Hip Abductor Muscle Weakness in Individuals with Gluteal Tendinopathy

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ABSTRACT

ALLISON, K., B. VICENZINO, T. V. WRIGLEY, A. GRIMALDI, P. W. HODGES, and K. L. BENNELL. Hip Abductor Muscle Weakness in Individuals with Gluteal Tendinopathy. *Med. Sci. Sports Exerc.*, Vol. 48, No. 3, pp. 346–352, 2016. **Purpose**: This study aimed to compare hip abductor muscle strength between individuals with symptomatic, unilateral gluteal tendinopathy (GT), and asymptomatic controls. **Methods**: Fifty individuals with GT age between 35 and 70 yr and 50 sex- and age-comparable controls were recruited from the community. Maximal isometric strength (torque normalized to body mass) of the hip abductors was recorded in the supine position using an instrumented manual muscle tester. A two-way mixed ANCOVA, with covariates of self-reported pain during testing and pain limiting maximum effort, was used to compare hip abductor strength of the symptomatic and asymptomatic hip between GT and control individuals. Data were expressed as mean and SD, with the pairwise comparisons expressed as mean differences and 95% confidence intervals. **Results**: Individuals with GT demonstrated significantly lower hip abductor torque of both their symptomatic and asymptomatic hip than healthy controls (both $P < 0.05$), with mean strength deficits of 0.35 $N \cdot m \cdot kg^{-1}$ (32%) on the symptomatic hip and 0.25 $N \cdot m \cdot kg^{-1}$ (23%) on the asymptomatic hip. In individuals with GT, the symptomatic hip was significantly weaker than the asymptomatic hip with a mean strength deficit of 0.09 $N \cdot m \cdot kg^{-1}$ (11%) ($P < 0.05$). **Conclusions**: People with unilateral GT demonstrate significant weakness of the hip abductor muscles bilaterally when compared with healthy controls. Although it is not clear whether hip weakness precedes GT or is a consequence of the condition, the findings provide a basis to consider hip abductor muscle weakness in the treatment plan for management of GT. **Key Words:** HIP, STRENGTH, MUSCLE TESTING, DYNAMOMETER, GREATER TROCHANTERIC PAIN

Gluteal tendinopathy (GT) is a debilitating musculoskeletal condition (2,21) that most frequently affects women age 40–60 yr (23,40,49). The condition is characterized by pain at or around the greater trochanter of the hip with distinct tenderness on palpation (4,50). Pain is typically aggravated by walking, stair climbing, and lying on the affected side (21,48), all of which can affect daily activities and physical activity levels. Treatment of GT and its results is variable, and the optimal management strategy for the condition has not been established (18,29).

GT involves tendinopathic change of the gluteus medius/and or minimus tendons with or without bursal distension (9,42,53). With respect to the aetiology of GT, it is hypothesized that excessive hip adduction during functional activities may compress the gluteus minimus and gluteus medius tendons against the greater trochanter, driving pathological changes within these tendons (3,7,10,11). The gluteus medius and minimus muscles constitute the deepest layer of the hip abductor muscle group whose primary function is eccentric control of hip adduction during single-leg loading tasks (1). Weakness of these muscles may result in excessive hip adduction during dynamic loading (25,28,31,44), thereby contributing to the development and exacerbation of GT and/or impeding recovery.

A greater understanding of muscle weakness associated with GT will likely assist with the design of targeted physiotherapy rehabilitation programs. Exercise and load modification seem to be effective strategies in managing tendinopathy (34,47). An informed exercise program for GT would rely on evidence of muscle weakness in the condition, which is currently lacking. The only available data of hip abductor strength indicate an association between reduced hip abductor muscle strength, defined as less than 5 on a nominal five-point scale on a manual muscle test, and a magnetic resonance imaging (MRI) diagnosis of GT in individuals with lateral hip pain (53). The failure to include a control group and the absence of objective strength measurement in that study limit the interpretation of the data. Thus, there is insufficient evidence upon which to design an exercise program.

The primary aim of this study was to compare strength of the hip abductor muscles between individuals with GT and asymptomatic controls. It was hypothesized that (a) participants

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with GT would have significantly lower hip abduction strength than asymptomatic controls and (b) the symptomatic side would be weaker than the asymptomatic side.

METHODS

Participants. Fifty people with unilateral GT and 50 asymptomatic controls age 35–70 yr were recruited for the present study over a 14-month period. To detect an effect size of 0.6 with 80% power, we required a sample size of 50 subjects per group. Individuals in the GT group were recruited from the community using local and national newspaper advertisements as well as online and radio advertising. Controls were recruited from the community using print and online advertising to be comparable in sex and age with the GT group. Ethical approval was obtained from the institutional human research ethics committee. All participants provided a written informed consent.

For this study, GT was defined as a clinical diagnosis of GT (22,48,53) with MRI confirmation of tendon pathology (14), that is, symptomatic MRI tendon pathology. Participants with GT first underwent phone screening to determine eligibility for physical screening for inclusion in the present study. The initial inclusion criterion for physical screening for the GT group was the presence of unilateral–lateral hip pain (22,48,53) greater than 4/10 on a numerical rating scale for longer than 3 months, absence of groin pain, low back pain, knee pain, known hip or knee osteoarthritis, or presence of any systemic diseases affecting the muscular or nervous systems. Participants who fulfilled the initial inclusion criteria attended a physical screening, performed by a qualified physiotherapist, to confirm a primary clinical diagnosis of GT and to exclude a primary clinical diagnosis of intraarticular hip pathology, the latter defined by reproduction of groin pain with or without reduced range of movement during end-range passive hip flexion, adduction, and internal rotation (hip quadrant) (38,51) compared with those on the other side. Clinical diagnosis of GT was defined as reproduction of trochanteric pain greater than 4/10 with palpation of the greater trochanter (22,37) and during at least one of the following diagnostic clinical tests for GT (22,27): 1) hip FADER: passive hip flexion to 90°, adduction, external rotation performed in supine (41); 2) hip FADER static derotation test: static muscle internal rotation test at 90° passive hip flexion and external rotation (35); 3) hip FABER: passive hip flexion, abduction, external rotation performed in supine (22); 4) modified Ober test: in sidelying with the lower extremity against the supporting surface in a position of 90° hip flexion, the uppermost leg moved to the end of the available range of hip adduction (33); and 5) static hip abduction at an end-range Ober test position (27). Participants with clinically diagnosed GT underwent a standard non–weight-bearing anterior–posterior hip x-ray in the supine position and noncontrast MRI to confirm evidence of GT and to exclude intraarticular hip pathology. The MRI protocol consisted of coronal T1-weighted series and a sagittal, coronal, and axial proton density fat saturation series. Primary MRI diagnosis of GT was defined by the classification criteria as per Blankenbaker et al. (14); tendinopathy was considered an area of intratendinous T2 hyperintensity with a thickened tendon without any irregularity, tendon thinning, or focal tendon discontinuity. The size of T2 hyperintensity was estimated by the radiologist to characterize the grade of tendinopathy, as follows: 1) mild (localized mild distension), 2) moderate (localized moderate distension), or 3) severe (localized marked distension). Exclusion criteria after radiological assessment were as follows: 1) radiological evidence of intraarticular hip pathology including fractures, avascular necrosis of the head of femur, and bony lesions; and 2) radiological evidence on plain x-ray of more than mild osteoarthritis defined as Kellgren and Lawrence grade 2 or above.

Control participants were free of any lateral hip or lower limb pain and were recruited to be comparable in age and sex with GT participants. The following exclusion criteria were implemented at phone screening: 1) any hip, lower limb, or lumbar pain that interfered with function such as walking or that caused the participant to seek treatment in the preceding 12 months, 2) any lumbar spine or lower limb surgery in the previous 6 months, and 3) any systemic diseases affecting the muscular or nervous systems. Fifty-four potential control participants were screened through phone, after which four were excluded (two individuals had low back pain at the time of screening, and two were not in the required age range of 35–70 yr old).

Hip abductor strength. Strength testing was performed by a physiotherapist. Standardized instruction and encouragement were provided to all participants. Isometric hip abduction strength was tested in supine (gravity eliminated) using a Lafayette Manual Muscle Tester 01160/01163/01165 (Lafayette Instrument Company, Lafayette, IN) placed above the lateral malleolus and fixed with an inelastic belt to the treatment table (Fig. 1). The tested limb was placed in midrange of rotation and 10° of hip abduction to minimize potential compression of the gluteal tendons against the greater trochanter. The lever arm was measured as the distance from the greater trochanter to the center of the dynamometer (D). The nontested lower extremity was positioned in 45° of hip and knee flexion, with the foot resting on the bed and the upper limbs resting lightly across the chest. A second fixation belt was placed around the participant's pelvis and the bed to prevent the participant from using trunk strategies during the procedure. After a single submaximal effort to familiarize participants with the procedure, three trials (each of 3-s duration) of maximal isometric hip abduction against the dynamometer were performed with a 60-s rest between trials. The maximum force output (N) from the three trials was recorded and converted to torque (N·m) by multiplying the lever arm (D) and then expressed relative to body mass ($N \cdot m \cdot kg^{-1}$). This procedure has been previously used in our laboratory, demonstrating excellent test–retest reliability (45), which was consistent with pilot testing of the current assessor (n = 20 hips; intraclass correlation coefficient, 0.96).

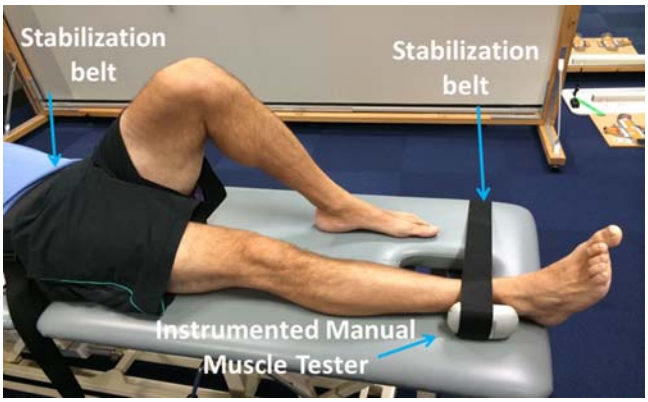

FIGURE 1—Maximal isometric hip abductor strength testing in supine position. A stabilization belt is placed over the pelvis of the participant, and a second stabilization belt was placed to stabilize the instrumented muscle tester above the lateral malleolus.

Pain during strength testing. Participants reported experiencing lateral hip pain during isometric hip abduction testing using an 11-point numerical rating scale (anchored with no pain at 0 and worst pain imaginable at 10). Participants indicated whether they considered that this pain limited their capacity to generate maximum effort during the test (yes or no).

GT-related lateral hip pain history and physical dysfunction. Participants in the GT group reported duration of lateral hip pain in months. Severity of average lateral hip pain and worst lateral hip pain experienced over the past week were reported using the 11-point numerical scale.

Data management and analysis. To balance the statistical model, the hips of control participants were arbitrarily designated as "symptomatic" and "asymptomatic" in a random manner using a coin toss. Data analysis was conducted with the Statistical Package for the Social Sciences statistical software version 22 (IBM, New York, NY). All data were explored for normality. Continuous descriptive data for each group were expressed as mean (SD) for normally distributed data and as median and interquartile range (IQR) for nonnormally distributed data. Independent *t*-tests were used to compare the normally distributed data between groups and Mann–Whitney *U* tests used for nonnormal data.

As torque data were found to be normally distributed, parametric tests were used. The torque data of the GT group were first analyzed to identify differences in hip abductor strength between individuals who reported pain limiting maximum effort and those who did not using a two-way mixed ANOVA. A two-way mixed ANCOVA was used to compare hip abductor strength of the symptomatic and asymptomatic hip within GT and control individuals using pain levels and the dichotomous variable of whether patients considered that pain limited their maximum effort (yes/no) during testing as covariates. Pairwise comparisons were expressed as mean differences and 95% confidence intervals.

RESULTS

Participant characteristics are shown in Table 1. The groups were comparable in age and sex distribution; however, the GT group was significantly heavier and had a higher body mass index (both $P < 0.05$). The median (IQR) reported duration of symptoms in the GT group was 24 (30) months, and the severity of lateral hip pain experienced over the past week was 5 (1) on the 11-point numerical pain

TABLE 1. Descriptive characteristics of the study sample.

	GT (*n* = 50)	Controls (*n* = 50)	*P* Value
Age (yr)	55.0 (8.6)	53.5 (8.1)	0.385
Height (m)	1.67 (0.09)	1.68 (0.09)	0.755
Mass (kg)	77.2 (15.2)	68.6 (11.1)	0.002
Body mass index ($kg \cdot m^{-2}$)	27.7 (5.4)	24.3 (2.5)	<0.001
Sex, *n* (%)			
Female	38 (76%)	38 (76%)	
Male	12 (24%)	12 (24%)	
Duration of lateral hip pain symptoms, median (IQR) (months)	24.0 (30.3)	0	<0.001
Symptomatic hip[b]	Right, 20; left, 30	Right, 22; left, 28	
Dominant limb	Right, 45; left, 5	Right, 47; left, 3	
Lateral hip pain severity (0–10)[a]			
Average over past week, median (IQR)	5 (1)	0	<0.001
Worst over past week, median (IQR)	8 (1)	0	<0.001
MRI diagnosis, location, and severity of tendinopathy, *n* (%)[c]			
Gluteus minimus	Mild, 6 (12%); moderate, 6 (12%); severe (0%)		
Gluteus medius	Mild, 4 (8%); moderate, 6 (12%); severe (0%)		
Gluteus minimus and medius	Mild, 12 (24%); moderate, 12 (24%); severe 4 (8%)		
Lateral hip pain during testing			
Pain reported symptomatic hip, *n* (%)	34 (68%)	0 (0%)	
Pain reported asymptomatic hip, *n* (%)	3 (6%)	0 (0%)	
Pain prevented maximal effort symptomatic hip, *n* (%)	10 (20%)	0 (0%)	
Pain prevented maximal effort asymptomatic hip, *n* (%)	0 (0%)	0 (0%)	
Pain level symptomatic hip (0–10)[a], median (IQR)	3 (4)	0 (0)	<0.001
Pain level asymptomatic hip (0–10)[a], median (IQR)	0 (0)	0 (0)	0.08

Data are presented as mean (SD) unless otherwise stated.
[a]Measured using an 11-point numerical rating scale, where 0 indicates no pain and 10 indicates the worst pain imaginable.
[b]*Symptomatic hip* in control participants as designated by a coin toss.
[c]MRI diagnosis as per location of T2 hyperintensity were subjectively graded by radiologist as follows: 1) mild (localized mild distension), 2) moderate (localized moderate distension), or 3) severe (localized marked distension).

TABLE 2. Maximal isometric hip abductor strength ($N \cdot m \cdot kg^{-1}$) and pain during testing (mean (SD) or 95% CI) for individuals with GT categorized as those who reported pain limiting maximum effort on the symptomatic hip and those who did not.

	Pain Limiting Maximum Effort GT ($n = 10$)	No Pain Limiting Maximum Effort GT ($n = 40$)	Mean Difference (95% CI)
Strength symptomatic hip	0.76 (0.21)	0.76 (0.26)	0.00 (−0.17 to 0.17)
Strength asymptomatic hip	0.81 (0.30)	0.85 (0.25)	0.04 (−0.22 to 0.14)
Mean strength difference between symptomatic and asymptomatic hip (95% CI)	0.05 (0.04 to 0.14)	0.09 (0.05 to 0.13)	
Pain level (0–10)[a] reported in symptomatic hip during testing[b]	5 (2)	2 (4)	1.9 (0.34 to 3.5)*
Pain level (0–10)[a] reported in asymptomatic hip during testing[b]	0 (0)	0 (0)	0.2 (−0.66 to 0.26)

[a]Measured using an 11-point numerical rating scale, where 0 indicates no pain and 10 indicates the worst pain imaginable.
[b]Data are presented as median (IQR).
*$P < 0.05$.

rating scale. Thirty-four (68%) participants in the GT group reported lateral hip pain during testing, but pain was less than that reported over the past week, and was at a relatively low median (IQR) level of 3 (4). Only 10 (20%) participants in the GT group considered that pain limited their maximum effort. No control participant experienced any lateral hip pain during testing. There was no significant difference in strength (either in symptomatic and asymptomatic hips) for the GT group between those who did and did not report that pain limited their maximum effort (Table 2). Consequently, the whole GT group was considered for the between-group analysis, but pain levels and the dichotomous variable of whether pain limited their maximum effort during testing were used as covariates.

There was a significant side-to-side difference in hip abductor strength within the GT group (0.09 $N \cdot m \cdot kg^{-1}$ (11%); effect size, 0.36) and a significant difference between groups in both the symptomatic and asymptomatic hip (both $P < 0.05$) (Table 3). The magnitude of difference between groups was greater than the difference between sides for the GT group. The mean between group difference was 0.35 $N \cdot m \cdot kg^{-1}$ (32%; effect size, 1.4) for the symptomatic hip and 0.25 $N \cdot m \cdot kg^{-1}$ (23%; effect size, 1.0) for the asymptomatic hip.

DISCUSSION

Few studies have investigated the physical impairments in individuals with GT. This study investigated hip abductor muscle strength because of the involvement of the hip abductor muscle tendons in GT. The results of this study demonstrated significant weaker hip abductor muscles, bilaterally, in individuals with unilateral GT than those of healthy asymptomatic controls. Strength differences were also apparent between the symptomatic and asymptomatic hip in individuals with unilateral GT, although the difference between limbs was smaller than the difference between groups.

TABLE 3. Maximal isometric hip abductor strength ($N \cdot m \cdot kg^{-1}$) in individuals with GT and control participants (mean (SD)) adjusted for covariates and pairwise comparison mean differences (95% CI).

	GT ($n = 50$)	Controls ($n = 50$)	Mean Difference (95% CI)
Symptomatic hip	0.76 (0.25)	1.11 (0.25)	0.35* (0.25 to 0.45)
Asymptomatic hip	0.85 (0.25)	1.10 (0.25)	0.25* (0.15 to 0.35)
Mean difference (95% CI)	0.09* (0.05 to 0.11)	0.00 (−0.03 to 0.07)	

*$P < 0.05$.

Several case studies of GT have reported reduced cross-sectional area (atrophy) of the gluteus medius (17,43,52) and minimus (43) muscles on the basis of visual report of a radiologist in the presence of MRI-confirmed symptomatic gluteal tendon pathology. Atrophy of these muscles would contribute to hip abductor weakness in GT. Similarly, in studies of hip osteoarthritis, unilateral gluteus medius muscle atrophy is consistently reported as the mechanism underpinning hip abductor weakness when contrasted to the asymptomatic side (36). Limited evidence for gluteal muscle atrophy in GT precludes conclusions regarding whether muscle atrophy is unilateral, whether it is a consistent feature of GT, or whether atrophy precedes or results from GT. However, it is plausible that gluteal muscle atrophy and hip abductor weakness could result from unilateral symptomatic GT as a result of disuse or inhibition of these muscles in the presence of lateral hip pain, particularly given the typically chronic nature of the condition and aggravation of symptoms with day-to-day activities such as walking and stair climbing (9,53). Certainly, reduced physical activity has been reported in individuals with GT (21), and this could underpin more widespread muscle wasting including the presentation of bilateral hip abductor weakness.

The reported strength deficit in the GT group is likely to reflect a functional impairment. To control the position of the pelvis in the frontal plane during gait, the hip abductor muscles must generate sufficient torque to match the magnitude of the external hip adduction moment, influenced primarily by body mass (39). Thus, in the present study, strength data were normalized to body mass to reflect the functional demand on the hip abductors. This method of normalization is used conventionally, as it accounts for differences in available muscle mass with increasing body size, which in general is the major factor that explains differences in muscle strength between individuals (32). However, variations in body composition, such as the increased adiposity common in GT (20), imply that a muscle may not represent a consistent proportion of total body mass when comparing the GT group with asymptomatic controls. Certainly, in the present study, individuals in the GT group were significantly heavier and had higher body mass index (within the range defined by the World Health Organization as overweight) than those in the control group. Increased adiposity (and associated implications on systemic inflammatory processes, e.g., cytokine release) is also considered to be a risk factor

for tendon pathology (24). Nonetheless, increasing body mass increases the demand on the hip abductors in body weight-supported functional activities, and thus, the reported hip abductor strength deficit may contribute to gluteal tendon overload and the development and perpetuation of GT.

Tendinopathic mechanisms in GT may contribute to hip abductor weakness of the symptomatic and asymptomatic hips in the GT group as a result of pain-related reduction in activation. At a cellular level, tendinopathy is characterized by local changes in tenocyte activity and local cell signaling (12,13), which leads to production of neuropeptides that influence the excitability of nociceptive nerve fibers (5,13). Persistence of such local tissue changes may have wider effects on sensory and motor thresholds of both the symptomatic and asymptomatic hip in GT. Heales et al. (30) propose that there is evidence of sensory and motor changes bilaterally in unilateral tendinopathy perhaps contributed to by the CNS. The presence of weakness of the asymptomatic hip in the GT group may reflect centrally mediated processes in the presence of unilateral GT.

Resisted hip abduction in side-lying has been investigated as a clinical test for pain or symptom provocation in individuals with GT (9,22,35). Test sensitivity (positive for evoked pain) has been reported to be between 46% (9) and 95% (35). Although 68% of our participants with GT reported pain during muscle strength testing of the symptomatic hip, pain intensity was low to moderate and no difference in maximum strength was identified between individuals with GT who reported that pain limited their maximum effort and those who did not. Given that hip abductor weakness was present in the asymptomatic hip in individuals with GT in the absence of pain, it is unlikely that pain experienced during testing was the primary mechanism underpinning the bilateral hip abductor weakness observed here.

In addition to hip abductor weakness being a potential consequence of pathology, hip abductor weakness may play a role in GT disease pathogenesis. The primary function of the hip abductor muscles is to maintain alignment of the pelvis in the frontal plane and thereby control hip adduction during single-leg weight bearing (1,25,44). Excessive hip adduction may compress the gluteal tendons against the greater trochanter and predispose an individual to the development of GT (8,16). Bilateral weakness may be a reflection of poor functional movement patterns including failure to control hip adduction during gait (25). Bilateral presentation of GT is relatively uncommon (9,35,53). Although we found differences in abductor muscle strength between sides in individuals with unilateral symptomatic GT, the difference was smaller than the difference between groups. It may be possible in the presence of bilateral weakness and poor functional movement patterns; the unilaterality of GT is a result of asymmetrical static postural habits that increase compression of the gluteal tendons against the greater trochanter, such as sitting cross-legged or lying on the affected side (26).

These results have several implications for clinical practice. Bilateral hip abductor weakness in individuals with GT suggests that the asymptomatic side should not be used as a "normal" comparison in clinical assessment. In addition, our data raise considerations regarding weight reduction and control in individuals with GT to prevent body mass generated overload of the hip abductor tendons. This study provides preliminary evidence to support the inclusion of hip muscle abductor strength testing in the management of GT, with the potential to use isometric hip abductor exercise as a treatment. Strengthening exercise is recommended for treatment of tendinopathy to address muscle strength and atrophy, promote tendon remodeling, and potentially provide an analgesic effect (47). To date, treatment for GT has involved corticosteroid injection (15), bursectomy (6), rest (19), and varied prescription of home exercises including stretching and strengthening exercises for the lower limb (46). Given that maximal isometric hip abduction only resulted in low-to-moderate pain provocation on the GT participants, hip abductor strengthening might be an appropriate exercise for this group. Randomized controlled trials are required to evaluate the efficacy of hip abductor strengthening exercises in GT.

This study has some limitations. As the present study had a cross-sectional design, it cannot be ascertained whether hip abductor weakness is a consequence of GT and/or a precipitating factor for its development. Further research is required to establish this relationship and how it might affect management of GT. Although participants in the present study were recruited from the general public and met a minimum severity of lateral hip pain, we did not ascertain whether participants were seeking treatment and so we would suggest that caution must be used in this regard when inferring to patients attending a health care clinic. We did not have available data to compare physical activity levels between groups, so although the groups were matched for age and gender, varying physical activity levels might have influenced the results. However, given that hip pain in GT is typically reproduced with weight-bearing activities (21,48), individuals with GT are likely to have negative or altered responses to physical activity that might differ from those of healthy controls. Regardless of physical activity level, our results suggest that strengthening exercises may be indicated for individuals with GT.

In conclusion, this study showed significant weakness of the hip abductor muscles of both the symptomatic and asymptomatic hip in individuals with GT. The study provides preliminary evidence that hip abductor muscle strength testing ought to be considered as part of the management plan for GT.

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The authors declare no conflicts of interest.

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