“EXTENSION OF PLATELET STORAGE:
FACT OR FANTASY”

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Introduction.

In 1986, the dating period for platelet concentrates was reduced from 7 days to 5 days because of an increase in the number of reported cases of post-transfusion sepsis since the 1984 extension of the storage period to 7 days.\(^1\) The incidence of bacterial overgrowth increases exponentially during platelet storage at 22°C, and it is estimated that 0.3% to 1.6% of platelet components are bacterially contaminated. The rate of transfusion-associated sepsis may be \(\leq 50,000\) transfusions with a fatality rate of 20% which may translate into 50 to 100 fatalities/year in the U.S.\(^2\)

There are several possible causes of bacterial contamination: 1) failure to adequately reduce the bacterial contamination of the donor’s skin at the time of the venapuncture; 2) inapparent donor bacteremia; and 3) contamination during collection and processing of platelets. Of these potential causes of bacterial contamination, the overwhelming majority of cases are due to inadequate skin preparation. The methods of correcting this problem are to: 1) improve skin preparation/disinfection; 2) remove the first aliquot of blood (this has been estimated to reduce the risk by 0.21% to 0.34%); 3) utilize a method of pre-transfusion detection of bacteria; or 4) decontaminate the platelets prior to transfusion. It is these latter two approaches that have led to the possibility of again extending the storage duration of platelets. However, if a platelet decontamination procedure is used to extend platelet storage, it must be documented that the decontamination procedure itself does not compromise the quality of long-term stored platelets.

Evaluation of Platelet Quality.

A multi-tiered approach is usually used to evaluate platelet quality starting with a panel of \textit{in vitro} measurements.\(^3\) Some investigators have been able to demonstrate a relationship between these assays and post-transfusion platelet viability measurements. However, even using those \textit{in vitro} tests that have shown some ability to predict post-transfusion platelet viability, the correlation is often not very good, and some of the \textit{in vitro} lesions are reversible following transfusion.\(^4,5\) Therefore, it is extremely important to ultimately document platelet quality with \textit{in vivo} measurements of platelet recovery, platelet survival, and hemostatic efficacy; these measurements will be the focus of this review. Specifically, this review data will concentrate on those \textit{in vivo} studies that have directly compared two different types of platelet products to determine if one platelet preparation and/or storage process is better than another.

\textit{In vivo} assessments are frequently performed using a two-step sequential process: 1) radiolabeled autologous platelet recovery and survival measurements in normal volunteers; and 2) transfusion experiments in thrombocytopenic patients. In thrombocytopenic patients, it is possible to assess both platelet viability by determining platelet increments and days to next transfusion, as well as platelet hemostasis by documenting the relationship between bleeding time and platelet count,\(^6\) determining hemorrhagic morbidity and mortality rates, and recording red cell transfusion requirements.

For reasons that have never been explained, there is a substantial amount of variability in the post-transfusion viability of platelets from different donors. Thus, the best experimental design to detect differences in platelet quality is to directly compare platelet product “A” to platelet product “B” using autologous platelets collected from the same donor. In normal volunteers, this is done by either simultaneously or sequentially preparing the two different autologous platelet products. For the simultaneous transfusion studies, one platelet product is labeled with \(^{51}\)Cr and
the other with $^{111}$In. In prior to transfusion so that concurrent measurements can be done. For the sequential studies, the same isotope can be used. In patients, it is impractical to use the same donors’ platelets for simultaneous or even for sequential measurements. However, it is important to perform the sequential transfusions within a relatively short time period as changes in the patient’s clinical condition or medications may influence platelet transfusion outcomes.

There is usually a good correlation between the platelet viability studies in normal volunteers and those in thrombocytopenic patients so that not every change in platelet processing or storage has to be evaluated by transfusion studies in thrombocytopenic patients. However, the introduction of new methods of preparing platelets for transfusion or making major changes in storage conditions should be assessed in thrombocytopenic patients to document both platelet viability and, as importantly, platelet hemostasis.

**Types Of Platelets Available For Transfusion.**

There are two basic approaches to harvesting platelets for transfusion: 1) they are prepared from donated units of whole blood; or 2) they are collected using apheresis techniques. Within these two approaches, different methods are available. In the U.S., platelets are prepared from whole blood by the so-called “platelet-rich plasma method” (PRP-PC) which involves a soft spin of the whole blood to separate the PRP from the red cells. After transfer of the PRP to a satellite bag, a hard centrifugation is performed to sediment the platelets, the majority of the supernatant plasma is transferred to another satellite bag, and the platelets are re-suspended in a small amount of residual plasma. In contrast, in many European countries, platelets are made by the so-called “buffy coat method” (BC-PC). In this method, the initial spin of the whole blood is at a high $g$ force which sediments the platelets into the white cell layer (buffy coat) on top of the red cells. The plasma is transferred to a satellite bag, and the buffy layer containing the platelets is transferred to another satellite bag. Several buffy coats are then pooled, and usually a platelet additive solution is added. During a second soft centrifugation procedure, the supernatant platelets are transferred to a satellite bag for storage in the additive solution.

For collection of platelets by apheresis, several devices are available which differ in how platelets are processed to obtain the final product. The Haemonetics machine collects platelets by an elutrimination procedure, the Cobe machine by dual-stage channels for separation, and the Fenwal machine uses an initial chamber to separate the red cells from the platelet-rich plasma and subsequent separation of platelets from plasma that involves continuous spinning of the platelets against the walls of the collection bag during harvesting.

If high $g$ forces are used during the final centrifugation of PRP to produce the platelet concentrate, the platelets may be damaged, and the injury may be potentiated during platelet storage. However, platelets are susceptible to a progressive decrease in platelet viability during storage even in the absence of a collection injury. It has been suggested, because the only hard spin of the platelets during the preparation of buffy coat platelets is against a red cell layer, that this technique may be less injurious to platelets during collection. Thus, the available data suggests that platelets are susceptible to both a collection injury and a separate storage injury. Since the processes used to prepare platelets from both whole blood and during apheresis collection may vary substantially, it may be important to document the viability of each of these types of platelets during extended storage. This may be particularly important if a substantial change...
has been introduced in platelet production such as a decontamination procedure or during storage such as the use of a platelet additive solution.

**In Vivo Evaluation Of Stored Platelets.**

Two major advances have made long-term platelet storage feasible; i.e., the development of platelet storage bags that allow adequate gas exchange across the walls of the container (O\textsubscript{2} ingress and CO\textsubscript{2} egress) and the formulation of platelet additive solutions that may be better able to meet the metabolic needs of platelets during storage than can be achieved with plasma.\(^{(17)}\)

As the normal platelet lifespan averages 8 to 10 days, if this lifespan is intrinsic to the cell, then long-term platelet storage may not even be possible. However, in a series of experiments, Holme and Heaton demonstrated that platelet aging is significantly less at the *in vitro* storage temperature of 22°C *versus* the *in vivo* temperature of 37°C; i.e., *in vitro* platelet aging occurred at a rate that was only 0.42 to 0.44 of that at 37°C.\(^{(18)}\) Their data suggest that a storage time of at least 7 if not 10 days may be possible. If other ways besides reduction in storage temperature can be found to decrease platelet metabolic requirements as 20°C is the minimum acceptable storage temperature,\(^{(19)}\) then storage intervals even longer than 7-10 days may be possible.

Most investigators have shown a progressive loss in platelet viability using the current conditions of platelet storage, and, with the current 5-day storage period, platelet viability is about 70% of fresh.\(^{(18)}\) The question then becomes what is an acceptable loss of platelet viability during extended storage? Furthermore, does platelet viability during storage necessarily equate with maintenance of platelet hemostatic efficacy or might there be situations where the two parameters become disparate?

Although there has been little recent data on the viability of platelets stored for 7 days or longer once the FDA reduced platelet storage to 5 days, there are some prior studies which are relevant to the issue of extended platelet storage. In addition, data comparing 5-day storage of the different platelet products will be presented that may be pertinent to how these products will perform at longer storage intervals.

**Platelet Storage In Plasma.**

**Viability Studies in Normal Volunteers.**

Data from six studies that performed paired autologous radiolabeled platelet recovery and survival measurements in normal volunteers to compare either the effects of storage time or type of product transfused on platelet viability are shown in Table 1. These studies demonstrated no significant differences between platelet products (PRP-PC, BC-PC, and apheresis platelets) when stored for 5 days.\(^{(20-24)}\) In addition, in the one reported study, there were no differences between 5-day and 7-day stored PRP-PC.\(^{(25)}\)

**Transfusion Studies In Thrombocytopenic Patients.**

**Comparison of Types of Platelet Products.**

Fifty-one patients were randomly assigned to receive all of their transfusions as PRP-PC (n=162), BC-PC (n=158), or Cobe apheresis platelets (n=117). Platelets were stored for 1 to 5 days, and the mean age of the platelets at transfusion was 3.0 ± 1.3, 3.0 ± 1.2, and 3.1 ± 1.2 days, respectively. Post-transfusion corrected count increments (CCI’s) were 12 ± 1.2, 11.1
± 1.3, and 9.2 ± 1.5 (S.E.) at 1 hour and 8.6 ± 1.7, 6.5 ± 1.8, and 6.2 ± 2.2 at 24 hours, respectively. None of these differences were statistically significant.(26)

Effect of Storage Time.

There is conflicting data on patients’ responses to fresh and stored platelets in thrombocytopenic patients. In three studies, no differences were observed between apheresis platelets stored for 1 day versus 4-5 days in thrombocytopenic patients.(27-29) In contrast, in six other studies when platelets were stored for similar time periods, increasing platelet storage time decreased post-transfusion CCI’s.(5,9,30-32) Two of these studies are of particular interest because of the findings and the experimental design used. Owens, et al.(5) simultaneously transfused radiolabeled PRP-PC from the same donor either fresh or after storage for 5 days to 12 thrombocytopenic patients. Platelet recoveries averaged 58% ± 10% versus 47% ± 10% (p<0.001), and survivals were 8.5 ± 0.8 days versus 6.5 ± 1.0 days (p<0.001) for 1-day versus 5-day stored platelets, respectively. Furthermore, platelet function - as measured by platelet retention in a glass bead column and by platelet aggregation measurements - showed poor post-transfusion function for the 5-day stored platelets that reversed to the values found for 1-day stored platelets by 24 hours after transfusion.

Norol, et al.(9) evaluated 141 patients given 2 transfusions of apheresis platelets from different donors. One transfusion was given within 8 hours of collection (fresh) and one after 2 days of storage (stored). Half of the patients received the fresh transfusion first, and the order was reversed in the other half. In 48 patients who were clinically stable at the time of transfusion, there was no difference between the platelet recoveries at 1-hour post-transfusion or in the number of days until their next transfusion. However, if they had an adverse clinical condition or were given specific drugs at the time of transfusion [i.e., infection (n=27), Amphotericin B (n=16), GVHD (n=18), palpable spleen (n=9), or veno-occlusive disease (n=4)], then both the 1-hour post-transfusion platelet recoveries and interval to next transfusion were statistically significantly less for the stored versus fresh platelet transfusions.

In the only study which assessed 7-day stored platelets, 16 patients received PRP-PC stored for 1 day, 3 days, or 7 days in that order over 1 to 2 weeks.(33) CCI’s averaged 20.1 ± 8.4, 12.2 ± 8.1, and 10.0 ± 7.2 at 1 hour post-transfusion, and 24 hour CCI’s averaged 10.8 ± 4.4, 7.5 ± 5.6, and 7.0 ± 5.5 for platelets of increasing storage age, respectively. Although there were no differences in patients’ responses to platelets stored for 3 days versus 7 days, the fresh platelets always gave statistically significantly higher responses than platelets stored for either time period.

Platelet Storage In Additive Solutions.

Several different types of platelet additive solutions (PAS) have been used for platelet storage.(34) These PAS are generally composed of different combinations and concentrations of glucose, acetate, citrate, and phosphate which have been found to have both separate and interactive effects on platelet metabolism during storage.

Viability Studies In Normal Volunteers.

In four different studies, paired radiolabeled autologous platelet storage studies were performed in normal volunteers (Table 2). The most interesting study(37) demonstrated progressive decreases in both platelet recoveries and survivals with increasing storage time whether the
platelets were stored in PAS or plasma, but viability was generally better maintained in PAS at storage intervals of ≥7 days.\(^{37}\) With increasing storage, the platelet survivals became not only shorter but also more curvilinear, suggesting that platelets were aging \textit{in vitro}. The T\(\frac{1}{2}\) of the lifespan of platelets stored in plasma was 7.2 days and in PAS 8.8 days, a marked improvement. Further improvement during 14 days of storage\(^{38}\) was achieved by adding platelet function inhibitors (PGE-1 and theophylline) and reducing the surface area of the storage bag.

These data clearly suggest that reasonable platelet viability can be maintained for platelets stored for at least 7 days, particularly if a PAS is used.\(^{37}\) Furthermore, additional increases in platelet viability may be achieved with other modifications to the storage conditions.\(^{38}\)

**Transfusion Studies In Thrombocytopenic Patients (Table 3).**

In two separate studies,\(^{35,39}\) a total of 54 thrombocytopenic patients received 148 transfusions of either fresh (stored up to 2 days) or stored (stored 3-5 days) pooled buffy coats in PAS-1 or apheresis platelets (Fenwal CS-3000) stored in plasma. Apparently, patients could be transfused with either type of platelets fresh or stored, perhaps depending on availability. Major conclusions were that there were no differences in post-transfusion CCI’s at either 1 or 24-hours following transfusion with either type of platelets stored for a comparable time period. However, fresh \textit{versus} stored BC-PC gave better CCI’s while there were no differences for the fresh \textit{versus} stored apheresis platelets.

BC-PC were stored for up to 5 days in either plasma or PAS-2 (T-SOL).\(^{40}\) Patients were randomly assigned to receive all their platelets stored in plasma or in PAS-2. The post-transfusion CCI’s were significantly better for BC-PC stored in plasma than PAS-2.

Using pooled BC-PC, 9 patients were transfused in random order with platelets stored in Plasmalyte and PAS-2.\(^{41}\) Results were the same for both products.

In another study, pools of BC-PC in 0.5 CPD solution were suspended in PAS-2, and the results of these transfusions were compared to pools of BC-PC in PAS-1 given to the same 20 thrombocytopenic patients.\(^{42}\) Patients received each type of platelets both fresh (0-2 days) and stored (3-5 days). The CCI results did not differ between the two types of platelets, but CCI’s were reduced at 24-hours post-transfusion, but not at 1-hour post-transfusion for both types of platelets when the results of the fresh and stored platelet transfusions were compared.

**Pathogen-Inactivated Platelets.**

Only one pathogen-inactivation procedure for platelets has been evaluated in normal volunteers and thrombocytopenic patients. This process involves adding a novel psoralen compound (S-59) to platelets collected in 35% plasma and 65% PAS-3 to allow adequate exposure of the platelets to UV-A light, the effects of which are markedly reduced by plasma.\(^{43}\) After UV-A exposure, intercalated psoralens form monadducts and interstrand crosslinks with the DNA and RNA of bacteria and viruses that prevents their replication. In paired experiments in normal volunteers, platelets were collected using Fenwal’s Amicus machine and half of the platelets were treated with UV-A and S-59, and the other half were stored in plasma. After 5 days of storage, platelet recoveries averaged 43% ± 9% \textit{versus} 50% ± 8% \((p<0.001)\), and survivals averaged 4.8 ± 1.3 days \textit{versus} 6.0 ± 1.2 days \((p<0.001)\) for treated \textit{versus} control platelets, respectively.\(^{44}\)
Two transfusion trials in thrombocytopenic patients using pathogen inactivated compared to control platelets have now been completed; i.e., a European study (euroSPRITE) using buffy coat platelets\(^{(45)}\) and a U.S. Trial (SPRINT) using Amicus collected apheresis platelets.\(^{(46)}\) In the euroSPRITE trial, post-transfusion platelet increments were significantly less at both 1 and 24-hours following transfusion, and also CCI’s at 24 hours post-transfusion for the treated compared to the control platelet transfusions (Table 4).\(^{(45)}\) In the SPRINT trial, all measurements (increments, CCI’s, and days to next transfusion) were significantly less for the treated compared to the control platelet transfusion. However, the hemostatic efficacy of the treated platelets was comparable to the control platelets.\(^{(46)}\) Both a loss in platelet viability as well as fewer platelets recovered for transfusions following treatment probably accounts for the differences in transfusion responses.

As with any major advance in medical therapy, there are often trade-offs. Certainly, the majority of the data available on the S-59 UV-A pathogen-inactivation process would suggest some loss of platelet viability and decrease in the number of platelets available for transfusion due to processing losses. This is reflected in decreased platelet recovery and survival measurements in normal volunteers as well as decreased post-transfusion platelet increments, CCI’s, and days to next transfusion in thrombocytopenic patients. As the platelet count needed to maintain hemostasis is very low, only 5,000-10,000 platelets/µl,\(^{(47)}\) a reduced platelet increment is not a concern for most patients because the majority of them will be receiving prophylactic platelet transfusions to prevent bleeding. However, for actively-bleeding patients or surgical patients where the actual post-transfusion platelet counts may be very important for hemostasis, larger numbers of treated platelets may be required. Overall, patients receiving treated transfusions required on average about 25% more platelet transfusions during their thrombocytopenic period compared to patients who received control platelet transfusions.

As previously discussed, one of the potential advantages of a pathogen-inactivation process is to extend platelet storage. However, it remains to be determined whether the S-59 UV-A pathogen-inactivation process will show further disparities between treated and control platelet transfusions with extended platelet storage. It is possible that the differences in platelet viability following the treatment process will be further magnified by extending the storage of these cells, limiting their use to a 5-day dating period. Well-designed transfusion studies with extended storage of pathogen-inactivated platelets are needed to determine the full potential of this process.
References:

1. FDA memo dated June 2, 1986 to all registered blood establishments entitled, “Reduction of the Maximum Platelet Storage Period to 5 Days in an Approved Container”. United States Food and Drug Administration, Washington, D.C.


### Table 1

**PAIRED RADIOLABELED AUTOLOGOUS PLATELET STORAGE STUDIES IN PLASMA IN NORMAL VOLUNTEERS**

<table>
<thead>
<tr>
<th>Author</th>
<th>PRP-PC Storage Time (Days)</th>
<th>BC-PC Storage Time (Days)</th>
<th>Apheresis Platelets Storage Time (Days)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>0</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Keegan, et al.</td>
<td>9</td>
<td>60±7%*</td>
<td>49±10%</td>
<td>6.8±1.2 d</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8.8±0.9 d*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mitchell, et al.</td>
<td>8</td>
<td>35±11%</td>
<td>6.9 d</td>
<td>(5.4–7.7)</td>
</tr>
<tr>
<td>Read, et al.</td>
<td>8</td>
<td>46±12%</td>
<td>5.6±1.0 d</td>
<td></td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Turner, et al.</td>
<td>10</td>
<td>37±5%</td>
<td>4.2±2.6 d**</td>
<td></td>
</tr>
<tr>
<td>Shanwell, et al.</td>
<td>8</td>
<td>58±12%</td>
<td>5.5±1.5 d</td>
<td></td>
</tr>
<tr>
<td>Simon, et al.</td>
<td>9</td>
<td>39±5%</td>
<td>42±7%</td>
<td>6.8±1.2 d</td>
</tr>
</tbody>
</table>

* Recovery data reported on first line (%) and survival data on second line (days).
** Estimated from data in a figure.

Data reported as average ±1 S.D. or average with a range.
PRP-PC-Platelets prepared from platelet-rich plasma; BC-PC-Platelets prepared from buffy coat platelets; and Apheresis Platelets-Machine used for preparation given in Comments section.
**Table 2**

### PAIRED RADIOLabeled AUTOLOGOUS PLATELET STORAGE STUDIES IN PLATELET ADDITIVE SOLUTION (PAS) VERSUS PLASMA

<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>Storage Time (Days)</th>
<th>PLASMA</th>
<th>Storage Time (Days)</th>
<th>PAS</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>1</td>
<td>5</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>Erickson, et al.</td>
<td>9</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>55±9%*</td>
<td>52±10%</td>
<td>7.8±2.0 d*</td>
<td>5.8±0.5 d</td>
</tr>
<tr>
<td>Holme, et al.</td>
<td>18</td>
<td></td>
<td>50±8%</td>
<td>55±9%</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>6.8±0.9 d</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Holme, et al.</td>
<td>10</td>
<td>55±10%*</td>
<td>7.9±1.0 d</td>
<td></td>
<td></td>
<td></td>
</tr>
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<td></td>
<td></td>
<td>41±11%</td>
<td>6.1±1.7 d</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>37±11%</td>
<td>4.5±1.6 d</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>23±9%</td>
<td>3.1±1.8 d</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>9±8%</td>
<td>2.1±1.0 d</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Holme, et al.</td>
<td>5</td>
<td>PRP-PC in PAS with added PGE-1 and theophylline in a small bag.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>23±9%</td>
<td>4.5±2.5 d</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>9±8%</td>
<td>0.80±9 d</td>
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</table>

Recovery data reported on first line (%) and survival data on second line (days).
Data reported as average ±1 S.D.
Type of platelets evaluated given in Comments section: PRP-PC-Platelets prepared from platelet-rich plasma; BC-PC-Platelets prepared from buffy coat platelets; and Machine used for apheresis platelet collection.
NS=No significant differences.
# Table 3

**RESULTS OF PLASMA VERSUS PAS STORED PLATELET TRANSFUSIONS IN THROMBOCYTOPENIC PATIENTS**

<table>
<thead>
<tr>
<th>Author</th>
<th>Storage Time (Days)</th>
<th>Patients (n)</th>
<th>Platelet Transfusions (n)</th>
<th>Platelets Transfused</th>
<th>POST-TRANSFUSION CCI</th>
<th>1 Hour</th>
<th>24-Hour</th>
</tr>
</thead>
<tbody>
<tr>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eriksson, <em>et al.</em>(35)</td>
<td>≤2</td>
<td>18 total</td>
<td>12</td>
<td>BC-PC (PAS-1)</td>
<td>20.9±11.8</td>
<td>13.5±8.3</td>
<td>17.4±15.5 p=0.06 p=0.04</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6</td>
<td>6 Apheresis (Plasma)</td>
<td></td>
<td>15.0±7.0</td>
<td>8.0±6.4</td>
<td>15.5±9.2 7.1±6.0</td>
</tr>
<tr>
<td></td>
<td>3-5</td>
<td>23</td>
<td>9 Apheresis (Plasma)</td>
<td></td>
<td>15.0±7.0</td>
<td>8.0±6.4</td>
<td>15.5±9.2 7.1±6.0</td>
</tr>
<tr>
<td>de Wildt-Eggen, <em>et al.</em>(40)</td>
<td>≤5</td>
<td>12</td>
<td>192 BC-PC (Plasma)</td>
<td></td>
<td>20.7±8.5 p&lt;0.001</td>
<td>11.5±8.0 p&lt;0.05</td>
<td>17.1±6.6 9.5±8.0</td>
</tr>
<tr>
<td>van Rhenen, <em>et al.</em>(41)</td>
<td>4.6±0.4</td>
<td>9</td>
<td>9 BC-PC (Plasmalyte)</td>
<td></td>
<td>22.4 (15.2-29.4)</td>
<td>11.3 (4.1-18.4)</td>
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<tr>
<td></td>
<td>4.1±0.6</td>
<td>9</td>
<td>9 BC-PC (PAS-2)</td>
<td></td>
<td>24.0 (16.9-31.2)</td>
<td>14.2 (7.1-21.3)</td>
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<tr>
<td>Hogman, <em>et al.</em>(42)</td>
<td>≤2</td>
<td>20</td>
<td>20 BC-PC (PAS-2)</td>
<td></td>
<td>16.9±2.1 p=0.005</td>
<td>13.7±2.7 p=0.04</td>
<td>17.8±2.6 15.0±3.2</td>
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<tr>
<td></td>
<td>≤2</td>
<td>20</td>
<td>20 BC-PC (PAS-1)</td>
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<td>13.5±2.2</td>
<td>7.4±1.8</td>
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<tr>
<td></td>
<td>3-5</td>
<td>20</td>
<td>20 BC-PC (PAS-2)</td>
<td></td>
<td>14.0±2.0</td>
<td>6.6±1.4</td>
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<tr>
<td></td>
<td>3-5</td>
<td>20</td>
<td>20 BC-PC (PAS-1)</td>
<td></td>
<td>13.5±2.2</td>
<td>7.4±1.8</td>
<td></td>
</tr>
</tbody>
</table>

All data reported as average ±1 S.D. or range, except for Hogman, *et al.*, reported as average ±1 S.E.
Table 4

RESULTS OF PATHOGEN INACTIVATION COMPARED TO CONTROL PLATELET TRANSFUSIONS IN THROMBOCYTOPENIC PATIENTS

<table>
<thead>
<tr>
<th></th>
<th>euroSPRITE TRIAL (Buffy Coat Platelets)</th>
<th>SPRINT TRIAL (Amicus Apheresis Platelets)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Treated ( (n=51) )</td>
<td>Control ( (n=51) )</td>
</tr>
<tr>
<td><strong>Platelet Increment:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Hour ( (x 10^3/\mu l) )</td>
<td>( 27.6 \pm 13.3 )</td>
<td>( 35.8 \pm 23.2 )</td>
</tr>
<tr>
<td>24 Hour ( (x 10^3/\mu l) )</td>
<td>( 16.0 \pm 9.8 )</td>
<td>( 24.7 \pm 17.4 )</td>
</tr>
<tr>
<td><strong>CCI:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Hour ( (x 10^3) )</td>
<td>( 13.1 \pm 5.3 )</td>
<td>( 14.8 \pm 6.2 )</td>
</tr>
<tr>
<td>24 Hours ( (x 10^3) )</td>
<td>( 7.3 \pm 5.4 )</td>
<td>( 10.6 \pm 7.1 )</td>
</tr>
<tr>
<td>Platelet Transfusion Interval (Days)</td>
<td>( 3.0 \pm 1.2 )</td>
<td>( 3.4 \pm 1.2 )</td>
</tr>
<tr>
<td>Platelet Transfusion Events</td>
<td>( 7.5 \pm 5.8 )</td>
<td>( 5.6 \pm 5.5 )</td>
</tr>
</tbody>
</table>