Journal of Human Hypertension (2007) 21, 297–306

www.nature.com/jhh

ORIGINAL ARTICLE

Coenzyme Q_{10} in the treatment of hypertension: a meta-analysis of the clinical trials

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Our objective was to review all published trials of coenzyme Q_{10} for hypertension, assess overall efficacy and consistency of therapeutic action and side effect incidence. Meta-analysis was performed in 12 clinical trials (362 patients) comprising three randomized controlled trials, one crossover study and eight open label studies. In the randomized controlled trials ($n=120$), systolic blood pressure in the treatment group was 167.7 (95% confidence interval, CI: 163.7–171.1) mm Hg before, and 151.1 (147.1–155.1) mm Hg after treatment, a decrease of 16.6 (12.6–20.6, $P<0.001$) mm Hg, with no significant change in the placebo group. Diastolic blood pressure in the treatment group was 103 (101–105) mm Hg before, and 94.8 (92.8–96.8) mm Hg after treatment, a decrease of 8.2 (6.2–10.2, $P<0.001$) mm Hg, with no significant change in the placebo group. In the crossover study ($n=18$), systolic blood pressure decreased by 11 mm Hg and diastolic blood pressure by 8 mm Hg ($P<0.001$) with no significant change with placebo. In the open label studies ($n=214$), mean systolic blood pressure was 162 (158.4–165.7) mm Hg before, and 148.6 (145–152.2) mm Hg after treatment, a decrease of 13.5 (9.8–17.1, $P<0.001$) mm Hg. Mean diastolic blood pressure was 97.1 (95.2–99.1) mm Hg before, and 86.8 (84.9–88.8) mm Hg after treatment, a decrease of 10.3 (8.4–12.3, $P<0.001$) mm Hg. We conclude that coenzyme Q_{10} has the potential in hypertensive patients to lower systolic blood pressure by up to 17 mm Hg and diastolic blood pressure by up to 10 mm Hg without significant side effects.
Journal of Human Hypertension (2007) **21,** 297–306. doi:10.1038/sj.jhh.1002138; published online 8 February 2007

Keywords: Coenzyme Q_{10}; blood pressure; therapeutics; meta-analysis

Introduction

Hypertension represents an increasing global burden of disease.[1] It is currently managed by the use of a variety of medications. These medications are effective in reducing blood pressure, but many have undesirable side effects such as renal or cardiac dysfunction, cough and depression. Coenzyme Q_{10} (CoQ_{10}), also known as ubiquinone because of its ubiquitous distribution in nature, is an antioxidant and an integral component of the mitochondrial respiratory chain for energy production.[2] It is found in all tissues and organs of the body but in highest concentration in the heart. Blood and tissue levels of CoQ_{10} are reduced by aging and cardiovascular disease.[3] There is evidence of CoQ_{10} deficiency in hypertension,[4] heart failure[5] and in statin-treated hypercholesterolemic individuals.[6] Since 1975, many studies have described the potential of CoQ_{10} to lower blood pressure in hypertensive patients. Few side effects have been reported even with high doses of CoQ_{10}. In a study of subjects with type II diabetes, we showed that CoQ_{10} therapy lowered blood pressure and improved glycemic control.[7] However, despite these reports, the current role of CoQ_{10}, if any, in the treatment of hypertension is unclear. We therefore set out to review the published clinical trials of CoQ_{10} in the management of hypertension in terms of its therapeutic effect and side effect profile.

The aims of the present study were to (1) review all published trials of the use of CoQ_{10} in the treatment of hypertension, (2) assess the overall efficacy and consistency of the therapeutic effect and (3) assess the incidence of side effects.

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Received 22 May 2006; revised 6 October 2006; accepted 14 November 2006; published online 8 February 2007

Methods

Careful searching of the literature was performed to ensure complete coverage of all available publications and minimization of publication bias.[8,9] We used an Evidence Based Medicine (EBM) review including Cochrane Database of Systematic Reviews, American College of Physicians Journal Club, Database of Abstracts of Reviews of Effects, Cochrane Central Register of Controlled Trials, Medline search (1966–2005), EMBASE and PubMed (1966–2005) to identify clinical trials of CoQ_{10} in the therapy of hypertension. Bibliographies of all retrieved articles were consulted for additional publications. Each search strategy included keywords related to trial design (double blind, random allocation, randomized, clinical trial, placebo controlled), which were then cross-linked with terms referring to CoQ_{10} (coenzyme Q_{10}, ubiquinone) and search terms of interest, including hypertension and blood pressure.

A meta-analysis was performed by pooling results in the STATA v.8.2 software[10] with the Cohen method of meta-analysis for weighted mean difference with respect to continuous variables and presented as effect size with 95% confidence limits. STATA assigns relative weights to each individual study according to the contribution of each study to the meta-analysis performed[8,11] on the basis of the size of the treatment group and the precision of the confidence interval (CI). For internal consistency, we used the same weighting technique (inverse variance) for calculating the before and after treatment mean values as were used to calculate the weighted mean differences shown in the Forest plots. Tests of heterogeneity were performed. Statistical significance was set at the 0.05 level on the basis of two-way *Z*-tests. Values are given as mean and 95% CIs unless otherwise stated.

Results

The reasons for inclusion and exclusion of studies from consideration in this meta-analysis, in accordance with the QUOROM statement,[12] are shown in Figure 1. We identified 12 studies that reported a total of 362 patients in which CoQ_{10} had been used in the therapy of hypertension.[4,13–23] These comprised three randomized controlled clinical trials,[20–22] one crossover study[23] (Table 1) and eight open label observational studies without a control group[4,13–19] (Table 2). Owing to the wide differences in methodology and presentation of the results in these three groups of trials, we elected to analyse the results separately (Table 3).

Randomized controlled trials

The mean systolic blood pressure in the pooled treatment group was 167.7 mm Hg (95% CI: 163.7–171.1 mm Hg) before treatment and 151.1 mm Hg (95% CI: 147.1–155.1 mm Hg) after treatment, with a mean decrease of 16.6 mm Hg (95% CI: 12.6–20.6 mm Hg, $P<0.001$). The mean systolic blood pressure in the pooled placebo group was 166 mm Hg (95% CI: 162.1–170.0 mm Hg) before treatment and 163.9 mm Hg (95% CI: 159.9–167.9 mm Hg) after treatment, with a mean decrease of 2.1 mm Hg (95% CI: 6.1 mm Hg decrease to 1.9 mm Hg increase, $P=0.295$) (Figure 2, Table 1).

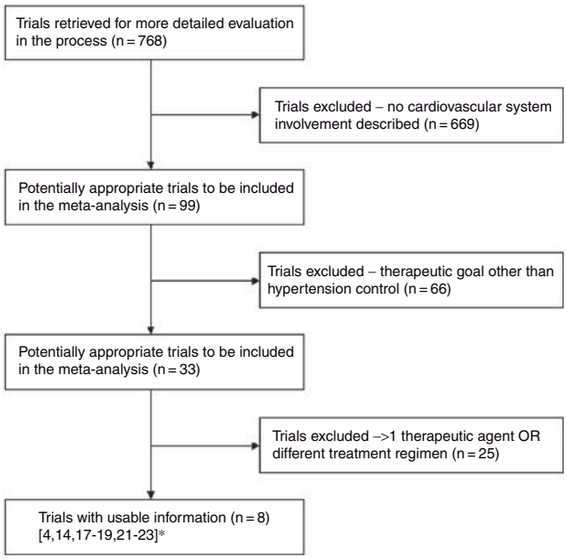

Figure 1 Flow diagram of exclusions for trials under consideration for meta-analysis.

The mean diastolic blood pressure in the pooled treatment group was 103 mm Hg (95% CI: 101–105 mm Hg) before treatment and 94.8 mm Hg (95% CI: 92.8–96.8 mm Hg) after treatment, with a mean decrease of 8.2 mm Hg (95% CI: 6.2–10.2 mm Hg, $P<0.001$). The mean diastolic blood pressure in the pooled placebo group was 102.5 mm Hg (95% CI: 100.2–104.7 mm Hg) before treatment and 100.5 mm Hg (95% CI: 98.3–102.8 mm Hg) after treatment, with a mean decrease of 1.9 mm Hg (95% CI: 4.2 mm Hg decrease to 0.3 mm Hg increase, $P=0.094$) (Figure 3, Table 1).

Crossover study

There was one crossover study with 18 patients. In the treatment phase, the mean systolic blood pressure was 167 ± 2.6 (mean $\pm$ s.e.m.) mm Hg before treatment and 156 ± 2.3 mm Hg after treatment, with a mean decrease of 11 mm Hg ($P<0.001$). The mean diastolic blood pressure was 103 ± 1.2 mm Hg before treatment and 95 ± 1.0 mm Hg after treatment, with a mean decrease of 8 mm Hg ($P<0.001$). During the placebo phase of the crossover study, systolic blood pressure was 166 ± 2.4 mm Hg and diastolic blood

Table 1 Randomized controlled trials and crossover study

Randomized controlled trials	N	*Patient characteristics (age/% male)*	*Anti-HT treatment?*	*CoQ_{10} dose (mg/day)*	*Period (weeks)*	*Treatment/ placebo*	*SBP (mm Hg)*			*DBP (mm Hg)*		
							Baseline	*Final*	*△SBP*	*Baseline*	*Final*	*△DBP*
Yamagami, 1986[20]	20	Essential HT, low Q_{10}, SDH-Q (60.4/40)[a]	No	100	12	Treatment – $N=10$	167 (161.9–172.1)	148 (139.4–156.6)	−19	97 (93.5–100.5)	91 (83.8–98.3)	−6
						Placebo – $N=10$	168 (158.6–177.4)	164 (152.8–175.2)	−4	96 (92.7–99.4)	93 (83.8–102.2)	−3
Singh, 1999[21]	59	HT and CAD (48.2/88)[b]	Yes	120	8	Treatment – $N=30$	168 (164.6–171.4)	152 (149.1–154.9)	−16	106 (104.3–107.7)	97 (95.5–98.5)	−9
						Placebo – $N=29$	166 (162.9–169.1)	164 (161–167)	−2	105 (103.3–106.8)	103 (101.3–104.7)	−2
Burke, 2001[22]	41	Isolated systolic HT (68.0/55)[c]	Ceased	120	12	Treatment – $N=23$	165.1 (155.5–174.7)	147.3 (132.4–162.2)	−17.8	80.9 (77.8–84.1)	78.3 (72.6–84)	−2.6
						Placebo – $N=28$	164.4 (154.2–174.6)	162.7 (151.3–174.1)	−1.7	82 (77.7–86.3)	81.5 (75.4–87.6)	−0.5
Crossover study												
Digiesi, 1990[23]	18	Essential HT (55.9/78)	Ceased	100	10[d]	Treatment	167±2.6	156±2.3	−11	103±1.2	95±1.0	−8
						Placebo		166±2.4			103±1.0	

Abbreviations: DBP, diastolic blood pressure; HT, hypertension; SBP, systolic blood pressure; SDH-Q, succinate dehydrogenase – CoQ_{10} reductase activity.
[a]Mean age of 59.5 years for treatment group and mean age of 61.3 years for placebo group.
[b]Mean age of 48.3 years for treatment group and mean age of 48.0 years for placebo group.
[c]Mean age of 69.7 years for treatment group and mean age of 67.3 years for placebo group.
[d]Two weeks washout between treatments in this crossover study.

Table 2 Open label observational studies of Coenzyme Q_{10} in hypertension

	N	*Patient characteristics (age/% male)*	Anti-HT treatment?	*CoQ10 dose (mg/day)*	*Period (weeks)*	*SBP (mm Hg)*			*DBP (mm Hg)*		
						Baseline	*Final*	*△SBP*	*Baseline*	*Final*	*△DBP*
Yamagami, 1975[4]	4	Essential HT (58.3/75)	No	33.8±7.5 (s.d.); 30 mg (median)	4–16	185.5 (174.0–197.0)	166.1 (151.3–180.9)	−19.4	101.8 (90.9–112.7)	94.8 (86.0–103.6)	−7
Yamagami, 1976[13]	5	Essential HT, low SDH-Q (54.4/60)	No	57.0±24.6 (s.d.); 75 mg (median)	1–20	180 (171.1–188.9)	164 (143.8–184.3)	−16	108 (95.6–120.4)	101.2 (82.1–120.3)	−6.8
Yamagami, 1977[14]	29	Essential HT (56.0/N/A)[a]	No	1–2 mg/kg	8–12	188 (180.9–195.1)	176 (167.2–184.8)	−12	104 (98.7–109.3)	98 (92.3–103.7)	−6
Folkers, 1981[15]	16	HT (55.9/69)	Yes: 10No: 6	60	8–16	166.5 (154.0–179.0)	145.4 (135.2–155.6)	−21.1	98.4 (91.4–105.4)	82.4 (72.7–92.2)	−16
Montaldo, 1991[16]	15	Borderline HT (48.0/N/A)	No	100	12	148.4 (143.1–153.7)	137.9 (133.2–142.6)	−10.5	98 (93.2–102.8)	91 (87.4–94.6)	−7
Digiesi, 1992[17]	10	Essential HT (61.5/50)	Ceased	100	10	161.5 (151.5–171.5)	142.2 (131.8–152.6)	−19.3	98.5 (95.2–101.9)	83.1 (79.2–87.0)	−15.4
Digiesi, 1994[18]	26	Essential HT (61.3/50)	Ceased	100	10	164.5 (158.4–170.6)	146.7 (138.7–154.7)	−17.8	98.1 (94.8–101.4)	86.1 (83.6–88.6)	−12
Langsjoen, 1994[19]	109	Essential HT (62.1/46)[b]	Yes	75–360; 225 mg (mean)	56	159.2 (154.4–164.0)	147.8 (143.8–151.8)	11.4[b]	94.4 (92.1–96.8)	85.4 (83.5–87.3)	−9[b]

Abbreviations: DBP, diastolic blood pressure; HT, hypertension; SBP, systolic blood pressure; SDH-Q, succinate dehydrogenase – CoQ_{10} reductase activity.
[a]Age details only provided for all 64 cases that were considered for the study.
[b]In addition, more than 50% of subjects ceased taking at least one of their antihypertensive medications.

Table 3 Summary of trials as groups

	Studies (n)	*Patients* (n)	*SBP (mm Hg)*			*DBP (mmHg)*		
			Before	*After*	*Difference*	*Before*	*After*	*Difference*
Randomized controlled trials								
Treatment group	3	63	167.7 (163.7 to 171.1)	151.1 (147.1 to 155.1)	−16.6 (−20.6 to −12.6)	103 (101.0 to 105.0)	94.8 (92.8 to 96.8)	−8.2 (−10.2 to −6.2)
Control group		57	166 (162.1 to 170.0)	163.9 (159.9 to 167.9)	−2.1 (−6.1 to 1.9)	102.5 (100.2 to 104.7)	100.5 (98.3 to 102.8)	−1.9 (−4.2 to 0.3)
Crossover study								
Treatment phase	1	18	167±2.6	156±2.3	−11	103±1.2	95±1.0	−8
Control phase				166±2.4			103±1.0	
Open label studies	8	214	162 (158.4 to 165.7)	148.6 (145.0 to 152.2)	−13.5 (−17.1 to −9.8)	97.1 (95.2 to 99.1)	86.8 (84.9 to 88.8)	−10.3 (−12.3 to −8.4)

DBP, diastolic blood pressure; SBP, systolic blood pressure.

pressure was 103±1.0 mm Hg – both readings were not significantly different to baseline (Table 1).

Open label observational studies
The mean systolic blood pressure was 162 mm Hg (95% CI: 158.4–165.7 mm Hg) before treatment and 148.6 mm Hg (95% CI: 145–152.2 mm Hg) after treatment, with a weighted mean decrease of 13.5 mm Hg (95% CI: 9.8–17.1 mm Hg, $P<0.001$). The mean diastolic blood pressure was 97.1 mm Hg (95% CI: 95.2–99.1 mm Hg) before treatment and 86.8 mm Hg (95% CI: 84.9–88.8 mm Hg) after treatment, with a mean decrease of 10.3 mm Hg (95% CI: 8.4–12.3 mm Hg, $P<0.001$), (Figure 4, Table 2).

Overall effects
All three types of study (randomized controlled, crossover and open label) showed decreases in systolic blood pressure ranging from 11 to 17 mm Hg and in diastolic blood pressure ranging from 8 to 10 mm Hg (Table 3).

Side effects
In all 12 studies, side effects were minimal. One trial reported a 37% occurrence of gastrointestinal side effects in 30 CoQ_{10} treated patients, but this was not significantly different to the rate in the placebo group (21%; $P=0.29$).[21] Another trial reported a 13% incidence of side effects, three out of 23 treated patients, one each with nausea, flatulence and headache.[22] Two further trials reported an absence of side effects,[15,23] whereas the remaining eight trials did not comment on side effects.

Discussion

This meta-analysis encompassed all published studies of CoQ_{10} in the treatment of hypertension and found a blood pressure lowering effect across all studies. There was also a similar consistency in the absence of serious adverse effects of therapy. In nine studies, anti-hypertensive medication was not in use or was ceased 2 weeks before commencement of CoQ_{10} therapy. In three studies, CoQ_{10} was given in addition to the existing anti-hypertensive medication and in one of these more than 50% of patients were able to cease taking at least one of their anti-hypertensive medications during the trial.

The prospective randomized studies and the crossover study showed similar results. Among treated patients, decreases in systolic blood pressure ranged from 11 mm Hg in the crossover study to 17 mm Hg in the group of randomized studies (Table 3). A decrease of 8 mm Hg in diastolic blood pressure was observed in the treated patients of both the crossover study and in the randomized trials (Table 3).

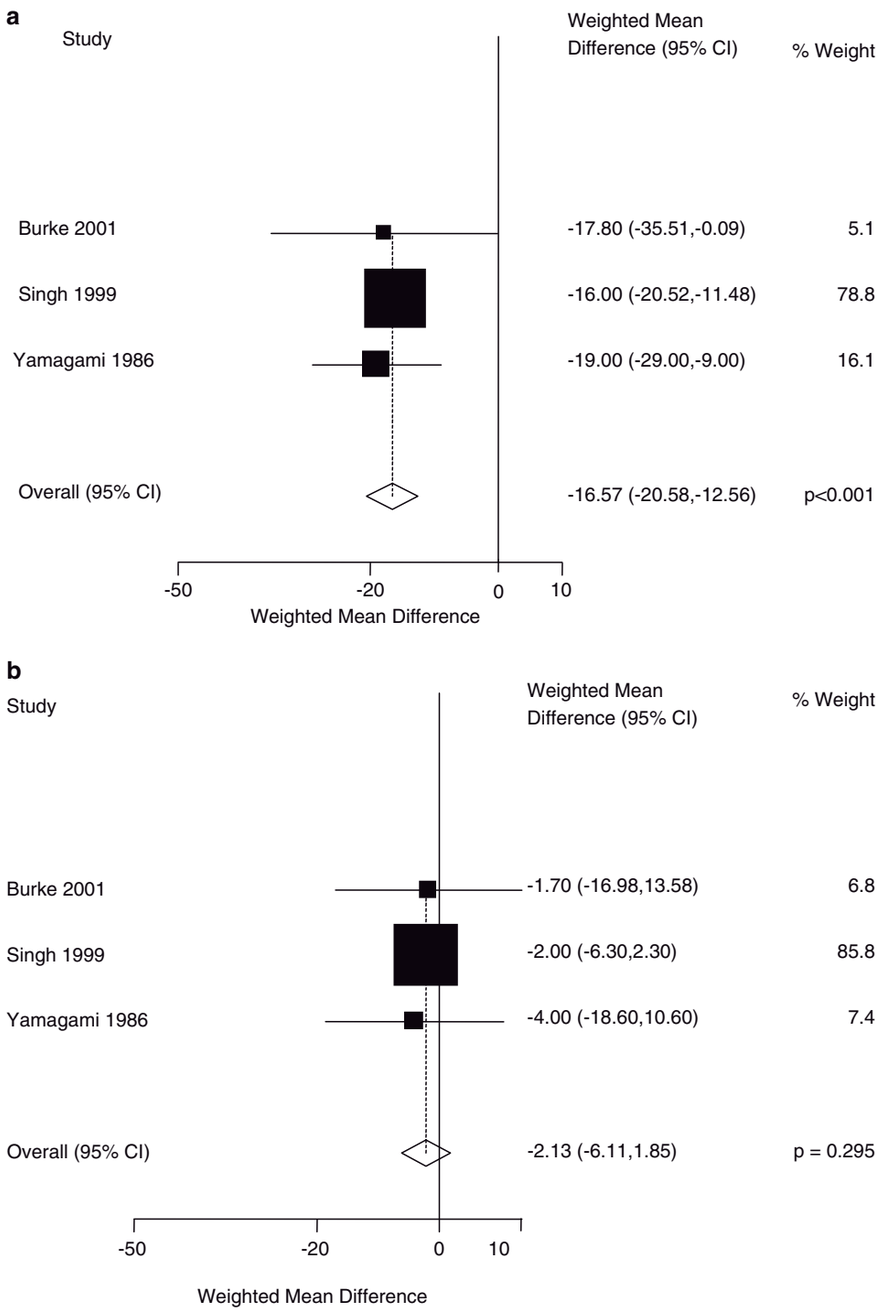

Figure 2 Randomized controlled trials, systolic blood pressure weighted mean differences from before to after – treatment group (**a**), placebo group (**b**).

The open label observational studies, of which the largest study[19] comprised 109 patients, were before-and-after studies without a placebo control group and included the earliest studies dating from 1975. All these studies showed significant decreases in blood pressure ranging from 10 to 21 mm Hg systolic and from 7 to 16 mm Hg diastolic that were similar in magnitude to the randomized studies. These represent credible therapeutic effects given that the placebo group in the randomized studies showed decreases in systolic blood pressure of only 1–4 mm Hg systolic and 0–3 mm Hg diastolic.

CoQ_{10} dosage

Doses used in the trials reported here varied from 34 mg/day in the early trials to 225 mg/day in the later ones. In the largest study, control of blood

pressure was said to come about gradually over several months and required varying doses in different patients (75–360 mg/day) to attain what was considered a therapeutic blood level of CoQ_{10} ($>2.0\,\mu g/ml$).[19] This is a common experience for those using CoQ_{10} therapy for a variety of therapeutic purposes. It is always desirable to monitor CoQ_{10} levels in the blood to guide dosage, as baseline levels are variable, absorption of CoQ_{10} varies with the preparation used[24] and variability in patient response also exists.

Another factor complicating CoQ_{10} therapy in the current era is the widespread use of statins to lower serum cholesterol. Statins inhibit the synthesis not only of cholesterol but also of CoQ_{10} because both substances share the mevalonate synthetic pathway beginning with acetyl-CoA and ending with cholesterol, CoQ_{10} and dolichol. Statin therapy has been shown to lower CoQ_{10} levels in plasma,[6] but this effect has not been demonstrated in tissue. However, lipid-soluble statins such as simvastatin have been shown experimentally to reduce myocardial adenosine triphosphate[25] most likely owing to reduced CoQ_{10}-mediated oxidative phosphorylation. Thus, higher dosages of CoQ_{10} may be required in patients receiving concomitant statin therapy.

Side effects

In these 12 studies, as in most studies of naturally occurring vitamin-like substances such as CoQ_{10}, side effects were not of concern, being reported in four trials as either absent,[15,23] approximately 13%[22] or no different from placebo.[21] The remaining eight trials provided no comment, suggesting that adverse effects from CoQ_{10} therapy were not significant. The low incidence of side effects accords with the published studies of CoQ_{10} therapy for cardiac failure where the reported side effects have been minimal. In a study of 3500 patients given CoQ_{10} for up to seven years, adverse effects were minor and occurred in only 0.8%.[26]

Study limitations

Differences in exclusion criteria, age, therapy duration and use of concomitant therapy. In the studies included in the meta-analysis, there were differences between patient populations with respect to age, underlying disease and comorbidities. Most patient populations were described as having *hypertension* or *essential hypertension*, one had *isolated systolic hypertension*,[22] another *borderline hypertension*[16] and still another *hypertension and coronary artery disease*.[21] The more recent studies applied stricter exclusion criteria to their selection processes than did the early ones. Variations in the age of the participants in the different studies (range of averages of 48–68 years) could conceivably influence interpretation of results. Usage of other anti-hypertensive therapy was also variable: no pre-existing therapy in five studies, therapy ceased in four studies and therapy continued in three studies.

Differences in the duration of therapy were also noted in the open-label study data, with Langsjoen's 1994 study[19] spanning 13 months contrasting with the average of 2–4 months for the other open-label studies.[4,13–18] However, we considered it clinically appropriate to combine these results statistically, as the magnitude of effect was comparable among the studies (no significant heterogeneity in overall weighted mean difference for systolic or diastolic blood pressure was observed throughout the meta-analysis, with all heterogeneity χ^2 calculations resulting in *P*-values well in excess of 0.05) and the weighting of the large Langsjoen study did not skew the results. Despite these differences in composition between the various study populations and in duration of therapy, the hypotensive effect was surprisingly uniform, suggesting a consistent and predictable hypotensive action.

No assessment of major-associated cardiac events. These trials did not include the assessment of major-associated cardiac events such as death, heart attack and stroke, and were mostly underpowered to detect these important end points. However, for anti-hypertensive therapy in general, the magnitude of reduction of cardiovascular risk parallels the magnitude of lowering of blood pressure.[27] Therefore, it is reasonable to infer that the blood pressure lowering observed by CoQ_{10} in the studies included in this meta-analysis should lead to meaningful reductions in major cardiovascular events when applied across a relevant patient population, assuming that this blood pressure lowering effect can be sustained over prolonged periods with a non-adverse safety profile.

Older trials not up to modern standards. Many of the trials were performed a long time ago (one 30 years ago) and some, especially the open label studies, do not conform to the standards of modern clinical trials. Clearly in open label studies, the observer (of blood pressure) would not be blinded to the treatment received, hence bias could occur. Within some of these older trials,[4,13–15] there are variations in the duration of therapy and in the dosage used (Table 2). Two of the earliest trials enroled less than 10 patients.[4,13] In many trials, the incidence of side effects was not specifically given, although it is clear that side effects were not of concern.

In uncontrolled observational studies, the real possibility exists of 'regression toward the mean', particularly where patients are entered into a trial based on levels being above a certain cutoff value for blood pressure. Therefore, for this reason, considerable caution needs to be exercised with interpretation of trials of this type.

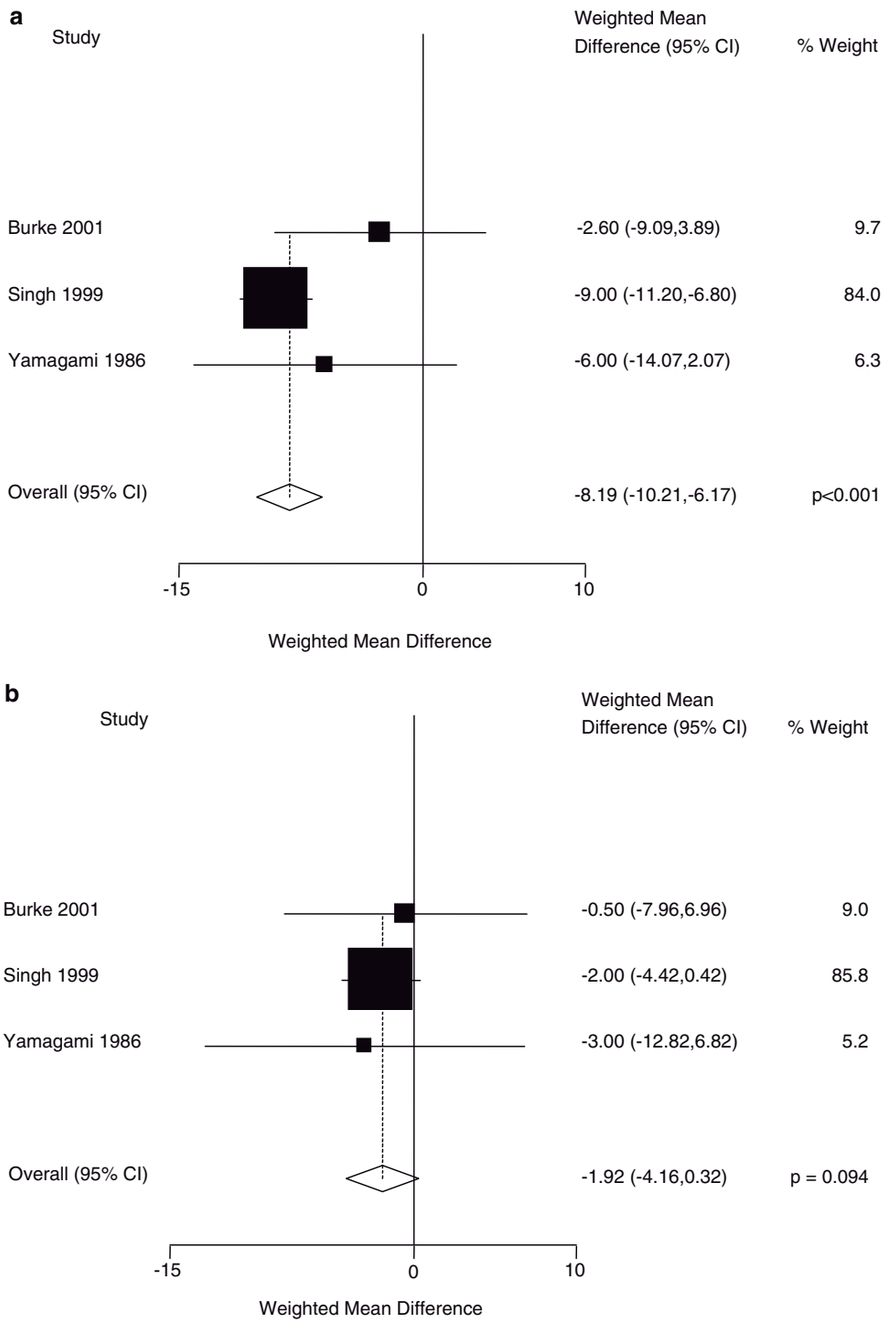

Figure 3 Randomized controlled trials, diastolic blood pressure weighted mean differences from before to after – treatment group (**a**), placebo group (**b**).

Publication bias. This meta-analysis potentially suffers from the drawback of all meta-analyses, that of publication bias favouring the reporting of positive studies and the non-reporting of negative (or neutral) ones. However, this meta-analysis conforms to the recommendation regarding careful searching of the literature to ensure minimization of publication bias.[8,9] We believe these results to be the best possible summary of all currently available information regarding the effect of CoQ_{10} on blood pressure.

Mechanism of action

An increase in oxidative stress is well documented in hypertensive states.[28] In blood vessels, oxidative

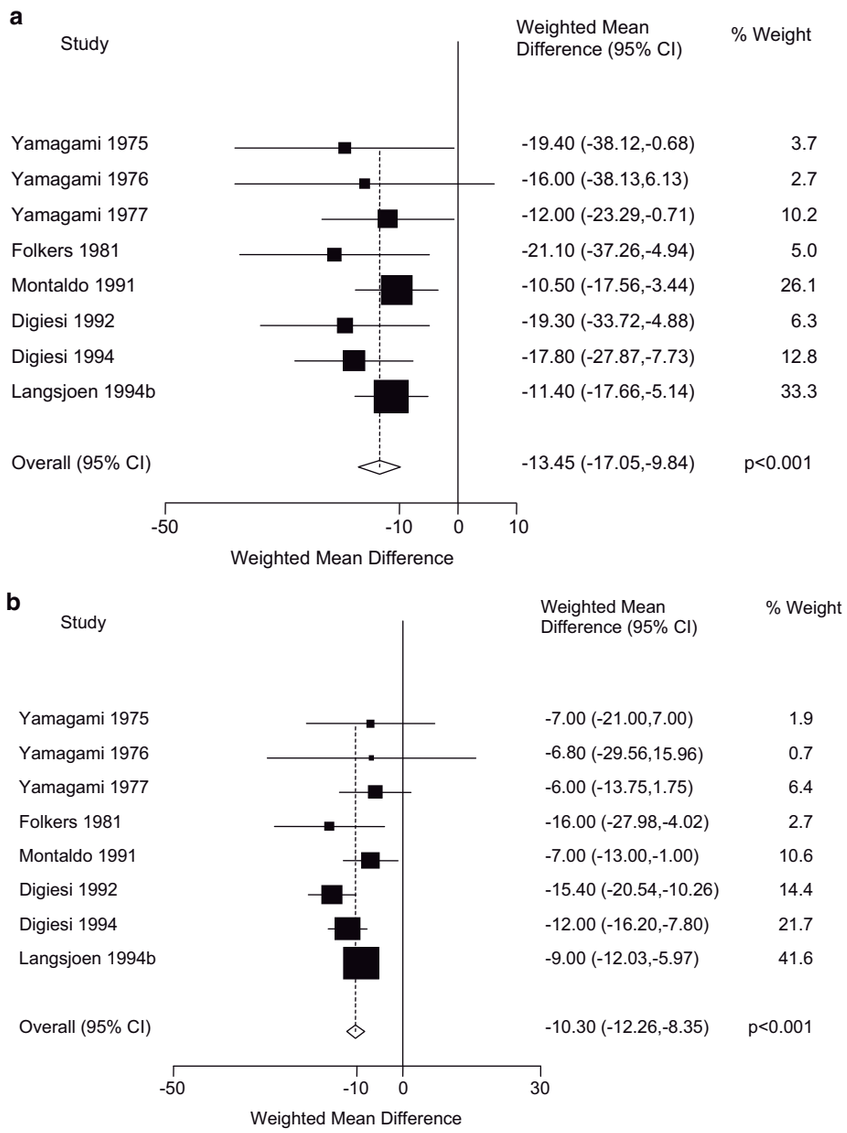

Figure 4 Open label observational studies, weighted mean differences from before to after treatment – systolic blood pressure, (**a**), diastolic blood pressure (**b**).

stress causes an increase in the production of the superoxide radical ($O_2^{\bullet -}$) that in turn rapidly reacts with endothelial nitric oxide (NO) to form peroxynitrite, thus reducing NO availability.[29] This reduction impairs the ability of endothelium to induce NO-mediated relaxation of underlying smooth muscle with resultant vasoconstriction and increased blood pressure. Coenzyme Q_{10} is a potent chain-breaking lipid-soluble antioxidant with the ability to counteract this vasoconstriction and thus lower blood pressure.

The primary action of CoQ_{10} in clinical hypertension is vasodilatation, via a direct effect on the endothelium and vascular smooth muscle.[15,17] In patients with diabetes or dyslipidemia, we have shown that CoQ_{10} improves endothelial function and lowers blood pressure.[30] Investigators of hypertensive patients treated with CoQ_{10} observed decreased peripheral resistance accompanying lowered blood pressure and unchanged cardiac output.[15,17] It should be noted however that in normal animals or humans, CoQ_{10} has no direct vasodilating or hypotensive effect. This confirms that the hypotensive effect of CoQ_{10} is specific to the state of enhanced oxidative stress occurring in hypertensive patients.

CoQ_{10} has also been shown to target the expression of multiple genes, particularly those involved in cell signalling and intermediary metabolism.[31] CoQ_{10} plays a role in energy dissipation by the

uncoupler proteins.[32] Thus gene regulation and control of metabolic flux may explain many of the cardiovascular and other actions of CoQ_{10}.

Clinical implications

Coenzyme Q_{10} has the potential in hypertensive patients to lower systolic blood pressure by up to 17 mm Hg and diastolic pressure by up to 10 mm Hg without significant side effects. CoQ_{10} appears effective as a hypotensive agent either on its own or in combination with conventional anti-hypertensive medication. We believe that there is now a convincing case for conducting a high quality prospective randomized trial of CoQ_{10} in order to validate the results of this meta-analysis. In the current era, it would be unethical to conduct a placebo-controlled trial in hypertensive patients. The ideal trial would be one comparing CoQ_{10} with an angiotensin converting enzyme (ACE) inhibitor or diuretic as in the ANBP2 trial to demonstrate non-inferiority of CoQ_{10}. Two types of trials would be useful. The more conventional type would be one with the primary end point of death and major cardiac events such as stroke. Such a trial would need to include several thousand patients for adequate statistical power. The second type of trial would be one with end points such as adequacy of blood pressure control, improvement in cardiac function, improvement in exercise tolerance and quality of life, as well as prevalence of adverse effects. Such a trial might include only several hundred patients.

Until the results of such trials are available, it would seem acceptable to add CoQ_{10} to conventional anti-hypertensive therapy, particularly in patients who are experiencing intolerable side effects of conventional anti-hypertensive therapy. CoQ_{10} may also have a particular therapeutic role in hypertensive patients with consistently increased levels of oxidative stress as in diabetes or renal failure.

What is known about topic
- In hypertension there is an increase in oxidative stress
- Therapy with coenzyme Q10 lowers oxidative stress
- Multiple individual studies have suggested that coenzyme Q10 lowers blood pressure

What this study adds
- A comprehensive review and meta-analysis of published studies of coenzyme Q10 and hypertension
- Discussion of the mechanism of the anti-hypertensive action of coenzyme Q10

Acknowledgements

Steven Joseph Haas previously received assistance via a National Health and Medical Research Council Public Health Postgraduate Research Scholarship, Scholarship application I.D. no. 237059. We acknowledge financial support for the Cardiac Surgical Research Unit from Pharma Nord Aps, Denmark, and Blackmores, Australia.

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