp: the glucose Rd-to-plasma insulin concentration ratio was 18 ± 7% greater after exercise than rest (P = 0.05).

Low-dose insulin infusion significantly suppressed palmitate Ra (by ~75%; P < 0.001), and the insulin-mediated decrease was not different after exercise and rest (Fig. 2).

DISCUSSION

The insulin-sensitizing effect of aerobic exercise has been known for several decades and affects many tissues (muscle, liver, adipose tissue), but skeletal muscle insulin sensitivity is the most comprehensively studied feature (21, 23). It is now clear that the exercise-induced improvement in insulin sensitivity is transient and lasts for only ~48 h after an exercise session (2, 5, 10, 25, 29, 30, 32); the magnitude of the effect, in muscle, appears to be directly related to exercise volume and/or intensity (3, 11, 12, 28). In this study we evaluated the effect of a brisk evening treadmill walk on multiorgan insulin sensitivity on the day after the exercise by using the two-stage hyperinsulinemic-euglycemic clamp procedure in conjunction with stable isotope-labeled glucose and palmitate tracer infusions in nonobese, 60- to 75-yr-old women. The walk, which was consistent with current public health recommendations for physical activity in terms of intensity and duration, improved
insulin-stimulated glucose disposal but had no effect on insulin-stimulated suppression of glucose and palmitate Ra into plasma. These results demonstrate that brisk walking has a beneficial effect on muscle insulin sensitivity but does not improve hepatic or adipose tissue insulin sensitivity in older women.

Several studies have evaluated the effect of exercise on skeletal muscle insulin sensitivity by using the gold-standard hyperinsulinemic-euglycemic clamp or a hyperglycemic clamp procedure in older adults, but the results are inconclusive. Most studies report improved insulin-mediated muscle glucose disposal after the last session of exercise training (11, 13, 22, 26, 27, 38); but some did not (11, 15). The discrepancy in results is likely due to a combination of the timing of the metabolic measurements in relation to the exercise, exercise intensity and statistical power. All of the studies that report a significant improvement on insulin-mediated glucose disposal focused on high-intensity (i.e., ≥70% V\text{O}_{2\text{peak}} or ≥80% maximal heart rate) exercise (11, 13, 22, 26, 27, 38), and in three of these studies, insulin-mediated glucose disposal was evaluated within 48 h after the last exercise session (13, 26, 27) by using either the hyperinsulinemic-euglycemic clamp technique (insulin infusion rate: 40 mU/m² BSA per min) (27) or the hyperglycemic clamp technique (13, 26); in the other three studies (11, 22, 38) measurements were made several days after the exercise session by using the hyperinsulinemic-euglycemic clamp procedure (insulin infusion rate ranging from 40 –200 mU/m² BSA per min), and the reported differences were statistically significant but only very small (~10%). Goulet et al. (15) and DiPietro et al. (11) failed to show a significant effect of 1 h of running or cycling (~65% V\text{O}_{2\text{peak}}) on insulin-mediated glucose disposal (evaluated by using the hyperinsulinemic-euglycemic clamp procedure; insulin infusion rate of 40 mU/m² BSA per min) when measurements were made 72–120 h (3–5 days) after the exercise session when the insulin-sensitizing effect of exercise has most likely nearly vanished (5, 25, 30). The results from our study demonstrate that even a 1-h-long brisk walk can significantly improve skeletal muscle insulin sensitivity for at least up to 20 h in older adults. Regular walking may therefore be sufficient and should be recommended to help preserve muscle insulin sensitivity in older adults.

We found no effect of exercise on hepatic and adipose tissue insulin sensitivity. To our knowledge, only one study to date has evaluated adipose tissue insulin sensitivity after exercise in older adults. From this study, it was reported that insulin-stimulated suppression of adipose tissue lipolysis was increased after high-intensity aerobic exercise (80% V\text{O}_{2\text{peak}}) but not after moderate-intensity exercise (50–65% V\text{O}_{2\text{peak}}) (11).
Thus the lack of change in adipose tissue insulin sensitivity in our study is likely due to the moderate intensity of exercise performed, and more strenuous exercise may be needed to improve adipose tissue insulin sensitivity in older adults. Three studies have evaluated the effect of exercise on hepatic insulin sensitivity in older adults, and two report an increase in hepatic insulin sensitivity (19, 27) whereas the other (11) found no difference in hepatic insulin sensitivity between the exercise and control groups. One of the two studies that demonstrate a beneficial effect of exercise on hepatic insulin sensitivity (19) included obese older men and women with impaired glucose tolerance who were studied before and after 7 days of daily exercise for 1 h at 65% $\dot{V}O_2$peak; the other (27) included overweight and obese diabetic men and women who were studied before and after 7 days of daily exercise for 1 h at 80–85% maximum heart rate ($\sim$70% $\dot{V}O_2$peak). We and DiPietro et al. (11), on the other hand, studied nonobese, nondiabetic older adults. It is therefore possible that the failure of exercise to improve hepatic insulin sensitivity in our study was due to the already near-maximal suppression of hepatic glucose production without exercise in rather insulin-sensitive subjects. However, we cannot rule out the possibility that repeated bouts of exercise and/or higher-intensity exercise are necessary to improve hepatic insulin sensitivity.

In summary, we have demonstrated that a single session of brisk walking that is consistent with public health recommendations improves skeletal muscle insulin sensitivity in older women when measured within ~24 h after completion of the exercise. Based on these results, regular brisk walking may be recommended to maintain metabolic health in older adults.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


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