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Delayed onset of electromyographic activity of the vastus medialis relative to the vastus lateralis may be related to physical activity levels in females with patellofemoral pain

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ABSTRACT

The aims of this study were to examine group differences in muscle activation onset of the vastus medialis (VM) in relation to the vastus lateralis (VL) and pain level during stair ascent in females with patellofemoral pain (PFP) who maintain high and moderate levels of physical activity; to determine the association between physical activity level and muscle activation onset. Forty-three females with PFP and thirty-eight pain-free females were recruited and divided into four groups based on their level of physical activity: females with PFP ($n = 26$) and pain-free females ($n = 26$) who practiced a moderate level of physical activity and females with PFP ($n = 17$) and pain-free females ($n = 12$) who practiced an intense amount of physical activity. Participants were asked to ascend a seven-step staircase and the VM and VL activation onset was determined. Females with PFP who practiced high level of physical activity demonstrated delayed onset of VM (4.06 ms) compared to healthy females (−14.4 ms). Conversely, females with PFP who practiced moderate level of physical activity did not present VM delay (−2.48 ms) in comparison to healthy females (−9.89 ms). Furthermore, physical activity significantly correlated to the muscle activation onset difference ($p = 0.005$; $R = 0.60$). These findings may explain why controversial results regarding VM and VL muscle activation onset have been found.

1. Introduction

Patellofemoral pain (PFP) is often seen in physically active men, women and adolescents, although women are 2.23 times more likely to develop PFP than men (de Oliveira Silva et al., 2015c). PFP is characterized by anterior, retro- or peripatellar pain and aggravated by activities that increase patellofemoral joint compressive forces, such as squatting, ascending and descending stairs and prolonged sitting (Willson et al., 2014; de Oliveira Silva et al., 2015a). Although this disorder accounts for 25–40% of all knee disorders in sports medicine (Freedman et al., 2014), its etiology remains unclear.

Several contributing factors have been proposed in order to explain the mechanisms underlying PFP development. Vastus medialis (VM) delayed onset in relation to vastus lateralis (VL) has arisen as a promissory hypothesis due to the influence of these muscles on patellar stabilization (Santos et al., 2008; Uliam Kuriki et al., 2011). However, despite the apparent consolidation of this hypothesis in theoretical models, onset has yielded controversial results (Chester et al., 2008). Some studies (Cowan et al., 2002; Van Tiggelen et al., 2009) have verified an onset difference between individuals with PFP and comparison groups while others have not (Cavazzuti et al., 2010; Briani et al., 2015). Therefore, the contribution of VM delayed onset to PFP remains unclear (Sheehan et al., 2012; Toumi et al., 2013).

Recently, Rathleff et al. (2013) suggested that VM delayed onset may not always be changed in individuals with PFP due to the influence of pain on muscle motor control. Studies have shown that knee pain may alter biomechanical responses during dynamic activities (Dierks et al., 2008; Tucker and Hodges, 2010). For instance, the onset of pain in PFP may result in a decreased ability of the gluteus medius and maximus to control hip adduction and internal rotation, factors highly related to PFP (Dierks et al., 2008, 2011). As PFP is a condition related to physical activity practice and the average highest level of daily pain is associated with increased physical activity (Fairbank et al., 1984), it has been

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suggested that oscillation exists in the knee pain level according to the exposure of the individuals to pain exacerbating activities during the preceding days (Thomeé et al., 1999). Therefore, it is possible that individuals with PFP who practice higher levels of physical activity could demonstrate elevated levels of pain and, thereby, may present VM delay, while individuals who practice lower levels of physical activity may not. As the majority of studies (Brindle et al., 2003; McClinton et al., 2007; Cavazzuti et al., 2010) in this area do not discriminate the sample according to the level of physical activity, individuals with different levels of physical activity and pain may be gathered in the same sample, which could produce heterogeneous data. To the best of our knowledge, such a hypothesis has not yet been investigated and may be a reasonable explanation for the controversial results regarding this subject.

Therefore, the aims of this study were to (1) examine group differences in muscle activation onset of the VM in relation to the VL and pain level during stair ascent in females with PFP who maintain high and moderate levels of physical activity, (2) determine the association between physical activity level and muscle activation onset. We hypothesized that females with PFP who practice elevated levels of physical activity would present VM delay compared to healthy females while females with PFP who practice moderate levels of physical activity would not. In addition, higher levels of pain would be found in females who maintain high levels of physical activity than in the other group.

2. Methods

2.1. Subjects

Forty-three females with PFP and thirty-eight pain-free females were recruited from the graduate student population via advertisements placed at the university, parks and gyms. Only females were included due to the high prevalence of PFP in this population (Silva et al., 2015). Based on calculations made in sample-power using Statistical Software for Social Sciences (SPSS) Version 18.0 (SPSS Inc. Chicago, IL, USA) with data from Cowan et al. (2002), a minimum sample size of 12 females per group was indicated to evaluate VM and VL onset differences with a statistical power of 85%, observing a minimum difference of 18.42 ms between means, a standard deviation of 17.16 ms and a significance level of 5%. Prior to the data collection, all participants provided written informed consent and the experimental protocol was approved by the Institutional Review Board of the University of São Paulo State Human Ethics Committee (306.729).

Diagnosis of PFP was confirmed following consensus from two experienced clinicians (>5 years' experience) and based on definitions used in previous studies (Garcia et al., 2010; Silva et al., 2014; Briani et al., 2015). The inclusion criteria were (1) anterior knee pain during at least 2 of the following activities: prolonged sitting, squatting, kneeling, running, climbing stairs, and jumping; (2) pain during patellar palpation; (3) symptoms of insidious onset and duration of at least 1 month; (4) worst pain level in the previous month at least 3 cm on a 10 cm VAS; and (5) 3 or more positive clinical signs in the following tests: Clarke's sign, McConnell test, Noble compression, Waldron test and patellar pain on palpation. The participants were required to fulfill all five requirements to be included in the study. The presence of any of the following conditions were carefully screened as exclusion criteria: events of patellar subluxation or dislocation, lower limb inflammatory process, patellar tendon or meniscus tears, bursitis, ligament tears or the presence of neurological diseases. Those who had undergone knee surgery; or received oral steroids, opiate treatment, acupuncture or physiotherapy during the preceding 6 months were excluded from this study.

Table 1
Anthropometric data of the subjects.

	MACG mean (SD)	IACG mean (SD)	MAPFPG mean (SD)	IAPFPG mean (SD)
Age (y)	21.33 (2.62)	22.21 (3.12)	21.79 (1.01)	22.77 (2.41)
Height (m)	1.64 (0.07)	1.65 (0.05)	1.66(0.08)	1.65 (0.04)
Mass (kg)	59.48 (8.13)	63.87 (10.81)	60.01 (7.10)	61.98 (9.13)
N	26	12	26	17

Abbreviations: MACG, moderate activity control group; IACG, intense activity control group; MAPFPG, moderate activity patellofemoral pain group; IAPFPG, intense activity patellofemoral pain group; SD, standard deviation.

After the screening process, the females with PFP and pain-free females were divided into four groups based on their level of physical activity. Such separation was realized through the self-administered International Physical Activity Questionnaire long form (IPAQ), a valid and reliable form for classifying level of physical activity (Craig et al., 2003). The levels of physical activity were determined by the total amount of physical activity done in the previous week involving the lower limbs and classified according to Craig et al. (2003) and Dyrstad et al. (2014). With respect to our sample, four groups were formed: females with PFP (MAPFPG = 26) and pain-free females (MACG = 26) who practiced a moderate level of physical activity and females with PFP (IAPFPG = 17) and pain-free females (IACG = 12) who practiced an intense amount of physical activity. Anthropometric data from all groups are presented in Table 1.

2.2. Instrumentation

The experimental design included a seven step staircase, each step being 28 cm deep and 18 cm high, with a walkway in front of and at the top of it. EMG data were collected using a conditioner module (Lynx®, Sao Paulo, BRA; model 1000-8-4I) with a fourth-order, zero-lag, Butterworth digital filter with cutoff frequencies of 20–500 Hz and an amplifier with a gain of 50. The preamplifier circuit on the electrode cable had a gain of 20, a common mode rejection ratio greater than 80 dB and an impedance of 1012 Ω. The raw EMG signal was recorded at a sampling rate of 4000 Hz (Ferrari et al., 2014; Briani et al., 2015). Two pairs of bipolar surface-capture Ag/AgCl electrodes (Kendall, Mansfield, MA, USA; model Medi-Trace) with diameters of 10 mm were used to obtain VM and VL EMG data. The data were collected using AqdAnalysis software (Lynx®, Sao Paulo, SP, BRA; model EMG 1000-8-4I). An electrostimulation device (Quark®, Piracicaba, SP, BRA; model Nemesys 942) was used to find the VM and VL motor points. A force plate (AMTI, OR6, Watertown, MA, USA) was positioned in the center of the fourth step and used to obtain ground reaction force data and, thus, to establish the moment when the subject was passing over the step. The force plate acquisition sampling rate was 4000 Hz and data were collected using the same software AqdAnalysis (Lynx®, Sao Paulo, SP, BRA; model EMG 1000-8-4I) (Briani et al., 2015). The EMG and force plate data were synchronized and analog-to-digital converted into 16-bit digital format using a signal conditioner module (Lynx®, Sao Paulo, SP, BRA; model ADS 1000-AC1160).

2.3. Procedure

After finding the VM and VL motor points, the skin over the anterior portion of the thigh was cleaned with rubbing alcohol. The electrodes were placed 2 cm below the motor point in the

direction of the muscle belly, with a 20 mm interelectrode distance (Pazzinatto et al., 2015). This motor point method for positioning the electrodes is in accordance with the Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM) (Hermens et al., 2000). The reference electrode was placed over the tibial tubercle.

Prior to data collection, the participants rated their pain during the previous month and at that moment on a VAS and were familiarized with the protocol. Once they felt comfortable and the investigators deemed they were ascending the stairs with consistent velocity, the sEMG data collection commenced.

Each participant was asked to ascend the stairs at their natural comfortable speed across the staircase and five successful trials were collected and averaged to be analyzed. As demonstrated by Jordan et al. (2007), controlling the timing of the stair negotiation can change the sEMG signal for gait in healthy subjects, thus, the speed of stair ascent was not controlled in this study. To ensure a natural stair ascent pattern, the participants were not made aware of the force plate, which was hidden within the fourth step covered by a rubberized fabric, making it impossible to distinguish the force plate from the other steps. In addition, the investigators were blinded concerning the groups.

2.4. EMG analysis

The analyzed EMG signals were referenced by the vertical component of ground reaction force measured by the force plate. Therefore, the EMG signal was considered only while the participant was crossing the fourth step; the vertical component of ground reaction force being a marker of the beginning and end of the EMG data collection. All processing was performed in MATLAB® (TheMathWorks, Inc, Natick, MA).

The automatic algorithm (Cowan et al., 2002), a commonly used method, was used to determine EMG onset. Initially, a linear envelope was applied to the signal and the data were full-wave rectified and low-pass filtered at 50 Hz. An automatic muscle contraction was quantified as more than three standard-deviations of signal alteration for a minimum of 25 ms above the baseline level of each muscle (Cowan et al., 2001). After identifying the respective values from the described technique, an algorithm subtracted the VL onset from the VM, where negative differences indicated previous activation of the VM and positive differences indicated previous activation of the VL (see Fig. 1).

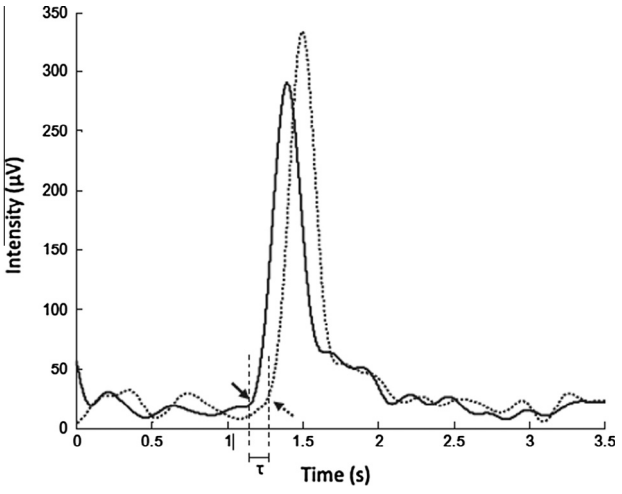

Fig. 1. The continuous line shows the VL signal, and the dotted line shows the VM signal. **t** indicates the delay between the VM and VL onsets. The signals were intentionally delayed for illustration.

2.5. Statistical analysis

Descriptive values (means (SDs)) of onset and pain were obtained and the Shapiro–Wilk test was used to analyze normal data distribution. The electromyographic variables of interest were compared between groups using univariate analysis of variance (ANOVA), with the dependent variable (onset) having 4 levels/groups (IAPFP, IACG, MAPFP, MACG). The Bonferroni *post hoc* test was performed for multiple pairwise comparisons. The data reported from ANOVA were the F values (with degrees of freedom), p values and the eta squared (η^2), whereby this last value can range from 0 to 1 and represents the proportion of variance in the dependent variable that is explained by the independent variable. The guidelines for interpreting the η^2 are: 0.01 = small effect, 0.06 = moderate effect, 0.14 = large effect. A Pearson product-moment correlation matrix was used to examine the relationships between the dependent (difference between VM and VL onset) and independent (physical activity level) variables. All statistical tests were performed using SPSS Version 18.0 statistical software (SPSS Inc, Chicago, IL), with an alpha level of 0.05.

3. Results

Independent t-tests for subject demographics revealed similar age, height, and body mass characteristics (Table 1). There was a significant interaction of groups on muscle activation onset ($F_{(3,77)} = 3.65$, $p = 0.016$, $\eta^2 = 0.13$) (Table 2). Females with PFP who practiced a high amount of physical activity demonstrated VM delayed onset compared to healthy females with the same level of physical activity ($p = 0.016$) and compared to healthy females with a moderate amount of physical activity ($p = 0.049$). On the other hand, females with PFP who practiced a moderate amount of physical activity did not present VM delay in comparison to matched physically active healthy females ($p = 0.857$) and in comparison to healthy females with a high level of physical activity ($p = 0.338$). Interestingly, differences in muscle activation onset were not found when compared IAPFPG and MAPFPG ($p = 0.720$). Furthermore, physical activity significantly correlated ($p = 0.005$) to the muscle activation onset difference in the IAPFPG, a fact not corroborated by the MAPFPG (Table 4).

When comparing the pain level, differences were found between females with PFP who maintain distinct levels of physical activity (Table 3). The IAPFPG presented higher levels of pain either for the worst pain in the previous month while ascending stairs ($p = 0.001$) or for the pain at the moment of data collection ($p = 0.02$).

Table 2
Temporal delay between VM and VL during stair ascent comparing groups with different levels of physical activity.

Groups	Mean (SD)	F-value	Eta squared
MAPFPG	−2.48 (18.8)	3.653	0.13
MACG†	−9.89 (15.3)		
IAPFPG*,†	4.06 (13.1)		
IACG *	−14.4 (13.4)		

Abbreviations: MAPFPG, moderate activity patellofemoral pain group; MACG, moderate activity control group; IAPFPG, intense activity patellofemoral pain group; IACG, intense activity control group; VM, vastus medialis; VL, vastus lateralis.

* Indicates statistical difference between IAPFPG and IACG.

† Indicates statistical difference between IAPFPG and MACG.

Table 3
Pain characteristics in individuals with PFP who present distinct levels of physical activity.

Referred pain	Groups	Mean (SD)	*p*-value
Worst pain in previous month while ascending stairs (VAS) (cm)	MAPFPG IAPFPG	4.38 (1.1) 5.86 (1.5)	0.001*
Knee pain at the moment of data collection (VAS) (cm)	MAPFPG IAPFPG	1.19 (1.3) 2.93 (2.1)	0.02*

Abbreviations: MAPFPG, moderate activity patellofemoral pain group; IAPFPG, intense activity patellofemoral pain group; PFP, patellofemoral pain.
* Indicates significant statistical difference.

Table 4
Pearson correlation matrix between onset and physical activity level.

Pearson correlation	Parameters	*R*	*p*-value
Physical activity MAPFPG	Onset	0.18	0.184
Physical activity IAPFPG	Onset	0.60	0.005*

Abbreviations: MAPFPG, moderate activity patellofemoral pain group; IAPFPG, intense activity patellofemoral pain group.
* Statistical significance in Pearson correlation coefficients.

4. Discussion

Controversial results have been reported with respect to VM and VL activation onset in individuals with PFP (Chester et al., 2008). This lack of agreement hinders the understanding of the contribution of muscle activation onset to PFP development (Wong, 2009). Our findings indicate that females with PFP who practice high amounts of physical activity present VM delay in relation to VL, while females with PFP who practice moderate amounts of physical activity do not. In addition, physical activity significantly correlated to muscle activation onset in females with PFP who practice high levels of physical activity.

Although relatively delayed onset of the VM when compared to the VL in females with PFP has not been found in some studies (Brindle et al., 2003; McClinton et al., 2007), the results from the current and past studies (Cowan et al., 2002; Van Tiggelen et al., 2009) support the theoretical model which suggests VM delay in females with PFP. However, only a portion of females with PFP seem to present this alteration. Our findings demonstrated that only females who practice a high amount of physical activity present VM delayed onset. These findings may shed some light on the controversial results in the literature. Witvrouw et al. (2000) and Earl et al. (2005) both included a physically active population in their samples and differences between groups were found toward VM delayed onset. In addition, Van Tiggelen et al. (2009) prospectively accompanied men submitted to 6 weeks basic military training (BMT) and found that 57.6% of the men with PFP presented VM delayed onset before the BMT and, interestingly, after the BMT, 100% of the men with PFP demonstrated VM delay. On the other hand, the majority of the studies that did not show differences in muscle activation onset (Brindle et al., 2003; McClinton et al., 2007; Cavazzuti et al., 2010) provide limited demographic data in terms of recreational pursuits and activity levels, which precludes further conclusions about the physical activity level of the participants.

However, without considering physical activity as a sample discriminator, it is probable that individuals with different levels of physical activity were included in the studies, which may have led to heterogeneous results regarding VM delayed onset. Therefore, a relationship between physical activity and muscle activation onset seems to exist and the controversy regarding VM delayed onset may be a consequence of distinct physical activity levels of the participants included in the studies. This hypothesis is supported by the Pearson correlation matrix analysis of our study who found a positive correlation (0.60) between high levels of physical activity and delayed onset. Nonetheless, care should be taken in interpreting this correlation because, in spite of being significant, the correlation was not strong. This may be partially explained due to the restricted sample size.

Reported pain levels were measured in order to confirm differences between females with PFP who maintain distinct levels of physical activity. As expected, females with higher levels of physical activity also presented higher levels of reported pain both in the previous month and at the moment of data collection. Physical activity has been linked to pain in females with PFP and, in turn, changes in normal muscle recruitment in a number of musculoskeletal conditions, including PFP, have been proposed as a consequence of pain (Cowan et al., 2002; Steenbrink et al., 2006; Park and Hopkins, 2013). Thus, it seems that females with PFP who practice a great amount of physical activity and who may frequently be exposed to activities that increase patellofemoral joint compressive forces present muscle activation onset alterations compared to healthy females in contrast to females with PFP who are not frequently exposed to such activities.

Interestingly, differences in muscle activation onset were not found when compared individuals with PFP in both groups. This may be related to the knee pain reported by the MAPFPG at the moment of data collection. As changes in normal muscle recruitment have been proposed as a consequence of pain (Park and Hopkins, 2013), albeit small, a knee pain level of 1.19 cm on VAS may have influenced VM and VL recruitment leading to the lack of difference between IAPFPG and MAPFPG. This influence can even be seen looking at the small difference between VM and VL onset activation (−2.48 ms). Actually, these findings may support the study's hypothesis. As these females were not exposed to a high amount of physical activity only a trend toward VM delayed onset can be seen, on the other hand, females in the other group practiced an elevated amount of physical and presented VM delayed onset.

The influence of higher levels of pain during testing on VM–VL results was not supported by Brindle et al. (2003) or McClinton et al. (2007). Significant levels of pain were reported on a VAS during stair ascent, 4.4 ± 3.0 and 2.4 ± 0.3, respectively, yet VM delayed onset was not found. However, some potential confounding factors must be acknowledged. Brindle et al. (2003) used a point at which mean voltage of a moving 25 ms window exceeded mean plus five standard deviations of baseline level to detect muscle activation onset, which is a non-standard way to perform this analysis. Except for this study, all other studies ($n = 5$) included in the most recent meta-analysis that addressed VM–VL onset during stair ascent (Chester et al., 2008) used plus two or three standard deviations of baseline level to detect muscle activation onset. This fact might be a confounding factor as it is possible that the point of muscle contraction analysis used may not correspond to the muscle activation onset due to the higher number of standard deviations. In addition, McClinton et al. (2007) reported a 2.4 ± 0.3 pain level during stair ascent and, despite this, higher knee flexion angles were found in individuals with PFP compared to pain-free individuals. In contrast, studies have shown decreased knee flexion as a consequence of knee pain in an attempt to reduce patellar pressure (Crossley et al., 2004; de Oliveira Silva et al., 2015b). Therefore, despite pain reports during stair ascent, it seems that the individuals with PFP in this sample did not present the common compensatory strategies related to PFP. One can attribute this lack of compensatory strategies to the minor exposure of these individuals to pain provoking activities during the preceding days,

which leads to unusual responses during the experimental procedure. As such, more studies are needed to investigate the exact effects of pain on muscle activation onset.

Two systematic reviews have proposed methodological differences as reasons for the lack of agreement between studies (Chester et al., 2008; Wong, 2009). To overcome these methodological concerns, Cavazzuti et al. (2010) collected electromyographic (EMG) data from both healthy and PFP individuals during a set of tasks using an onset detection algorithm with a greater accuracy than the methods previously described on this topic (Cavazzuti et al., 2010). In spite of this, there was a lack of differences between groups in the onset of VM and VL. However, why VM motor control improvement relative to VL (onset) is associated with a positive clinical outcome (Cowan et al., 2002) remains obscure. Results from the present study may clarify why VM delayed onset was not found even when methodological care was taken. Obviously, Cavazzuti et al. (2010) did not overcome all methodological differences that could affect onset determination, therefore, together with this, different levels of physical activity may account for the different results among studies. Therefore, physical activity may be a factor to be taken into account in PFP investigations.

There are some limitations of the present study that must be acknowledged. Firstly, the sample included only women. Although this subgroup is important to study, as these individuals are the most likely to suffer from PFP, the results may not be generalizable to the entire population of people with PFP. Secondly, in spite of PFP being a physical activity related condition, there was no group considered inactive, which may render the results unable to be generalized to this population of individuals with PFP. Lastly, it is unknown whether submitting females with PFP who maintain moderate levels of physical activity to a pain exacerbating protocol would stimulate the appearance of VM delayed onset. Therefore, future studies should include men and inactive individuals to compare with other physical activity levels and submit these individuals to a pain exacerbating protocol.

5. Conclusions

Females with PFP who practiced high levels of physical activity present VM delayed onset compared to controls and have higher levels of pain than those females with PFP who practiced a moderate amount of physical activity. In addition, a correlation between physical activity and muscle activation onset exists in females with PFP who maintain high levels of physical activity. These findings may explain why controversial results regarding VM and VL muscle activation onset have been found.

Conflict of interest

The authors declared that there is no conflict of interest.

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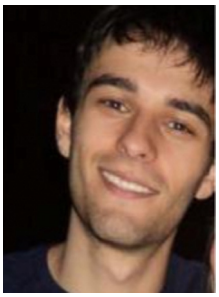

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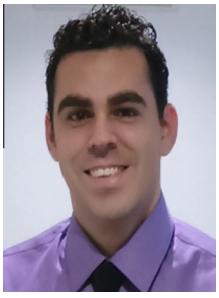

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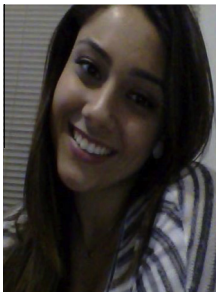

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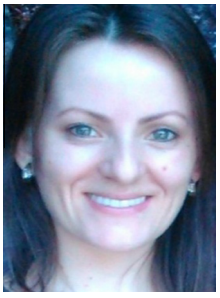

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