Osteoarthritis is a chronic degenerative joint disease characterized by pain, immobility, and impairment of joint function.1–3 In humans, osteoarthritis is a major cause of physical disability and reduced quality of life. This debilitating joint disease also affects other species, including canids, felids, and equids. In equine athletes, osteoarthritis seriously impairs performance and ultimately leads to premature retirement.4,5

Biochemical events associated with the pathological changes in osteoarthritic joints include excess production of proinflammatory cytokines (particularly IL-1β and TNF-α) and PGE2, as well as upregulation of the expression of degradative enzymes.5–7 Prostaglandin E2 is a proinflammatory lipid mediator that is locally increased in synovial membranes and synovial fluid.
during osteoarthritis. Prostaglandin E₂ upregulates metalloproteinases that degrade cartilage, suppresses aggrecan synthesis and total proteoglycan accumulation, promotes bone resorption and osteophyte formation, and contributes to the development of pain during osteoarthritis.  

Similar to horses, camels are used for transportation, trained as athletes for racing, and afflicted with lameness presumably attributable to osteoarthritis.  

Limited information is available about the pathogenesis of osteoarthritis in camels. Radiographic evaluations have been used to identify osteoarthritic lesions in the metacarpophalangeal and metatarsophalangeal joints of a camel sent to slaughter because of lameness, and chronic osteoarthritis has also been detected during necropsy. Homology evaluations that involved the use of molecular cloning as well as phylogenetic analysis of inflammatory cytokines have revealed that camels are more closely related to horses and cattle than to other mammalian species. On the basis of these observations, it is possible that horses and camels share pathological markers characteristic of osteoarthritis. 

The sole cellular constituents of cartilage are chondrocytes that are responsible for the synthesis and breakdown of ECM. The ECM components consist primarily of type II collagen and proteoglycans that aggregate to form high–molecular-weight aggrecan. Chondrocytes also degrade cartilage components as part of physiologically normal turnover and remodeling. In cartilage, the balance between anabolic and catabolic processes is maintained by a network of regulatory molecules, including proinflammatory mediators. Specifically, cytokines and PGs are critical players in regulating the homeostasis of this network. In contrast to the situation in horses and humans, biological processes in articular cartilage and chondrocytes of camels have not been investigated. Studies of the cellular mechanisms associated with the pathogenesis of osteoarthritis in camels would be facilitated if chondrocytes from camel articular cartilage could be maintained in tissue culture. Camel chondrocytes could be used in disease-relevant cell-based assays to characterize their biological response to proinflammatory stimuli. They could also be used for testing of drugs and nutraceuticals aimed at the management of osteoarthritis in camels and other species. 

In the study reported here, we evaluated the isolation and propagation of camel articular chondrocytes in vitro and determined whether cultured camel chondrocytes would respond to cytokine stimulation by producing the proinflammatory mediator PGE₂. We tested the hypothesis that cytokine-induced production of PGE₂ will be attenuated by the combination of ASU + glucosamine + CS and PPS + NG.

**Materials and Methods**

**Sample population**—Articular cartilage was obtained from 4 carpal joints of 2 healthy adult camels (Camelus dromedarius) after they were slaughtered at an abattoir in Northern Australia. Cartilage pieces were fixed in paraformaldehyde for histologic and immunohistochemical analyses. The remaining cartilage samples were aseptically diced into pieces (approx 5 mm²) and digested by incubation in type II collagenase (110 U/mL) for 12 to 18 hours at 37°C in an atmosphere of 5% CO₂. Chondrocytes were filtered through a 0.4-µm mesh screen to remove debris and were rinsed 4 times with Hank’s balanced salt solution. Cells were counted and assessed for viability by use of the Trypan blue dye exclusion method. Pools of chondrocytes were cryopreserved until subsequent use. 

**Chondrocyte propagation**—Molecular cloning studies have identified homology between camels, cattle, and horses. Thus, we first tested whether media successfully used to culture bovine and equine chondrocytes would support camel chondrocytes. Chondrocytes (1 × 10⁶ cells) were plated in 175-mm flasks containing medium of Dulbecco modified Eagle medium—F12 medium supplemented with 10% (vol/vol) fetal bovine serum, 50 µg of ascorbic acid/mL, and 50 µg of gentamicin/mL, which has been used for culture of bovine chondrocytes, or medium composed of Dulbecco modified Eagle medium supplemented with 10% (vol/vol) fetal bovine serum, 300 µg of L-glutamine/mL, 30 µg of antimicrobial-antimycotic/mL, and 3.7 µg of sodium bicarbonate/L, which has been used for culture of equine chondrocytes. Preliminary testing revealed that camel chondrocytes proliferated favorably in medium used for equine chondrocyte culture. In contrast, camel chondrocytes did not proliferate beyond the second passage when grown in medium used to culture bovine chondrocytes. Therefore, for all subsequent experiments, camel chondrocytes were cultured in medium used for culture of equine chondrocytes. 

**Immunohistochemical analysis**—Chondrocytes (1 × 10⁴ cells) were incubated in 8-well chamber slides overnight. They then were fixed in 10% (vol/vol) paraformaldehyde for 15 minutes, washed 3 times with PBS solution, and processed for immunohistochemical analysis. For staining sections of cartilage, blocks of cartilage were embedded in paraffin and sectioned at a thickness of 7 µm. Sections were deparaffinized and processed for histologic examination or immunohistochemical analysis. For staining of cells, chamber slides seeded with cultured chondrocytes were incubated overnight at 4°C with goat anti-type I human collagen, anti-type II human collagen, anti–NF-κB, and anti-human aggrecan antibodies. Cells in chamber slides were washed in PBS solution 3 times and incubated with fluorescein isothiocyanate–labeled anti-goat antibodies. Chondrocyte immunostaining was evaluated by use of a fluorescence microscope.

**Experimental procedures**—We evaluated whether cytokine-induced stimulation of PGE₂ production can be inhibited by preparations known to have anti-inflammatory activity. Two such preparations, both of which have been found to be efficacious for the management of osteoarthritis in horses, were used in the study. One preparation was a mixture of ASU, glucosamine, and CS (ie, ASU + GLU + CS). The ASU was dissolved in and diluted with 100% ethanol to achieve a final concentration of 8.3 µg/mL, which has been reported to exert significant anti-inflammatory effects in vitro. Glucosamine and CS were dissolved in Hank’s bal-
anced salt solution to achieve working concentrations that have been found in other studies\(^5,6\) to be detectable in the plasma of dogs and horses after administration. Concentrations of ASU, glucosamine, and CS in the ASU + GLU + CS mixture were 8.3, 11, and 20 µg/mL, respectively. The second preparation was a mixture of PPS and NG (ie, PPS + NG).\(^7\) Concentrations of PPS ranged from 25 to 125 µg/mL, and concentrations of NG ranged from 40 to 200 µg/mL.

Chondrocytes (5 × 10\(^5\) cells) harvested from monolayer cultures were seeded into 6-well plates and cultured for 24 hours. They then were incubated for another 24 hours with control medium alone, the ASU + GLU + CS mixture, the PPS + NG mixture, or a combination of the ASU + GLU + CS and PPS + NG mixtures. Chondrocyte cultures were subsequently incubated with medium alone or with the combination of IL-1\(\beta\) (10 ng/mL) and TNF-\(\alpha\) (1 ng/mL) for another 24 hours to determine PGE\(_2\) production.

**High-sensitivity PGE\(_2\) immunoassay**—A commercial PGE\(_2\) immunoassay\(^8\) was used in accordance with the manufacturer’s instructions to quantify secreted PGE\(_2\) concentrations in the cellular supernatant. A PGE\(_2\) standard was assayed in parallel with the supernatant samples. Optical density was measured immediately by use of a microplate reader\(^9\) at 450 nm with wavelength correction set at 540 nm.

**Immunofluorescent localization of NF-\(\kappa\)B**—Camel chondrocytes (1 × 10\(^4\) cells/well) were seeded into 8-well chamber slides.\(^10\) To determine the effect of treatments on the nuclear translocation of NF-\(\kappa\)B in response to IL-1\(\beta\) or TNF-\(\alpha\) exposure, cells were incubated for 24 hours with control medium alone, the ASU + GLU + CS mixture, the PPS + NG mixture, or a combination of the ASU + GLU + CS and PPS + NG mixtures. Cytokines were added, and cells were incubated for 1 hour. Cells then were fixed by incubation in neutral-buffered 10% formalin\(^11\) for 15 minutes and washed 3 times with PBS solution. Chondrocytes were then incubated overnight at 4°C with rabbit anti–NF-\(\kappa\)B (diluted 1:100) in PBS solution containing 0.1% Triton X-100. Cells were incubated with donkey anti-rabbit IgG labeled with green fluorescent dye\(^12\) (diluted 1:100) for 2 hours at 23°C and then were washed 4 times with PBS solution. Cells were viewed on an inverted fluorescence microscope\(^13\) equipped with a digital camera.\(^14\) Digital images for 5 frames were recorded for each experimental condition and were imported into an image-processing program.\(^15\) The number of cells that had nuclei stained or not stained was analyzed by use of a software program.\(^16\)

**Statistical analysis**—Data were reported as the mean ± SD. Statistical software\(^17\) was used to perform multiple comparisons by use of a 1-way ANOVA with a Tukey post-hoc test. Values of \(P < 0.05\) were considered significant.

**Results**

Isolation, growth, and phenotype characterization of camel articular chondrocytes—Articular cartilage retrieved from camel carpal joints appeared glassy and smooth and was 2 to 4 mm in thickness. Examination of cartilage sections stained with H&E revealed the classical hyaline cartilage structure with primarily ECM surrounding lacunae containing 1 to 3 chondrocytes. The superficial cartilage layer had chondrocytes with flattened, spindlelike morphology. Cells in the intermediate layer were spherical with eccentric nuclei. Immunohistochemical analysis confirmed localization of type II collagen and aggrecan in the cartilage ECM and chondrocytes (Figure 1). Sections stained to detect type I collagen were indistinguishable from sections in which the primary antibody was omitted in the staining process.

![Photomicrographs of tissue sections of camel (Camelus dromedarius) carpal cartilage deparaffinized and immuno-stained with monospecific antibodies against type II collagen (arrow) and aggrecan (arrowhead; A). Notice the aggrecan immunostaining (B) and the immunostaining in chondrocytes and the ECM. Bar = 50 µm.](image)

![Phase-contrast photomicrographs of camel chondrocytes cultured in medium used to culture bovine (A) and equine (B) cartilage. Not stained; bar = 100 µm.](image)
Camel chondrocytes did not propagate well in medium used to culture bovine chondrocytes and did not survive beyond passage 2. At passage 1, chondrocytes cultured in that medium appeared stellate in shape with a granular cytoplasm when viewed by use of phase-contrast microscopy (Figure 2). In contrast, chondrocytes propagated in medium used to culture equine chondrocytes proliferated well. These chondrocyte cultures proliferated with an estimated doubling time of 3 to 4 days. Chondrocytes grown in that medium had heterogeneous shapes that ranged from spheroid to elongated. Most of the cells had 1 nucleus, but a few were binucleated. Immunohistochemical analysis confirmed that the chondrocyte cultures continued to produce the ECM components type II collagen and aggrecan (Figure 3).

Effect of cytokine stimulation on PGE₂ production in camel chondrocytes—Nonstimulated camel chondrocytes incubated with control medium alone secreted undetectable or low amounts of PGE₂, compared with the amount of PGE₂ produced by cytokine-stimulated cells (Figure 4). Chondrocytes responded to cytokine activation with dramatic logarithmic increases in PGE₂ synthesis. The robust response to cytokine stimulation was similar with subsequent passages, although the magnitude of the response was more variable in later passages. Activated chondrocytes produced greater amounts (20,000- to 80,000-fold) of PGE₂, although the degree of variability was most pronounced for passage 4. Mean PGE₂ concentrations after cytokine stimulation of chondrocytes in passages 2, 3, and 4 did not differ significantly.

Inhibition of cytokine-induced PGE₂ production in camel chondrocytes—Activated chondrocytes pretreated with the ASU + GLU + CS combination had a significant (P < 0.001) decrease in PGE₂ production (Table 1). Similarly, pretreatment with PPS + NG at the concentrations tested resulted in a significant (P < 0.001) suppressive effect on cytokine stimulation of PGE₂ production. Combining the ASU + GLU + CS and PPS + NG mixtures at a wide range of concentrations

![Figure 3](image-url) Photomicrographs of cultures of camel chondrocytes immunostained with monospecific antibodies against type II collagen (A) and aggrecan (B). Notice that the chondrocytes have intense green immunostaining of the cytoplasm and blue counterstaining of the nucleus. Bar = 20 µm.

![Figure 4](image-url) Mean ± SD PGE₂ concentration in cultures of camel chondrocytes incubated in medium alone (control medium), medium containing ASU + GLU + CS, medium containing PPS + NG, or medium with a combination of both followed by incubation with IL-1β and TNF-α.

<table>
<thead>
<tr>
<th>Incubation</th>
<th>PGE₂ concentration (pg/mL)</th>
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<tbody>
<tr>
<td>Control medium</td>
<td>6,227 ± 219.3</td>
</tr>
<tr>
<td>IL-1β + TNF-α</td>
<td>93,634 ± 6,384</td>
</tr>
<tr>
<td>ASU + GLU + CS + IL-1β + TNF-α</td>
<td>67,880 ± 5,075.7</td>
</tr>
<tr>
<td>PPS + NG + IL-1β + TNF-α</td>
<td>57,999 ± 3,838.8</td>
</tr>
<tr>
<td>25 + 40</td>
<td>54,045 ± 4,391.9</td>
</tr>
<tr>
<td>50 + 80</td>
<td>68,073 ± 4,979.2</td>
</tr>
<tr>
<td>75 + 120</td>
<td>68,358 ± 1,472.1</td>
</tr>
<tr>
<td>100 + 160</td>
<td>60,837 ± 6,222.7</td>
</tr>
<tr>
<td>125 + 200</td>
<td>67,680</td>
</tr>
<tr>
<td>ASU + GLU + CS + PPS + NG + IL-1β + TNF-α</td>
<td>4,979.2 ± 6,394.7</td>
</tr>
<tr>
<td>25 + 40</td>
<td>3,838.8 ± 3,953.4</td>
</tr>
<tr>
<td>50 + 80</td>
<td>3,456 ± 4,391.9</td>
</tr>
<tr>
<td>75 + 120</td>
<td>2,807.1 ± 4,905.5</td>
</tr>
<tr>
<td>100 + 160</td>
<td>3,953.4 ± 4,979.2</td>
</tr>
<tr>
<td>125 + 200</td>
<td>2,838.8 ± 6,384.8</td>
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*Concentrations of ASU, glucosamine, and CS were 8.3, 11, and 20 µg/mL, respectively. †Concentrations of IL-1β and TNF-α were 10 and 1 ng/mL, respectively. ‡Value differs significantly (P < 0.001) from the value for IL-1β + TNF-α. §Value differs significantly (P < 0.001) from the value for ASU + GLU + CS + IL-1β + TNF-α. ¶Value differs significantly (P < 0.001) from the value for ASU + GLU + CS + PPS + NG + IL-1β + TNF-α. ‖Values reported represent concentrations (µg/mL) of PPS and NG, respectively.
Activation of NF-κB and translocation of NF-κB from the cytoplasm to the nucleus—Nonstimulated control chondrocytes had strong immunostaining for NF-κB throughout the cytoplasm, whereas the nuclei of chondrocytes appeared unstained (Figure 5). After cytokine stimulation, cytoplasmic immunostaining for NF-κB decreased, whereas the nuclei were intensely stained. This nuclear translocation of NF-κB was identified by intense immunostaining after incubation for 1 hour. Quantitation of nuclear immunostaining for NF-κB confirmed translocation after cytokine activation. Pretreatment of chondrocytes with ASU + GLU + CS or PPS + NG significantly (P < 0.001) reduced NF-κB translocation from the cytoplasm to the nucleus (Figure 6).

Discussion
Analysis of results of the study reported here revealed that chondrocytes retrieved from the articular cartilage of the carpal joints of camels can be propagated for several passages in medium used for culture of equine chondrocytes without loss of phenotype or the ability to respond to proinflammatory stimuli. It is unclear why culture medium that supports growth of equine chondrocytes also supports proliferation of camel chondrocytes. However, it is suggestive that the chondrocytes from these 2 species may have similar growth requirements.

The biological responses of cultured camel chondrocytes to the proinflammatory cytokines IL-1β and TNF-α described here were similar to the observed responses of cultured chondrocytes from other species, including equids and humans. Results of our experiments indicated that nonstimulated camel chondrocytes produce undetectable or low concentrations of PGE₂. This finding is in contrast to observations with cultured equine, bovine, and human chondrocytes, which secrete measurable concentrations of PGE₂. The low production of PGE₂ in cultured camel chondrocytes may indicate a distinct species difference or may reflect a lack of inflammation in the carpal joints used in the present study. In contrast to their low production of PGE₂, camel chondrocytes responded with a dramatic increase (up to 4 orders of magnitude) in PGE₂ synthesis when stimulated with the proinflammatory cytokines IL-1β and TNF-α. Increased production of PGE₂ during osteoarthritis has been traced to the effect of proinflammatory cytokines on chondrocytes in cartilage and synoviocytes in the lining of the joint capsule. The importance of the observable increase in the production of PGE₂ by camel chondrocytes in response to cytokine exposure is unclear but suggests that there may be a similar response in vivo.

The key finding in this study was that the mixture of ASU + GLU + CS significantly reduced the cytokine-induced production of PGE₂ by camel chondrocytes. This is consistent with results from our earlier in vitro studies with equine, canine, feline, and bovine chondrocytes as well as cultures of human monocyte-macrophage-like cells. Recent studies in which investigators used cultured feline cells also confirmed the anti-inflammatory activity of ASU + GLU + CS, compared with that for the NSAID meloxicam. In human and equine cell cultures, the mixture of ASU + GLU + CS was more potent than was ASU alone or the combination of glucosamine and CS. The human and equine cultures were used to determine the inhibitory potency of ASU + GLU + CS, ASU alone, or the combination of glucosamine and CS on cytokine, inducible nitric oxide, and cyclooxygenase-2 expression and PGE₂ production. The cytokine-induced production of PGE₂ was inhibited by the mixture of ASU + GLU + CS, which indicated the responsiveness of camel chondrocytes was similar to that of other species.

In the study reported here, the mixture of PPS + NG also exerted inhibitory effects on PGE₂ production by cytokine-stimulated camel chondrocytes. This finding was not unexpected because each of these agents reportedly has both anti-inflammatory and anabolic effects.

Figure 5—Photomicrographs of cultures of camel chondrocytes immunostained by use of monospecific antibodies to detect NF-κB translocation. Immunostaining (arrows) of the cytoplasm is evident in control chondrocytes (A), and cytokine activation—Nonstimulated chondrocytes had strong immunostaining from the cytoplasm to the nucleus (B). Treatment with ASU + GLU + CS (C) or PPS + NG (D) before activation via incubation with IL-1β and TNF-α inhibited NF-κB translocation, as indicated by immunostaining of predominantly the cytoplasm. Concentrations of ASU, glucosamine, and CS were 8.3, 11, and 20 µg/mL, respectively; concentrations of IL-1β and TNF-α were 10 and 1 ng/mL, respectively; and concentrations of PPS and NG varied from 25 to 125 µg/mL and 40 to 200 µg/mL, respectively. Bar = 20 µm.

Figure 6—Mean ± SD percentage of cultured camel chondrocytes immunostained for NF-κB. Cultures were incubated in medium alone (control medium) or with IL-1β and TNF-α (with or without ASU + GLU + CS or PPS + NG). Cultures were activated via incubation with IL-1β and TNF-α. See Figure 5 for key.
activity. The agent PPS belongs to the class of polysulfated glycosaminoglycans, which have been found to promote chondrocyte anabolic activity. Polysulfated glycosaminoglycans are cited in the International League of Associations for Rheumatology guidelines as disease-modifying osteoarthritic drugs in humans. These classes of agents can prevent, retard, or reverse lesions in cartilage. The biosynthetic activity of PPS is supported by results of in vitro experiments, which have revealed the ability of PPS to stimulate synthesis of ECM components (including proteoglycans, hyaluronic acid, and collagen) in several species, including horses. The agent NG can reduce cartilage degradation and also suppresses synovitis in rabbits, thereby suggesting anti-inflammatory activity.

Interestingly, PGE\textsubscript{2} synthesis was more profoundly inhibited by the combination of the ASU + GLU + CS and PPS + NG mixtures than when the 2 mixtures were used separately. This suggested that superior anti-inflammatory activity may be achieved with the combination of the 2 mixtures. The notion of combining 2 or more agents with similar or complementary biological activities to improve clinical outcome has been explored in other studies. In some cases, this strategy has yielded greater efficacy in animals while minimizing adverse effects, such as life-threatening gastrointestinal complications. The responsiveness of chondrocytes to the combination of the 2 mixtures reinforced their potential use as a treatment option for the management of joint inflammation in camels. These 2 preparations are currently being investigated for use in horses.

The cytokine-induced production of PGE\textsubscript{2} in camel chondrocytes was associated with activation of NF-\kappaB (a response characterized by the translocation of NF-\kappaB from the cytoplasm to the nucleus). This observation suggested that camel chondrocytes responded to stimulation by the proinflammatory cytokines IL-1\beta and TNF-\alpha through the NF-\kappaB signaling pathway. Activation of the NF-\kappaB pathway by cytokines has been reported for rabbit and human chondrocytes. Analysis of our data indicated that NF-\kappaB was a regulator of several proinflammatory cytokines in camel chondrocytes, which is a biological mechanism shared with chondrocytes of other species. Diminishing the translocation of NF-\kappaB in camel chondrocytes has long been considered a promising target for pharmacological agents intended for the treatment of various chronic inflammatory conditions. Our results indicated that inhibition of NF-\kappaB translocation can be achieved by either mixture tested in the study.

Inhibition of NF-\kappaB by products used for the treatment of osteoarthritis has been reported. Glucosamine inhibits IL-1\beta-induced NF-\kappaB activation in osteoarthritic chondrocytes. Similar inhibition of cytokine-induced NF-\kappaB activation has been observed after treatment of cells with CS or ASU. The role of NF-\kappaB as the key transcriptional regulator of many proinflammatory genes, including genes for cytokines, chemokines, and cyclooxygenase-2, which controls the production of PGE\textsubscript{2}, has been recognized. In addition, NF-\kappaB regulates other cellular functions, including proliferation, differentiation, and synthesis of ECM components. Because of its multiple functions, it has been proposed that NF-\kappaB is pivotal in the pathogenesis of osteoarthritic and synovitis in the knee joint of humans. The in vitro study reported here revealed that the ASU + GLU + CS mixture suppressed NF-\kappaB activation, which provided further support that this mixture modulates a critical site of control in the inflammatory cascade. The inhibition of NF-\kappaB translocation by PPS + NG could also be attributed to similar pathways affected by ASU + GLU + CS.

Results from this study revealed that cultured camel chondrocytes can be used in a disease-relevant cell-based assay system to test drugs and nonpharmacological products intended for use in the treatment of osteoarthritis in camels. The technique described here may also be useful in determining the mechanisms of action of agents used for the treatment of osteoarthritis in camels. Similar to other described cell culture techniques, the effective concentrations observed in vitro may provide the basis for estimating dose ranges that can be used in vivo.

a. Sigma-Aldrich, St Louis, Mo.
b. Gibco-Invitrogen, Frederick, Md.
c. BD Falcon, Baltimore, Md.
d. American Type Culture Collection, Manassas, Va.
e. Gemini Bio-Products, Woodland, Calif.
g. Southern Biotechnology Association, Birmingham, Ala.
h. EMD Biosciences Inc, La Jolla, Calif.
i. Santa Cruz Biotechnology, Santa Cruz, Calif.
j. US Biological, Swampscott, Mass.
k. Nikon Eclipse TE200, Nikon Instruments, Melville, NY.
l. NMX 1000, Nutramax Laboratories Inc, Edgewood, Md.
m. FCHG49, Nutramax Laboratories Inc, Edgewood, Md.
n. TR1H22, Nutramax Laboratories Inc, Edgewood, Md.
o. AUPEN 3000, Nutramax Laboratories Inc, Edgewood, Md.
p. R&D Systems, Minneapolis, Minn.
q. SpectraMax M5 microplate reader, Molecular Devices, Sunnyvale, Calif.
r. LabChem Inc, Pittsburgh, Pa.
s. Alexa Fluor-488, Gibco-Invitrogen, Frederick, Md.
t. Nikon Spot camera, Nikon Instruments, Melville, NY.
w. NIH ImageJ, National Institutes of Health, Bethesda, Md.
x. SigmaStat 3.3 for Windows, version 3.11, Systat Software Inc, Chicago, Ill.
y. Demko JL, Phan PV, Kramer EA, et al. Inhibition of prostaglan-
din E-2 production in chondrocyte microcarrier spinner cul-
tures by the combination of avocado soybean unsaponifi-
aa. Au RY, Au AY, Rashmir-Raven AM, et al. The combination of av-
ocado soybean unsaponifiables, glucosamine, and chondroitin sulfate inhibits pro-inflammatory gene expression in chondro-
ab. Grzanna MW, Heinicke LF, Au AY, et al. Down-regulation of prostaglandin E2 production in cytokine activated feline chondrocytes by the combination of avocado/soybean unsa-
bb. Au RY, Au AY, Rashmir-Raven AM, et al. Inhibition of pro-in-
fammatory gene expression in chondrocytes, monocytes, and fibroblasts by the combination of avocado soybean unsaponi-
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