

University of Arkansas, Fayetteville

ScholarWorks@UARK

Health, Human Performance and Recreation
Undergraduate Honors Theses

Health, Human Performance and Recreation

5-2015

Effects of Dehydration on Changes in Arterial Stiffness with Passive Heating

AZ Satterfield

University of Arkansas, Fayetteville

Follow this and additional works at: <https://scholarworks.uark.edu/hhpruht>



Part of the [Health and Physical Education Commons](#), [Health Services Research Commons](#), and the [Science and Mathematics Education Commons](#)

Citation

Satterfield, A. (2015). Effects of Dehydration on Changes in Arterial Stiffness with Passive Heating. *Health, Human Performance and Recreation Undergraduate Honors Theses* Retrieved from <https://scholarworks.uark.edu/hhpruht/55>

This Thesis is brought to you for free and open access by the Health, Human Performance and Recreation at ScholarWorks@UARK. It has been accepted for inclusion in Health, Human Performance and Recreation Undergraduate Honors Theses by an authorized administrator of ScholarWorks@UARK. For more information, please contact scholar@uark.edu, uarepos@uark.edu.

Effects of Dehydration on Changes in Arterial Stiffness with Passive Heating

AZ Satterfield

University of Arkansas

Major Advisor: Matthew S. Ganio, Ph.D.

Committee Members: Stavros A. Kavouras, Ph.D. & Inza L. Fort, Ed.D

A thesis submitted to the Honors College at the University of Arkansas in

partial fulfillment of the requirements for the degree

Bachelor of Science with Honors

© 2015

Abstract

Context: There is an inverse relationship between baseline arterial stiffness and the change in arterial stiffness with passive heating. However, it is unknown whether this relationship is affected by dehydration. **Objective:** To investigate the effect of acute dehydration on arterial stiffness during passive heat stress. **Design:** Two randomized counter-balanced trials. **Setting:** Laboratory. **Patients or Other Participants:** Eleven healthy males (age= 24.5 ± 2.8 years, body mass= 76.6 ± 9.1 kg, body fat= $16.8 \pm 6.4\%$). **Interventions:** In one trial subjects were dehydrated (DE) and in another euhydration (EU) was maintained during passive heating to a 1.5°C increase in body temperature. Subjects were euhydrated prior to each trial confirmed via urine specific gravity (USG) <1.021 . During the EU trial, water (15 ml/kg body mass) was given during passive heating, but no water was given during the DE trial so that the subjects became dehydrated. At baseline, 0.5, 1.0, and 1.5°C core temperature increases, arterial stiffness was measured by calculating central (cPWV) and peripheral (pPWV) pulse wave velocity by using a Doppler ultrasound at the radial, carotid and femoral arteries. **Main Outcome Measures:** Primary measures were cPWV, pPWV, T_c , and body mass (BM) pre- and post-trial. A two-way repeated measures ANOVA (hydration status x core temperature increase) was used to assess the effect of acute dehydration on arterial stiffness during passive heating. Regression analyses were used to assess the relationship between baseline PWV and the change with heating (both centrally and peripherally). An alpha <0.05 defined significance for all tests. **Results:** As designed, BM was maintained in the EU trial (BM loss= $0.08 \pm 0.74\%$, $p=0.54$) and decreased in the DE trial (BM loss= $1.58 \pm 0.63\%$, $p <0.001$). Hydration differences between trials did not affect cPWV ($p = 0.634$) or pPWV ($p = 0.189$). Independent of hydration status, heat stress decreased pPWV (711.3 ± 76.4 to 652.0 ± 54.3 cm/s; $p = 0.003$), but not cPWV (494.3 ± 87.9 to

488.2 ± 71.9 cm/s; $p = 0.651$). The effect of heat stress on cPWV and pPWV was not further affected by hydration status (non-significant interaction $p > 0.05$). A significant negative relationship ($p < 0.01$) was observed between baseline arterial stiffness and changes during heating in EU cPWV ($r = -0.61$), pPWV ($r = -0.70$), and DE pPWV ($r = -0.55$), but not DE cPWV ($r = -0.09$; $p = .61$). The relationships between baseline stiffness and changes in heating were not significantly different between EU and DE trials ($p > 0.05$). **Conclusions:** Passive heat stress decreases peripheral, but not central arterial stiffness independent of hydration status. Acute dehydration that occurs with passive heating does not affect arterial stiffness in young, healthy males.

Introduction

Increased arterial stiffness (i.e., decreased compliance) alters arterial pressure and flow dynamics and impacts cardiac performance and coronary perfusion (Zieman et al. 2005). This can increase one's risk for a cardiovascular event (e.g., heart attack) and premature death. Arterial stiffening develops from complex interactions between stable and dynamic changes within structural and cellular elements of the vessel wall (Zieman et al. 2005). Diseases such as hypertension, diabetes, and aging lead to increased arterial stiffness. It is important to investigate factors that might affect arterial stiffness in order to gain knowledge that may help decrease the number of preventable cardiovascular deaths in the United States.

Chronic exercise training reduces resting arterial stiffness. This is likely a result of an accumulation of the arterial benefits reaped from each acute exercise bout. For example, exercise at 65% of maximal oxygen uptake elevates aortic flow and carotid pressure for about 30 min post-exercise with a return to baseline one hour after exercise (Kingwell et al. 1997). This leads to parallel changes in the arteries such that arterial stiffness is acutely reduced with moderate

intensity exercise. Overall it is well established that decreased arterial stiffness during exercise (and immediately after) reduces cardiovascular risk in the immediate post exercise period and chronically with regular exercise (Kingwell et al. 1997).

During exercise, or increased physical activity, the human body's core internal temperature will increase. In an attempt to maintain homeostasis, the body thermoregulates to maintain the body's internal body temperature. During thermoregulation, heat is released through the evaporation of sweat and increased skin blood flow. Blood flow to the skin helps remove the heat from the body via convection while the evaporation of sweat removes heat from the body. However the loss of sweat without adequate replacement via fluid ingestion leads to dehydration. Dehydration reduces aerobic endurance performance and creates an independent increase in body temperature, heart rate, and perceived exertion (Barr et al. 1999).

Previously we have shown that passive heat can acutely decrease arterial stiffness (Ganio et al. 2011). Subjects with the greatest normothermic arterial stiffness had the greatest decreases in stiffness when heat-stressed (Ganio et al. 2011). In other words those with poor stiffness at baseline had greater improvements in stiffness with heating. In fact, because we have found differences, we need to know if these are in part due to dehydration that occurs during heat stress (see above). Changes in arterial stiffness during heat stress are likely because of increased blood flow (shear stress) during heat stress. Shear stress releases nitric oxide, which leads to vasodilation and subsequent reductions in arterial stiffness. So with dehydration, it is possible there is not as much blood flow and thus not as much shear stress and consequently not as much improvement in arterial stiffness occurs with heating. However this hypothesis needs to be tested. Therefore, the purpose of this study was to test the hypothesis that dehydration leads to less improvement in arterial stiffness compared to when euhydrated during passive heating.

Materials and Methods

Subjects.

This study involved 11 (n=11), non-obese, healthy, moderate activity males (ages 18-39). Subjects' mean \pm SD age, mass and body fat percentage were 24.5 ± 2.8 years, 76.6 ± 9.1 kg, $16.8 \pm 6.4\%$. Females were excluded because of the effects of the menstrual cycle on body water balance and arterial stiffness; however future studies need to investigate them. Individuals with previous heat illness, serious medical conditions, or taking medications altering cardiovascular or thermoregulatory function also were excluded.

Experimental Controls.

To evaluate how changes in arterial stiffness with passive heating are affected by dehydration, subjects completed two trials: one dehydrated and one euhydrated. Each trial was separated by at least 72 hours. Prior to each trial, each subject did not exercise or drink alcohol for 24 hours and consume caffeine for eight hours. For the both trials, subjects were asked to drink 1000 mL of extra water the night before the trial and 500 mL of water the morning of the trial. Fluid was provided during the passive heating of the euhydrated trial to offset sweat losses and maintain hydration. The amount of fluid given was 15 ml/kg body mass. This fluid was provided after recording the baseline, 0.5°C , and 1.0°C measurements. During the dehydrated trial, subjects were not provided fluid during passive heating in order for the subject to become dehydrated. Through pilot testing, we have found this will lead to a dehydration of approximately 1.5 % body mass loss; a magnitude of dehydration that is safe and often experienced by individuals during daily activities.

Experimental Procedures.

Upon arrival for each trial, body weight was measured (Health-o-meter digital scale, model 349KLX, Pelster LLC, Alsip, IL, USA) and a urine sample was provided in which urine specific gravity was measured to assess hydration status (clinical refractometer 30005, SPER Scientific, Scottsdale, AZ, USA). At that time, subjects inserted a rectal thermistor 16 cm past the anal sphincter. This was used to validly monitor and measure core body temperature during passive heating.

Next, blood pressure was measured with an automated sphygmomanometer blood pressure cuff (Tango+; SunTech Medical, Inc., Morrisville, NC, USA). Subjects were fitted with a heart rate monitor (Polar Electro Inc., Lake Success, NY, USA). Skin thermistors were placed on the right anterior thigh, chest, lateral calf, and triceps to record and calculate mean skin temperature (Omega Engineering, Stamford, CT, USA). Subjects dressed in a water-perfused suit (Allen Vanguard, Ottawa, ON, Canada) that covered the whole body except for hands, head, and feet. After this, subjects were asked to put on rain pants and a blanket over the water-perfused suit; this helped facilitate heating. After the subject was dressed, they laid supine for 30 minutes. As the subject was lying down, water at 34°C ran through the suit. After 30 minutes, the baseline measurements were recorded. Next, the heating phase began by running hot water (49°C) through the suit. Measurements were recorded at each 0.5° C core temperature increase until core temperature increased 1.5 °C from baseline. After heating, cold water was run through the suit.

The main dependent variable, arterial stiffness, was measured at each 0.5°C core temperature increase using pulse wave velocity (PWV). Pulse wave velocity is the preferred method to evaluate arterial stiffness (Laurent et al. 2006). It was done using Doppler ultrasound

combined with a 3-lead ECG [LOGIQ e; GE Healthcare, Milwaukee, WI, USA (Laurent et al. 2006)]. PWV was measured on the carotid, radial, and femoral arteries on the right side of the body. For the reference point, the peak R wave on the ECG to the foot of the pulse wave for each site was used. At least ten cardiac cycles was used at each site and time point (Laurent et al. 2006). From these measurements central (carotid-femoral) and peripheral (carotid-radial) arterial stiffness were calculated using standard techniques (Ganio et al. 2011).

Statistical Analysis.

Arterial stiffness was compared at each time point (i.e., each 0.5° C core temperature increase) between the dehydrated and euhydrated trials. SPSS v. 20.0 was used to analyze the mean \pm standard deviation (SD) data (IBM Corporation, Somers, NY). A 2-way repeated analysis of variance (ANOVA) was utilized to evaluate the change in arterial stiffness between trials (dehydrated vs. euhydrated) during heating. Bonferroni alpha corrections were made when appropriate. Alpha was set at 0.05.

Results

Subjects started all trials euhydrated and remained euhydrated in the euhydrated (EU) trial (urine specific gravity; USG = 1.008 ± 0.007). As designed, body mass (BM) was maintained in the euhydrated (EU) trial (BM loss = $0.08 \pm 0.74\%$, $p=0.54$) and decreased in the dehydrated (DE) trial (BM loss = $1.58 \pm 0.63\%$, $p < 0.001$).

For the euhydrated trial, core temperature increased by 0.54 ± 0.09 , 1.01 ± 0.07 , $1.51 \pm 0.09^\circ\text{C}$ from baseline to each measurement ($p < 0.05$). For the dehydrated trial, core temperature increased by 0.51 ± 0.09 , 0.97 ± 0.09 , $1.49 \pm 0.08^\circ\text{C}$ from baseline to each measurement ($p < 0.05$). For both euhydrated and dehydrated trials, skin temperature and heart rate increased

over time ($p < 0.05$; Table 1). The only significant change in mean arterial pressure was from baseline to 0.5°C ($p = 0.175$).

Hydration differences between trials did not affect central pulse wave velocity (cPWV) ($p = 0.634$) or peripheral pulse wave velocity (pPWV) ($p = 0.189$). Independent of hydration status, heat stress decreased pPWV (711.3 ± 76.4 to 652.0 ± 54.3 cm/s; $p = 0.003$), but not cPWV (494.3 ± 87.9 to 488.2 ± 71.9 cm/s; $p = 0.651$). The effect of heat stress on cPWV and pPWV was not further affected by hydration status (non-significant interaction $p > 0.05$). A significant negative relationship ($p < 0.01$) was observed between baseline arterial stiffness and changes during heating in EU cPWV ($r = -0.61$), pPWV ($r = -0.70$), and DE pPWV ($r = -0.55$), but not DE cPWV ($r = -0.09$; $p = 0.61$). The relationships between baseline stiffness and changes in heating were not significantly different between EU and DE trials ($p > 0.05$).

Discussion

There is an inverse relationship between baseline arterial stiffness and the change in arterial stiffness with passive heating. Individual changes in central and peripheral arterial stiffness are negatively correlated with baseline normothermic arterial stiffness (Ganio et al. 2011). However, it is unknown whether this relationship is affected by dehydration, which is what this study examined. The primary findings of this study were that hydration status had no significant effect on changes in cPWV and pPWV during heating. Independent of hydration status, heat stress decreased pPWV but not cPWV. Overall, these results suggest that hydration status from baseline to 1.5°C core temperature increase has similar affects while being passively heated.

Studies have shown that dehydration does impair muscle blood flow in athletes during prolonged exercise in hot conditions (González-Alonso et al. 2004). In the aforementioned study, subjects cycled for a long period of time and had one euhydrated and one dehydrated trial. During the dehydrated trial, blood flow to the legs declined significantly during the 20 minute to exhaustion period of exercise. Forearm blood flow was also significantly lower during the dehydrated trial (González-Alonso et al. 2004). With exercise, studies have shown that blood flow is reduced. This could imply that less shear stress is also occurring. This is important because increases in shear stress is hypothesized as the reason for why heat stress reduces arterial stiffness. Although cardiac output was elevated during heat stress in the present study, the magnitude of dehydration was probably not great enough to result in cardiac output differences between the euhydrated and dehydrated trials. If the cardiac output stayed the same between the trials, it would mean that shear stress was probably similar between trials. Therefore it is not too surprising that arterial stiffness was not different between the euhydrated and dehydrated trials.

A study found that skin blood flow and nitric oxide concentrations both increased at similar internal temperatures during body heating (Kellogg et al. 2003). It is possible that during heat stress, release of neurotransmitters from the cholinergic cotransmitter system increases skin blood flow. The increase in skin blood flow could then increase shear stress on the endothelial cells that in turn generate more nitric oxide (Kellogg et al. 2003). Due to homeostasis, the body thermoregulates in response to heat stress and thus increases skin blood flow. Nitric oxide is a major vasodilator, which causes the blood vessels to relax and decrease arterial stiffness. Regardless of hydration status, there is a large release of nitric oxide from passive heating, which, along with the other reasons described above, may be the reason for similar PWV during the euhydrated and dehydrated trials.

Changes in the stiffness of the large arteries, such as the aorta and its major branches, largely account for the changes in SBP, DBP, and PP that occur from 50 years of age onward (Oliver et al. 2003). Since this research only had young, healthy male subjects, it is very unlikely that any of them showed signs of significant changes of arterial stiffness. For most, showing signs of arterial disease or damage is a chronic and long process. It is plausible that the subjects in this experiment are simply too healthy to show signs of increased arterial stiffness from the dehydrated trial. Studies have shown, in older people, large artery stiffness, has emerged as an even stronger predictor of chronic heart disease (Oliver et al. 2003). It would be wise to do future studies on older subjects to see if hydration status may have an effect on arterial stiffness with passive heating.

The increase in arterial stiffness that occurs with age is largely the result of progressive elastic fiber degeneration (Oliver et al. 2003). Elasticity is a major indicator of arterial stiffness. The more elastic arteries are, the slower the PWV and vice versa. The elasticity of a given arterial segment is not constant but instead depends on its distending pressure. As distending pressure increases, there is greater recruitment of relatively inelastic collagen fibers and, consequently, a reduction in elasticity (Oliver et al. 2003). Distending pressure is determined by mean arterial pressure (MAP). The subjects in this study were young, healthy males and none of them had histories of hypertension or showed signs of elastic fiber degeneration. Once again, the fact these were young and healthy subjects with no prior heart or heat illness made it very unlikely to show differences in cPWV and pPWV between the euhydrated and dehydrated trials.

In conclusion, hydration status had no effect on cPWV and pPWV during passive heating. There was a similar effect on arterial stiffness during both trials. Independent of hydration status, heat stress decreased pPWV but not cPWV. The decreases and similarities

between the euhydrated and dehydrated trials in PWV are likely connected to increased production of nitric oxide due to passive heating. This study simulated only mild dehydration on the subjects. Since the dehydration was so mild, it would have made it less likely to see differences between the two trials. The subjects were in supine position, which also would have made it difficult to find differences in cPWV and pPWV between the euhydrated and dehydrated trials. Arterial stiffness increases with age, which leads to decreased elasticity and high PWV. The subjects in this study were all young and healthy with no prior heat or heart related illness. This might have been a factor why they did not show signs of arterial stiffness or increased PWV between the two trials.

References

- Ashton, K. (2013). The effect of passive heat stress on arterial wave reflection, pulse wave velocity, and cerebral blood flow. University of Delaware, Department of Exercise Science.
- Barr, S. (1999). Effects of dehydration on exercise performance. *Applied Physiology, Nutrition, and Metabolism*, 24(2), 164-172.
- Ganio, M., Brothers, R., Shibata, S., Hastings, J., & Crandall, C. (2011). Effect of passive heat stress on arterial stiffness. *Experimental Physiology*, 96(9), 919-926.
- Gonzalez-Alonso, J., Calbet, J., & Nielsen, B. (1998). Muscle blood flow is reduced with dehydration during prolonged exercise in humans. *The Journal of Physiology*, 3, 895-905.
- Kellogg, D., Zhao, J., Friel, C., Roman, L. (2003). Nitric oxide concentration increases in the cutaneous interstitial space during heat stress in humans. *Journal of Applied Physiology*, 94 (5), 1971-1977.
- Kinlay, S., Creager, M., Fukumoto, M., Hikita, H., Fang, J., Selwyn, A., & Ganz, P. (2001). Endothelium-derived nitric oxide regulates arterial elasticity in human arteries in vivo. *Hypertension*, 38, 1049–1053.
- Laurent, S., Cockcroft, J., Bortel, L., Boutouyrie, P., Giannattasio, C., Hayoz, D., & Struijker-Boudier, H. (2006). Expert consensus document on arterial stiffness: Methodological issues and clinical applications. *European Heart Journal*, 2588-2605.
- Moyen, N., Ganio, M., Burchfield, M., Tucker, M., Gonzalez, M., Dougherty, E., Robinson, F., Ridings, C., & Veilleux, J. Effect of Passive heat-stress on arterial stiffness in smokers versus non-smokers. Human Performance Laboratory, Department of Health, Human

Performance and Recreation, University of Arkansas, Fayetteville, AR.

Oliver, J. (2003). Noninvasive assessment of arterial stiffness and risk of atherosclerotic events.

Arteriosclerosis, Thrombosis, and Vascular Biology, 554-566.

Roberts, M., Wenger, C., Stolwijk, J., & Nadel, R. (1977). Skin blood flow and sweating changes following exercise training and heat acclimation. *Journal of Applied Physiology*, 43(1), 33-137.

Zieman, S. (2005). Mechanisms, pathophysiology, and therapy of arterial stiffness.

Arteriosclerosis, Thrombosis, and Vascular Biology, 932-943.

Table 1. Euhydrated (EU) and dehydrated (DE) thermal and hemodynamic responses at normothermia and after subjects' core temperatures were increased by approximately 0.5, 1.0 and 1.5°C

		Baseline	0.5°C	1.0°C	1.5°C
Mean skin temperature (°C)	EU	33.0± 2.4	37.9± 2.8	38.2± 2.6	38.7± 2.5
	DE	33.2± 3.6	37.5± 4.2	37.8± 4.8	38.2± 4.8
Core temperature (°C)	EU	36.1± 1.2	37.2± 0.3	37.7± 0.4	38.2± 0.3
	DE	36.5± 0.9	37.2± 0.3	37.7± 0.3	38.2± 0.3
Systolic blood pressure (mmHg)	EU	125± 10	128±12	133±16	138±20
	DE	120± 11	129± 13	131±18	133± 15
Diastolic blood pressure (mmHg)	EU	66± 7	56 ±16	50± 8	52.3± 8
	DE	63± 8	58± 10	56± 14	52.0± 12
Mean arterial pressure (mmHg)	EU	85.5± 5.8	73.3± 25.8	77.4±5.9	81± 5.7
	DE	82.6± 6.4	78.0± 16.9	81.4±13.0	79± 9.2
Heart rate (beats min ⁻¹)	EU	59± 15	82± 16	92± 18.8	103± 17
	DE	60± 12	83± 18	94± 19.1	102± 19

* Significant difference from the previous temperature stage ($p < 0.05$).

Figure 1. Dehydration (DE, red) did not affect central pulse wave velocity (cPWV) compared to euhydrated (EU, blue) ($p=0.634$) during passive heating ($+1.5^{\circ}\text{C}$ core temperature increase).

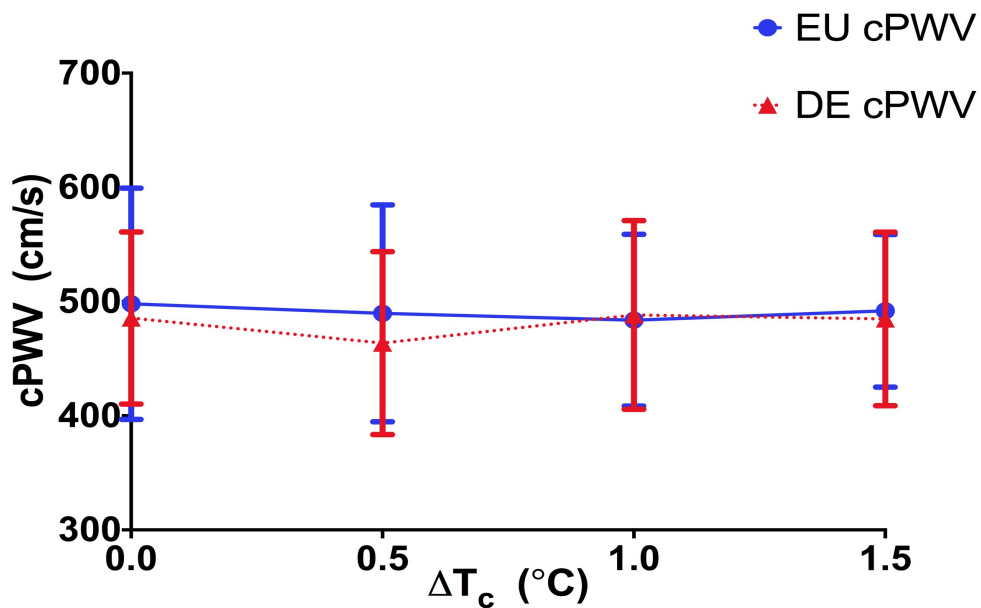


Figure 2. Dehydration (DE, red) did not affect peripheral pulse wave velocity (pPWV) compared to euhydrated (EU, blue) ($p=0.189$) during passive heating ($+1.5^{\circ}\text{C}$ core temperature increase).

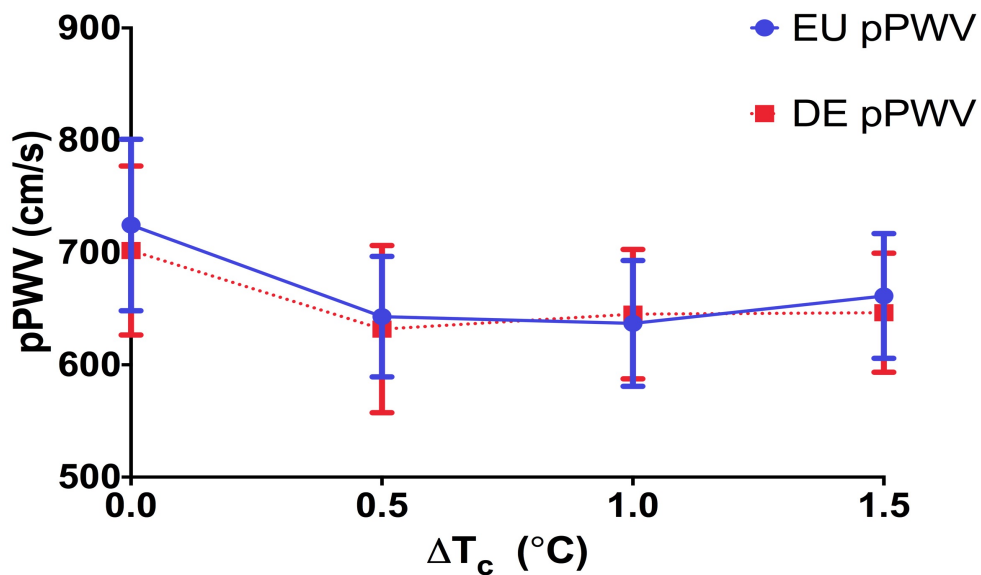


Figure 3. Relationship of change between baseline central pulse wave velocity (cPWV) and cPWV at 1.5°C core temperature increase. A significant negative relationship was observed between baseline arterial stiffness and changes during heating in euhydrated (EU, blue) cPWV ($r = -0.61$) but not dehydrated (DE, red) cPWV ($r = 0.09$)

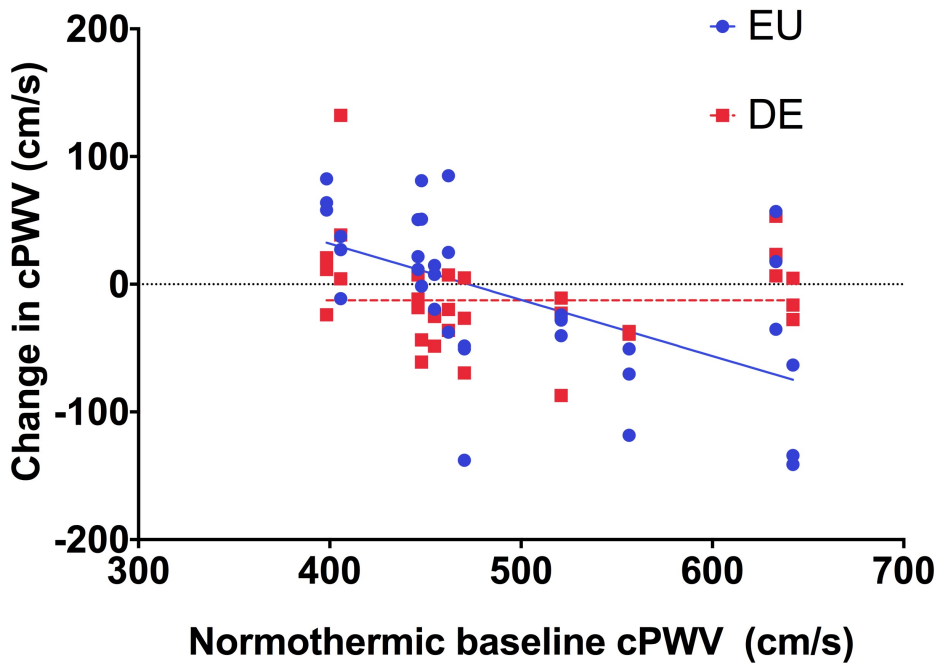


Figure 4. Relationship of change between baseline peripheral pulse wave velocity (pPWV) and pPWV at 1.5 °C core temperature increase. A significant negative relationship was observed between, euhydrated (EU, blue) pPWV ($r = -0.70$), and dehydrated (DE, red) pPWV ($r = -0.55$).

