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Telmisartan or Valsartan Alone or in Combination with Hydrochlorothiazide: A Review

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Abstract

The aim of this review was to compare telmisartan and valsartan in the treatment of hypertension. PubMed searches were conducted to identify randomized trials ($n = 14$) comparing the two agents, alone or combined with hydrochlorothiazide. With one exception, all studies with blood pressure reduction as primary endpoint showed significantly greater reductions with telmisartan than with valsartan. Other studies showed that telmisartan was associated with greater improvements in metabolic measures and inflammatory markers than valsartan. These findings suggest that pharmacologic differences between telmisartan and valsartan may translate into clinically relevant differences between the two drugs in the management of hypertension.

Keywords: angiotensin receptor blockers, antihypertensive agents, blood pressure, clinical trials, telmisartan, valsartan

INTRODUCTION

The central place of antihypertensive therapy in cardiovascular (CV) risk reduction strategies is founded on extensive epidemiologic data and evidence from major intervention studies, which together show that the risk of CV events is linearly related to blood pressure (BP), and that lowering elevated BP decreases the incidence of such events (1,2). In addition, studies such as Hypertension Optimal Treatment (HOT), Antihypertensive and Lipid-Lowering Therapy to prevent Heart Attack Trial (ALLHAT), and Valsartan Antihypertensive Long-term Use Evaluation (VALUE) (3–5) have shown that the reduction in CV risk is directly related to the final BP achieved, with no lower threshold. Such findings highlight the need for effective control of BP in hypertensive patients. Even though effective antihypertensive drugs are available and widely used, adequate BP control is achieved with monotherapy in only a minority of patients, and hence most patients will require combination therapy (6,7).

Angiotensin II receptor blockers (ARBs) offer a number of advantages in the treatment of hypertension because they combine effective BP lowering and a placebo-like tolerability profile (8). Furthermore, a number of major trials have shown that these agents have cardioprotective and renoprotective effects, and reduce the risk of type 2 diabetes mellitus (T2DM) in high-risk hypertensive patients and patients with conditions such as heart failure or diabetic nephropathy (9–12). Angiotensin II receptor blockers have also been shown to prevent the development of T2DM (13,14). In patients who require more than one antihypertensive agent to control their BP, the combination of ARBs and diuretics such as hydrochlorothiazide (HCTZ) has been shown to be effective and well tolerated, and is recommended in current hypertension management guidelines (6,7).

Two ARBs of particular interest are telmisartan and valsartan. Both have been shown to be effective antihypertensive agents (15,16) and to reduce CV events in major outcome trials (5,11). However, the two drugs differ markedly in their pharmacokinetic and pharmacodynamic properties. Telmisartan has a higher affinity for the angiotensin II AT_1-receptor than other ARBs, resulting in a long dissociation half-life; it acts as an insurmountable receptor antagonist, thereby decreasing the maximum response to angiotensin II (17,18). In addition, telmisartan has a long elimination half-life (approximately 20–30 h) and is more lipophilic than other ARBs, and hence readily distributes into tissues (19,20). By contrast, valsartan has a relatively short elimination half-life, of approximately 7 hours (20). As a result, telmisartan might be expected to provide more effective 24-hour BP control than valsartan; this is an important consideration because BP and CV risk peak during the early morning hours, when plasma concentrations of many antihypertensive agents are declining (21,22). This article reviews comparative studies of the

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antihypertensive efficacy of telmisartan and valsartan, alone or in combination with HCTZ.

METHODS

A PubMed search with the search terms "telmisartan" and "valsartan" was conducted to identify primary reports of clinical trials comparing the two agents alone or combined with HCTZ, published up to 2010. Searches were confined to articles published in English, with the limits "human," "clinical trial," or "randomized clinical trial." Retrieved articles were selected for inclusion in the review if they described randomized parallel-group or crossover trials. Additional material for inclusion was selected based on the author's personal knowledge.

RESULTS

A total of 27 articles were initially identified, of which only 14 (Table 1) remained after the selection criteria were applied. Of these, 11 (23–33) described studies in which telmisartan and valsartan were used as monotherapies (with or without add-on therapy to control BP) in a variety of patient populations, and 3 (34–36) described studies using combinations of the ARBs and HCTZ. One article (24) was excluded from the review because it described a previously reported study (23). Blood pressure reductions were the primary endpoints in five studies; the remaining trials focused on metabolic endpoints such as microalbuminuria, insulin resistance, or markers of inflammation such as high-sensitivity C-reactive protein (hs-CRP).

Monotherapy

Antihypertensive Efficacy

Several studies (23,25,27) have reported that once-daily treatment with telmisartan produces significantly greater reductions in BP than valsartan. In one study, for example, 24-hour BP was measured by ambulatory BP monitoring (ABPM) in 490 hypertensive patients (mean seated diastolic BP [DBP] $\geq$95 mm Hg but <110 mm Hg), who received telmisartan and valsartan for 8 weeks each in a randomized crossover design (25). Treatment was started at doses of 40 mg for telmisartan and 80 mg for valsartan, and the doses were increased to 80 and 160 mg, respectively, after 2 weeks. The mean reductions from baseline in systolic BP (SBP) and DBP during the last 6 hours of the dosing interval were -11.0 ± 0.8 and -7.6 ± 0.6 mm Hg, respectively, with telmisartan and valsartan -8.7 ± 0.8 and -5.8 ± 0.6 mm Hg, respectively ($P = .02$ for SBP; $P = .01$ for DBP). In this study, patients were randomized to receive either an active dose or placebo at the end of each treatment period, to mimic the effect of a missed dose. On the day of the missed dose, there was a trend toward greater reductions in early morning BP with telmisartan than with valsartan (mean reductions $-9.0 \pm 0.7/-6.3 \pm 0.6$ mm Hg vs. $-7.4 \pm 0.7/-5.1 \pm 0.4$ mm Hg, respectively; $P = .09$ for SBP, $P = .06$ for DBP).

The results of this study were pooled with those of a second study with identical design (published only in abstract form). The combined analysis (27) confirmed that telmisartan produced significantly greater reductions in BP throughout the 24-hour dosing interval, and particularly during the last 6 hours (Figure 1). Furthermore, following a missed dose, telmisartan produced significantly greater reductions, compared with valsartan, in 24-hour mean SBP (-10.7 vs. -8.7 mm Hg, $P = .0024$), and 24-hour mean DBP (-7.2 vs. -5.5 mm Hg, $P = .0004$) (Figure 2). The proportion of patients who showed a response to treatment (defined as 24-h mean DBP <80 mm Hg or a reduction of at least 10 mm Hg from baseline) after a missed dose was significantly higher with telmisartan than with valsartan (27.0% vs. 20.9%, respectively, $P = .0387$).

A further ABPM study (23) compared the antihypertensive efficacy of telmisartan 80 mg and valsartan 80 mg in 436 patients with mild-to-moderate essential hypertension. After 8 weeks, the reduction in mean DBP during the last 6 hours of the dosing interval was significantly greater with telmisartan than with valsartan (-7.5 ± 0.6 vs. -5.2 ± 0.6 mm Hg, $P < .01$). Telmisartan was also associated with significantly greater reductions in daytime (06:00 to 21:59) and morning (06:00 to 11:00) ambulatory BP, and greater decreases in trough cuff BP, compared with valsartan. The DBP response rate (24-h mean DBP <80 mm Hg or a reduction of at least 10 mm Hg from baseline) was significantly higher with telmisartan than with valsartan (45.7% vs. 30.0%, respectively, $P < .01$). Similarly, the proportion of patients in whom DBP control (seated mean DBP <90 mm Hg) was achieved was significantly higher in the telmisartan group (47.2% vs. 32.1%, $P < .01$).

In contrast to the above studies, one small open-label unblinded exploratory trial has shown significantly greater reductions in ambulatory BP with valsartan 160 mg than with telmisartan 80 mg (26). This study involved 70 patients with mild-to-moderate essential hypertension, who were treated for 3 months. The mean reductions in 24-hour mean SBP and DBP were 18.6 and 12.1 mm Hg, respectively, with valsartan, compared with 10.8 and 8.4 mm Hg, respectively, with telmisartan (both $P < .001$). However, the baseline demographics, although classed as not significantly different in the publication, did vary between the groups and were not adjusted for. In particular, the valsartan group had on average a baseline SBP of 5 mm Hg and a DBP of 2.6 mm Hg greater than the telmisartan group. Baseline BP could have been responsible for the greater BP decrease seen with valsartan.

The VIVALDI® (a trial to inVestigate the efficacy of telmIsartan versus VALsartan in hypertensive type 2 DIabetic patients with overt nephropathy) study (32) compared the effects of telmisartan 80 mg and valsartan

Table 1. Studies comparing telmisartan and valsartan, alone or in combination with hydrochlorothiazide (HCTZ).

Study	Patient population	Number of patients (telmisartan/ valsartan)	Doses	Add-on therapy (yes/no)	Primary endpoint or other measures	Principal findings
Monotherapy						
Littlejohn et al. (23)[a]	Patients with mild-to-moderate essential hypertension	426 (214/212)	Telmisartan: 80 mg o.d. Valsartan: 80 mg o.d.	No	Change from baseline in BP (ABPM) during the last 6 h of the dosing interval	Greater decrease in DBP during the last 6 h of the dosing interval with telmisartan than valsartan
Bakris (24)[a]	As above	As above	As above	As above	As above	As above
White et al. (25)	Hypertensive patients (seated DBP $\geq$ 95 mm Hg but <110 mm Hg; ambulatory DBP $\geq$ 85 mm Hg)	490 (244/246)	Telmisartan: 40–80 mg o.d. Valsartan: 80–160 mg o.d.	No	BP reduction after missed dose	Trend toward more sustained BP reduction with valsartan
Calvo et al. (26)	Patients with mild-to-moderate essential hypertension	70 (crossover design)	Telmisartan: 80 mg o.d. Valsartan: 160 mg o.d.	No	24-h BP reduction	Significant BP reductions with both drugs; greater decrease in 24-h mean BP and arterial pulse pressure with valsartan
Lacourcière et al. (27)[b]	Hypertensive patients	930 (468/462)	Telmisartan: 40–80 mg o.d. Valsartan: 80–160 mg o.d.	No	BP reduction after missed dose	More sustained BP reduction with telmisartan than with valsartan after a missed dose
Yano et al. (28)	Patients with metabolic syndrome, previously treated with valsartan	51 (30/21)	Telmisartan: 20–80 mg o.d. Valsartan: 40–160 mg o.d.	No	Reduction in microalbuminuria	Significant reductions in microalbuminuria and C-reactive protein with telmisartan, compared with valsartan
Ichikawa (29)	Hypertensive patients with metabolic syndrome	53 (26/27)	Telmisartan: 20 mg o.d. Valsartan: 40 mg o.d.	No	BP reduction; insulin sensitivity	Both treatments significantly reduced BP; significant increase in insulin sensitivity only with telmisartan
Hong et al. (30)	Hypertensive patients receiving sirolimus-releasing stents	159 (79/80)	Telmisartan: 40 mg o.d. Valsartan: 80 mg o.d.	No	Late lumen loss and inflammatory markers	Telmisartan significantly reduced late lumen loss and inflammatory markers, compared with valsartan
Tomiyama et al. (31)	Hypertensive patients previously treated with ARBs other than telmisartan or valsartan	40 (crossover design)	Telmisartan: 40 mg o.d. Valsartan: 80 mg o.d.	No	Mean reactive hyperemia ratio	Significantly higher mean reactive hyperemia ratio, and more favorable effects on other markers of endothelial function, with telmisartan than with valsartan

Table 1. (Continued)

Study	Patient population	Number of patients (telmisartan/ valsartan)	Doses	Add-on therapy (yes/no)	Primary endpoint or other measures	Principal findings
Galle et al. (32)	Hypertensive patients with T2DM and overt nephropathy	885 (443/442)	Telmisartan: 80 mg o.d. Valsartan: 160 mg o.d.	Yes	24-h proteinuria	Similar reductions in 24-h proteinuria with both treatments; less add-on antihypertensive therapy with telmisartan
Georgescu et al. (33)	Patients with mild-to-moderate hypertension and nonalcoholic steatohepatitis	54 (28/26)	Telmisartan: 20 mg o.d. Valsartan: 80 mg o.d.	No	Liver biopsies, BP, metabolic markers	Similar reductions in BP with both treatments; greater improvement in insulin sensitivity with telmisartan
Combination with HCTZ						
White et al. (34)	Patients with stage 1 or 2 hypertension (DBP $\geq$ 95 mm Hg)	1066 (485/498/ 126 placebo)	Telmisartan: 80 mg/HCTZ 25 mg o.d. Valsartan: 160 mg/HCTZ 25 mg o.d.	No	Changes in seated BP	Significantly greater reductions in BP with telmisartan/HCTZ than with valsartan/HCTZ
Sharma et al. (35)	Overweight or obese hypertensive patients with T2DM	840 (428/412)	Telmisartan: 80 mg/HCTZ 12.5 mg Valsartan: 160 mg/HCTZ 12.5 mg	No	Change from baseline in ambulatory SBP and DBP during the last 6 h of the dosing interval	Significantly greater reductions in BP with telmisartan/HCTZ, compared with valsartan/HCTZ
White et al. (36)	Patients with stage 1 or 2 hypertension (DBP $\geq$ 95 mm Hg)	1181 (528/523/ 130 placebo)	Telmisartan: 80 mg/HCTZ 12.5 mg Valsartan: 160 mg/HCTZ 12.5 mg	No	Changes in seated BP	Significantly greater reductions in BP with telmisartan/HCTZ than with valsartan/HCTZ

Abbreviations: o.d. – once daily; BP – blood pressure; ABPM – ambulatory blood pressure monitoring; DBP – diastolic blood pressure; ARB – angiotensin II receptor blocker; HCTZ – hydrochlorothiazide; T2DM – type 2 diabetes mellitus.

[a]Reporting the same study.

[b]Pooled analysis of two identical studies, including the study by White et al. (25).

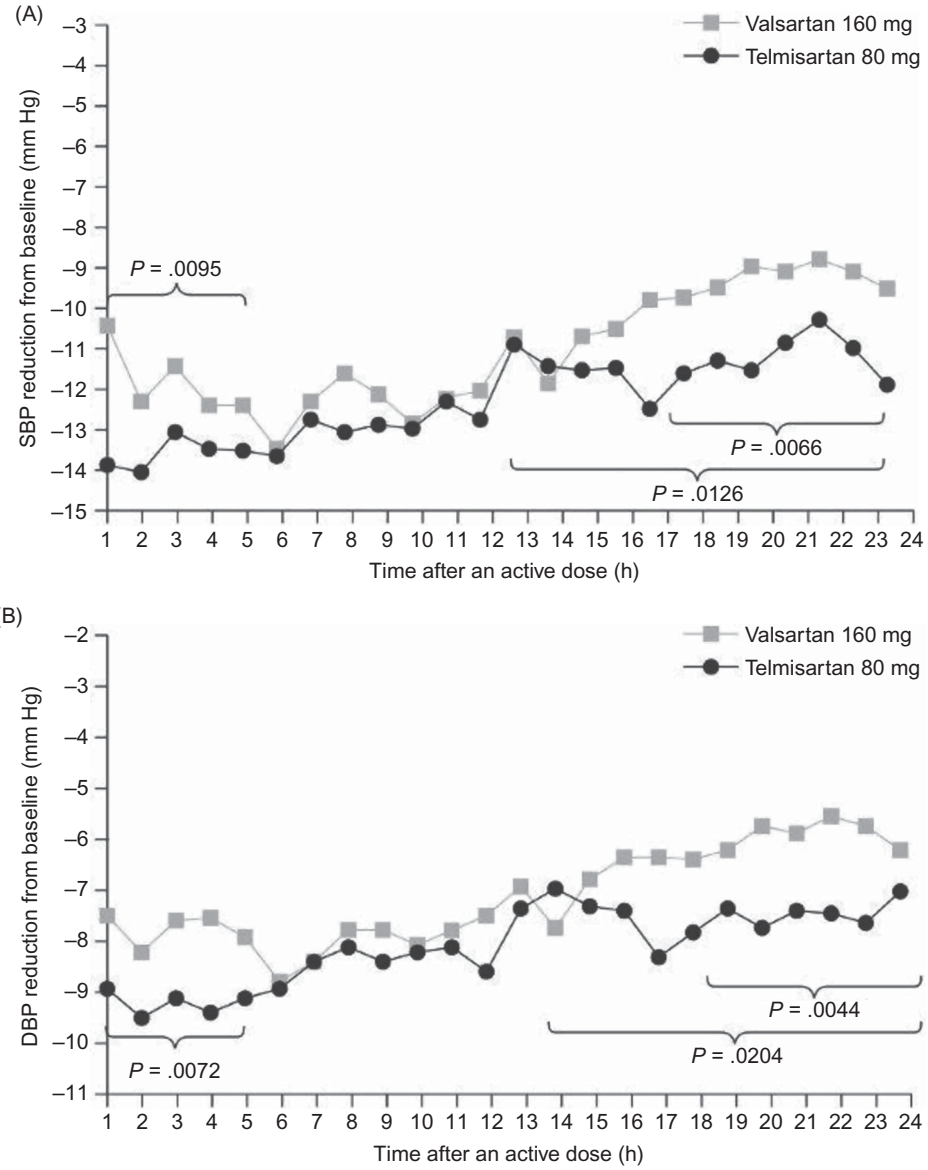

Figure 1. Mean reductions in (**A**) SBP and (**B**) DBP (measured by ABPM) over 24 h after dosing in two identical trials comparing telmisartan 80 mg ($n = 447$) and valsartan 160 mg ($n = 430$). *P* values relate to the differences between telmisartan and valsartan during the time periods indicated. Reproduced with permission (27).

160 mg in 885 hypertensive patients with T2DM and overt nephropathy. In this study, additional antihypertensive medication could be given if needed to control BP. After 12 months, the BP reductions achieved did not differ significantly between the groups; however, there was a trend toward less use of add-on antihypertensive medication in the telmisartan group, although this was not statistically significant. Decreases in 24-hour urinary protein excretion and urinary albumin excretion were similar in the two groups; in both cases, the greatest reductions were seen in patients with the largest decreases in BP. No significant changes in asymmetric dimethylarginine (ADMA, a marker of endothelial function) or CRP were noted in either group after 12 months, but urinary 8-iso-PGF2α levels decreased by 14% with telmisartan and by 7% with valsartan ($P = .040$).

Effects on Metabolic and Inflammatory Markers

A number of studies have suggested that telmisartan produces greater improvements in metabolic variables or inflammatory markers than valsartan at comparable levels of BP control. In one study, 53 hypertensive Japanese patients with metabolic syndrome were treated with telmisartan 20 mg or valsartan 40 mg for 4 weeks; all other antidiabetic, lipid-lowering, and antihypertensive therapies were kept constant throughout the study (29). Both treatments produced significant reductions in SBP, of approximately 10 mm Hg ($P < .05$); DBP decreased by approximately 4 mm Hg in both groups, but this change was only significant ($P < .05$) in the telmisartan group. Telmisartan was associated with a 16% ($P = .031$) decrease from baseline in the homeostasis model assessment of insulin resistance (HOMA-IR), whereas

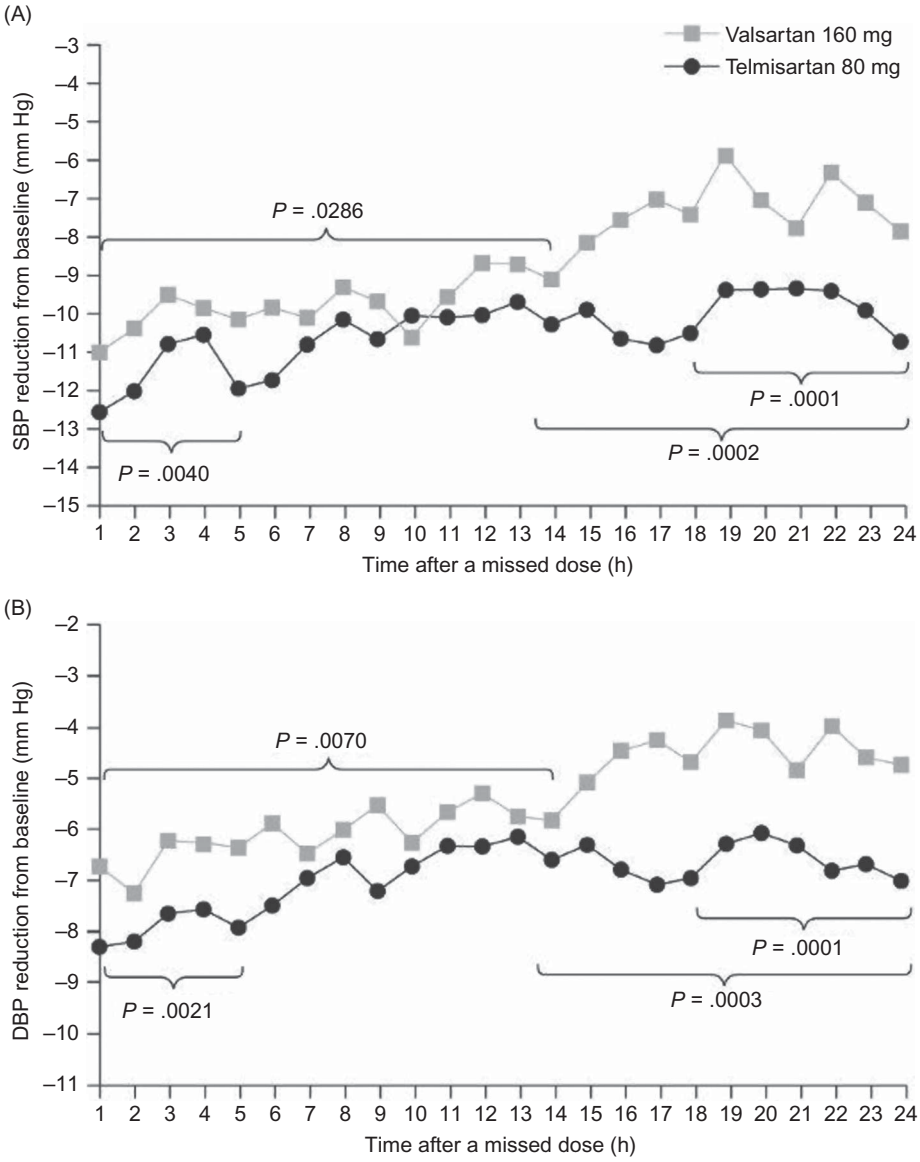

Figure 2. Mean reductions from baseline in (**A**) SBP and (**B**) DBP over 24 h after a missed dose in two trials with identical design comparing telmisartan 80 mg ($n = 447$) and valsartan 160 mg ($n = 430$). *P* values relate to the differences between telmisartan and valsartan during the time periods indicated. Reproduced with permission (27).

valsartan-treated patients showed no significant change. There was a significant correlation between the decrease in HOMA-IR in telmisartan-treated patients and baseline values ($r = 0.475$, $P < .05$). These findings suggest that, in contrast to valsartan, telmisartan improves insulin sensitivity in hypertensive patients with metabolic syndrome.

In a further study, 53 hypertensive patients with metabolic syndrome, who had previously been treated with valsartan at mean daily doses of approximately 70 mg, were randomized to continue valsartan or to switch to telmisartan for 12 weeks (28). There were no significant changes in BP in either group. However, telmisartan-treated patients showed significant reductions from baseline in microalbuminuria (28.1 vs. 18.9 mg/g creatinine; $P = .001$) and hs-CRP (0.77 vs. 0.60 mg/L; $P = .022$), consistent with suppression of low-grade inflammation; by contrast, no such effects were seen in the valsartan group. The reduction in microalbuminuria in telmisartan-treated patients was significantly correlated to the decrease in hs-CRP ($r = 0.413$, $P = .003$), but not to changes in BP. Patients receiving telmisartan also showed a significant (8.7%) increase in high molecular weight adiponectin, a hormone that regulates a number of processes in glucose and lipid metabolism.

The anti-inflammatory effects of telmisartan are demonstrated further in a study of 159 hypertensive patients who received sirolimus-eluting coronary stents (30). After 8 months of treatment, patients receiving telmisartan showed significantly greater decreases in total and low-density lipoprotein cholesterol, and inflammatory markers such as hs-CRP and tumor necrosis

factor-α, than valsartan-treated patients. Moreover, late lumen loss in coronary arteries was significantly lower with telmisartan than with valsartan (0.1 ± 0.4 vs. 0.3 ± 0.5 mm; $P = .001$).

The effects of telmisartan 40 mg and valsartan 80 mg on endothelial function were compared in a randomized crossover study involving 40 patients with essential hypertension (31). All patients had previously been treated with ARBs other than telmisartan or valsartan. There were no significant differences in BP with the two treatments, but telmisartan was associated with significantly greater reactive hyperemia and higher concentrations of ADMA than valsartan, and a significant decrease in HOMA-IR (Figure 3). There was a significant correlation between the increase in ADMA and forearm postischemic hyperemia, but not between ADMA and HOMA-IR. Telmisartan, therefore, appears to have a more favorable effect on functional parameters related to endothelial function than valsartan.

Combination Therapy

Three studies (34–36) have investigated the antihypertensive efficacy of combinations of telmisartan or valsartan with HCTZ. In the SMOOTH® (Study of Micardis® in Overweight/Obese patients with Type 2 diabetes and Hypertension) study, 840 overweight or obese hypertensive patients with T2DM were randomized to receive telmisartan 80 mg or valsartan 160 mg for 4 weeks, after which HCTZ 12.5 mg was added and treatment continued for a further 6 weeks (35).The primary endpoint was the change from baseline in mean ambulatory SBP and DBP during the last 6 hours of the dosing interval. Telmisartan produced significantly greater BP reductions than valsartan throughout the 24-hour dosing interval. During the last 6 hours of the dosing interval, the mean treatment differences in favor of telmisartan were 3.9 mm Hg (95% confidence interval [CI] −5.8, −2.1 mm Hg; $P < .0001$) for SBP and 2.0 mm Hg (95% CI −3.2, −0.8 mm Hg; $P = .0007$) for DBP (Figure 4). Similar treatment differences were seen for mean 24-hour BP and morning, daytime, and nighttime BPs (Figure 4). The mean BP reductions during the last 6 hours of the dosing interval in patients under 65 years were similar to those in older patients. Blood pressure reductions were greater in women than in men, but the observed treatment differences in favor of telmisartan were seen in both sexes. Measurement of trough office BP during the study showed that the greater efficacy of telmisartan compared with valsartan (SBP 3.2 mm Hg, $P = .0017$; DBP 1.2 mm Hg; $P = .0446$), which became apparent by the end of the 4-week monotherapy period (SBP 2.5 mm Hg: $P = .0106$; DBP 0.8 mm Hg: $P = .1370$).

Superior BP control with a combination of telmisartan and HCTZ, compared with valsartan plus HCTZ, was also shown in two large, randomized, placebo-controlled trials with identical designs (34,36). Both involved patients with Stage 1 or Stage 2 hypertension (seated DBP ≥ 95 mm Hg) who received telmisartan/HCTZ (80/25 mg) or valsartan/HCTZ (160/25 mg) for 8 weeks. The primary endpoint was the change from baseline in-clinic SBP and DBP at the end of the study. In both studies, decreases in BP were significantly greater with telmisartan/HCTZ than with valsartan/HCTZ. The data from these two studies were pooled in a combined analysis, which included a total of 2121 randomized patients (37). The mean BP reductions in patients receiving telmisartan/HCTZ were −24.5/−18.0 mm Hg, compared with −22.3/−16.8 mm Hg with valsartan/HCTZ ($P = .0004$ for SBP; $P = .0019$ for DBP) and −4.1/−6.5 mm Hg in placebo-treated patients ($P < .0001$ for both SBP and DBP). Consistent treatment differences in favor of telmisartan/HCTZ were seen irrespective of age group (<65 vs. ≥65 y), gender, or race (non-Black vs. Black). Response rates (SBP response: <140 or >20 mm Hg reduction from baseline; DBP

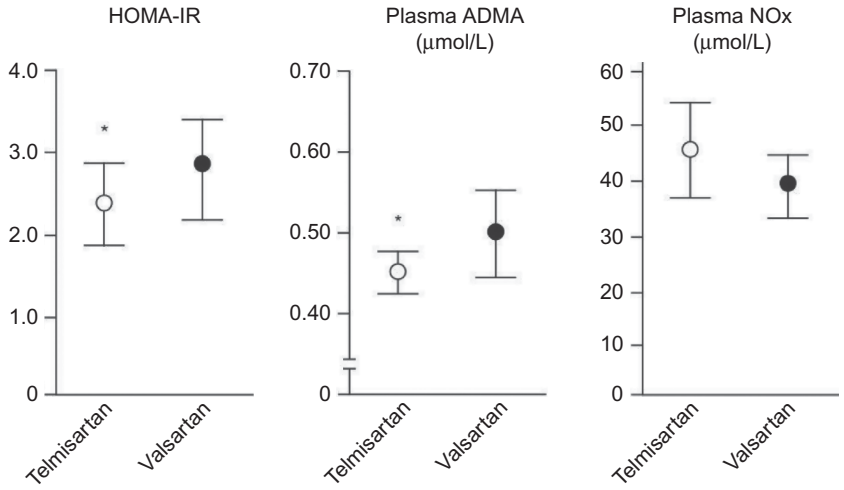

Figure 3. Changes in the homeostasis model assessment of insulin resistance (HOMA-IR), plasma asymmetric dimethylarginine (ADMA), and serum nitric oxide (NO_x) in 40 hypertensive patients treated with telmisartan 40 mg or valsartan 80 mg for 12 weeks each in a randomized crossover study. $P < .05$ versus treatment with valsartan. Reproduced with permission (31).

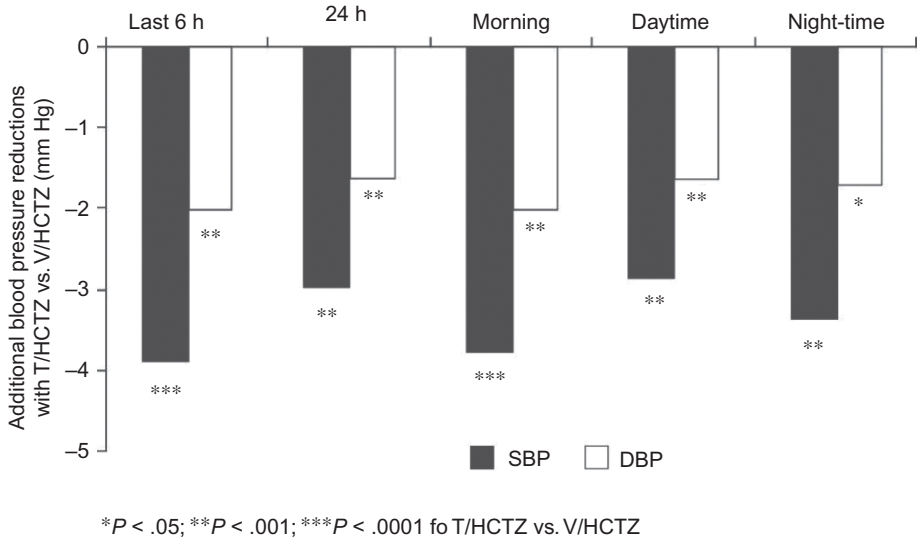

Figure 4. Additional BP reductions with telmisartan/HCTZ 80/12.5 mg, compared with valsartan/HCTZ 160/12.5 mg in overweight or obese hypertension patients with type 2 diabetes in the SMOOTH® study. Reproduced with permission (35).

response: <90 or >10 mm Hg reduction from baseline) were significantly ($P = .02$ for both SBP and DBP response) higher with telmisartan/HCTZ than with valsartan/HCTZ. At the end of the study, 63.9% of patients receiving telmisartan/HCTZ had achieved BP control (<140/90 mm Hg), compared with 59.9% of those receiving valsartan/HCTZ. Logistic regression analysis showed that the probability of achieving BP control was significantly greater with telmisartan/HCTZ than with valsartan/HCTZ (Figure 5).

CONCLUSIONS

The studies reviewed here show that telmisartan offers a number of advantages over valsartan for the management of hypertensive patients, in terms of both antihypertensive efficacy and potentially beneficial effects on other measures, such as insulin sensitivity and inflammation.

The antihypertensive benefits were seen when both telmisartan and valsartan were given as monotherapies, and when the two agents were combined with HCTZ. With the exception of one small single-blind exploratory trial by Calvo et al. (26), studies have consistently shown that telmisartan produced greater reductions in office or 24-hour BP than valsartan, either as monotherapy or in combination with HCTZ. Of particular interest is the finding that telmisartan was superior to valsartan in controlling BP during the early morning hours, when the risk of CV events is highest (21,22). The finding that telmisartan provides more effective control of 24-hour ambulatory BP (23,25,27,35) is important because ABPM measurements show a better correlation with target organ damage, such as left ventricular hypertrophy (38) and microalbuminuria (39) than office BP measurements. Ambulatory BP is therefore a more reliable indicator of prognosis than office BP; indeed, the risk of CV events in patients with high office BP but normal ambulatory BP is comparable with that in normotensive individuals (40).

The differences between the BP reductions achieved with telmisartan and valsartan are likely to be clinically relevant. Several major trials (3–5) and meta-analyses (41) have shown that benefits of BP lowering are not restricted by a minimum threshold. Furthermore, a meta-analysis involving 1 million patients in 60 prospective studies showed that a decrease in SBP of only 2 mm Hg was associated with 7%–10% reductions in mortality from vascular diseases at BPs down to 115/75 mm Hg (42); similarly, a further study showed that a 1 mm Hg decrease in DBP was associated with 5%–8% reductions in CV morbidity (43).

The differences between telmisartan and valsartan observed in the studies reviewed here are likely to reflect differences in the pharmacological properties of the two drugs. The greater antihypertensive efficacy of telmisartan, compared with valsartan, may be attributable to telmisartan's longer duration of action, based on its prolonged AT_1-receptor binding and long elimination half-life.

In summary, the available evidence suggests that telmisartan, alone or in combination with HCTZ, is superior to valsartan-based regimens in the management of hypertension. Additionally, the benefits of telmisartan in preventing CV events in high-risk patients, beyond that of BP lowering alone, have recently been demonstrated in The ONgoing Telmisartan Alone and in combination with Ramipril Global Endpoint Trial (ONTARGET®) Programme (12).

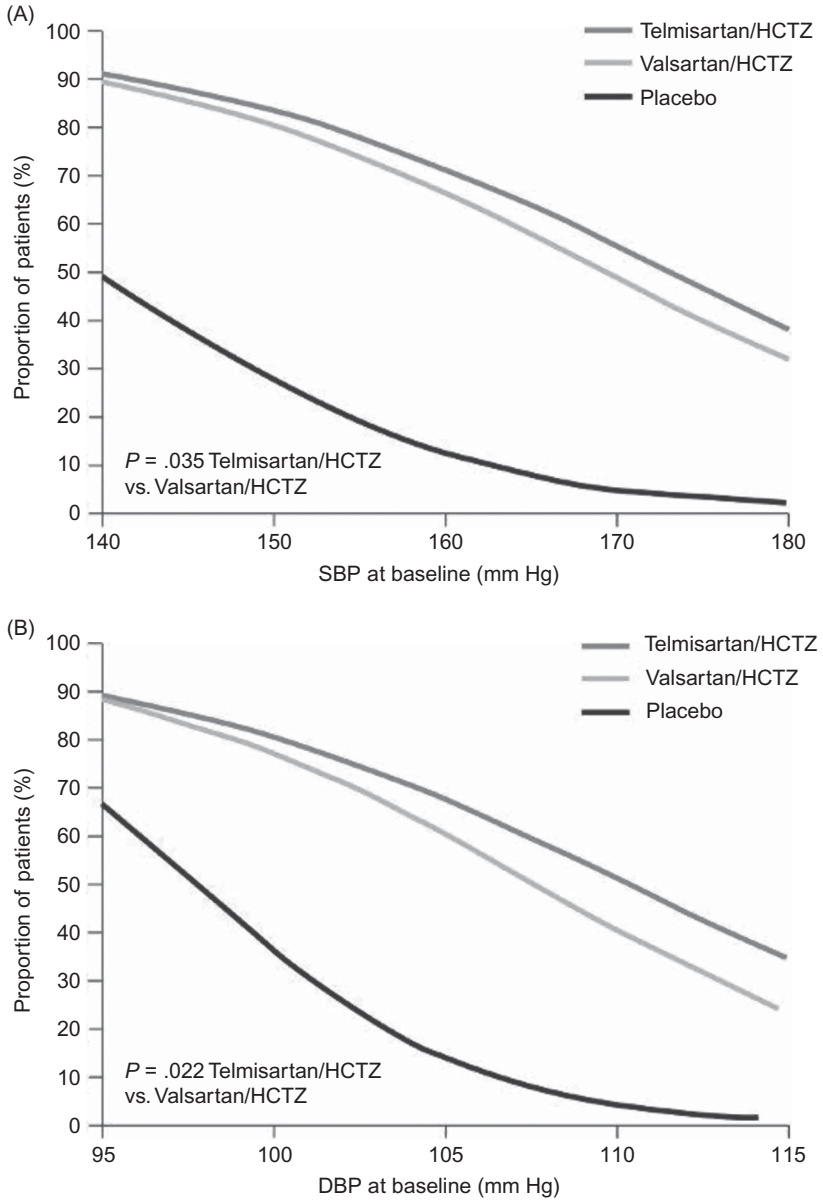

Figure 5. Probability of achieving BP control (<140/90 mm Hg) in relation to baseline BP and treatment in two randomized trials comparing telmisartan/HCTZ 80/25 mg and valsartan/HCTZ 160/25 mg (36). For example, a patient with baseline BP of 165/105 mm Hg would have a 63.7% (59.4–67.7) probability of achieving SBP control (**A**) with telmisartan/HCTZ versus 57.6% (53.3–61.8) with valsartan/HCTZ or 8.3% (4.7–14.3) with placebo. For DBP control (**B**), the corresponding probabilities are 67.8% (95% CI: 63.6–71.3), 60.2% (56.1–64.3), and 14.0% (8.6–22.2), respectively. Reproduced with permission (36).

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