How to Interpret Serum Amyloid A Concentrations

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1. Introduction
The role of the clinical veterinarian encompasses many features of medical practice, but in many cases starts with a simple dichotomization: is this animal normal or abnormal? This can include the question, “is it lame or sound,” but often also includes, “is it sick or well?” A thorough history and physical examination will often reveal how to stratify the patient, but in cases of subtle disease especially for horses in high level competition, mild and early signs of infection and inflammation may be occult yet significant. A reliable test for infection or inflammation, therefore, can have an extremely valuable place in the clinician’s armamentarium. Good tests allow for some degree of quantification both to allow the practitioner to assess the severity of the process and also to follow and document its response to therapy and track its resolution over time. The earliest of these tools was rectal temperature, in which fever signified a secondary indicator of increased cytokines such as tumor necrosis factor (TNF)-α and interleukin (II)-1. However, over the past century, blood analysis has allowed us to quantify multiple inflammatory markers including the acute phase proteins such as fibrinogen, haptoglobin, α1-acid glycoprotein, C-reactive protein (mainly in humans), serum amyloid A (SAA), and many others, as well as secondary indicators such as white blood cell count and serum iron levels. Of these, fibrinogen has probably been the most heavily relied on for horses. It can be easily and inexpensively measured, but may be confounded by in vitro preanalytical microclot formation. However, its concentration only slowly increases in the 24 hours after induction of inflammation and often does not peak until 48 hours. In addition, there is often only a small increase (often only a 1–2-fold difference) from baseline, and thus mild inflammation cannot reliably be distinguished from normal values. Nonetheless, any method that detects inflammation in the horse probably must outperform fibrinogen in one or more of these factors: accuracy, ease of interpretation, cost, and ease of use.

SAA is the major acute-phase protein of the horse (and most other mammals), and is produced predominantly by the liver as a systemic manifestation of the body’s response to inflammation. It exists in equine plasma as one of three isoforms of apolipoprotein and is complexed to high-density lipoprotein in circulating blood. First investigated in horses in the 1980s, its clinical use as a marker of inflammation is probably eclipsed by fibrinogen as a function of assay availability rather than diagnostic inferiority. The advantages of SAA over fibrinogen include that it has both low/undetectable constitutive expression in normal animals but reaches levels of 100-1000-fold baseline values in clinical disease
states. In addition, its rapid increase in concentration over 6–12 hours combined with a 30–120-minute half-life means that serum values track disease severity closely, and subsequent relapse or secondary infections result in similar response to primary infections. SAA is stable both at room temperature and refrigerated, can be measured using a relatively inexpensive stallside test or a variety of laboratory-based assays, can be performed using plasma as well as serum, and may be assessed using noninvasive samples such as saliva. Although there is some difference in precision and accuracy between assays, most available tests seem to be accurate enough within clinically relevant ranges to be acceptable to the practitioner.

2. Materials and Methods

A review of the literature reveals many publications that evaluate the use of SAA as a tool for distinguishing healthy horses from those with local or systemic inflammation, and as a diagnostic and monitoring tool for specific conditions. To maximize utility of this compilation of clinical equine veterinary publications, they are presented by body system or disease process, and the review focuses on the most clinically relevant studies. The astute reader will notice that these references refer to SAA in mg/L, μg/mL, and ng/mL; the first two of these units are equivalent and the third represents one thousandth the concentration of the first. The authors’ original units are maintained throughout.

3. Results

SAA to Determine Infectious/Inflammation Versus Normal

Horses that are “not quite right” or performing poorly are often diagnostic challenges, and identifying mild inflammation and distinguishing it from noninflammatory differential diagnoses before its clinical signs declare themselves can stymie even the most meticulous clinician. A recent large study evaluated the SAA concentrations of hospitalized horses that had either local inflammation (gastric ulceration, abscesses, Streptococcus equi subsp equi infection), systemic inflammation (disease accompanied by fever, tachycardia, leukopenia/leukocytosis) or were otherwise healthy or had noninflammatory conditions. Patients with systemic inflammation had significantly higher SAA (mean, 1583 mg/L; range, 688–4000 mg/L) than horses with local or no inflammation, which had mean SAA concentrations of 343 mg/L (range, 37–1609 mg/L) and 5.6 mg/L (range, 1.8–14.5 mg/L) respectively. This discrimination was more distinct than that of fibrinogen, in which the mean values of the three groups were 224, 181, and 128 mg/dL, respectively. Using receiver operator curve analysis, SAA had the highest accuracy for diagnosing inflammation (Fig. 1), but predictive modeling failed to generate useful algorithms. A similar study dichotomized horses into “clinically normal” and “clinically abnormal,” the latter of which included conditions as diverse as pneumonia, cholangiohepatitis, Streptococcus equi subsp equi infection, meningitis, enterocolitis, various forms of colic and neoplasia, and orthopedic infections. The clinically normal horses had a mean SAA of 6.8 mg/L (range, 0.1–26.6 mg/L), whereas the clinically abnormal horses had a mean SAA of 71.7 mg/L with a range of 0.1–3,800. In the same cases, mean fibrinogen values (ranges) were 349 mg/dL (100–800 mg/dL) and 514 mg/dL (100–1200 mg/dL), respectively. For discrimination of clinically normal horses from clinically abnormal horses, SAA had sensitivity of 53% and specificity of 94% (diagnostic accuracy, 75%), whereas using white blood cell count, and plasma fibrinogen concentration and mean albumin:globulin ratio, accuracy ranged from 59 to 62%. The authors also showed data from six cases comparing the resolution of inflammatory markers over time (Fig. 2) and concluded that “SAA concentration can provide valuable information regarding the clinical state of horses and may be more useful for patient monitoring and as a prognostic indicator than are traditional markers of inflammation.”

Foals have been shown to have similar baseline values of SAA compared with adults, the kinetics of its rise and resolution seems grossly similar, and SAA is higher in animals with bacterial infections than in those with nonbacterial or uncertain diagnoses. In the largest study looking at SAA in foals, 226 healthy Thoroughbred neonates had me-
dian SAA concentrations of 0.9, 4.5, and 2.5 mg/L on
days 1, 2 and 3 of life, with the values on day 2 being
significantly higher than baseline. In 136 foals
with clinical disease, median SAA concentrations of
cases with focal infections such as omphalitis were
195 mg/L and those with septicemia higher still at
280 mg/L. Foals with noninflammatory abnormalities
such as failure of passive transfer and noninfectious
disease had low SAA concentrations at 5.1
and 3.1 mg/L, respectively. These data suggest
that SAA can be used in foals even at young ages as
an indicator of infectious or inflammatory processes,
especially given that its quicker rise allows abnor-
malities to be identified in the first few days of life.
Increased fibrinogen levels in neonatal foals often
indicate intrauterine inflammation; whether SAA
rises during prepartum exposure to infectious or
inflammatory agents is not known.

SAA and Gastrointestinal Disease
The main applications for the use of SAA in colic
would be to assist in the ability to distinguish sur-
gical vs nonsurgical disease, to identify infectious
complications, and to gauge prognosis and response
to therapy. A study assessing SAA in colic cases
admitted to two university teaching hospitals found
that concentrations of SAA were significantly higher
at admission in horses with colic attributable inflam-
matory causes (e.g., enteritis, colitis, peritonitis,
or abdominal abscesses). This was most useful in
separating enteritis cases (median, 65.5 μg/mL;
interquartile range, 3–500 μg/mL) from strangulating
obstructions (median, 4.8 μg/mL; interquartile
range, 0.3–58.6 μg/mL). A significant difference
was also seen in the SAA value between horses that
survived the colic episode (median, 1.4 μg/mL) and
nonsurvivors (median, 10.8 μg/mL). Looking at
SAA levels in the peritoneal fluid of horses with
abdominal pain, cases with various etiologies of colic
had a mean SAA concentration of 249 mg/L in serum
and 97 mg/L in peritoneal fluid, compared with less
than 1 mg/L in both samples obtained from a
healthy control population. Furthermore, SAA
was elevated in horses with equine grass sickness
(median, 50 mg/mL) compared with surgical lesions
and noninflammatory colics (median, ~0 mg/
ml). A small experimental study found that inoc-
ulation with equine coronavirus resulted in SAA
levels that mirrored clinical disease; although all
three challenged horses shed large quantities of vi-
rus, only those that showed clinical signs of diar-
rhea, fever, and anorexia had elevated SAA, which
peaked at 200–400 μg/mL. Interpreting these
data is tricky given that there is not clear consensus
on the value of SAA in colics. However, based on
these studies and clinical experience, SAA does not
offer clear guidance in making the decision if a colic
is surgical, nor should it be relied upon for prognos-
ticating survivability when euthanasia is being con-
sidered. However, colic cases that are admitted
with SAA well above the reference range (> 20
mg/L) should have inflammatory gastrointestinal les-
sions such as enteritis and colitis higher on the
differential diagnosis, and SAA values in the hun-
dreds should prompt a search for an infectious
etiology.

SAA and Respiratory Disease
SAA has been investigated as a diagnostic tool for
both noninfectious inflammatory airway disease and
viral and bacterial infectious diseases. Horses with
severe bacterial pneumonia often present with SAA
values well into the thousands, and even road
transportation, the primary risk factor for pneu-
nia in horses, increases SAA anywhere from \( \sim \) 30 to 500 \( \mu \)g/mL for 24 to 48 hours after shipping healthy Thoroughbreds 1200 km by road over 26 hours. This increase was abrogated by administration of antimicrobials,\textsuperscript{24} and shorter-distance shipping (4 h) did not have an effect on SAA concentrations.\textsuperscript{25} In equine influenza, serum SAA increases during the first 48 h of clinical signs and returns to baseline values in 11 to 22 days barring secondary infections, with maximum values of \( \sim \) 450 mg/L during the acute phase.\textsuperscript{26} In ponies experimentally infected with EHV-1, serum levels peaked at 100 to almost 1000 mg/L in the week after inoculation.\textsuperscript{5}

Various methods have been evaluated to screen foals for sub-clinical *Rhodococcus equi* pneumonia including sonographic examination, hematology, and physical examination, but each have been found to be imperfect with regard to either diagnostic accuracy or cost effectiveness. Acute phase proteins, especially fibrinogen, have been used as moderately effective screening tools for foals on endemic farms. It would seem that with its greater sensitivity and more labile kinetics, SAA would be a better biomarker for this disease that often exhibits an extended preclinical latency where detection is often difficult. Two recent studies have investigated SAA as a possible predictor of *R. equi* pneumonia in at-risk populations. The first of these studies used a large, well-selected population of affected foals and age-matched controls from endemic farms. No predictive value was found in SAA levels in 212 foals 7 to 14 days and 196 foals 21 to 28 days of age, nor at the onset of clinical signs of pneumonia.\textsuperscript{27} The authors conclude that “monitoring concentration of SAA is not useful as a screening test for early detection of *R. equi*.”\textsuperscript{27} A subsequent smaller study also assessing weekly screening with SAA to identify pre-clinical *R. equi* infections found similar results. SAA concentrations did not show an association with the development of sonographic evidence of lung abscessation, and of six foals on an endemic farm diagnosed with *R. equi* pneumonia, only two demonstrated elevated SAA concentrations and this was around the time that the disease became clinically evident.\textsuperscript{28} These results are surprising given that *R. equi* generally results in a robust increase in fibrinogen and leukocyte count, which for other inflammatory diseases tends to be less sensitive than SAA. In conclusion, one must wonder why SAA is not a better aid in early identification of *R. equi* pneumonia; no completely satisfactory explanation is apparent for these results.

Recurrent airways obstruction (“heaves”) is a disease of the small airways characterized by neutrophilic exudate within the alveoli and bronchi. Although it is not associated with explicit signs of inflammation such as fever or peripheral neutrophilia, SAA has the potential to be more sensitive for subtle alterations. A prospective, observational study used six healthy and six heaves-affected horses challenged with hay and straw to examine a variety of acute-phase proteins.\textsuperscript{29} Although haptoglobin concentrations were higher in the heaves horses both before and during an exacerbation, the SAA did not reliably increase, although there was a small but significant difference between heaves-affected horses and controls on day 7 of exacerbation (15.75 vs 3.22 \( \mu \)g/mL, respectively).\textsuperscript{29} Nonetheless, probably the main use of SAA in horses with recurrent airways obstruction is distinguishing them from pneumonia cases, in which the SAA is likely to be much higher.

### SAA and Surgery

However much attention is paid to careful tissue handling and correct technique, surgery is an inflammatory stimulus and this fact is revealed by elevations in SAA after even minor, uncomplicated procedures.\textsuperscript{4,5,30,31} Therefore, its more useful application is probably identifying postoperative infections both earlier and with more accuracy than other methods. A study looking at standing castrations, for example, found that all horses had elevations of SAA to the 400–600-mg/L ranges at day 3 postoperatively, but those that went on to develop infections (as evidenced by fever, swelling, serohemorrhagic or purulent discharge) still had SAA values in this range at the eighth day whereas horses recovering without complication were in the \( \sim \) 200-mg/L range by this point. The increased SAA values associated with infection were not reliably reflected by increases in rectal temperature, leukocyte count, or fibrinogen, suggesting that SAA was a superior marker for infection.\textsuperscript{31}A subsequent study found that perioperative treatment with penicillin reduced the SAA in horses undergoing castration from a mean of 708 to 543 mg/L at day 3 postcastration, and from 515 to 125 mg/L on day 8,\textsuperscript{32} supporting the idea that even mild infections result in appreciable differences in SAA concentration. Looking beyond orchiectomy, the effect of minor surgical procedures on SAA shows that levels of 100 to 400 mg/L that peak at approximately day 3 after surgery can be expected in cases uncomplicated by infection. These include tibiotarsal arthroscopy and osteochondrosis fragment removal, laryngoplasty, and ventriculectomy (peaked at 50–150 mg/L at d 2; normal by d 7)\textsuperscript{30} carotid exteriorization and flexor tendon division (peaked at 100–400 mg/L at d 2; normal by 7–14 d)\textsuperscript{3} and a variety of elective procedures including minor airway and orthopedic surgeries (peaked at 16.4 \( \mu \)g/mL at 24 h, 15.5 on d 2).\textsuperscript{16} SAA was also significantly lower in elective (defined as noninfamed) vs nonelective (pre-existing inflammatory foci) cases\textsuperscript{16} as well as being able to delineate differing levels of surgical trauma based on the invasiveness of the procedure.\textsuperscript{30} In several of these studies\textsuperscript{6,30} SAA response was found to be a more sensitive indicator of inflammation than a variety of other acute-phase protein or leukocyte responses, and decreased more quickly in response to resolution than fibrinogen (Fig. 3). This is particularly useful to the practitioner who must decide whether hematologic ev-
idence of inflammation is simply a holdover from the effects of surgery or indicative of postoperative infection that requires further diagnostic evaluation or treatment.

SAA and Joint/Synovial Disease

SAA seems to be a sensitive marker of septic arthritis and tenosynovitis in adult horses. Healthy control horses have serum and synovial concentrations of SAA that are generally less than 1 mg/L. Repeated arthrocentesis (which increases cell count and total protein) and intra-articular amikacin injection do not affect SAA, and this may be one of its important uses in these cases given that the effects or repeated sampling can confound assessment of treatment efficacy and resolution.33,34 Although much of the SAA found within synovial fluid may be an ultrafiltrate from plasma, a joint-specific isoform of SAA is also produced by synoviocytes.35 As in other diseases, bacterial infection of joints and other synovial structures seems to be the most potent stimulant of SAA production. SAA concentrations in horses with bacterially contaminated (but not necessarily septic) synovial structures ranged from <1 to 402 mg/L (serum) and 94.5 mg/L (synovial fluid), whereas those with confirmed septic arthritis or tenovaginitis had varied SAA values that were possibly confounded by prior treatment; the highest value in these horses was a serum SAA of ~1700 mg/L and a synovial fluid SAA of ~1100 mg/L. In horses with noninfectious arthropathies such as osteoarthritis, SAA values were not different from control horses.33 In experimentally induced chemical arthritis (amphotericin B, midcarpal joint), serum SAA peaked at day 2 with values of ~50–300 mg/L and returned to normal between days 7 and 14.36 Although septic arthritis is rarely a diagnostic challenge, SAA may provide the clinician with supporting information regarding degree of inflammation and allow monitoring of resolution of time in conjunction with traditionally measured variables such as synovial fluid analysis.

SAA and Laminitis/Endocrinopathy

The question of how SAA changes in laminitis is complicated by the myriad inflammatory as well as noninflammatory etiologies of laminitis, as well as its varied chronicity and severity. A review of the literature in this area does not show a consistent story. For example, although SAA mRNA is increased in laminic hoof tissue samples,37 SAA levels in previously laminic ponies currently in remission showed no elevation from baseline.38 In obesity (which is associated with metabolic diseases and laminitis), increases in SAA were correlated with higher body condition score and higher plasma insulin.39 However, all of the horses had SAA levels that would fall within the reference range of the clinical assay, with highest values obtained topping out at 3845 ng/mL, or 3.8 mg/L. However, even at these low values, the authors concluded that “SAA concentration is a better marker of obesity-associated inflammation and laminitis [and] ... is possible that SAA is a component of laminitis pathophysiology.”39

SAA and Exercise

In Standardbred trotters and Arabian racehorses, SAA levels were unchanged in response to racing.40,41 However, significant findings have been identified looking at the acute phase response of horses undergoing long-distance endurance riding competitions and training. Although horses evaluated before an endurance ride all had SAA values within normal limits, SAA was lower in horses that successfully finished a 120–160-km competition compared with those that were eliminated based on intra- and postrace lameness and metabolic examinations (411.7 ng/mL in finishers vs 5809.5 ng/mL in eliminated horses; N.B. these units are equivalent to 0.4 and 5.8 mg/L). This study found that “serum SAA level was the only laboratory parameter that indicated most (66.6%) of the eliminated horses before entering the competition.”42 After long-distance (> 100 km) but not shorter (30–60 km) rides, a 10-fold or greater increase in SAA to ~13,000 ng/mL (13 mg/L) was found,42 although other acute phase proteins such as C-reactive protein and haptoglobin remained unchanged.43 Looking beyond the massive efforts of competitions at this level, there were no changes in SAA concentrations after race and endurance training sessions in experienced horses. Interestingly, SAA values also increased during training levels for inexperienced, early-career endurance horses (from ~1 to ~3.5 mg/L), but not in experienced competitors.41 This suggests that using SAA after training sessions may be a method of determining preparedness for advanced competition, but this has not yet been explicitly evaluated.
SAA and Reproductive Disease
There is conflicting evidence on SAA in the periparturient period of healthy mares. One study found that SAA levels were low in the 8 weeks prepartum although increased slightly in some mares in the week preceding foaling, whereas other studies have found no change in SAA associated prior to normal parturition. At 12 and 36 hours postpartum, mean SAA increases to 62 mg/L (range, 0.7–305 mg/L) and 189 mg/L (range, 0–1615 mg/L) and returns to basal concentrations by 60 hours. In mares with experimentally induced placentitis, SAA values peaked between 274 and 4385 mg/L within 2 to 6 days after intracervical inoculation with Streptococcus equi subspecies zooepidemicus; mares generally aborted within 2 to 6 days of the increase in SAA. Abortion was more likely in mares with high SAA compared with mares where the SAA remained within the reference range, and values increased steadily until abortion, when they then decreased rapidly. In comparison, fibrinogen and white blood cell count were not found to be useful markers of placentitis.

SAA and Parasites
In a study using horses experimentally infected with both small and large strongyles, acute-phase proteins were monitored over 161 to 164 days. Although haptoglobin, iron, and albumin/globulin ratios were associated with strongyle burden, SAA was not and remained low throughout the study. In addition, no significant change in SAA was seen after anthelmintic treatment in a group of heavily parasitized horses. This provides the practitioner with useful information as larval cyathostomiasis is a diagnosis difficult to make antemortem, and low SAA may steer the differential diagnosis away from inflammatory causes of colopathy.

SAA and Vaccination
After vaccination using two different influenza and tetanus toxoid products, horses showed variable acute-phase responses with six of 10 developing SAA concentrations greater than ~5 mg/L which peaked at 48 hours after vaccination. Maximum values ranged from ~30 to 175 mg/L, and increased white blood cell counts and fibrinogen concentrations, and decreased serum iron were also noted. By 96 hours, SAA levels were returning to normal but had not quite reached baseline values.

4. Discussion
SAA is a sensitive predictor of early inflammation, and due to its rapid onset and short half-life, tracks the course of disease closely. In most studies it outperforms the common variables of both fibrinogen and white blood cell count, and also often the other measured acute-phase reactors of haptoglobin, C-reactive protein, and serum iron. In a general sense, SAA can be used in most situations to obtain early identification of an inflammatory process, to assess the effectiveness of a chosen antimicrobial or other treatment, to monitor the rate of improvement, and to mark resolution of disease. SAA should be considered as an adjunct to diagnosis of a wide variety of inflammatory conditions in the horse, and may well replace fibrinogen in coming years as the main acute phase protein monitored in clinical medicine. However, it is not useful to diagnose specific diseases, and should not replace careful physical examination and identification of the etiologic causes of the inflammatory response. Table 1 shows a compilation of approximations of SAA values for common scenarios encountered in both primary care and referral equine practice.

The main disadvantages of SAA at this time are a lack of complete standardization of assay techniques, which imposes on the practitioner the need to commit to the same assay throughout evaluation of a particular case; values obtained from different assays may not be completely interchangeable. Although there are differences in results and precision between laboratory-based and in-clinic or stall-side tests, studies comparing the different forms of assay have found that the results given by each are clinically comparable. A newer stall-side assay can be read visually for semiquantitative measurement or using a handheld reader for a quantitative results. No peer-reviewed data have been published demonstrating the accuracy of this kit.

Although SAA has many advantageous characteristics, it is still not a diagnostic panacea. It seems to have no validity in assessing foals for R. equi pneumonia, although it often increases to extremely high concentrations in pleuropneumonia in adults and can be very valuable in assessing response to treatment in these cases. SAA does not reliably distinguish surgical from nonsurgical colic cases, although it is probably superior to fibrinogen in identifying postoperative infections because it follows a standard increase-and-decrease pattern of peaking at day 2 postoperatively and then returning to normal after approximately 1 week. Any deviations from a steady decrease after the first 2–3 days after surgery might prompt a search for infectious complications.

Although published reference ranges of SAA vary somewhat, the vast majority of normal, healthy horses tested in all these studies had SAA concentrations of 0 mg/L, or at the most, low single digits; control horses that had SAA levels greater than this may have been suffering from subclinical conditions that were not evident on physical examination. Most instances of infectious or inflammatory disease show SAA levels greater than 50 mg/L, which leaves a gray area between where interpretation may be difficult. SAA is still a relatively new technique compared with other measured indices of inflammation, so less is known about how to interpret these values than older, more familiar parameters. Studies correlating SAA concentrations to a large number of diseases have been published every year over the last decade, and currently the body of liter-
ature is expanding rapidly in ways that assist veterinarians in the application of this parameter. Overall, practitioners should feel comfortable using SAA in lieu of fibrinogen for most cases in which infectious or inflammatory disease is suspected, although ordering both for a period of time is probably wise until the clinician develops the experience necessary to interpret the wide range of values seen with this marker.

Acknowledgments

Declaration of Ethics

The Author declares that she has adhered to the Principles of Veterinary Medical Ethics of the AVMA.

Conflict of Interest

The Author declares no conflicts of interest.

References and Footnote


### Table 1. Approximate Peak SAA Values Seen in Common Equine Diseases With Their Time to Peak and Resolution

<table>
<thead>
<tr>
<th>Disease State</th>
<th>Approximate Peak Value, mg/L</th>
<th>Time to Peak; Time to Resolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAA in normal horses and foals</td>
<td>0–7 mg/L*</td>
<td>NA</td>
</tr>
<tr>
<td>SAA, inflammatory colic (enteritis, colitis, coronavirus)</td>
<td>50–500</td>
<td>Depends; decreases in tandem with disease resolution</td>
</tr>
<tr>
<td>SAA, surgical colic</td>
<td>5–50</td>
<td>NA</td>
</tr>
<tr>
<td>SAA, grass sickness</td>
<td>~50</td>
<td>NA</td>
</tr>
<tr>
<td>SAA, major bacterial infections (peritonitis, pleuropneumonia)</td>
<td>Up to 3000–5000 depending on severity</td>
<td>Depends; decreases in tandem with disease resolution</td>
</tr>
<tr>
<td>SAA, viral lung disease</td>
<td>500–1000</td>
<td>Peaks in first wk; 11–22 d</td>
</tr>
<tr>
<td>SAA, <em>R. equi</em> pneumonia</td>
<td>Variable; not reliable indicator</td>
<td>NA</td>
</tr>
<tr>
<td>SAA, recurrent airway obstruction (&quot;heaves&quot;)</td>
<td>≤15</td>
<td>Increased &gt; baseline only during acute exacerbation</td>
</tr>
<tr>
<td>SAA, castration</td>
<td>400–700</td>
<td>2–3 d; to ~200 by 8 d if no complications</td>
</tr>
<tr>
<td>SAA, elective surgery</td>
<td>50–400</td>
<td>2 d; 7–14 d</td>
</tr>
<tr>
<td>SAA, septic arthritis</td>
<td>Up to 1500 in serum, 1100 in synovial tissue</td>
<td>Not reported</td>
</tr>
<tr>
<td>SAA, chemical arthritis</td>
<td>50–300</td>
<td>48 h</td>
</tr>
<tr>
<td>SAA, laminitis</td>
<td>No change in chronic, metabolic etiologies</td>
<td>NA</td>
</tr>
<tr>
<td>SAA, post-race (STBs, Arabians)</td>
<td>No change</td>
<td>NA</td>
</tr>
<tr>
<td>SAA, long-distance endurance ride (&gt; 120 km)</td>
<td>~13</td>
<td>No data; no data</td>
</tr>
<tr>
<td>SAA, shorter-distance endurance ride (&gt; 40 km)</td>
<td>No change</td>
<td>NA</td>
</tr>
<tr>
<td>SAA, before normal parturition</td>
<td>No change</td>
<td>NA</td>
</tr>
<tr>
<td>SAA, after normal parturition</td>
<td>50–600 at 12–36 h postpartum</td>
<td>36 h; 60 h</td>
</tr>
<tr>
<td>SAA, placentitis</td>
<td>250–4500</td>
<td>Rises until abortion; rapidly drops after abortion</td>
</tr>
<tr>
<td>SAA, parasitism</td>
<td>No change</td>
<td>NA</td>
</tr>
<tr>
<td>SAA, after anthelminthic treatment</td>
<td>No change</td>
<td>2 d; probably between 4–7 d</td>
</tr>
<tr>
<td>SAA after vaccination</td>
<td>30–175</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: STB, Standardbred.
* Suggested reference ranges vary from <1.3 and <20 mg/L.

*StableLab SAA assay, StableLab, Ballinodre, Sligo, Ireland.