Abstract

Previous evidence indicates that chronic consumption of dairy whey proteins has beneficial effects on CVD risk factors. The present study investigated the postprandial effects of whey protein isolate on blood pressure, vascular function and inflammatory markers in overweight and obese postmenopausal women. This was a randomised, three-way cross-over design study where twenty overweight and obese postmenopausal women consumed a breakfast meal in conjunction with one of three supplements: 45 g whey protein isolate, 45 g sodium caseinate or 45 g of a glucose control. Fasting and postprandial blood samples, blood pressure and pulse wave analysis readings were taken for up to 6 h. After consumption of the meal, both systolic and diastolic blood pressure, and augmentation index (AI) decreased initially for all interventions and gradually returned to baseline levels by 6 h. However, there were no significant differences in AI, systolic or diastolic blood pressure within or between the glucose control, casein or whey groups. There were also no significant group effects on plasma inflammatory markers (IL-6, TNF-α and C-reactive protein). The health effects previously seen with chronic whey protein ingestion were not seen in the acute 6 h postprandial period in relation to blood pressure, vascular function or inflammatory markers when compared with casein and a glucose control. This suggests that such effects are better observed from the long-term consumption of whey proteins.

Key words: Whey protein: Obesity: Overweight: Postmenopausal women: Postprandial: Blood pressure: Vascular function: Inflammatory markers

CVD is the leading cause of death in industrialised countries such as the USA and Australia(1,2). Risk factors for CVD include hypertension, high blood cholesterol, obesity, poor nutrition and smoking(3). A decrease in blood pressure (BP) of 2–5 mmHg in small populations results in a reduction in total mortality as a result of a lower prevalence of stroke and CHD(4). Arterial stiffness is an independent risk factor for CVD, particularly atherosclerosis, and its severity may increase with the presence of other CVD risk factors(5,6). Evidence has indicated correlations between arterial stiffness and CVD risk factors such as hypertension, hypercholesterolaemia and obesity(7). Chronic subclinical inflammation is also thought to influence the pathogenesis of atherosclerosis(8). Postmenopausal women are at a particularly high risk of developing CVD, primarily due to the cessation of oestrogen production which occurs during menopause(9,10). Oestrogen confers protection against CVD, as it is a potent stimulator of NO synthase, vascular endothelial growth factor and LDL-receptor activity. The common atherogenic risk profile observed in postmenopausal women includes increased total- and LDL-cholesterol levels, decreased HDL-cholesterol levels, elevated fasting apoB-containing lipoproteins, predominance of small, dense lipoproteins, elevated BP, endothelial dysfunction and insulin resistance(9,10).

Dietary interventions often form part of treatment strategies in dealing with CVD and its associated risk factors such as hypertension and arterial stiffening. Epidemiological studies have shown an inverse relationship between the incidence of stroke and the consumption of milk and milk products(11). The renin–angiotensin system is an important regulator of BP and is often the target of hypotensive medication. Therefore, drugs that inhibit the renin–angiotensin system, either by inhibiting angiotensin-converting enzyme (ACE) or by blocking angiotensin (AT1) receptors, are widely used in the treatment of hypertension(12). Recent evidence also indicates that the administration of ACE inhibitors may also have beneficial effects on inflammatory markers(13,14). Previous evidence indicates that dairy milk proteins inhibit ACE activity(15–18), with in vitro studies suggesting that dairy whey proteins, in particular, have an anti-hypertensive effect(15–17). A study by
Kawase et al.\(^{(16)}\) has shown that systolic blood pressure (SBP) was significantly reduced after ingestion of fermented milk enriched with whey proteins in healthy men after 8 weeks. Interestingly, a recent study by Lee et al.\(^{(19)}\) found that consumption of 125 ml of a milk drink daily which was supplemented with whey peptides, for 12 weeks, did not reduce BP and/or inflammation markers (IL-6 and C-reactive protein (CRP)) in mildly hypertensive subjects. However, they acknowledge that a higher dose of whey protein may have been required to see a measurable biological effect. A previous study in our laboratory has shown that chronic ingestion of 54 g of whey/d improved arterial stiffness measures when compared with casein and a glucose control and 54 g of either whey or casein reduced diastolic blood pressure (DBP) when compared with the control after 12 weeks in overweight and obese participants, suggesting that a higher dose of whey is required for observable effects\(^{(20)}\). However, it remains to be elucidated whether the beneficial effects of chronic whey consumption can be observed acutely in the overweight and obese.

Given that chronic consumption of whey proteins can affect BP, arterial stiffness and potentially inflammation, we hypothesise that these pathways would also be affected acutely in overweight and obese postmenopausal women, a population highly susceptible to CVD. Therefore, the aim of the present study was to investigate the postprandial effects of whey protein isolate on BP, vascular function and inflammatory markers compared with casein and glucose supplementation in overweight and obese postmenopausal women.

**Methods and procedure**

**Subjects**

Healthy, overweight and obese postmenopausal women with a BMI between 25 and \(40 \text{ kg/m}^2\), and aged between 40 and 65 years, were recruited from the community. There were a total of 125 respondents, of which seventy-seven respondents were suitable to participate in the study after screening and subsequently commenced the study. Menstrual status was assessed by measured plasma oestradiol levels, with menopause being defined as those with <10 pg/ml and amenorrhoea in the previous twelve consecutive months before the study. Exclusion criteria included regular medications (such as lipid lowering and hypotensives) that were likely to affect the study outcomes, hormone replacement therapy, cancer within the last 5 years, smoking, major illnesses, eating disorders, over two standard alcoholic drinks per day, over two standard alcoholic drinks per day for 24 h before each intervention day and were instructed to consume a standardised evening meal supplied frozen before each visit. On each intervention day, the subjects attended at 08.00 hours at the Health Clinic at Curtin University after a 12–14 h fast. An indwelling catheter was placed in the antecubital fossa or in a suitable vein in the forearm. Fasting blood samples and measures of BP and augmentation index (AI) were then obtained. Subjects were randomised to consume one of three supplements: 45 g whey protein isolate, 45 g sodium caseinate or 45 g of a glucose control (provided by MG Nutritional, Brunswick, VIC, Australia) mixed with water and consumed with a breakfast meal. The composition of the protein supplements is shown in Table 1. Subsequent 10 ml of blood samples were then taken at 15, 30, 60, 90, 120, 180, 240 and 360 min, and BP and AI readings were taken at 30, 60, 90, 120, 180, 240 and 360 min after consumption of the test meal. Based on our previous experience, greater than 4 h is required to measure a complete postprandial response of inflammatory markers\(^{(22)}\). Subjects remained in the semi-recumbent position during the intervention day and could read, listen to music or do puzzles. They were encouraged to drink water to maintain hydration throughout the day. The catheter was removed after the final blood sample, and the subjects were given something to eat.

**Dietary protocol and test meal**

All study subjects were instructed not to take any vitamins or minerals, or other supplements and to minimise dairy intake to 1 serving/d, for 4 weeks leading up to, and during the study period, to limit confounding factors. They were provided with instructions as to what constitutes a serving of dairy. The frozen meal consumed on the evening before each intervention day (McCain’s Roast Turkey and Vegetable) contained 1245 kJ, which comprised 26 % protein, 59 % carbohydrates and 15 % fat. The test meal consisted of a 50 g bread roll with 45 g of dairy-free, rapeseed margarine and 10 g of Vegemite or Promite plus whey protein isolate or sodium caseinate supplements or the glucose control (Table 2). All supplements were mixed with 400 ml water and had equal energy content at 875 kJ. The bread roll plus spreads and the supplement were consumed together within 15 min. Subsequent measurements were timed from the subject’s completion of the test meal. The supplements were served chilled.
in dark containers and were matched for appearance, sweetness and palatability with sucralose and flavouring. The total energy content of the complete test meal was 2587 kJ and fat intake was 45% of total energy intake. The whey- and casein-supplemented meals contained 38.3% energy from protein and 16.7% from carbohydrates, and the glucose-supplemented meal contained 5.2% energy from protein and 49.8% from carbohydrates. Data were analysed using FoodWorks 2007 (Xyris Software, Highgate Hill, QLD, Australia) based on the Australian food composition tables.

Measurement of blood pressure and vascular function

BP was measured with an automated, calibrated sphygmomanometer (Dinamap, Compact T; Critikon, Norderstedt, Germany), with subjects in a supine position after resting for at least 10 min. The measurements were taken on the same arm three times at 1 min intervals. These readings were then averaged.

Vascular measurements were performed by using the non-invasive SphygmoCor system (AtCor Medical Private Limited, Sydney, NSW, Australia). This system has been validated in a population at risk of CVD and has been used previously in our laboratory (23–25). The SphygmoCor system produces a waveform analysis based on measuring the aortic root pressure wave, and is represented by the AI. All measurements were performed by the same operator. These measurements were performed by the same operator. These measurements were then averaged.

Measurement of plasma inflammatory markers

Blood samples (10 ml) were taken to measure circulating CRP, IL-6 and TNF-α. Serum was isolated by centrifugation at 3000 rpm at 4°C for 10 min and stored at −80°C until the end of the study. Plasma IL-6, TNF-α and CRP levels were measured by a solid-phase enzyme-amplified sensitivity immunocassay performed on microtitre plates according to the manufacturer’s instructions (TNF-α ELISA and IL-6 ELISA kits: BioSource, Nivelles, Belgium; CRP kits: Alpha Diagnostics International, San Antonio, TX, USA). These are established methods in our laboratory (20,22).

Statistical analysis

Sample size calculations were based on a minimum predicted 15% change in area under the curve (AUC) for BP, AI and inflammatory markers between the intervention and control groups, with an expected standard deviation of 15%. A sample size of twenty participants per group was predicted to provide sufficient power (80%) to detect significant changes at the 5% significance level. However, we aimed to recruit twenty-five participants to accommodate for a 20% attrition rate.

All subjects who completed the study were included in the data analysis. Statistical analysis was conducted using SPSS 17 for Windows (SPSS, Inc., Chicago, IL, USA). Data are expressed as means with their standard errors and assessed for normality. Differences between groups were tested by ANOVA. Postprandial changes in SBP, DBP, AI, TNF-α, IL-6 and CRP were tested by ANOVA for repeated measures, taking into account the group and time effect, followed by Tukey’s post hoc test. Significant changes for each time point t: baseline values were tested by paired t tests. The incremental AUC was determined by subtracting the area below the baseline concentration from the area under the plasma curve between 0 and 6 h. The AUC represents the increase in area following the consumption of a fat load, corrected for fasting concentration. The AUC data were analysed using a general linear model to assess the effects of the groups after adjusting for the following covariates: age and baseline values, with post hoc comparisons made using Tukey’s test. An α level of P<0.05 was considered statistically significant for all analysis.

This clinical trial has been registered with the Australian New Zealand Clinical Trials Registry. The registration number is ACTRN12609000178246, and the trial web address is http://www.anzctr.org.au/trial_view.aspx?ID=83681

Results

Subject characteristics

A total of twenty subjects completed the study, with seven subjects being withdrawn due to unsuitable vein integrity for
an indwelling catheter. The results were reported on those that completed the study. Table 3 shows the mean and range of clinical characteristics of the subjects.

### Nutritional assessment

The mean 3 d dietary intake of subjects before each visit is shown in Table 4. There were no significant differences in energy, fibre and macronutrient intake.

### Blood pressure, vascular function and inflammatory markers

Postprandial SBP, DBP, AI and various inflammatory markers are shown in Fig. 1. There was no significant difference in fasting BP, vascular function or inflammatory markers between the three groups (Fig. 1; Table 5). After consumption of the meal, SBP, DBP and AI decreased initially for all interventions and gradually returned to baseline levels by 6 h (Fig. 1(a)–(c)). Postprandial SBP, DBP and AI followed a similar pattern for each intervention and were not significantly different between groups at any time point.

The inflammatory marker IL-6 increased for 6 h postprandially (Fig. 1(d)), but there was no significant group effect. However, IL-6 was significantly higher than baseline in all three groups at 3, 4 and 6 h postprandially (P<0.05). There were no significant group effects on plasma TNF-α and CRP concentrations (Fig. 1(e) and (f)). There was also no significant change within groups in TNF-α and CRP concentrations over the 6 h following the meal.

There was no significant difference in AUC for SBP, DBP or AI between the control, casein or whey groups (Table 6). There was also no difference in AUC for the inflammatory markers IL-6, CRP and TNF-α between the groups.

### Discussion

The present study demonstrated that the ingestion of 45 g whey protein isolate did not reduce BP, arterial stiffness or inflammatory markers for up to 6 h postprandially when compared with 45 g casein and 45 g of a glucose control in overweight and obese postmenopausal women. The hypotensive effects and improvement in measures of arterial stiffness from whey ingestion observed in previous feeding studies indicate that such effects are observed from intake over a longer time period.

Previous research has demonstrated that milk proteins contain peptides which inhibit ACE activity, a key enzyme in the regulation of peripheral BP. Casokinins are casein-derived inhibitors of ACE, and lactokinins are whey-derived inhibitors. Previous studies in hypertensive rats have indicated that casokinins and lactokinins can significantly reduce BP, with decreases in SBP ranging from 2 to 34 mmHg. Most of the clinical studies have examined the hypotensive effects of casokinins, with studies showing a reduction of SBP and DBP after consumption of casein and fermented milk. In addition, a recent study in our laboratory has revealed that consumption of 54 g whey or 54 g casein/d reduced DBP in overweight individuals after 12 weeks. However, there is still uncertainty as to the relative significance of reducing diastolic BP. In contrast, the study by Lee et al. has found that the daily consumption of 125 ml of a milk drink containing whey peptides (2·6 g) over 12 weeks...
did not reduce BP compared with a lactose control drink, suggesting that higher doses as used in our previous studies are required to observe BP reductions. They have proposed that the lack of an in vivo effect could be due to the degradation of the peptides by intestinal or plasma peptidases before they could exert an effect on BP or there was an insufficient reabsorption of the peptides in the milk drink. However, they have suggested that a greater dose of whey may have been required to exert a observed biological effect (19). Despite the previous evidence indicating the hypotensive effects of whey and casein in our laboratory (20), there were no changes in BP postprandially over 6 h after consuming whey and casein when compared with a glucose control in the present study, even with a high dose of 45 g. This may indicate that the effect of whey on BP may need to be undertaken over a longer period of time to see any effect and that a one-off dose of whey protein is inadequate to regulate or inhibit ACE activity.

Arterial stiffening is an independent risk factor for CVD (31) and is associated with ageing, as the central arteries gradually stiffen over time (32). The rate of progression is influenced by hypertension, diabetes and atherosclerosis (33). Arterial stiffness did not reduce BP compared with a lactose control drink, suggesting that higher doses as used in our previous studies are required to observe BP reductions. They have proposed that the lack of an in vivo effect could be due to the degradation of the peptides by intestinal or plasma peptidases before they could exert an effect on BP or there was an insufficient reabsorption of the peptides in the milk drink. However, they have suggested that a greater dose of whey may have been required to exert a observed biological effect (19). Despite the previous evidence indicating the hypotensive effects of whey and casein in our laboratory (20), there were no changes in BP postprandially over 6 h after consuming whey and casein when compared with a glucose control in the present study, even with a high dose of 45 g. This may indicate that the effect of whey on BP may need to be undertaken over a longer period of time to see any effect and that a one-off dose of whey protein is inadequate to regulate or inhibit ACE activity.

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Table 5. Fasting blood and vascular measurements in each group

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Casein</th>
<th>Whey</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean</strong></td>
<td><strong>SEM</strong></td>
<td><strong>Mean</strong></td>
<td><strong>SEM</strong></td>
</tr>
<tr>
<td>Fasting TNF (pg/ml)</td>
<td>2.67±0.23</td>
<td>2.63±0.24</td>
<td>2.55±0.23</td>
</tr>
<tr>
<td>Fasting IL-6 (pg/ml)</td>
<td>1.21±0.13</td>
<td>1.24±0.15</td>
<td>1.23±0.12</td>
</tr>
<tr>
<td>CRP (mg/l)</td>
<td>1.21±0.13</td>
<td>1.30±0.16</td>
<td>1.34±0.18</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>119.32±3.91</td>
<td>117.32±3.14</td>
<td>118.34±3.45</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>68.40±1.89</td>
<td>67.08±1.16</td>
<td>68.94±1.76</td>
</tr>
<tr>
<td>Augmentation index (%)</td>
<td>35.05±1.79</td>
<td>35.42±1.72</td>
<td>35.30±2.14</td>
</tr>
</tbody>
</table>

CRP, C-reactive protein; SBP, systolic blood pressure; DBP, diastolic blood pressure.
can be assessed using pulse wave analysis, a non-invasive method that indirectly measures arterial stiffness using radial
applanation tonometry\(^\text{57}\). This is represented by the AI. It has been proposed that an increase in arterial stiffening may be
associated with increases in SBP and DBP\(^\text{57,54–56}\). While mechanisms of age- and disease-related arterial stiffness are not fully understood, previous studies have indicated that components of the renin–angiotensin system, matrix metallo-
proteinases, intracellular signalling and extracellular matrix components may be involved in the process\(^\text{54–56}\). Given
the potential effects of dairy protein on the angiotensin system\(^\text{28–30,37}\), it could be expected that dairy proteins may also have a beneficial effect on AI. A previous study in our laboratory found that supplementation with 54 g of whey protein a day for 12 weeks resulted in a 21% decrease in AI compared with the control group and a 23% decrease when compared with the casein group in overweight and obese individuals\(^\text{20}\).

The mechanism for the effect of whey on AI was not known, but it was proposed that differences between whey and casein
digestion and absorption may provide an insight. Whey protein consists of a heterogeneous group of proteins containing lactalbumin, \(\beta\)-lactoglobulin, immunoglobulins and lacto-
peroxidase. Whey generally also has a higher content of branched-chain amino acids (such as leucine, isoleucine and
valine) when compared with casein, which is thought to be
responsible for its efficient metabolism after consumption\(^\text{38}\). The greater content of amino acids, in particular branched-chain
amino acids, after whey consumption, and peptide fractions or the synergistic actions between them, may explain the
physiological effects of whey. In the present acute study, however, there was no decrease in AI postprandially after
consumption of whey when compared with casein and control. This may indicate that consumption of whey would
need to be prolonged to see a reduction in AI and therefore
arterial stiffening. In our previous feeding study, a significant
reduction in AI was only observed after 12 weeks of whey consumption but not after 6 weeks\(^\text{20}\). In contrast to the pre-
cent study, Ballard et al\(^\text{40}\) found that consumption of 5 g/d of a novel whey-derived peptide (NOP-47) after 2 weeks resulted in no change in vascular function in healthy individuals; interestingly, however, 5 g of the whey-derived peptide improved vascular function postprandially when compared with a
placebo. They suggested that ACE-inhibitory activity of the
novel whey peptide may have been responsible, as previous
clinical trials had shown that patients taking ACE inhibitors
had resulted in improvements in vascular function\(^\text{40}\). The
effects of whey protein components such as novel whey-
derived peptides should be further investigated in both the
longer feeding and acute settings.

Chronic inflammation is thought to play an important role in
the pathogenesis of atherosclerosis\(^\text{41}\). We have previously
demonstrated that the type of fat in a meal can regulate the
concentration of inflammatory cytokines, CRP, TNF-\(\alpha\) and
IL-6 postprandially\(^\text{42}\). It has been shown that ACE inhibitors
can have beneficial effects on inflammatory markers\(^\text{13,14}\),
CRP, IL-6 and TNF-\(\alpha\), so the potential ACE-inhibitory activity of
dairy proteins was investigated postprandially. The present study revealed that CRP, IL-6 and TNF-\(\alpha\) levels were not
influenced by whey or casein supplementation postpran-
dially. These findings are consistent with previous feeding
studies examining whey and milk peptides on inflammatory
markers\(^\text{10,20,40}\).

It is possible that the lack of the effect of whey protein in
the present study on BP, vascular function or inflammatory
markers may be related to the confounding effects of the test
meal. Consuming a test meal with the supplements may have
slowed down gastric emptying, which could have delayed or
inactivated bioactive components once they reached the small
intestine. This may have attenuated the beneficial effects of
whey on BP and vascular function in the present study. Future
research could investigate these effects after consumption of
the supplements only, thereby limiting possible confounding
effects from the meal.

In conclusion, the present study demonstrated that consump-
tion of 45 g whey protein isolate did not alter BP, vascular
function or inflammatory markers in a 6 h postprandial

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### Table 6. Effect of the test meal on postprandial blood pressure, vascular function and inflammatory markers

(Mean values with their standard errors)

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Casein</th>
<th>Whey</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>703±47</td>
<td>14±1</td>
<td>704±15</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>389±67</td>
<td>6±2</td>
<td>388±37</td>
</tr>
<tr>
<td>Augmentation index (%)</td>
<td>180±33</td>
<td>5±3</td>
<td>172±32</td>
</tr>
<tr>
<td>CRP (mg/l)</td>
<td>14±6</td>
<td>2±4</td>
<td>17±34</td>
</tr>
<tr>
<td>IL-6 (pg/ml)</td>
<td>116±70</td>
<td>12±47</td>
<td>126±50</td>
</tr>
<tr>
<td>TNF-(\alpha) (pg/ml)</td>
<td>22±25</td>
<td>3±08</td>
<td>16±49</td>
</tr>
</tbody>
</table>

**SBP, systolic blood pressure; DBP, diastolic blood pressure; CRP, C-reactive protein.**

\(\ast\) Data are expressed as postprandial incremental area under the curve.
period when compared with casein and a glucose control in overweight and obese postmenopausal women. It is difficult to speculate whether the same effects would have been observed in men of the same BMI. These trials need to be conducted in future studies. Previous feeding studies have demonstrated the hypertensive effects of whey and casein and improvements in arterial stiffness after whey consumption, suggesting that intake over a longer time is required. However, the potential of whey in improving CVD risk factors such as BP and arterial stiffness should be investigated further, exploring the effects of whey postprandially with higher doses of whey than the 45 g used in the present study. Further investigations into novel whey peptides or studies in a population group that is hypertensive are also recommended. Thus, there is a need for future research investigating the potential benefits of both acute and chronic whey protein ingestion on metabolic risk factors.

Acknowledgements
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References


