Motor imagery of gait in non-demented older community-dwellers: performance depends on serum 25-hydroxyvitamin D concentrations

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Abstract Hypovitaminosis D has been associated with poorer physical and cognitive performances in older adults. The objectives of this study were (1) to measure and compare the time to perform (pTUG) and to imagine (iTUG) the Timed "Up & Go" test (TUG) test, and the time difference between these two performances (i.e., TUG delta time) in non-demented community-dwelling older adults with and without lower serum 25-hydroxyvitamin D (25OHD) concentrations and (2) to examine the association between the TUG delta time and serum 25OHD concentrations. Durations of pTUG, iTUG and TUG delta time, and serum 25OHD concentrations (severe insufficiency <10 ng/mL; moderate insufficiency: 10–30 ng/mL; normal status >30 ng/mL) were measured in 359 non-demented participants (mean

age 70.4 ± 4.8 years; 40.7 % women). Participants with severe 25OHD insufficiency (15.6 %) had higher TUG delta time compared to those with moderate insufficiency ($P=0.010$) and normal status ($P=0.048$). TUG delta time was negatively associated with serum 25OHD concentrations ($P<0.010$). Accurate motor imagery of gait was explained in part by serum 25OHD concentrations, increased discrepancy between pTUG and iTUG being associated with lower serum 25OHD concentrations.

Keywords Timed "Up and Go" test · Motor imagery · 25-Hydroxyvitamin D · Gait control · Older adults

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Introduction

Motor imagery (MI) is a mental representation of body movement without its actual execution (Jeannerod and Decety 1995; Jeannerod 1995). MI is used in clinical practice to access to higher levels of motor control by examining the temporal correspondence between the time taken to perform and to imagine the same movement (Bakker et al. 2007a). A close temporal correspondence is interpreted as a normal and efficient motor control (Jeannerod and Decety 1995; Jeannerod 1995; Bakker et al. 2007a). Using the Timed "Up & Go" test (TUG) (Podsiadlo and Richardson 1991)—measuring the time needed to rise from a chair, walk 3 m, turn around and return to a seated position—we adapted an imagined version to assess higher levels of gait control (Beauchet et al. 2010; Lallart et al. 2012; Bridenbaugh

et al. 2013; Beauchet et al. 2014). The findings showed that older adults imagined TUG faster than actually performing it in comparison to young adults. This temporal discrepancy measured by the time difference between these two performances suggested an age-related disturbance in higher levels of gait control (Beauchet et al. 2010; Bridenbaugh et al. 2013).

Vitamin D is a neurosteroid hormone that exhibits multiple biological targets mediated by the vitamin D receptor (VDR) present in many cells (Annweiler et al. 2010a). Specific actions on target organs such as muscles and central nervous system (CNS), directly influencing motor and cognitive performances, have been previously reported (Annweiler et al. 2010b, c; Annweiler et al. 2014a, b). Hypovitaminosis D, usually defined as a serum 25-hydroxyvitamin D (25OHD) concentration ≤ 30 ng/mL, is highly frequent among older adults with a prevalence estimated around 50 % in community-dwelling adults aged 65 years and older in Europe (Annweiler et al. 2010b; Mithal et al. 2009). Epidemiological studies showed that lower serum 25OHD concentrations are associated with poorer motor and cognitive performances among older adults (Annweiler and Beauchet 2014; Annweiler et al. 2009a, b; Annweiler et al. 2010c; Annweiler et al. 2014a; Beauchet et al. 2011a). In particular, the specific action of vitamin D on the lower components of gait control has been described (Beauchet et al. 2011a). In this study, low serum 25OHD concentrations were associated with gait instability; this association being explained by an action of vitamin D on the different subsystems involved in the lower levels of gait control including muscles and the neurofeedback system. However, the consequences of low serum 25OHD on the higher levels of gait control in non-demented older adults remain yet unknown. Recently, it has been reported that lower serum 25OHD concentrations were associated with increased white matter abnormalities, especially in the periventricular regions, as well as with brain atrophy (Annweiler et al. 2014b, c). In addition, lower level of vitamin D was associated with reduced neuronal functioning in the caudal primary motor cortex (Annweiler et al. 2014c). All these brain structures are involved in higher levels of gait control (Allali et al. 2014).

Because low serum 25OHD concentrations affect cognitive performances, brain structures, and brain functioning (Annweiler et al. 2009a, b; Annweiler et al. 2010c; Annweiler et al. 2014a, b, c; Beauchet

et al. 2011a), we hypothesized that they could be associated with impaired higher levels of gait control (assessed by motor imagery of gait) in older adults. The objectives of this study were (1) to measure and compare the time to perform (pTUG) and to imagine (iTUG) TUG test, and the time difference between these two performances (i.e., TUG delta time) in non-demented older adults with and without lower serum 25OHD concentrations and (2) to examine the association between the TUG delta time and serum 25OHD concentration among non-demented community-dwelling older adults.

Material and methods

Participants

Three hundred fifty-nine participants (mean age 70.4 ± 4.8 years, 40.7 % women) were enrolled in this cross-sectional study between August and December 2009 from a free medical examination performed in the French health examination centre (HEC) of Lyon, France. Exclusion criteria for this analysis included age below 65 years, inability to understand and speak French, previous history of acute medical illness during the past month, dementia, inability to walk 6 m unassisted, neurological diseases including Parkinson's disease, cerebellar disease, myelopathy, peripheral neuropathy, and major orthopedic diagnoses (e.g., osteoarthritis) involving the lumbar vertebra, pelvis, or lower extremities.

Clinical assessment

Participants underwent a comprehensive medical examination. The use of psychoactive drugs (i.e., benzodiazepines, antidepressants, or neuroleptics) and the number of drugs taken each day were recorded. Body mass index (BMI; in kg/m^2) was calculated based on anthropometric measurements (i.e., weight in kg and height in m). Lower limb proprioception was evaluated with the use of a graduated tuning fork placed on the tibial tuberosities (Beauchet et al. 2011a, b). The mean value obtained from left and right sides was used in the present data analysis. Far binocular vision was measured at 5 m with a Snellen letter test chart (Lord et al. 1994). Vision was assessed with corrective lenses on if usually used by the participant. The maximal isometric voluntary

contraction (MVC) strength of handgrip was measured with computerized hydraulic dynamometers (Martin Vigorimeter, Medizin Technik, Tutlingen, Germany) (Beauchet et al. 2011a, b). The test was performed one time on each side. The highest MVC value was retained for this analysis. An abnormal (i.e., any error made in the execution of the circle of the clock, or in the position of the hands and numbers of the clock) Clock Drawing Test (CDT) defined cognitive impairment (Cahn et al. 1996). Depression symptoms were defined by a four-item Geriatric Depression Scale (GDS) score ≥ 1 (Shah et al. 1997).

TUG assessment

This study used the TUG as described by Podsiadlo and Richardson (1991). Participants were asked to perform TUG at their self-selected usual speed in a well-lit environment. Two TUG conditions were successively measured in non-randomized order: first executing TUG and then imaging TUG while sitting in a chair. Time for each condition was recorded with a stopwatch to the nearest 0.01 s. Before testing, a trained evaluator gave standardized verbal instructions regarding the test procedure. Participants were seated, allowed to use the armrests to stand up, and instructed to walk 3 m, turn around, walk back to the chair and sit down. The stopwatch was started on the command “ready-set-go” and stopped as the participants sat down. For the imagined condition, participants sat on the chair and were instructed to imagine executing the TUG and to say “stop” out loud when they were finished. Participants could choose to do the iTUG with their eyes opened or closed. The stopwatch was started on the command “ready-set-go” and stopped when the participants pronounced the word “stop.”

Serum 25 OHD measurement

Venous blood was collected from resting participants for the measurement of serum 25OHD. Serum 25OHD concentrations were measured by radioimmunoassay (Incstar Corp., Stillwater, Minn., USA). With this method, interference of lipids is avoided that is often observed in other non-chromatographic assays of 25OHD. The intra- and interassay precisions were respectively 5.2 and 11.3 %, (range in normal adults aged 20–60 years, 30–125 ng/mL). Normal vitamin D status was

defined by a serum 25OHD concentration >30 ng/mL, moderate insufficiency by serum 25OHD between 10 and 30 ng/mL, and severe insufficiency by serum 25OHD <10 ng/mL (Annweiler et al. 2010a, b). All 25OHD measurements were performed in the same laboratory at Lyon, France.

Outcome variables

The main outcome variables were (1) the time taken to perform and to imagine TUG (mean $\pm$ SD) and the time difference between pTUG and iTUG (i.e., TUG delta time) expressed in percentage and calculated according to the following formula: $(pTUG - iTUG) / [(pTUG + iTUG) / 2] \times 100$ and serum 25OHD concentrations. Age, gender, BMI, the number of drugs taken each day, the use of psychoactive drugs, far visual acuity, lower limb proprioception, handgrip strength, depressive symptoms, and cognitive impairment were used as covariates in the data analysis.

Statistical analysis

The participants' characteristics were summarized using means and standard deviations or frequencies and percentages, as appropriate. The normality of the data distribution was verified with skewness and kurtosis tests before and after applying usual transformations to normalize non-Gaussian variables. As the number of observations was higher than 40 for each group, no transformation was applied. Participants were assigned into three groups based on serum 25OHD concentration cutoff values (i.e., severe insufficiency <10 ng/mL; moderate insufficiency: 10–30 ng/mL; normal status >30 ng/mL). Between-group comparisons were performed using one-way analysis of variance (ANOVA) with Bonferroni corrections or chi-square, as appropriate. Multiple linear regression analyses (i.e., full adjusted model and backward model) were performed to specify the association between TUG delta time (dependent variable) and serum 25OHD (independent variable) adjusted for participants' baseline characteristics. *P* values less than 0.05 were considered as statistically significant. All statistics were performed using SPSS (version 15.0; SPSS, Inc., Chicago, IL).

Standard protocol approvals, registrations, and patient consents

Participants in the study were included after having given their written informed consent for research. The study was conducted in accordance with the ethical standards set forth in the Helsinki Declaration (1983). The study protocol was approved by Lyon Sud-Est III local Ethical Committee, France.

Results

A total of 15.6 % ($n=56$) of participants presented a severe 25OHD insufficiency, 71.3 % ($n=256$) a moderate insufficiency, and 13.1 % ($n=47$) a normal status. The mean 25OHD concentration was 4.3 ± 2.5 ng/mL within the group with severe 25OHD insufficiency, 19.4 ± 5.0 ng/mL within the group with moderate 25OHD insufficiency, and 36.0 ± 5.3 ng/mL within the group with normal status ($P < 0.001$ for all between-group comparisons). Time to perform TUG was higher (i.e., worse performance) among participants with severe 25OHD insufficiency compared to those with moderate 25OHD insufficiency ($P=0.009$), whereas iTUG was similar among the three groups ($P=0.884$) (Table 1). However, participants with severe 25OHD insufficiency had higher TUG delta time (i.e., worse performance) compared to those with a moderate insufficiency ($P=0.010$) and with a normal 25OHD status ($P=0.048$). No significant between-group difference for the other clinical characteristics, except for lower limb proprioception (reduced among participants with a severe 25OHD insufficiency) was noticed.

As shown in the multiple (i.e., full and backward) linear regression models, TUG delta time was negatively associated with serum 25OHD concentration ($P=0.008$ and $P=0.006$, respectively) and lower limb proprioception ($P=0.038$ for full adjusted model), and positively associated with BMI ($P=0.035$ and $P=0.020$, respectively) and cognitive impairment ($P=0.004$ and $P=0.002$, respectively; Table 2).

Discussion

In this study, we tested, in a large group of non-demented older community-dwellers, the hypothesis that serum 25OHD concentrations may influence higher

levels of gait control assessed by the time difference (i.e., TUG delta time) between pTUG and iTUG. The results showed that participants with severe 25OHD insufficiency presented an increased time delay between the actual time performance of the TUG and its imagination in comparison to the other participants. In addition to its association with serum 25OHD concentrations, TUG delta time was associated with obesity and cognition.

The main finding of our study is that the lowest serum 25OHD concentration was associated with an increase in time taken to perform TUG, but not with an increased time to imagine it. The association between hypovitaminosis D and pTUG may be explained by the adverse effects of hypovitaminosis D on the neuromuscular system (Annweiler et al. 2009a, b; Annweiler et al. 2010c; Annweiler et al. 2014a; Beauchet et al. 2011a). Muscle is indeed a target tissue for vitamin D, which acts on myocytes via genomic and non-genomic pathways, and thus influences motor performance (Annweiler et al. 2009a; Annweiler et al. 2010a, b, c). For instance, chronic low serum 25OHD concentration has been associated with muscle weakness of lower limb (Annweiler et al. 2010b; Annweiler et al. 2014a). In our study, the absence of association between muscle strength and serum 25OHD concentration can be explained by the fact that only handgrip strength was explored. Reduced nerve conduction has been described in individuals with serum 25OHD concentrations below 30 ng/mL (Annweiler et al. 2010b), explaining the alteration of lower limb proprioception among participants with severe 25OHD insufficiency shown in our sample. These combining effects of vitamin D on the sensory and the motor systems contribute to the increased time to perform TUG reported in our study.

Increased pTUG duration should be accompanied by a parallel increased time of iTUG since actual execution of gait is normally associated to its mental representation (Jeannerod and Decety 1995; Jeannerod 1995; Bakker et al. 2007a). This absence of adaptation of the iTUG performance—whose main consequence was to make iTUG faster than TUG—may be interpreted as another adverse effect of hypovitaminosis D. Indeed, a growing number of evidence supports the idea that MI involves higher levels of motor control, which are themselves under the influence of vitamin D (Annweiler et al. 2009b; Annweiler et al. 2010a; Annweiler et al. 2014a). In terms of neuroimaging, MI of gait has been associated with the activations of neural networks

Table 1 Clinical characteristics of participants according to serum 25OHD concentrations ($n=359$)

	Serum 25OHD concentrations (ng/mL)			P value ^a			
	<10 ($n=56$)	10–30 ($n=256$)	>30 ($n=47$)	Overall	<10 versus 10–30	<10 versus >30	10–30 versus >30
Age, mean $\pm$ SD (years)	70.9 $\pm$ 5.3	70.5 $\pm$ 4.9	69.4 $\pm$ 4.1	0.268	–	–	–
Female, n (%)	23 (41.1)	101 (39.5)	22 (46.8)	0.639	–	–	–
Number of drugs daily taken, mean $\pm$ SD	3.0 $\pm$ 2.7	2.6 $\pm$ 2.4	2.7 $\pm$ 3.1	0.548	–	–	–
Use of psychoactive drugs ^b , n (%)	48 (85.7)	215 (84.0)	36 (76.6)	0.399	–	–	–
Body mass index (kg/m ²), mean $\pm$ SD	27.3 $\pm$ 5.4	26.5 $\pm$ 4.1	25.3 $\pm$ 3.5	0.057	–	–	–
Visual acuity ^g (/10), mean $\pm$ SD	6.8 $\pm$ 2.6	7.6 $\pm$ 2.3	7.6 $\pm$ 2.3	0.080	–	–	–
Lower limb proprioception ^f (/8), mean $\pm$ SD	5.6 $\pm$ 1.6	5.9 $\pm$ 1.3	6.3 $\pm$ 1.2	<i>0.042</i>	0.398	<i>0.035</i>	0.240
Handgrip strength ^e (N/m ²), mean $\pm$ SD	30.8 $\pm$ 11.1	31.4 $\pm$ 10.9	30.6 $\pm$ 11.2	0.867	–	–	–
Depression symptoms ^c , n (%)	17 (30.4)	59 (23.0)	9 (19.1)	0.373	–	–	–
Cognitive impairment ^d , n (%)	20 (35.7)	78 (30.5)	11 (23.4)	0.399	–	–	–
Timed "Up & Go" test (seconds), mean $\pm$ SD							
Performed	11.2 $\pm$ 3.5	10.0 $\pm$ 2.6	10.0 $\pm$ 2.8	<i>0.010</i>	<i>0.009</i>	0.078	1.00
Imagined	6.3 $\pm$ 2.5	6.4 $\pm$ 2.4	6.5 $\pm$ 2.1	0.884	–	–	–
TUG delta time ^h (%), mean $\pm$ SD	56.6 $\pm$ 28.5	44.5 $\pm$ 27.5	43.3 $\pm$ 28.4	<i>0.010</i>	<i>0.010</i>	<i>0.048</i>	1.00

Significant P value (i.e., $P<0.05$) indicated in italics

25OHD 25-hydroxyvitamin D, SD standard deviation

^a Comparison based on one-way ANOVA, with Bonferroni corrections or chi-square test, as appropriate

^b Use of benzodiazepines or antidepressants or neuroleptics

^c Four-item Geriatric Depression Scale score ≥ 1

^d Participants with impaired Clock Drawing Test

^e Mean value of maximal isometric voluntary contraction strength measured with computerized dynamometers expressed in Newton per square meter

^f Mean value from left and right sides, based on graduated tuning fork placed on the lower limb

^g Binocular visual acuity at a distance of 5 m with a Snellen letter test chart

^h Calculated from the following formula: "[(Performed Timed "Up & Go" – Imagined Timed "Up & Go") / ((Performed Timed "Up & Go" + Imagined Timed "Up & Go") / 2)] $\times$ 100

involved in the higher level of gait control, like the supplementary motor area, the primary motor cortex, the left dorsal premotor cortex, the cingulate motor area, the bilateral precentral gyrus, and the inferior parietal cortex (Wang et al. 2008; Guillot et al. 2008; Bakker et al. 2007b), with an additional activation of the hippocampus in aging (Allali et al. 2014). In parallel, experimentation supports a direct action of vitamin D in the CNS as VDR have been found in brain neurons and glial cells (Kaluëff and Tuohimaa 2007). At this level, vitamin D is involved in the neurophysiology as a neurosteroid hormone regulating neurotrophs, neuro-immunomodulation, detoxification, and

neurotransmitters levels (Annweiler et al. 2009a; Kaluëff and Tuohimaa 2007). Its deficiency is instead associated with pathological dysfunction of the CNS such as impaired cognitive function (Annweiler et al. 2010b; Annweiler et al. 2014a). Based on our results, we thus propose that vitamin D deficiency induce dysfunction in the cerebral networks involved in MI, with subsequent abnormal MI performances.

Our findings also highlighted that cognitive impairment was associated with an important time difference between pTUG and iTUG, which was in concordance with the previous study involving older adults (Beauchet et al. 2010) or patients with neuropsychiatric

Table 2 Multiple linear regression models showing the association between Timed Up & Go delta time (dependent variable) and serum 25OHD concentrations (independent variable) adjusted for clinical characteristics ($n=359$)

	Full adjusted model			Backward model		
	β	95 % CI	<i>P</i> value	β	95 % CI	<i>P</i> value
Serum 25OHD concentration	<i>-0.408</i>	<i>[-0.707; -0.108]</i>	<i>0.008</i>	<i>-0.415</i>	<i>[-0.710; -0.121]</i>	<i>0.006</i>
Age	-0.177	[-0.869; 0.514]	0.604	–	–	–
Female	0.128	[-10.042; 10.297]	0.980	–	–	–
Number of drugs daily taken	-0.305	[-1.646; 1.036]	0.655	–	–	–
Use of psychoactive drugs ^a	0.542	[-8.926; 10.010]	0.910	–	–	–
Body mass index (kg/m ²)	<i>0.757</i>	<i>[0.052; 1.461]</i>	<i>0.035</i>	<i>0.795</i>	<i>[0.127; 1.463]</i>	<i>0.020</i>
Visual acuity ^f (/10)	0.730	[-0.608; 2.069]	0.284	–	–	–
Lower limb proprioception ^e (/8)	-2.407	[-4.677; -0.136]	<i>0.038</i>	–	–	–
Handgrip strength ^d (N/m ²)	-0.225	[-0.680; 0.230]	0.331	–	–	–
Depression symptoms ^b	0.543	[-6.655; 7.741]	0.882	–	–	–
Cognitive impairment ^c	<i>9.512</i>	<i>[3.083; 15.942]</i>	<i>0.004</i>	<i>9.755</i>	<i>[3.592; 15.918]</i>	<i>0.002</i>

Calculated from the following formula: "[(Performed Timed "Up & Go" – Imagined Timed "Up & Go") / ((Performed Timed "Up & Go" + Imagined Timed "Up & Go") / 2)] × 100. Coefficient of regression β and significant *P* value (i.e., $P < 0.05$) indicated in italics

β Coefficient of regression beta corresponding to an increase or a decrease of delta time, *CI* confident interval, 25OHD 25-hydroxyvitamin D

^a Use of benzodiazepines or antidepressants or neuroleptics

^b Four-item Geriatric Depression Scale score ≥ 1

^c Participants with impaired Clock drawing test

^d Mean value of maximal isometric voluntary contraction strength measured with computerized dynamometers expressed in Newton per square meter

^e Mean value from left and right sides, based on graduated tuning fork placed on the lower limb

^f Binocular visual acuity at a distance of 5 m with a Snellen letter test chart

conditions, like schizophrenia (Lallart et al. 2012) or multiple sclerosis (Allali et al. 2012). The executive functions seem to play a key role in the TUG delta time (Lallart et al. 2012; Allali et al. 2012). This involvement of the executive functions in the higher level of gait control has also been reported in studies showing that demented older adults presented with greater gait impairments than non-demented ones and specifically when the patients presented an alteration of the executive system (Beauchet et al. 2008; Allali et al. 2008, 2010). This last point explains the association between cognition and TUG delta time reported in our study.

High BMI was also associated with increased TUG delta time, which could be explained by adverse effects of obesity on motor performance. Indeed, obese participants present not only with changes in gait due to osteoarthritis of lower limbs joints and lower muscle strength but also with

changes in cognition, especially in executive functions, memory, and visuomotor speed (Segal et al. 2009; Arterburn et al. 2005; Al-Arfaj 2002; Walther et al. 2010). In addition, it has been observed that a graded relationship between vitamin D status and BMI, lower serum 25OHD concentrations being shown with higher BMI (Pourshahidi 2014). A number of hypotheses have been proposed to explain the potential mechanisms, whereby alterations in the vitamin D endocrine system occur in obesity. Plausible explanations include sequestration in the adipose tissue, volumetric dilution, or negative feedback mechanisms from increased circulating 1,25-dihydroxyvitamin D3.

Interestingly, participants with lower 25OHD concentrations (i.e., <10 ng/ml) seemed to have normal gait performance with an average score under 12 s. To date, although the TUG has been recommended as a validated test for fall risk screening, the optimal threshold value to

detect older adults with abnormal gait performance, and thus an elevated fall risk, remains controversial: a wide range of threshold values has been reported in the literature (from 10 to 33 s) (Beauchet et al. 2011b). Furthermore, in a systematic review, it has been noticed that although the retrospective studies have shown a significant association between time taken to perform the TUG and a past history of falls, conflicting results have been observed in the prospective cohort studies (Beauchet et al. 2011b). Actually, only one of the four prospective studies found a significant association between the pTUG time and future falls (Beauchet et al. 2011b). Thus, it remains difficult to provide an interpretation of this apparent good pTUG performance in the studied participants.

Assessing such a “high number” of older adults in motor imagery of gait in addition to the determination of their serum 25OHD concentrations represents one of the main strength of our study. However, this study cohort was restricted to non-demented participants and the sample size was still low. Therefore, the studied sample may not be representative of community-dwelling older adults. Moreover, the study participants may be more motivated with greater interest in personal health issues than the general population of older adults. The cross-sectional design of the present study may limit the exploration of the association between MI and serum 25OHD concentrations and does not allow any causal inference, compared with a prospective cohort design. As the exploration of cognitive function was limited to CDT in the present study, we were unable to assess which cognitive domain moderated the association between motor imagery and serum 25OHD concentrations. Finally, although we were able to control for many characteristics likely to modify this association, residual potential confounders may still be present.

Conclusions

In conclusion, serum 25OHD concentrations contribute to the performance of motor imagery of gait in non-demented older adults, suggesting an involvement of vitamin D in higher levels of gait control. These findings also suggest that supplementing older adults in vitamin D could contribute to improve higher levels of gait

control. This hypothesis should be assessed in a future randomized controlled trial.

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Sponsor's role None

Conflict of interest The authors report no conflicts of interest.

Authors' contributions Beauchet has full access to all of the data in the study, takes responsibility for the data, the analyses and interpretation and has the right to publish any and all data, separate, and apart from the attitudes of the sponsor. All authors meet all of the following criteria: (1) contributing to the conception and design or analyzing and interpreting data, (2) drafting the article or revising it critically for important intellectual content, and (3) approving the final version to be published.

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