Human Plasma ATP Concentration

MARK W. GORMAN,1* ERIC O. FEIGL,1 and CHARLES W. BUFFINGTON2

Background: Human plasma ATP concentration is reported in many studies as roughly 1000 nmol/L. The present study tested the hypothesis that the measured plasma ATP concentration is lower if ATP release from formed blood elements is inhibited during blood sample processing. A second hypothesis was that pretreatment with aspirin to inhibit platelets would reduce the measured plasma concentration of ATP.

Methods: Blood was sampled from the antecubital vein in 20 healthy individuals 30 and 60 min after ingestion of aspirin (325 mg) or placebo. Aliquots of each blood sample were added to the usual EDTA/saline solution to inhibit ATP catabolism, or to a new stabilizing solution designed to both stop ATP catabolism and inhibit ATP release from blood elements. The stabilizing solution contained NaCl, EDTA, tricine buffer, KCl, nitrobenzylthioinosine, forskolin, and isobutylmethylxanthine. Plasma ATP was measured with the luciferin–luciferase assay with standard additions in each sample to determine ATP content. Hemoglobin concentration was used as an index of sample hemolysis, and the plasma ATP concentration was corrected for the hemolysis component.

Results: Aspirin pretreatment had no effect on plasma ATP concentrations. However, use of the stabilizing solution resulted in mean (SD) ATP concentrations 8-fold lower than the use of EDTA alone [28 (16) vs 236 (201) nmol/L; P < 0.001].

Conclusion: When precautions are taken to inhibit ATP release from blood elements during sample preparation, human venous plasma ATP concentration is much lower than previously reported.

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ATP release can occur during various physiological and pathophysiological events such as ischemia, hypoxia, platelet aggregation, sympathetic nerve stimulation, or cellular damage (1–5). ATP can affect thromboregulation and stimulate the immune cells responsible for asthma attacks (6, 7). ATP has also been proposed as a mediator of vasodilation during ischemia, hypoxia, and exercise. According to this hypothesis, low oxygen tension provokes ATP release from erythrocytes, as has been demonstrated in vitro (8, 9). ATP (or its metabolite ADP) subsequently binds to endothelial P2Y1 receptors, resulting in vasodilation (10, 11). Recent studies have provided preliminary support for the hypothesis by demonstrating that venous plasma ATP concentration increases during exercise in human skeletal muscle (12) and in the canine heart (13).

A plasma ATP assay (14) developed for the aforementioned dog study measured mean dog plasma ATP concentrations of 25–50 nmol/L (13). These concentrations are far lower than reported human venous plasma ATP concentrations, which are generally in the 1 µmol/L range (Table 1). The purpose of the present study was to determine whether human plasma ATP concentration measured with the revised ATP assay is significantly below the micromolar range. Additional aims were to determine whether pretreatment with aspirin to inhibit platelets lowers measured plasma ATP concentration, and whether a 2nd blood sample, drawn at a later time, yields lower ATP concentrations than the first (24).

Sample treatment is critical to accurate measurement of plasma ATP concentration. EDTA arrests ATP catabolism and is routinely used in plasma ATP assays (27). However, EDTA does not prevent potential ATP release from erythrocytes or platelets during sample preparation. Gorman et al. (14) collected blood samples in a stabilizing solution designed to minimize ATP release by platelets and erythrocytes and prevent plasma ATP catabolism. The stabilizing solution contains EDTA to inhibit ATPases and nitrobenzylthioinosine (NBTI)3 to inhibit ATP release from erythrocytes (8). Platelets are stabilized by increasing intracellular cAMP with forskolin and by inhibiting cAMP phosphodiesterase with isobutylmethylxanthine.

1 Department of Physiology and Biophysics, University of Washington, Seattle, WA.
2 Department of Anesthesiology, University of Pittsburgh, Pittsburgh, PA.
*Address correspondence to this author at: Department of Physiology and Biophysics, Box 357290, University of Washington, Seattle, WA 98195-7290.
Fax 206-616-3685; e-mail mgorman@u.washington.edu.
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3 Nonstandard abbreviations: NBTI, nitrobenzylthioinosine; IBMX, isobutylmethylxanthine.
Materials and Methods

This study was approved by the University of Washington human subjects review committee. Subjects were 20 healthy adult paid volunteers [10 female, 10 male; mean (SD) age, 39.4 (14.1); range, 21–75 years] who had not taken aspirin, ibuprofen, or cold medications within the previous 2 weeks. After participants gave written informed consent, they were randomly given either aspirin (325 mg, orally) or a placebo. For each participant, a 9-mL blood sample was drawn from an antecubital vein after a 30-min sitting period, and then 30 min later a 2nd sample was drawn from the opposite arm. The arm chosen first was assigned randomly. Both the investigator drawing blood samples and the investigator measuring plasma ATP concentration were blinded to the aspirin status of the sample donor.

BLOOD SAMPLING PROTOCOL

A rubber tourniquet was placed on the upper arm to assist in venipuncture. The blood collection set (Becton Dickinson 367281) used to draw blood samples consisted of a 21-guage 3/4-inch needle attached to 12 inches of plastic tubing. Blood samples (~9 mL) were manually drawn during ~20 s into 10-mL plastic syringes (Becton Dickinson 301604) containing 60 μL heparin (60 units) for anticoagulation.

BLOOD SAMPLE TREATMENT

Immediately after a blood sample was drawn, sample volume in excess of 8 mL (~1 mL) was expressed from the syringe into a test tube and subsequently used for hematocrit determination. Using the volume markings on the syringe, we added 4-mL blood aliquots to each of 2 plastic tubes containing 5.4 mL of 2 different diluent solutions at room temperature. One tube, referred to as the stabilizing solution sample, contained the stabilizing solution developed by Gorman et al. (14), containing, per liter, 118 mmol NaCl, 5 mmol KCl, 40 mmol tricine buffer, 4.15 mmol EDTA, 5 mmol NBTI, 10 μmol forskolin, and 100 μmol IBMX, pH adjusted to 7.4 with 2 mol/L KOH. The other tube, referred to as the EDTA-only sample, contained 4.15 mmol/L EDTA in isotonic saline. Blood was gently ejected from the syringe down the side of the tubes to avoid hemolysis. The blood:diluent solution volume ratio matched the ratio used in prior dog studies (13, 14) and provided sufficient volume for 4 subsequent ATP measurements plus a hemoglobin assay (see below). Tubes were capped and gently inverted twice for mixing.

Table 1. Human plasma ATP studies. a

<table>
<thead>
<tr>
<th>Reference</th>
<th>Plasma ATP, nmol/L</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Forrester and Lind (15)</td>
<td>1150</td>
<td>EDTA, luciferase</td>
</tr>
<tr>
<td>Forrester (16)</td>
<td>1030</td>
<td>EDTA, luciferase</td>
</tr>
<tr>
<td>Parkinson (17)</td>
<td>~230</td>
<td>Luciferase, deproteinized plasma</td>
</tr>
<tr>
<td>Jabs et al. (18)</td>
<td>1250</td>
<td>EDTA, luciferase, deproteinized plasma</td>
</tr>
<tr>
<td>Moss et al. (19)</td>
<td>11 000</td>
<td>Heparin, HPLC, acid plasma extract</td>
</tr>
<tr>
<td>Harkness et al. (20)</td>
<td>10 900</td>
<td>EDTA, HPLC, acid plasma extract</td>
</tr>
<tr>
<td>Harkness et al. (21)</td>
<td>1500</td>
<td>EDTA, HPLC, corrected for hemolysis, acid extract</td>
</tr>
<tr>
<td>Born and Kratzer (22)</td>
<td>1800</td>
<td>Luciferase, blood sampled via bleeding time lancet</td>
</tr>
<tr>
<td>Capecechi et al. (23)</td>
<td>120</td>
<td>Heparin, dipyrindamide, EHNA, b HPLC</td>
</tr>
<tr>
<td>Ryan et al. (24)</td>
<td>655 (first samples), 380 (second samples)</td>
<td>EDTA, theophylline, luciferase, βTG index of platelet activation</td>
</tr>
<tr>
<td>Lader et al. (25)</td>
<td>1020</td>
<td>EDTA, luciferase, samples iced 2–24 h before study</td>
</tr>
<tr>
<td>Gonzalez-Alonso et al. (12)</td>
<td>~600</td>
<td>Femoral vein, EDTA, luciferase, plasma frozen for later analysis</td>
</tr>
<tr>
<td>Rosenmeier et al. (26)</td>
<td>900 (venous), 550 (arterial)</td>
<td>EDTA, luciferase, plasma frozen for later analysis</td>
</tr>
<tr>
<td>Current study</td>
<td>28</td>
<td>See Materials and Methods</td>
</tr>
</tbody>
</table>

a A compilation of studies reporting normal human plasma ATP concentrations. All concentrations have been converted to nmol/L. The Notes column indicates additives to the blood sample, the type of assay (luciferase or HPLC), and other details of sample preparation or results. Samples are venous unless indicated otherwise. Some concentrations (~) were estimated from figures.

b EHNA, erythro-6-amino-9-(2-hydroxy-3-nonyl)-purine hydrochloride, βTG, β-thromboglobulin.
Elapsed time from the beginning of blood sample withdrawal to mixing with stabilizing solution or EDTA was ~1 min. Blood/diluent tubes were immediately centrifuged (13 000g) for 2 min at room temperature, and then 5 mL of supernatant was pipetted from these tubes and immediately recentrifuged for 2 min to pellet any remaining erythrocytes caught by surface tension. The supernatant of the 2nd centrifugation was transferred to a new tube and used for ATP and hemoglobin measurements. ATP measurements were begun immediately. Although stabilizing solution greatly retards ATP degradation, it does not stop it indefinitely (14).

**ATP assay**

The firefly luciferin–luciferase assay was used for determination of plasma ATP concentration. The current assay has been described earlier in detail (14). Supernatant aliquots (300 μL) from the 2nd centrifugation were added to each of 4 test tubes containing 100 μL ATP standard solution. The ATP standard solutions contained 0, 10, 20, or 30 pmol ATP per 100 μL. The ATP standard solutions were prepared in stabilizing solution but with the pH previously adjusted to 8.75, so that after addition of all reagents including luciferase the sample pH was ~7.9, the optimal pH for the luciferase reaction (Fig. 1). For EDTA-only samples the ATP standard solutions were prepared in EDTA/saline without prior pH adjustment.

After a test tube was placed in the luminometer (Berthold model LB 9507), 25 μL Mg2+ solution (177 mmol/L MgCl2, 40 mmol/L tricine, pH 7.75) was added via an automatic injector in the luminometer to counteract the decrease of sample Mg2+ concentration by EDTA in the samples; 2 s later, 100 μL luciferase reagent (ATP Bioluminescence Assay Kit CLS II: Roche Diagnostics) was added via a 2nd luminometer injector. After 3 s for the reaction to reach a steady state, the cumulative light output in relative light units was measured for 10 s. A blank sample containing 300 μL of either stabilizing solution or EDTA/saline without plasma was used to determine luminescence in the absence of ATP, and the appropriate value was subtracted from all samples.

Sample ATP content was calculated using the method of standard additions. The cumulative relative light units in 10 s for each test tube (containing 0, 10, 20, or 30 pmol added ATP) was plotted vs the amount of added ATP (Fig. 2). A least-squares regression line was fit to the data, and the ATP content of the unenriched (native) sample was equal to the y intercept divided by the slope (or the absolute value of the x intercept). This ATP content was attributed to the 300-μL sample. Final concentrations of added ATP in the 4 assay tubes were 0, 19.0, 38.1, and 57.1 nmol/L.

**Correction for hemolysis**

Sample hemoglobin concentration was used as an index of hemolysis. An aliquot of the same supernatant used for ATP determination was used without dilution for hemoglobin concentration determination (29). Absorbance (A) was measured at 380, 415, and 450 nm with a Bausch and Lomb Spectronic 70 spectrophotometer. Hemoglobin concentration (milligrams per liter) was calculated as 10[16.72 A415 − 8.36 A380 − 8.36 A450] (13, 28). To determine the ATP concentration attributable to hemolysis, heparinized blood samples were collected from 2 individuals (without stabilizing solution) and immediately centrifuged. The plasma layer and buffy coat were removed by aspiration. The erythrocyte pellet was resuspended in isotonic saline and centrifuged again. The saline supernatant was removed by aspiration, and various volumes of the eryth-
rocyte pellet (2–40 μL) were lysed in 5 mL distilled water containing 3 mmol/L EDTA (to stabilize the ATP concentration); 25 μL of this lysate was then diluted in 3 mL stabilizing solution, and the ATP content in 300 μL of this solution was determined using the 4-point standard addition method described above. ATP concentration was calculated by attributing the ATP content to the 300 μL sample volume. Hemoglobin concentration was determined in the same solution. ATP concentration was plotted vs hemoglobin concentration to create a hemolysis correction curve (Fig. 3).

**Calculation of Plasma ATP Concentration**

Sample ATP content (pmol per 300 μL sample) was multiplied by 3.33 to arrive at the concentration (ATP_{total}, nmol/L). The concentration of ATP in the sample due to hemolysis (ATP_{hemol}, nmol/L) was determined from the sample hemoglobin concentration and the hemolysis correction curve and was subtracted from ATP_{total}. Plasma ATP concentration was arrived at after correcting for dilution. Thus, plasma [ATP] = (ATP_{total} - ATP_{hemol}) × (1.35 + 1 - HCT)/(1 - HCT), where 1.35 is the ratio of stabilizing solution (or EDTA/saline) volume to blood sample volume, and HCT is the fractional hematocrit.

**Statistical Analysis**

Three-way ANOVA using aspirin status, sample time, and diluent solution composition as factors revealed that the only significant differences in sample ATP concentration were between stabilizing solution and EDTA-only samples (P < 0.001). Because the data did not show a gaussian distribution, further paired comparisons (sample time, stabilizing solution vs EDTA) were made with the nonparametric Wilcoxon matched-pairs test. Placebo and aspirin results (unpaired) were compared with the nonparametric Mann–Whitney test. Correlation between stabilizing solution and EDTA-only results in the same samples was determined with the nonparametric Spearman correlation. P < 0.05 was considered statistically significant. Statistical tests were done with GraphPad Prism software. Results in the text and tables are presented as mean and SD.

**Results**

The effects of temperature and pH on the luciferase reaction are presented in Fig. 1 (14). Maximum light output is achieved at room temperature and a pH of 7.75–7.95. The graphical determination of plasma ATP concentration is illustrated in Fig. 2. Results are shown for the same blood sample with both stabilizing solution and EDTA only. The native samples are points on the y axis with zero added ATP. Light output as a function of sample ATP content is highly linear in both cases. Because of sample pH optimization, the slope is higher for the stabilizing solution sample than for the EDTA-only sample.

Results of the placebo vs aspirin groups and stabilizing solution vs EDTA only are presented in Fig. 4 and Table 2. The 2 samples drawn 30 min apart were not significantly different in any subgroup (Table 2) and have therefore been combined in Fig. 4, which illustrates that aspirin pretreatment had no effect on plasma ATP concentration. On the other hand, use of stabilizing solution resulted in...
ATP concentrations 8-fold lower than EDTA alone ($P < 0.001$).

Paired EDTA and stabilizing solution results from the same blood samples ($n = 40$, combined aspirin and placebo groups) are presented in Fig. 5. There was no correlation between the 2 values (Spearman $r = -0.23$; $P = 0.15$).

The ATP concentrations resulting from intentional hemolysis of small volumes of human erythrocytes are shown in Fig. 3. This relationship between supernatant hemoglobin concentration and ATP concentration was used to correct sample ATP concentrations for hemolysis. For all stabilizing solution samples ($n = 40$) the mean (SD) supernatant (plasma + stabilizing solution) hemoglobin concentration was $2.7 (1.6)$ mg/L. Without hemolysis correction the plasma ATP concentration was $64 (23)$ nmol/L. After individual correction for hemolysis the plasma ATP concentration was $28 (16)$ nmol/L. Thus, hemolysis correction reduced the ATP concentration in stabilizing solution samples by 56%. In 4 of the 20 study participants high plasma turbidity or coloration prevented an accurate plasma hemoglobin determination. These samples were assigned the mean hemoglobin concentration from all samples from the remaining 16 participants. The supernatant hemoglobin concentration in EDTA-only samples [$2.5 (1.5)$ mg/L] was not different from that of stabilizing solution samples [$2.7 (1.6)$ mg/L]. These values correspond to undiluted plasma hemoglobin concentrations of $8.3$ mg/L in EDTA-only samples and $9.0$ mg/L in stabilizing solution samples.

**Discussion**

The most important conclusion from this study is that addition of the stabilizing solution to human blood samples leads to measured plasma ATP concentrations 8-fold lower than the use of EDTA alone. Use of stabilizing solution and correction for ATP released by hemolysis resulted in measured plasma ATP concentrations [$28 (16)$ nmol/L] that are much lower than those reported in previous studies (see Table 1). Because many artifacts can increase plasma ATP, the lower values are more likely to be the correct ones. Other conclusions are that aspirin pretreatment does not influence the measurement of plasma ATP concentration, and a 2nd blood sample drawn 30 min after the first results in measurements that are not significantly different.

An accurate plasma ATP assay should prevent both plasma ATP catabolism and additional ATP release by blood formed elements during sample preparation. EDTA prevents plasma ATP breakdown ($14, 27$) and is routinely

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**Table 2. Plasma ATP concentrations.**

<table>
<thead>
<tr>
<th>Treatment and time</th>
<th>Plasma ATP, nmol/L</th>
<th>Uncorrected</th>
<th>Corrected for hemolysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>EDTA only</td>
<td>Stabilizing solution</td>
<td>EDTA only</td>
</tr>
<tr>
<td>Placebo</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sample 1</td>
<td>267 (235)</td>
<td>46 (27)</td>
<td>244 (236)</td>
</tr>
<tr>
<td>Sample 2</td>
<td>291 (232)</td>
<td>74 (19)</td>
<td>252 (230)</td>
</tr>
<tr>
<td>Aspirin</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sample 1</td>
<td>249 (234)</td>
<td>65 (21)</td>
<td>224 (220)</td>
</tr>
<tr>
<td>Sample 2</td>
<td>267 (141)</td>
<td>72 (14)</td>
<td>223 (131)</td>
</tr>
<tr>
<td>All Samples</td>
<td>269 (206)</td>
<td>64 (23)*</td>
<td>236 (201)</td>
</tr>
</tbody>
</table>

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Fig. 4. The effects of stabilizing solution and aspirin pretreatment on human plasma ATP concentration.

There were no significant differences between 30-min and 60-min samples, which have been consolidated in this figure. Aspirin pretreatment had no effect on ATP concentration with either stabilizing solution or EDTA only. However, stabilizing solution resulted in plasma ATP concentrations far lower than the use of EDTA alone ($P < 0.001$ for both placebo and aspirin groups). Numbers within the bars are the number of samples. Error bars indicate SE.
added to blood samples in plasma ATP assays. The stabilizing solution in the current assay includes the nucleoside transport inhibitor NBTI, which also inhibits erythrocyte ATP release (8). Platelets are stabilized by inclusion of forskolin to increase cAMP concentration and IBMX to inhibit cAMP phosphodiesterase (30, 31). Use of a stabilizing solution is analogous to the approach developed for plasma adenosine assays in which blood is mixed with inhibitors of adenosine formation, uptake, and deamination (32–34).

Although the use of EDTA alone instead of stabilizing solution increased the measured ATP concentration 8-fold on average, the correlation between EDTA-only and stabilizing solution ATP concentrations in the same blood samples was nonexistent (Fig. 5). This result may be explained by centrifugation of EDTA-only samples releasing a large amount of ATP from platelets or erythrocytes. Assuming that the ATP concentrations with stabilizing solution are correct, Fig. 5 demonstrates that using an EDTA-only assay and dividing the resulting concentration by 8 will not provide a reliable estimate of ATP concentration in individual samples.

A potentially important difference between the stabilizing solution and EDTA/saline was that the stabilizing solution was buffered to pH 7.4. The EDTA/saline solution typically had a pH of 4.6. It is unlikely, however, that a lower pH induced the release of ATP in the EDTA/saline samples. In dog blood samples in which pH 7.4 buffered stabilizing solution was compared with and without forskolin/IBMX, forskolin and IBMX decreased measured ATP values at a constant stabilizing solution pH (14). Hemoglobin concentrations in EDTA-only and stabilizing solution samples in the present study were not different, indicating that pH differences did not induce hemolysis.

Sample buffering clearly has an influence on the light output from the luciferase reaction, with buffered samples having higher output for a given ATP concentration (Figs. 1 and 2). Most of the buffering for optimum luciferase reaction pH (≈7.9) was achieved in stabilizing solution samples by adjusting the pH of the ATP standard solution added to the luminometer tubes. Although sample pH and possibly other components of the stabilizing solution (forskolin, IBMX, etc.) influence the light output of the luciferase reaction, this does not explain the lower ATP concentrations in stabilizing solution samples. Both EDTA-only and stabilizing solution samples exhibited highly linear standard curves when light output was plotted vs ATP content (Fig. 2). The virtue of the standard addition technique is that all 4 samples in these plots are identical except for ATP content. Any effect of sample composition on light output (other than ATP) is therefore common to all samples and does not influence the assay result. The effect of stabilizing solution on sample ATP concentration occurs during sample processing and is independent of effects on the luciferase reaction.

Another factor contributing to low plasma ATP concentrations in the current study is correction for hemolysis. The high cytosolic ATP concentration in erythrocytes means that hemolysis invisible to the naked eye can significantly increase plasma ATP concentration. Plasma hemoglobin concentration was used as an index of hemolysis. Harkness et al. (21) plotted plasma ATP concentrations vs plasma hemoglobin concentrations in a group of samples and extrapolated to zero hemoglobin for an estimate of hemolysis-free plasma ATP concentration. The hemolysis correction technique in the current study has the virtue of being applicable to individual samples. In samples treated with stabilizing solution, hemolysis was on average responsible for roughly half of the sample ATP concentration (56%). It is uncertain, however, whether the low plasma hemoglobin concentrations in this study represent fresh hemolysis. The hemoglobin concentration in circulating plasma may be greater than zero; if so, the present method overcorrects for hemolysis, and the true plasma ATP concentration may be somewhat higher. Thus, the true mean plasma ATP concentration in the current study may be between 28 nmol/L (with hemolysis correction) and 64 nmol/L (no hemolysis correction).

The sample hemoglobin concentrations in the current study [overall mean, 2.6 (1.5) mg/L] incorporated dilution of plasma with stabilizing solution or EDTA/saline. When corrected for this dilution, mean hemoglobin concentration in the undiluted plasma was 8.6 mg/L, which represents ~0.006% hemolysis (assuming 140 g hemoglobin per L blood). Even modest hemolysis can clearly have a large influence on plasma ATP concentration, an effect that may account for some of the high values reported in the literature (Table 1). It is essential to measure the hemoglobin concentration in every sample. Samples with >10 mg/L hemoglobin after dilution with stabilizing solution (~32.5 mg/L in undiluted plasma) should probably be discarded because of the large hemolysis correction that would be required.

A given plasma hemoglobin concentration in human samples required roughly twice the plasma ATP correction that was necessary in dog plasma (14). This finding is consistent with the high ATP content in human erythrocytes compared with those of dogs (35). The slope of the plot in Fig. 3 indicates an erythrocyte ATP content of 3.94 μmol ATP per g hemoglobin, similar to human erythrocyte ATP contents detected by other laboratories [3.7 (8), 4.24 (36), and 3.94 (37)]. Other plasma hemoglobin assays (28) may be used in place of the Harboe (29) technique chosen for this study and may prove to be superior in samples with low hemolysis. Each laboratory should use the chosen hemoglobin assay to generate a hemolysis ATP correction curve similar to Fig. 3.

One hypothesis was that the negative effects of aspirin on platelet aggregation might reduce platelet ATP release and lead to lower plasma ATP concentrations, especially in samples treated with EDTA only. The results clearly do
not support this hypothesis, because aspirin pretreatment had no effect on plasma ATP concentration in either saline/EDTA or stabilizing solution. Aspirin had no effect in samples taken either 30 or 60 min after aspirin ingestion. Thirty min after oral administration is sufficient for saline/EDTA or stabilizing solution. Aspirin had no effect on plasma ATP concentration in either study are not the result of low recoveries. Plasma ATP concentration did not change in blood samples drawn 30 min apart. Correction for hemolysis-induced ATP release from erythrocytes may result in different ATP concentrations. Local ATP release from erythrocytes other locations with different venous oxygen tensions武川 financing. All blood samples in this study were drawn from the antecubital veins. Because oxygen tension influences ATP release from erythrocytes, samples drawn from other locations with different venous oxygen tensions may result in different ATP concentrations. Local ATP concentrations, particularly near aggregating platelets or nerve endings, may be considerably higher than the circulating venous concentrations reported here.

In summary, addition of blood samples to a stabilizing solution designed to stabilize plasma ATP concentration resulted in human plasma ATP concentrations 8-fold lower than treatment with EDTA alone. Aspirin pretreatment did not affect plasma ATP concentration. ATP concentration did not change in blood samples drawn 30 min apart. Correction for hemolysis-induced ATP release decreased plasma ATP concentration by 56%. These results indicate that the true resting human venous plasma ATP concentration is far lower than previously reported and is ~28 nmol/L.

References