

Research Article

The Effects of Glucosamine on Equine and Canine Platelet Aggregation

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Abstract

Glucosamine supplementation is a commonly administered therapy in horses and dogs, but has been shown to inhibit platelet aggregation in humans and guinea pigs. The aim of this study was to determine the *in vitro* effects of glucosamine on equine and canine platelet aggregation. Platelet-rich plasma, created from blood from eight healthy horses and eight healthy dogs, was incubated for 5 minutes at room temperature with four glucosamine concentrations: 0 µg/ml, 1 µg/ml, 10 µg/ml, and 100 µg/ml. Platelet aggregation was measured, using ADP (40 µM) and collagen (10 µg/ml) as agonists. In horses, there was no change in the mean maximum aggregation at any glucosamine concentration for either ADP ($p=0.096$) or collagen ($p=0.86$). In dogs, there was an increase in maximum aggregation for the 100 µg/ml samples compared to the 0 µg/ml ($p=0.0013$) and 1 µg/ml ($p=0.0244$) samples when ADP was used as an agonist. There was no change ($p=0.2925$) in maximum aggregation when collagen was used as an agonist. Our study suggests that glucosamine does not exert an *in vitro* anti-platelet effect on equine and canine platelet aggregation.

Keywords

- Glucosamine
- Aggregometry
- Dog
- Horse

ABBREVIATIONS

ADP: Adenosine Diphosphate; PPP: Platelet-poor Plasma; PRP: Platelet-Rich Plasma

INTRODUCTION

Osteoarthritis is a multi-factorial syndrome that ultimately leads to cartilage degeneration within diarthrodial joints [1-3]. Unfortunately, there is no cure for osteoarthritis and treatments are intended to minimize or reduce the pain and discomfort associated with this condition. In veterinary medicine, glucosamine is commonly administered to both horses and dogs to maintain joint health and reduce the pain associated with osteoarthritis.

Glucosamine is believed to slow cartilage degradation and alleviate pain associated with osteoarthritis [4]. Clinical and experimental studies in both horses and dogs have demonstrated positive clinical effects of oral glucosamine administration [5-9]. The oral supplementation of glucosamine is considered to be very safe, and toxicology studies in animals and clinical trials in human patients have failed to demonstrate significant adverse effects of glucosamine supplementation on hematologic, biochemical, or physical exam parameters [10]. Additionally, human patients receiving glucosamine supplementation reported fewer side effects than those receiving nonsteroidal anti-inflammatory drugs or a placebo [10,11]. Despite the apparent safety of these

supplements, glucosamine supplementation may have some unintended consequences. In humans, the *in vitro* treatment of platelets with glucosamine caused a significant inhibition of platelet activation [12]. Also, when glucosamine was administered orally to guinea pigs, there was a decrease in platelet aggregation when platelets were activated with adenosine diphosphate (ADP), but not collagen [13]. In both humans and guinea pigs treated with glucosamine, synthesis of thromboxane A₂, a potent platelet activator and vasoconstrictor, was inhibited, suggesting that this nutraceutical may have an inhibitory effect on platelet function [12,13].

Since glucosamine is commonly administered to equine and canine patients, it is necessary to determine if this nutraceutical could adversely inhibit platelet function. The objectives of this study were to determine the *in vitro* effects of glucosamine on platelet aggregation and in horses and dogs at clinically relevant doses. Our hypothesis was that, similar to other species, the *in vitro* treatment of equine and canine platelets with glucosamine would cause a decrease in platelet aggregation.

MATERIALS AND METHODS**Study population**

Blood from eight healthy Quarter Horses (4 geldings and 4 mares) and 8 healthy Walker Hound dogs (4 castrated males and 4 intact females) was used for the study. The mean age of the

horses was 10 years (range, 5-15 years) and the mean age of the dogs was 3 years (range, 1.5-6.5 years). The mean platelet counts for the horses and dogs were 218 K/ μ L (range, 160-368 K/ μ L) and 233.7 K/ μ L (range, 160-352 K/ μ L), respectively. The horses and dogs did not receive any medications for at least 2 weeks prior to or during the study. The health status was established via physical examination, complete blood count, and serum chemistry analysis. Animal use was approved by the University Institutional Animal Care and Use Committee.

Study Design and Sample Collection

Blood samples were collected via jugular venipuncture with a 20 gauge needle into a 4.5 mL vacutainer tube containing 3.2% sodium citrate anticoagulant (Becton Dickinson, Franklin Lakes, NJ). Platelet-rich plasma (PRP) was created from whole blood via centrifugation and collected for *in vitro* treatment with three concentrations of glucosamine plus a control. The three treatment concentrations were selected based on approximate plasma concentrations of glucosamine in these species [11,14-16].

To create equine PRP, whole blood samples were centrifuged at 450 x g at room temperature for 5 minutes. The PRP supernatant was removed and the remaining blood samples centrifuged at 1,800 x g at room temperature for 8 minutes to create platelet poor plasma (PPP) [17]. A previously published technique was used to create canine PRP [18]. Briefly, whole blood was centrifuged at 1,200 x g at room temperature for 3 minutes, the PRP supernatant was removed, and the remaining blood sample was centrifuged at 1,800 x g at room temperature for 8 minutes to create PPP.

Glucosamine incubation

Previously published protocols [19,20] were modified to evaluate canine and equine platelet function following the *in vitro* treatment of PRP using 3 concentrations of D-(+)-glucosamine (BioReagent, Sigma Aldrich, St. Louis, MO) plus a control. Briefly, three concentrations of glucosamine working solutions, 1 μ g/mL, 10 μ g/mL, and 100 μ g/mL, were created from a stock solution containing 10 mg/mL of glucosamine in sterile water. To determine the dose effect on platelet aggregation, 5 μ L from each working solution was added to 245 μ L of PRP, inverted three times and incubated, at room temperature, for 5 minutes. For the control sample, 5 μ L of sterile water was added to 245 μ L of PRP. Following incubation, the treated PRP was transferred to a glass cuvette for analysis.

Platelet aggregometry

Turbidimetric aggregometry: A 2 channel turbidimetric platelet aggregometer (Chronolog Corporation Haverton, PA) was used to analyze platelet aggregation. Aggregation, for both horses and dogs, was assessed using ADP (40 μ M) or collagen (10 μ g/mL) as agonists, with a temperature of 37°C, and a stirring speed of 1,000 rpm for equine samples and 1,200 rpm for canine samples. Samples were analyzed based on the manufacturer's standard guidelines (Chronolog 700 Manual, Chronolog Corporation, Haverton, PA). Briefly, 247 μ L of glucosamine treated PRP was placed into a glass cuvette with a stir bar and 250 μ L of PPP were placed into a cuvette without a stir bar. Samples were incubated

for one minute at 37°C, placed into the aggregometer, and stable baseline values corresponding to 0% and 100% aggregation were obtained using PRP and PPP, respectively. ADP or collagen was added to the PRP, and platelet aggregation was monitored for 6 minutes and 8 minutes respectively. The maximal percentage aggregation was calculated and recorded using computer software (AGGRO/LINK 8, Chronolog Corporation, Haverton, PA). For each glucosamine concentration and agonist, 3 total samples were analyzed, and the results were averaged to yield a single value. All samples were analyzed within 4 hours of collection. Based on recommendations published by the International Society of Thrombosis and Haemostasis Platelet Physiology and Scientific and Standardization Committee, the platelet count in the PRP was not adjusted to a standardized count by dilution with PPP prior to analysis [21-23].

Statistical analysis

The effect of *in vitro* glucosamine supplementation on platelet aggregation in horses and dogs was analyzed by mixed model analysis using a computer program (SAS for Windows 9.4 SAS Institute, Inc., Cary, NC, USA). Separate models were made for each marker and species. The model included concentration as a fixed effect. Horse or dog was included as a random effect. Tukey's adjustment for multiple comparisons was made if concentration was found to have a significant effect. Conditional residual plots were assessed to ensure the assumptions of the statistical methods had been met. In the interpretation of results, P-values \leq 0.05 were considered significant.

RESULTS AND DISCUSSION

Turbidimetric aggregometry Results

Equine: Based on turbidimetric aggregometry, the mean and standard deviation of the maximum aggregation for ADP- and collagen-induced platelet aggregation is summarized (Table 1). Compared to the control sample, there were no significant changes in the maximum aggregation at any glucosamine concentration when ADP ($p=0.096$) or collagen ($p=0.86$) was used as an agonist.

Canine: The mean and standard deviation of the maximum aggregation for ADP- and collagen-induced platelet aggregation is summarized (Table 1). Compared to the control sample, with ADP-induced aggregation, there was a significant ($p=0.0013$) increase in maximum aggregation for 100 μ g/mL glucosamine concentration. Additionally, compared to the 1 μ g/mL concentration, there was a significant increase ($p=0.0244$) in maximum aggregation with 100 μ g/mL glucosamine concentration. Compared to the control sample, there was no significant changes in the maximum aggregation at any glucosamine concentration when collagen ($p=0.2925$) was used as an agonist.

DISCUSSION

The results of our study indicate that, contrary to our hypothesis, the *in vitro* treatment of platelets with glucosamine does not inhibit platelet aggregation in horses or dogs, even at supraphysiologic concentrations of glucosamine. Our results are in sharp contrast to similar studies performed in humans and

Table 1: The maximal platelet aggregation (mean +/- standard deviation) based on turbidimetric aggregometry after incubation of platelet-rich plasma with various concentrations of glucosamine.

	Glucosamine concentration (µg/mL)			
	0 µg/mL	1 µg/mL	10 µg/mL	100 µg/mL
Equine				
ADP-induced aggregation (%)	89.4 ± 9.7	84 ± 14.2	93.4 ± 8.9	94.7 ± 6.9
Collagen-induced aggregation (%)	67.6 ± 24.1	73 ± 32	75.6 ± 30.7	75.8 ± 31
Canine				
ADP-induced aggregation (%)	29.2 ± 15.1*	32.4 ± 13.8*	33.5 ± 9.2	40 ± 12.2
Collagen-induced aggregation (%)	37.4 ± 13.3	26.3 ± 16.5	28.7 ± 16.4	32.5 ± 17.2

Abbreviations: Equine and canine PRP was treated with 3 doses of glucosamine (final concentration, 1, 10, or 100 µg/mL) plus a sterile water control (glucosamine concentration, 0 µg/mL). Based on turbidimetric aggregometry, the maximum aggregation (%) was measured following ADP- or collagen-induced aggregation. Sample results that are labeled with a "*" indicate a significant (p<0.05) difference from the 100 µg/mL glucosamine concentration.

Table 2: The lag-time and slope (mean +/- standard deviation) of platelet aggregation based on turbidimetric aggregometry after incubation of platelet-rich plasma with various concentrations of glucosamine.

	Glucosamine concentration (µg/mL)			
	0 µg/mL	1 µg/mL	10 µg/mL	100 µg/mL
Equine				
Lag-time (seconds)				
ADP	32 ± 5.8	28.6 ± 6.4	29.4 ± 5.2	26.4 ± 3.7
Collagen	83.1 ± 31.4	84.3 ± 35.2	112.8 ± 48.5	117.3 ± 62.1
Slope				
ADP	77.4 ± 16.7	74.9 ± 24.6	82.6 ± 16.7	86.2 ± 15.4
Collagen	70.1 ± 20.1	81.7 ± 36	78.2 ± 27	74.9 ± 27.5
Canine				
Lag-time (seconds)				
ADP	16.8 ± 2.5	16 ± 2.2	15.3 ± 4.6	14.0 ± 2
Collagen	37.4 ± 13.3	26.3 ± 16.5	28.7 ± 16.4	32.5 ± 17.2
Slope				
ADP	56.1 ± 13.7	59.6 ± 14.6	60.1 ± 16.7	64.2 ± 13.8
Collagen	47.4 ± 23.3	29.4 ± 21.5	33.6 ± 21.2	37.5 ± 21.3

Abbreviations: Equine and canine PRP was treated with a 3 doses of glucosamine (final concentration, 1, 10, or 100 µg/mL) plus a sterile water control (glucosamine concentration, 0 µg/mL). Based on turbidimetric aggregometry, the lag-time (seconds) and slope was measured following ADP- or collagen-induced aggregation.

guinea pigs [12,13]. Lu-Suguro et al. demonstrated that compared to pretreatment aggregation, there was a 51% decrease in ADP-induced platelet aggregation in guinea pigs treated with glucosamine; however, glucosamine supplementation did not adversely inhibit collagen-induced platelet aggregation [13]. Additionally, in humans, following a week of glucosamine supplementation, there was a 29% decrease in ADP-induced platelet aggregation, without adversely inhibiting collagen-induced platelet aggregation [12]. In these studies, it was suggested that glucosamine antagonized the binding of low and high affinity ADP platelet receptors in a noncompetitive and dose-dependent manner [12,13].

In both humans and guinea pigs, the inhibition of ADP-induced aggregation was observed when platelets were activated

with low concentrations of ADP (2.5 µM and 2 µM respectively). However, similar results could not be reproduced when higher concentrations of ADP (10 µM) were used for platelet activation [12,13], suggesting that the ADP-associated anti-platelet effects of glucosamine were dose dependent and could be overcome with the addition of a higher concentration of ADP. In our study, 40 µM of ADP was used for platelet activation, which was the lowest concentration of ADP that consistently yielded 50% platelet aggregation in untreated horses and dogs. Therefore, it is possible that with the use of a lower ADP concentration, glucosamine-associated platelet dysfunction could be detected in both horses and dogs. However, with the use of lower ADP concentrations, it is possible that glucosamine associated platelet dysfunction could be mistaken for inadequate platelet activation.

One possible explanation for the differences between our study and those performed in humans and guinea pigs is a species difference in the response and sensitivity to platelet agonists. Previous studies have demonstrated species-related differences in platelet responsiveness to ADP [24,25].

Compared to the control sample and the 1 µg/mL concentration, there was a significant increase in the maximal aggregation when samples were incubated with 100 µg/mL of glucosamine. It is possible that in the supraphysiologic concentration, glucosamine could stimulate platelet activation and increase aggregation. However, additional studies are required to determine the effects of glucosamine on increased platelet aggregation.

Additional differences between our study and the previous studies were the length and method of glucosamine treatment. In both the human and guinea pig studies, glucosamine was administered *in vivo* for an extended length of time [12,13]. This long-term administration and exposure of glucosamine to platelets could have contributed to the inhibition in platelet aggregation. In our study, we utilized previously published protocols that were capable of detecting drug associated platelet inhibition following a brief *in vitro* incubation period [18-20]. Therefore, in horses and dogs, it is possible that oral administration of glucosamine over an extended period of time could have an accumulative anti-platelet effect. Additional studies are required to determine the anti-platelet effects of glucosamine following long-term, oral glucosamine therapy in horses and dogs.

Based on previous pharmacokinetic analysis of oral glucosamine administration in horses [11,14] and dogs [15,16], the concentrations of glucosamine used in this study were based on approximate plasma concentrations in horses and dogs. Unfortunately, there is a wide variation in plasma concentrations for both species in these studies. There are several factors that contribute to the wide variation in glucosamine plasma concentrations, such as differences in glucosamine bioavailability, methodology used to measure the plasma concentration, and glucosamine formulation. The 1 µg/mL and 10 µg/mL glucosamine concentrations represent the range of achievable plasma concentrations reported following oral glucosamine administration in dogs and horses [11,14,15]. The 100 µg/mL glucosamine concentration used in this study is 9.4 and 14.7 times greater than the documented mean peak plasma concentrations following oral glucosamine administration in horses [11] and dogs [15], respectively. In horses, plasma concentrations can be as high as 349 µg/mL after intravenous glucosamine administration [11], however given the variation in glucosamine oral bioavailability, approximately 5% in horses [11] and 12-26% in dogs [15,26], oral administration of standard glucosamine doses are unlikely to achieve plasma concentrations of 100 µg/mL.

McNamara et al. evaluated platelet aggregation in dogs treated with a chondroprotective agent that included glucosamine, and identified a significant decrease in platelet function [27], but there were several differences from the current study. First, along with glucosamine, the chondroprotective agent in the McNamara et al. study contained several additional compounds which could have inhibited platelet function. Based on the results of our

study, the glucosamine in the chondroprotective agent may not have been responsible for the inhibition of platelet function, but the additional components of the product may have caused the platelet dysfunction. Second, McNamara et al. used impedance aggregometry, which evaluated platelet function in whole blood, while our study used turbidimetric aggregometry and platelet-rich plasma. While impedance aggregometry provides a good assessment of platelet function, the non-platelet components of whole blood also affect platelet aggregation. In the current study, which used turbidimetric aggregometry, any glucosamine induced platelet dysfunction could only be contributed to platelets. Finally, in the McNamara et al. study, a luminance agent (Chrono-lume®) was included in the assessment of platelet function to measure ATP release during platelet aggregation. Unfortunately, this luminance agent will activate platelets and enhance aggregation in healthy and thrombocytopenic dogs [28]. The concurrent use of this luminance agent with a potential anti-platelet treatment could mask any glucosamine induced platelet dysfunction.

Our study had several limitations. First, this study was conducted *in vitro*, and not *in vivo*. Therefore, in horses and dogs, the oral administration of glucosamine, especially over an extended period of time, may have an accumulative anti-platelet effect that was not detected in our study. Secondly, our study included a small number of horses and dogs with a large standard deviation. Therefore, it is possible that with a larger population of animals different conclusions could be drawn from our study. However, the number of animals in our study were similar to previous studies that evaluated the *in vitro* effects of therapies on platelet function in both horses and dogs [21,22]. Finally, there is a wide variation in plasma glucosamine concentrations for both horses and dogs. There were several contributing factors to this variation in glucosamine plasma concentrations, including the methodology used to measure plasma concentration and glucosamine bioavailability and formulation. Except for extreme cases, the concentrations used in our study should adequately represent plasma glucosamine concentration in patients receiving oral glucosamine.

CONCLUSION

The results of our study indicate that, unlike humans and guinea pigs, glucosamine does not exert an *in vitro* anti-platelet effect on equine and canine platelet aggregation, even at supraphysiologic plasma concentrations. However, it is unknown if long-term oral glucosamine administration will inhibit platelet aggregation.

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