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Do Hypertensive Men Spy With an Angry Little Eye? Anger Recognition in Men With Essential Hypertension - Cross-sectional and Prospective Findings

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Abstract

Background Higher trait anger has inconsistently been associated with hypertension and hypertension development, but social context in terms of recognition of other persons' anger has been neglected in this context.

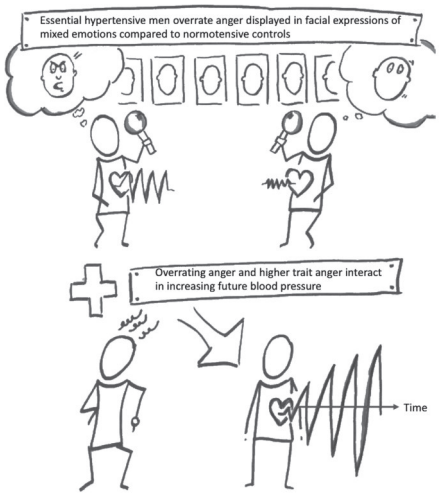
Purpose Here, we investigated anger recognition of facial affect and trait anger in essential hypertensive and normotensive men in addition to prospective associations with blood pressure (BP) increases.

Methods Baseline assessment comprised a total of 145 participants including 57 essential hypertensive and 65 normotensive men who were otherwise healthy and medication-free. Seventy-two eligible participants additionally completed follow-up assessment 3.1 (±0.08 *SEM*) years later to analyze BP changes over time. We assessed emotion recognition of facial affect with a paradigm displaying mixed facial affect of two morphed basic emotions including anger, fear, sadness, and happiness. Trait anger was assessed with the Spielberger trait anger scale.

Results Cross-sectionally, we found that with increasing BP, hypertensive men overrated anger displayed in facial expressions of mixed emotions as compared to normotensive men ($ps \leq .019$) while there were no differences in trait anger ($p = .16$). Prospectively, the interaction between mean anger recognition and trait anger independently predicted BP increases from baseline to follow-up ($ps \leq .043$), in that overrating displayed anger predicted future BP increases only if trait anger was high.

Conclusions Our findings indicate an anger recognition bias in men with essential hypertension and that overrating displayed anger in combination with higher trait anger seems to predict future BP increases. This might be of clinical relevance for the development and progression of hypertension and cardiovascular disease.

Graphical Abstract



Keywords Hypertension · Blood pressure · Trait anger · Anger recognition (bias) · Mixed emotions

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Introduction

Arterial hypertension is a major risk factor for cardiovascular disease. About 95% of patients with arterial hypertension are diagnosed with "essential" hypertension, meaning there is no clear medical explanation for their condition [1]. This stimulated research to investigate the potential role of psychosocial factors in essential hypertension. In particular, *anger* as an emotion-related psychosocial factor has been associated with hypertension and hypertension development (for reviews [2–4]). Anger is a multidimensional construct and in earlier research, the terms anger, hostility, and aggression were often used interchangeably without clear conceptual distinction [5]. In this context, an early review indicated associations between anger and hostility and the development of hypertension [4]. Later meta-analyses differentiated between anger experience and anger expression and reported small and highly variable or modest *cross-sectional* relationships between anger and blood pressure (BP) [3, 6] with hostility inconsistently assigned to anger experience [3, 6] and/or expression [6]. Prospectively, there was support for associations between anger expression and the development of hypertension [2]. Currently, a three-factor model considering trait anger to have distinct affective, behavioral, and cognitive dimensions has been proposed to represent the best conceptual structure [5]. The affective dimension refers to feelings of anger, the behavioral dimension to aggression and anger expression, and the cognitive dimension to hostility [5, 7].

An established scale with proven reliability and validity to assess "trait anger" is the Spielberger trait anger scale [8]. According to Spielberger [8], trait anger represents the disposition to frequently and intensively experience anger over a range of situations. Based on this definition, we refer in the following to the affective dimension of trait anger when using the term trait anger. With respect to associations with hypertension, we argue that feelings of anger are accompanied by heightened sympathetic activation [9, 10] and that frequent anger feelings and thus increased sympathetic activation may accumulate over time. According to the allostatic load model [11], this may result in chronic BP elevation. Some *cross-sectional* studies assessing trait anger accordingly found higher trait anger with increasing resting BP or hypertension [12–14], whereas others did not [15–18]. The vast majority of cross-sectional studies reported mixed results where associations between trait anger and BP or hypertension were not supported unequivocally with findings varying between the studies [19–27]. The above-mentioned meta-analyses summarized different anger scales to experience of anger either with [3] or without [6] inclusion of the Spielberger trait anger scale [8] and found positive associations between anger experience and diastolic BP (DBP) [6] and/or systolic BP (SBP) [3, 6]. With respect to *prospective* assessment, trait anger according to Spielberger [8] has not yet been investigated in manifest hypertension. However, few longitudinal studies enrolling normotensive or prehypertensive individuals assessed trait anger in the context of hypertension development. One epidemiological study in a cohort of middle-aged prehypertensive men and women followed for up to 8 years found that about 67% of participants with high baseline trait anger scores developed hypertension [28]. Two other studies in middle-aged normotensive women found increases in trait anger over time but not baseline trait anger to be predictive of BP increases [29] and hypertension incidence [30]. In sum, these cross-sectional and prospective findings remain inconclusive and the role of trait anger in hypertension warrants further elucidation.

The inconsistency of the above-summarized findings may relate to the assessment of trait anger by means of a self-report measure that does not capture anger within interpersonal or social context [31]. The personality in context theory states that personality does not reflect an individual in isolation but rather an individual within interpersonal or social context [32, 33]. Based on this theory, trait anger may require situational features that in particular elicit feelings of anger and thus the affective dimension of trait anger, and corresponding interrelations. Moreover, the perception-action model for empathy (PAM) posits that in social contexts a person's own emotions strongly interact with the perception and recognition of emotions of others [34]. In the sense of emotional contagion, individuals come to feel a similar emotional state as the one perceived [34]. To recognize a person's emotional state, facial expressions have been proposed to be of particular importance [35]. In line with the personality in context theory and the PAM, social context in terms of facial affect recognition may modulate the associations between personality traits, such as trait anger, and hypertension (development). More specifically, differences in the recognition of anger displayed in another person's face may interact with trait anger (in particular with its affective dimension), and thus modulate situational feelings of anger. This interaction may contribute to hypertension (development) in accordance with the allostatic load model [11].

So far, evidence from cross-sectional studies points to associations between higher resting BP or hypertension and impaired emotion recognition. A very early study suggests that hypertensive individuals tend to recognize hostility in notably non-standardized and non-validated pictures of facial affect more often than non-hypertensive patients [36]. Later studies using standardized, reliable, and valid pictures of facial affect reported impaired emotion recognition in borderline hypertensive participants as compared

to normotensive controls [37] and in bariatric surgery candidates with a history of hypertension [38]. Moreover, within the normotensive up to hypertensive range higher resting BP [39, 40], elevated DBP [41], and elevated SBP [42] were found to relate to reduced accuracy in recognition of facial affect. The hitherto only study in manifest hypertension similarly revealed reduced facial emotion recognition accuracy in prehypertensive and hypertensive participants as compared to normotensive participants [43]. Interestingly, notwithstanding the potential role of anger in hypertension, all of these studies investigated different emotions displayed separately and then averaged the proportion of correctly recognized emotions to total recognized emotions, that is, emotion recognition accuracy. So far, two studies considered recognition of separate displayed emotions in borderline hypertensive participants [37] or hypertensive heart disease patients [44] and controls. The recognition of the emotion anger did not differ between groups despite an impaired overall emotion recognition in patients with hypertensive heart disease and borderline hypertensive participants compared to controls [37, 44]. Meanwhile, sophisticated methods have been developed to assess recognition of facial affect more specifically by displaying mixed facial affect comprising blends of two morphed basic emotions [45]. This methodology allows to identify a recognition bias toward one emotion (e.g., anger) over others. To the best of our knowledge, neither recognition of mixed facial affect nor prospective associations regarding recognition of facial affect have been studied in the context of hypertension. Also, trait anger combined with anger recognition of facial affect has not yet been investigated in essential hypertension either cross-sectionally or prospectively.

We, therefore, set out to specifically investigate anger recognition and trait anger in otherwise healthy essential hypertensive and normotensive men and to determine prospective associations with BP changes. We assessed emotion recognition accuracy (i.e., underrating or overrating displayed emotion) of electronically morphed pictures of facial affect displaying two blended basic emotions (anger, fear, sadness, happiness). Cross-sectionally, we hypothesized that hypertensive men would differ from normotensive controls in emotion recognition accuracy of anger displayed in pictures of mixed facial affect. Moreover, we expected prospective associations between anger recognition, trait anger, and BP increases over an average 3-year follow-up period. In particular, given the personality in context theory [32, 33] and the PAM [34], we expected prospective associations between the interaction of anger recognition with trait anger and future BP increases.

Methods

Study Participants

This study is part of a series of studies assessing psychoneurobiological mechanisms in essential hypertension and coronary heart disease [46, 47]. The project was approved by the Ethics Committee of the State of Bern, Switzerland, and conducted in accordance with the Declaration of Helsinki principles. All participants provided written informed consent for both assessments and were financially compensated for each assessment with 20 Swiss Francs.

For the *baseline cross-sectional assessment* of the present study, we recruited apparently healthy participants with BP in the normotensive to hypertensive range. The final cross-sectional sample comprised 145 participants with 65 normotensive participants, 57 essential hypertensive participants, and 23 participants without clear classification (16 white coat hypertensive participants, 7 masked hypertensive participants) (see below).

Due to gender differences regarding cardiovascular risk and facial affect recognition [48], only men were included in the study. Recruitment was carried out with the aid of the Swiss Red Cross of the Canton of Bern. In detail, members of our study team accompanied the mobile blood-donation unit of the Swiss Red Cross that routinely records BP before blood donation. Male blood donors with elevated or normal BP expressing interest in participating in the study were given study information asking for the following inclusion criteria: age 18–80 years; nonsmoker (≤3 cigarettes per day; to exclude confounding influences of smoking on BP [49]); no current intake of medication. In addition, eligible hypertensive participants provided blood samples for routine assessment of serum creatinine, calcium, sodium, and potassium to test for potential cases with secondary hypertension. No eligible hypertensive participant showed critical values indicative of secondary hypertension post-hoc and all participants were included. Notably, calcium, sodium, and potassium could not be analyzed in eight hypertensive participants, in two of them, creatinine could not be analyzed either. To rule out diabetes, we measured HbA1c in all participants. HbA1c could not be assessed in two hypertensive, five normotensive, and three participants with white coat hypertension because of technical problems. Apart from hypertension-related criteria, normotensive controls had to meet the same inclusion and exclusion criteria as hypertensive participants verified by telephone interview using an extensive health questionnaire [46, 50]. Subjects who reported any chronic or acute clinical psychiatric disorders, current use of medication, heart disease, elevated cholesterol, elevated blood sugar and diabetes, liver and renal diseases,

chronic obstructive pulmonary disease, rheumatic diseases, HIV, cancer, current infectious diseases, regular strenuous physical exercise, smoking, alcohol, and illicit drug abuse were not eligible for study participation.

All 145 participants of the baseline assessment were invited for the *follow-up assessment* scheduled between 2 and 5 years later with a final sample of 72 participants completing both assessments (*mean* (*standard error of the mean* (*SEM*)) = 3.06 (0.08)). Apart from study exclusion due to start of regular BP medication intake (n = 11), reasons for drop-out comprised: severe illness (n = 7), no interest (n = 10), not reachable by phone (n = 11), lack of time (n = 17), or no reason given (n = 17). Notably, we initially intended to schedule for a 2-year follow-up period that had to be prolonged due to organizational reasons. Drop-out rates did not differ between study groups ($\chi^2(2) = 1.35$, $p = .51$).

Assessment of Essential Hypertension

Classification of essential hypertension and normotension was based on a two-step assessment procedure to allow for detection of white coat and masked hypertension and thus, to ensure essential hypertension status validity. (a) *Home BP measurement.* Following written instructions, each interested candidate was instructed to measure BP on three separate days at home using sphygmomanometry (Omron M6; Omron Healthcare Europe B.V., Hoofdorp, Netherlands). Home BP measurements were to be obtained in a seated position after a 15-minute rest twice per day (once in the morning and once in the evening). Based on a maximum of six home BP measurements, participants were preliminary classified as hypertensive if the average home SBP was ≥135 mm Hg and/or the average home DBP was ≥85 mm Hg according to recommendations for home or ambulatory BP measurements [51]. Correspondingly, participants were preliminary classified as normotensive if average home SBP was <135 mm Hg and average home DBP was <85 mm Hg. (b) *Study BP measurement*. To verify the home-measurement-based preliminary classification of each participant as hypertensive or normotensive, trained personnel obtained up to three additional seated baseline BP measurements each after a 15-minute rest (three measurements: n = 126; two measurements: n = 19). BP was again assessed by means of sphygmomanometry (Omron M6; Omron Healthcare Europe B.V., Hoofdorp, Netherlands). For classification regarding study BP measurements, we applied the regular definition of hypertension based on office or clinic BP measurements and classified participants as hypertensive if their average study SBP was ≥140 mm Hg and/or their average study DBP was ≥90 mm Hg [51, 52]. Participants were classified as normotensive if their average study SBP was <140 mm Hg and their average study DBP was <90 mm Hg.

Based on this two-step procedure, we recruited a total of 145 participants including 54 hypertensive and 65 normotensive participants with consistent group assignment based on both, home BP and study BP measurements. Three participants who were hypertensive based on self-report did not measure their BP at home due to technical problems and were classified according to study BP measurements only, rendering 57 hypertensive participants in total. Twenty-three participants deviated between home and study BP assessment with 16 participants displaying normotensive home BP and hypertensive study BP (white coat hypertension) and 7 participants displaying hypertensive home BP and normotensive study BP (masked hypertension).

Experimental Procedure

All participants abstained from caffeine and alcohol consumption for 24 hours and consumed a semi-standardized breakfast following written instructions prior to arrival at the lab at 8:00 A.M. Besides the assessment of height and weight as well as blood sampling for secondary hypertension screening, three seated *study baseline BP measurements* were performed each after a 15-minute rest. These study baseline BP measurements were used (a) for the final classification of essential hypertension or normotension as described above and (b) to calculate mean arterial pressure (MAP) (see below). The mixed emotion paradigm to assess emotion recognition (see below) started at 9:45 A.M. The experimental procedure was repeated at the follow-up assessment.

Mixed Emotion Paradigm

To assess emotion recognition, we used the mixed emotion forced-choice paradigm by Kaiser et al. [45]. Facial pictures of two male and two female actors were chosen from the "NimStim set of facial expression" [53] that we morphed from 30% to 70% of one emotional expression to another in 10% steps. This procedure resulted in a series of five different affect intensity pictures with blends of two morphed basic emotions, for example: 30%-anger/70%-sadness, 40%-anger/60%-sadness, 50%-anger/50%-sadness, 60%-anger/40%-sadness, and 70%-anger/30%-sadness (see Supplementary Fig. S1). Affect intensity pictures comprised six blended emotion-combinations: anger to sadness, anger to fear, anger to happiness, fear to sadness, fear to happiness, and happiness to sadness. All pictures were projected separately on a screen in a randomized order. In a forced-choice decision task, participants were asked to choose as quickly as possible which of the two possible emotions was displayed by the presented facial expression. A corresponding button press stopped the presentation and another face was displayed.

Pictures were randomly presented within two blocks for each blended emotion-combination (e.g., anger-fear). Each block contained 20 pictures of all four faces in all five affect intensities in a randomized order. In total, 12 blocks of the six blended emotion combinations were presented rendering a total of 240 pictures (four faces × five affect intensities × two blocks × six blended emotion-combinations). We averaged the percentage of anger recognition for each pairing of anger intensity (30/70%, 40/60%, 50/50%, 60/40%, 70/30%) with anger-other emotion combination (anger-fear, anger-happiness, anger-sadness), based on eight button presses (four faces × two blocks). We calculated *anger recognition accuracy* as the difference between an individual's percentage of anger recognition and the actually displayed anger intensity (30%–70%, see Fig. 1B and C displaying emotion recognition accuracy of five emotion intensities averaged over the different emotion combinations). The closer to zero the accuracy score is, the more accurate the facial expression had been recognized, with positive scores indicating overrating and negative scores indicating underrating of the respective emotion. We accordingly averaged the percentages of recognition of fear, happiness, and sadness and calculated emotion recognition accuracy of non-anger emotions.

Trait Anger

We used the *trait anger* subscale of the German version of the State Trait Anger Expression Inventory to assess the affective dimension of anger [8, 54]. The trait anger scale asks people to rate themselves with ten items such as "I am quick tempered" (German: "Ich werde schnell ärgerlich") on a four-point Likert scale (1 = never to 4 = almost always). We summarized items to compute the total trait anger score with higher scores indicating a greater level of trait anger. The trait anger scale has previously shown excellent psychometric quality [55]. In our study, n = 17 participants did not fill out the questionnaire and trait anger scores were available for n = 128 participants with acceptable internal consistency in our sample (Cronbach's α =.77).

Statistical Analyses

Data were analyzed using SPSS (Version 26.0) statistical software packages for Macintosh (IBM SPSS Statistics, Chicago, IL, USA) and presented as mean ± *SEM*. All tests were two-tailed with the significance level set at $p < .05$.

We a-priori calculated power-analyses using the statistical software G*Power for Macintosh (Version 3.1.9.6; Heinrich Heine University Düsseldorf, Germany). Expecting effects of small to medium effect size [43] of f = .15 with a power of .90 and an average correlation of the repeated measures of r = .11 in a conservatively calculated 2 (groups) × 5 (affect intensities) repeated measures analysis of variance (ANOVA) for one emotion combination, the required sample size is N = 138 in cross-sectional analyses. For prospective analyses, the required sample size to detect medium effects of R^2 = .13 in linear regression analyses is N = 73.

For all participants, we calculated MAP at baseline and follow-up assessment based on the up to three study BP measurements by the formula MAP = (2/3*mean study DBP) + (1/3*mean study SBP) as well as body mass index (BMI) by the formula BMI = kg/m^2. As MAP had greater predictive power for cardiovascular mortality in the general population as compared to SBP and DBP separately [56], we used MAP as combined BP measure in our linear analyses. Analyses considering trait anger were conducted threefold: first, with missings excluded (trait anger missings in cross-sectional analyses: n = 17 (nine normotensive participants, six hypertensive participants, two participants with white coat or masked hypertension); in prospective analyses: n = 3 (two normotensive participants, one hypertensive participant)) as depicted in tables and figures, second, by replacing missings with group means of trait-anger scores, and third, by replacing missings using multiple imputation as implemented in SPSS (Version 26.0).

All data were tested for normal distribution and homogeneity of variance using Kolmogorov–Smirnov and Levene's tests prior to statistical analyses. Statistical analyses were conducted with original untransformed data because (a) our sample size is larger than n = 30 [57], (b) we used AN(C)OVAs, that are in general relatively robust against violations of normality [58–62], and (c) data transformation did not improve distribution of skewed emotion recognition accuracies. Furthermore, effect size parameters f were calculated from partial η^2 (η^2_p) using G*Power for Macintosh (Version 3.1.9.6; Heinrich Heine University Düsseldorf, Germany). Effect size parameters f (effect size conventions f: .10 = small; .25 = medium; .40 = large [63]) and R^2 changes (ΔR^2; effect size conventions R^2: .02 = small; .13 = medium; .26 = large [63]) are reported where appropriate.

To compare group characteristics at baseline and test for differences between follow-up and drop-out participants, we used independent t-tests and χ^2-tests where appropriate.

Emotion recognition in essential hypertension

To test whether hypertensive participants differ from normotensive participants in anger recognition accuracy, we applied the following procedure: first, we calculated a repeated measures ANOVA with group as independent variable and two factors representing anger recognition accuracy as dependent variable. The first factor consists of the three displayed contrasting emotion combinations

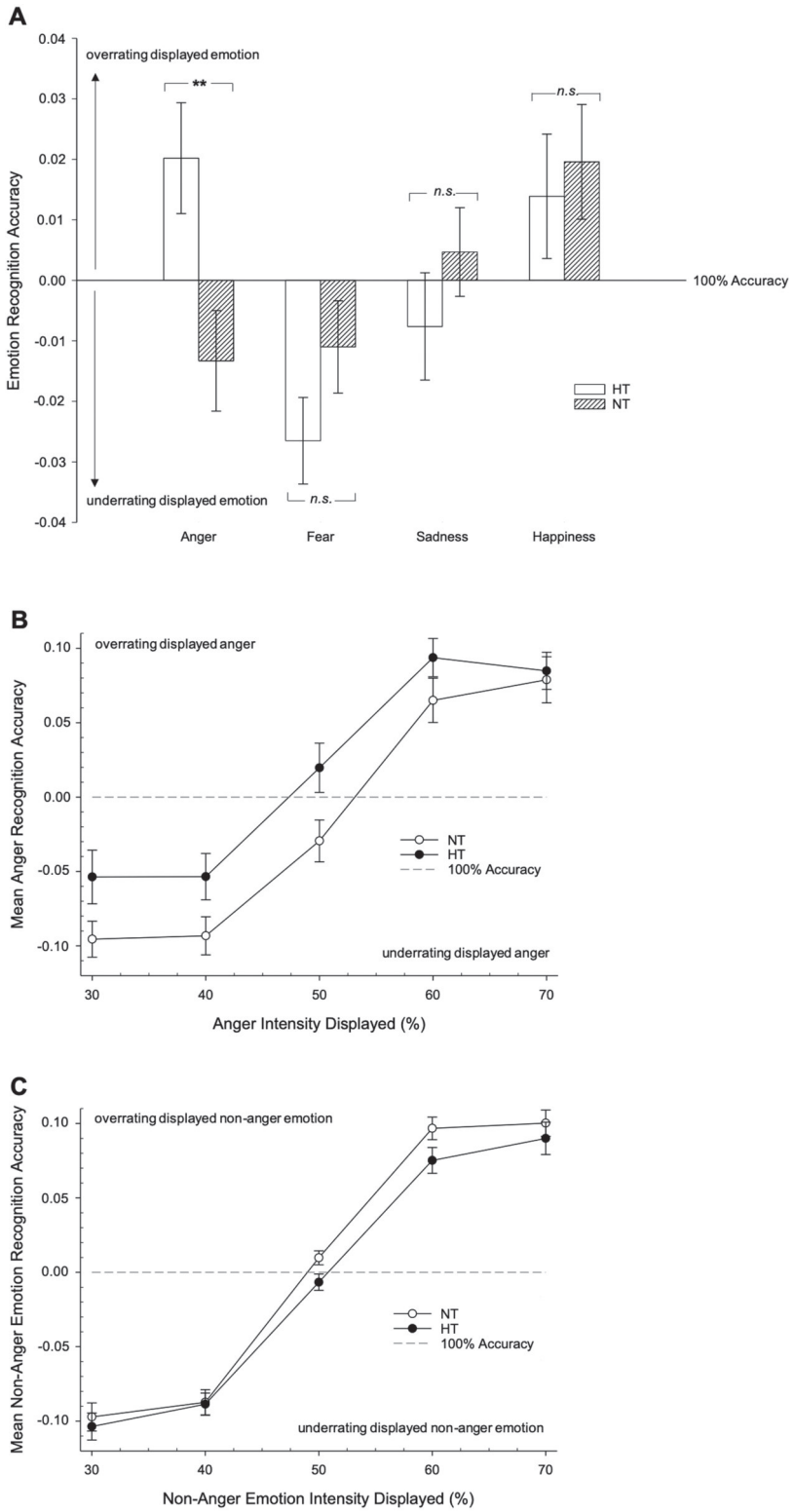

Fig. 1. Cross-sectional analyses: (A) Emotion recognition accuracy of anger, fear, sadness, and happiness averaged over all affect intensities and the different emotion combinations in hypertensive (HT) compared to normotensive (NT) participants ± standard error of the mean (*SEM*). Over all affect intensities and contrasting emotion combinations, HT significantly differed from NT in anger recognition accuracy ($p = .008$), but not in emotion recognition accuracy of other emotions. Asterisks indicate significant group differences between HT and NT (**$p < .01$; *n.s.* = not significant). (B) *Anger* recognition accuracy of the five displayed anger intensities averaged over three anger-other emotion combinations in HT compared to NT ± *SEM*. (C) *Non-anger emotion* recognition accuracy of the five displayed non-anger emotion intensities averaged over the different emotion combinations (i.e., average accuracy of displayed fear, sadness, and happiness per intensity) in HT compared to NT ± *SEM*.

(*anger-fear*, *anger-happiness*, *anger-sadness*), and the second factor comprises the five displayed affect intensities of anger (30/70%, 40/60%, 50/50%, 60/40%, 70/30%). Given age effects in emotion recognition [64] and computer-based tasks [65, 66], we conducted all analyses without and with controlling for age as covariate. Second, we tested for linear associations between anger recognition accuracy and MAP as a continuous measure of hypertension by recalculating repeated measures AN(C)OVAs but using MAP as the independent variable instead of group, again without and with covarying for age. Third, to test whether trait anger relates to the hypothesized associations between hypertension/MAP and anger recognition accuracy, either directly, or as a moderating factor, we repeated the above-described two-step procedure entering trait anger without and with the interaction trait anger-by-group (or trait anger-by-MAP).

We performed the above-described two-step procedure accordingly for *fear*, *happiness*, and *sadness* recognition accuracy as dependent variables.

Prediction of MAP change in prospective follow-up assessment

To test for potential *direct associations*, we calculated hierarchical linear regressions with MAP change (MAP follow-up minus baseline assessment) as dependent variable and *mean* anger recognition accuracy (i.e., anger recognition accuracy averaged over all affect intensities and anger other-emotion combinations) and/or trait anger as independent variable(s). To test for *moderation effects*, we additionally entered the corresponding interaction terms between *mean* anger recognition accuracy and trait anger as independent variables. All prospective analyses were conducted twice: first, including participants with consistent group assignment only (n = 62), and second, including all participants (n = 72) as depicted in the prospective analyses figure with missing trait anger scores excluded. Whereas participants with antihypertensive medication intake at follow-up were a priori excluded (n = 11), we controlled for blood lipids and diabetes medication intake (n = 6) as well as any other medication intake (n = 7) that started since baseline. Each analysis was performed without and with controlling for baseline age, baseline MAP [67], BMI changes (BMI follow-up minus baseline assessment), and medication intake as potential confounders as well as days between baseline and follow-up assessment. The latter covariate was needed to at least statistically control for the follow-up differences ranging from 2 up to 5 years.

Notably, all interaction terms were calculated after prior z-transformation of the respective composing variables, and moderation testing comprised z-transformed independent variables.

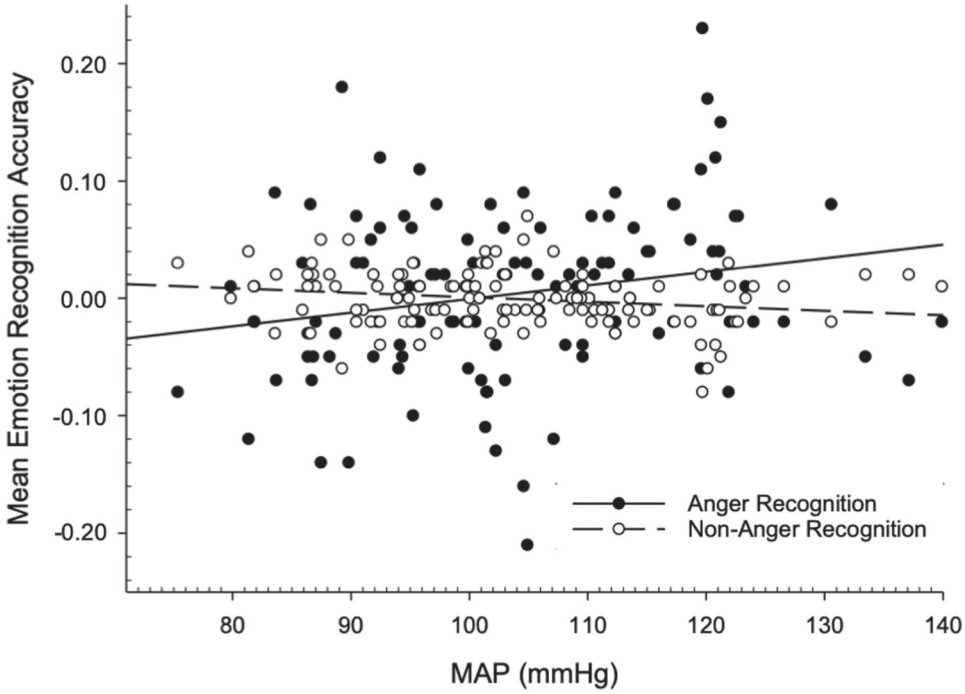

Fig. 2. Cross-sectional analyses: Linear associations between mean arterial pressure (MAP) and anger recognition accuracy averaged over all affect intensities and anger-other emotion combinations (solid line and black dots) and between MAP and non-anger recognition accuracy (i.e., accuracy of displayed fear, sadness, and happiness) averaged over all affect intensities and the different emotion combinations (dotted line and white dots). Over all affect intensities and contrasting emotion combinations, MAP significantly related to anger recognition accuracy (p = .019), but not to emotion recognition accuracy of other emotions.

Results

Group Characteristics

Table 1 depicts group characteristics of the 145 participants studied at baseline and group characteristics of the 72 participants studied during the follow-up assessment. As expected, hypertensive participants displayed higher average SBP, DBP, MAP, and BMI compared to normotensive participants ($ps < .001$) as well as comparable age ($p = .54$) at baseline. Furthermore, hypertensive and normotensive participants did not significantly differ in their trait anger scores ($p = .16$) or any other group characteristics ($ps \geq .42$). There were no significant group differences between the follow-up and the drop-out participants in any baseline characteristic (see Table 2).

Cross-sectional Analyses: Emotion Recognition in Essential Hypertension

Anger recognition

Over all affect intensities and contrasting anger emotion combinations, hypertensive participants significantly differed from normotensive participants in anger recognition accuracy ($F(1, 120) = 7.37$, $p = .008$, $\eta^2_p = .058$, $f = .25$; with age as covariate: $F(1, 119) = 7.40$, $p = .008$, $\eta^2_p = .059$, $f = .25$; see Fig. 1A and B). More precisely, hypertensive participants displayed lower accuracy in recognizing anger in terms of comparably overrating displayed anger. These group differences remained when additionally controlling for trait anger without and with missing trait anger scores replaced either with group means or multiple imputation ($ps \leq .011$) with trait anger not being related to anger recognition ($ps \geq .20$). Moreover, trait anger did not moderate the observed group differences in anger recognition ($ps \geq .22$).

Complementary analyses using MAP as linear independent variable instead of group confirmed the above findings: over all affect intensities and contrasting anger emotion combinations higher MAP significantly related to anger recognition accuracy in that displayed anger is overrated with increasing MAP ($F(1, 120) = 5.62$, $p = .019$, $\eta^2_p = .045$, $f = .22$; with age as covariate: $F(1, 119) = 5.70$, $p = .019$, $\eta^2_p = .046$, $f = .22$; see Fig. 2). Again, these associations were independent of trait anger without and with missing trait anger scores replaced either with group means or multiple imputation ($ps \leq .028$) while trait anger did not relate to anger recognition ($ps \geq .17$). Also, trait anger did not moderate the association between MAP and anger recognition ($ps \geq .17$).

Non-anger emotion recognition

We further tested whether hypertensive participants would also differ from normotensive controls in non-anger emotion recognition (i.e., fear, happiness, or sadness), again by comparing groups and complementary association testing with MAP. There were no significant findings except for a borderline significant finding regarding sadness recognition accuracy in relation to MAP, notably in the opposite direction as compared to anger recognition and not independent of age (without age as covariate $p = .087$; with age as covariate $p = .12$). Figure 1A and C depict non-anger emotion recognition accuracy in hypertensive and normotensive participants, and Fig. 2 depicts corresponding relations with MAP.

Prospective Analyses: Prediction of MAP Change by Anger Recognition, Trait Anger, and Their Interaction

Direct effects

We calculated hierarchical linear regression analyses with MAP change as dependent variable and mean anger recognition accuracy and/or trait anger without and with missing trait anger scores replaced (with group means/ multiple imputation) as independent variables. There were no direct associations with MAP change either when considering participants with consistent baseline group assignment only (mean anger recognition accuracy: $ps \geq .76$; trait anger: $ps \geq .55$) or when considering all follow-up participants including white coat and masked hypertension (mean anger recognition accuracy: $ps \geq .74$; trait anger: $ps \geq .75$). Controlling for the a-priori defined set of confounders (i.e., baseline age, baseline MAP, BMI change, days between baseline and follow-up assessment, and medication intake) did not significantly change these results (mean anger recognition accuracy: $ps \geq .46$; trait anger: $ps \geq .18$). Notably, of these confounders, higher baseline MAP ($\beta s \leq -.25$, $ps \leq .039$, $\Delta R^2 s \geq .05$) related to decreases or lower increases in MAP from baseline to follow-up. Higher BMI increases ($\beta s \geq .44$, $ps < .001$, $\Delta R^2 s \geq .19$) related to higher MAP increases from baseline to follow-up.

Moderation effects

Moderation testing revealed that the interaction between mean anger recognition accuracy and trait anger without missings replaced significantly predicted MAP change both, in participants with consistent baseline group assignment ($\beta = .38$, $p = .006$, $\Delta R^2 = .13$) and when considering all follow-up participants including white coat and masked hypertension ($\beta = .33$, $p = .009$, $\Delta R^2 = .10$). These associations remained when replacing missing trait anger scores with group means or multiple imputation ($ps \leq .009$). Notably, controlling for confounders did not change the significance of the observed moderation effects ($ps \leq .043$). Again, higher BMI increases ($\beta s \geq .46$, $ps < .001$, $\Delta R^2 s \geq .18$) related to higher MAP increases over time and higher baseline MAP (marginal

Table 1. Baseline and follow-up group characteristics of medication-free hypertensive participants, normotensive participants, and participants with white coat or masked hypertension

	Baseline group characteristics (N = 145)			p			Follow-up group characteristics (N = 72)		
	NT n = 65	HT n = 57	wc/m HT n = 23	NT vs. HT	NT vs. wc/m HT	HT vs. wc/m HT	NT n = 40	HT n = 22	wc/m HT n = 10
Age (years)	49.82 ± 1.50 (25–78)	51.14 ± 1.53 (26–74)	53.43 ± 3.08 (21–73)	.54	.25	.46	51.90 ± 1.91 (31–81)	56.14 ± 2.72 (31–77)	53.30 ± 4.88 (33–73)
BMI (kg/m^2)	25.27 ± 0.30 (19.78–30.85)	28.71 ± 0.55 (20.35–38.86)	26.96 ± 0.73 (21.55–35.26)	<.001***	.040*	.077	24.89 ± 0.38 (20.07–28.59)	29.14 ± 1.00 (23.40–40.44)	28.10 ± 1.13 (23.72–33.44)
BP (home)		n = 54							
SBP (mm Hg)	121.87 ± 0.82 (105.17–133.83)	145.26 ± 1.04 (132.17–162.33)	130.37 ± 2.16 (115.67–148.33)	<.001***	.001**	<.001***			
DBP (mm Hg)	71.72 ± 0.71 (60.00–82.33)	87.00 ± 0.89 (75.67–103.00)	77.27 ± 1.14 (68.33–84.83)	<.001***	<.001***	<.001***			
BP (study)									
SBP (mm Hg)	126.57 ± 1.05 (108.67–139.67)	153.60 ± 1.48 (129.33–189.67)	141.60 ± 2.67 (109.50–177.00)	<.001***	<.001***	<.001***	128.80 ± 1.85 (102.33–161.67)	151.98 ± 4.15 (102.67–181.67)	147.10 ± 4.27 (123.67–167.00)
DBP (mm Hg)	77.59 ± 0.87 (58.33–88.67)	95.47 ± 1.14 (73.67–115.00)	88.15 ± 1.26 (75.00–96.67)	<.001***	<.001***	<.001***	78.33 ± 1.22 (55.67–92.67)	91.06 ± 2.73 (65.67–112.00)	87.83 ± 1.59 (81.00–93.67)
MAP (mm Hg)	93.92 ± 0.86 (75.33–104.56)	114.85 ± 1.17 (95.78–139.89)	105.97 ± 1.54 (86.50–117.44)	<.001***	<.001***	<.001***	95.15 ± 1.34 (71.22–111.78)	111.37 ± 3.00 (79.56–135.22)	107.59 ± 2.05 (95.22–115.78)
MAP change (mm Hg)							1.35 ± 1.06 (−13.8–11.44)	−2.97 ± 2.39 (−40.00–14.22)	0.89 ± 1.83 (−7.00–8.72)
Trait anger	n = 56 16.63 ± 0.45 (10–29)	n = 51 17.69 ± 0.60 (11–29)	n = 21 18.14 ± 0.71 (12–24)	.16	.079	.66			
High school degree (Swiss "Matura" or "Fachhochschulreife") (% [N])	n = 57 49.12 (28)	n = 53 41.51 (22)	39.13 (9)	.42	.42	.85	n = 38 57.90 (22)	45.45 (10)	50.00 (5)
Working status							n = 39		
Working (% [N])	86.15 (56)	84.21 (48)	65.22 (15)	.76	.029*	.060	84.62 (33)	68.18 (15)	60.00 (6)
Retired (% [N])	13.85 (9)	14.04 (8)	34.78 (8)	.98	.029*	.036*	15.39 (6)	27.27 (6)	40.00 (4)
Days (baseline till follow-up)							1091.68 ± 40.65 (651–1923)	1102.45 ± 48.90 (875–1598)	1242.30 ± 75.70 (952–1678)

Values are means ± *SEM* (range) if not indicated differently.

BMI body mass index; *BP* blood pressure; *SBP* systolic blood pressure; *DBP* diastolic blood pressure; *MAP* mean arterial pressure; *MAP change* follow-up MAP minus baseline MAP; *NT* normotensive participants; *HT* hypertensive participants; *wc/m HT* white coat or masked hypertension; *n* sample size; deviating sample sizes of a parameter are indicated.

*p < .05.

**p < .01.

***p < .001.

Table 2. Comparison of baseline group characteristics between eligible participants completing follow-up assessment (n = 72) and participants who dropped out (n = 73)

	Follow-up sample (n=72)			Drop-out participants (n=73)			Follow-up vs. drop-out		
	NT n = 40	HT n = 22	wc/m HT n = 10	NT n = 25	HT n = 35	wc/m HT n = 13	p		
							NT	HT	wc/m HT
Age (years)	49.50 ± 1.92 (28–78)	53.73 ± 2.74 (28–74)	50.40 ± 4.85 (29–70)	50.32 ± 2.45 (25–78)	49.51 ± 1.78 (26–74)	56.77 ± 4.01 (21–73)	.79	.18	.40
BMI (kg/m^2)	24.89 ± 0.40 (19.78–28.87)	29.21 ± 1.00 (20.35–38.86)	27.53 ± 1.04 (23.25–32.99)	25.88 ± 0.45 (21.93–30.85)	28.40 ± 0.63 (22.67–37.74)	26.53 ± 1.03 (21.55–35.26)	.11	.47	.51
BP (study)									
SBP (mm Hg)	126.48 ± 1.31 (109.33–139.67)	154.06 ± 2.71 (135.67–188.00)	142.62 ± 5.38 (109.50–177.00)	126.73 ± 1.80 (108.67–138.00)	153.30 ± 1.74 (129.33–189.67)	140.81 ± 2.50 (121.33–157.00)	.91	.81	.75
DBP (mm Hg)	77.47 ± 1.13 (58.33–87.67)	94.47 ± 2.31 (73.67–113.67)	88.73 ± 1.80 (75.00–94.00)	77.79 ± 1.37 (62.67–88.67)	96.10 ± 1.18 (86.00–115.00)	87.71 ± 1.81 (78.67–96.67)	.86	.49	.70
MAP (mm Hg)	93.80 ± 1.11 (75.33–104.56)	114.33 ± 2.30 (95.78–137.11)	106.69 ± 2.68 (86.50–117.44)	94.10 ± 1.40 (79.83–104.56)	115.17 ± 1.26 (104.89–139.89)	105.41 ± 1.87 (93.11–115.89)	.87	.73	.69
Trait anger	n = 38 16.79 ± 0.60 (10–29)	n = 21 18.10 ± 1.05 (12–29)	n = 10 18.50 ± 0.90 (15–22)	n = 18 16.28 ± 0.61 (12–22)	n = 30 17.40 ± 0.73 (11–28)	n = 11 17.82 ± 1.13 (12–24)	.60	.58	.65

Values are means ± *SEM* (range).

BMI body mass index; *BP* blood pressure; *SBP* systolic blood pressure; *DBP* diastolic blood pressure; *MAP* mean arterial pressure; *NT* normotensive participants; *HT* hypertensive participants; *wc/m HT* white coat or masked hypertension; *n* sample size; deviating sample sizes of a parameter are indicated.

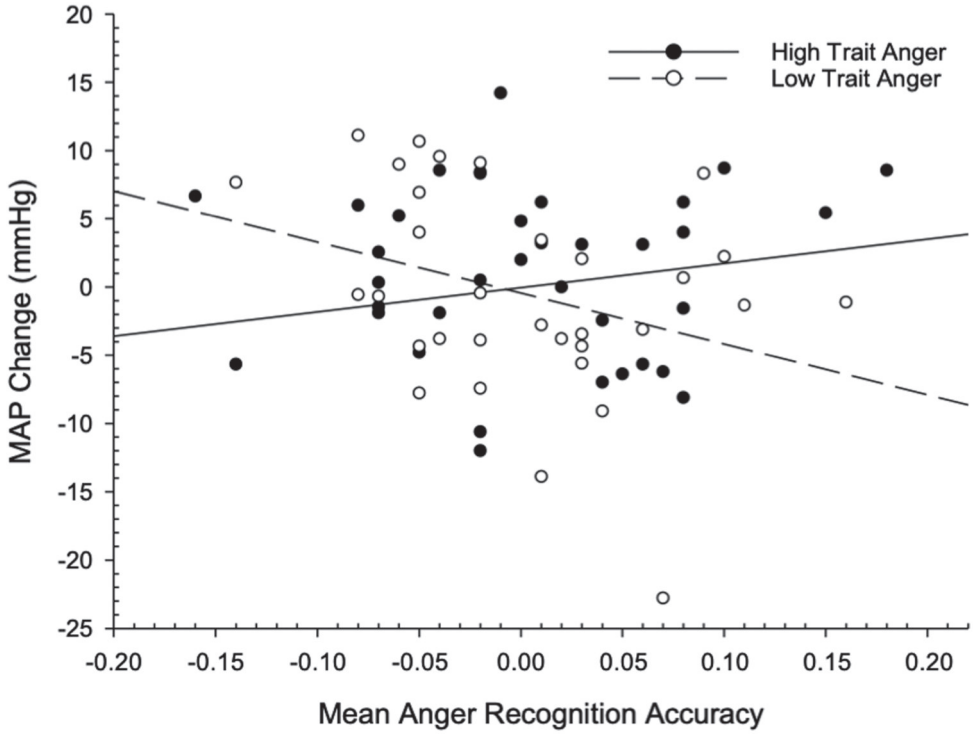

Fig. 3. Prospective analyses: Moderation by trait anger: Mean anger recognition accuracy predicting mean arterial pressure (MAP) change depending on trait anger. The interaction between mean anger recognition accuracy and trait anger significantly predicted MAP change (p = .009): Overrating of displayed anger predicted higher MAP increases from baseline to follow-up if trait anger was high (solid line and black dots), but not if trait anger was low (dotted line and white dots). For illustrative purposes (but not for modeling and testing), we categorized trait anger levels based on median split and depict levels below median as "low trait anger" and levels above median as "high trait anger".

significantly) related to decreases or lower increases in MAP over time ($\beta s \leq -.21$, $ps \leq .083$, $\Delta R^2 s \geq .03$).

Figure 3 depicts moderation by trait anger: overrating of displayed anger predicted higher MAP increases from baseline to follow-up only if trait anger was high but not if trait anger was low.

Discussion

Here, we investigated cross-sectionally whether essential hypertensive men differ from normotensive men in anger recognition and trait anger. We additionally examined prospective associations of anger recognition and trait anger with BP changes over an average 3-year follow-up period.

Cross-sectionally, we found that hypertensive men overrated displayed anger as compared to normotensive men. In other words, hypertensive men tend to recognize anger more often when evaluating ambiguous facial expressions comprising anger plus another emotion. These results were confirmed in complementary analyses using MAP as continuous hypertension measure, suggesting a linear relationship with increasing BP. We argue that the observed recognition bias toward anger in hypertension is in line with previous studies reporting impaired emotion recognition in the context of hypertension [37–44] due to the following: while we used pictures displaying mixed facial affect allowing to identify a recognition bias toward one emotion over others, these studies presented facial expressions displaying prototypical single basic emotions. Emotion recognition accuracy was then averaged over several emotions by calculating the proportion of correctly recognized emotions ("hits") to total recognized emotions ("hits + errors"). Notably, observers tend to perceive other emotions in addition to displayed prototypical basic emotion in facial expressions at least to a certain degree [68]. Given this, our findings suggest that hypertensive men correctly identify displayed anger, but may overrate anger in facial expressions of non-anger emotions leading to increased error rates in total, that is, over all emotions. As a consequence, emotion recognition accuracy as proportion of hits to hits + errors would be impaired as previously observed in the context of hypertension overall emotions [37–44], but not when considering anger separately [37, 44]. Notably, we could not observe relevant group differences in non-anger emotion recognition or relations to MAP.

Furthermore, we did not find group differences between hypertensive and normotensive men in trait anger, and trait anger was neither associated with anger recognition accuracy nor did it moderate the associations

between anger recognition and group or MAP. In line with previous studies that did not find higher trait anger with increasing resting BP or hypertension [15–18], our findings further question an independent role for trait anger in essential hypertension.

Prospectively, our analyses did not support expected direct associations between anger recognition or trait anger and BP increases over an average 3-year follow-up period. However, the interaction between mean anger recognition accuracy and trait anger predicted MAP changes from baseline to follow-up as expected. With regard to *clinical implications* these results suggest that in men, high trait anger together with a recognition bias toward anger contributes to further BP increases and thus progression of hypertension and related cardiovascular risk. This finding is in line with the personality in context theory that personality does not reflect an individual in isolation but rather an individual within interpersonal or social context [32, 33]. Our results indicate that trait anger seems to require situational features such as perceiving anger in facial expressions of interaction partners to elicit anger, or feelings of anger, respectively. Moreover, it is conceivable that trait anger predisposes to select anger-related situations and to modulate social situations [32]. The observed interaction can be interpreted based on the allostatic load model [11]: Given that feelings of anger are characterized by heightened sympathetic activation [9, 10], frequent anger feelings and thus increased sympathetic activation may accumulate over time, and result in chronic BP elevation. Notably, these implications should be treated with caution, as our participants were hypertensive and normotensive men. It remains to be elucidated whether our findings and thus the resulting implications also apply to women with hypertension.

We can only speculate about *mechanisms* underlying the observed anger recognition bias in hypertension in our study participants. On the one hand, the observed anger recognition bias could predispose to BP increases and thus hypertension development. According to the PAM [34], in the sense of anger contagion, perceiving anger in interaction partners likely results in own feelings of anger with concomitant increased sympathetic activation [9] that may accumulate over time [11]. At the same time, impaired emotion recognition that others observed in hypertension [37–44] may lead to inadequate social behavior [69] resulting in reduced quality of social relationships with lower social support, social distancing, and isolation. As a consequence, stress-buffering is reduced [70, 71], giving rise to more stressful experiences and further BP increases. On the other hand, an anger recognition bias could be a consequence of increased BP or hypertension. To respond quickly to potential environmental threats in social contexts, angry faces are in general detected more quickly and accurately than happy or other nonthreatening faces, referred to as anger superiority effect [72, 73]. Due to their BP hyperactivity, individuals with hypertension might be on higher alert and thus more sensitive to perceive threatening stimuli. This may result in an overestimation of threatful stimuli such as angry faces. It is also conceivable that both proposed mechanisms interact to increase BP. Additional mechanisms may comprise integrated neurophysiological pathways that control both, resting BP and affect [41, 42, 74, 75].

As outlined before, an anger recognition bias, especially if combined with trait anger, may contribute to the development and progression of essential hypertension and related cardiovascular risk. *Therapeutic implications* include to not only address anger conventionally with regard to individual's responses to anger [76] but to additionally target anger recognition by training the perception of the social environment, especially emotion recognition of facial affect. For example, experimental modification of biases in emotion recognition by promoting perception of happiness over anger in ambiguous facial expressions has been shown to decrease state anger [77]. Further positive effects of emotion recognition training may comprise improved social behavior [69] that in turn can increase social support with its beneficial effects on cardiovascular health [70, 71]. Again, the generalization of our findings to populations other than men with and without hypertension is unclear.

Strengths of our study include the prospective design, which allowed participant monitoring over an average follow-up period of 3 years. Second, we had a rigid hypertension assessment procedure ensuring hypertension status validity by including assessment of both, home and study BP combined with exclusion of the diagnosis of secondary hypertension. This procedure also allows to detect white coat and masked hypertension. Third, in real-life social contexts facial expressions of emotions are proposed to appear not as straightforward as prototypical facial expressions of basic emotions, but rather ambiguous [78, 79]. Additionally, depending on the situation, natural emotional expressions are more likely subtle or mixed. Therefore, our mixed emotion paradigm may be more ecologically valid than prototypical emotion recognition assessment. Fourth, combined investigation of both, trait anger and anger recognition of mixed facial affect, may shed light on how these factors interact in the development of essential hypertension, revealing more specific potential mechanisms. Last, we controlled for a variety of potential confounders. *Limitations* of our study are that the generalizability of our results is limited to normotensive and hypertensive but otherwise healthy and medication-free men. Also, we recruited blood donors, which however should not further limit the generalizability of our findings [80].

Second, ecological validity of our paradigm is unclear. Our displayed mixed facial expressions were morphed static pictures and do not necessarily reflect natural facial expressions. Also, our displayed facial expressions were isolated from body motion, posture, and prosody limiting the amount of perceivable information [81]. Third, modern conceptualizations consider trait anger to not only have an affective dimension as measured by the Spielberger trait anger scale [8] but also behavioral and cognitive dimensions with potentially different impacts on cardiovascular health [5, 82]. Finally, participant number of our prospective assessment was substantially reduced. Whereas start of antihypertensive medication intake was a major exclusion reason inherent in our study design, we feel that the reduced and less flexible offers for follow-up assessment time-points due to organizational reasons may have contributed to a higher drop-out rate. Notably, drop-out and follow-up participants did not substantially differ in their characteristics (see Table 2).

Taken together, our findings provide for the first-time evidence for an anger recognition bias in essential hypertension in terms of overrating anger displayed in facial expressions of mixed emotions. Noteworthy, in our study, normotensive and hypertensive men did not differ in trait anger. The proposed clinical significance of our findings is supported by our prospective data with anger recognition interacting with trait anger in the prediction of BP increases over an average period of 3 years. Future studies are needed to verify the observed results obtained in hypertensive men and whether they extend to women with hypertension since gender differences in facial affect recognition have been described [48]. In addition, the potential role of anger recognition and trait anger in hypertension development and ecological validity of the mixed emotions paradigm warrant further investigation.

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Compliance with Ethical Standards

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards: Authors Alisa Auer, Roland von Känel, Ilona Lang, Livia Thomas, Claudia Zuccarella-Hackl, Cathy Degroote, Angelina Gideon, Roland Wiest, and Petra H. Wirtz declare that they have no conflict of interest. The corresponding author signed the disclosure form on behalf of all authors.

Authors' Contributions: Conceptualization and design of the study as well as funding acquisition: Petra H. Wirtz; supervision of the project: Petra H. Wirtz and Roland von Känel; supervision of analyses: Petra H. Wirtz; data acquisition/investigation: Livia Thomas, Claudia Zuccarella-Hackl, Roland Wiest, and Roland von Känel; data curation: Alisa Auer, Cathy Degroote, Angelina Gideon; formal analysis and visualization: Alisa Auer and Ilona Lang; wrote the manuscript: Alisa Auer, Ilona Lang, and Petra H. Wirtz; critical revision of the manuscript: Roland von Känel, Ilona Lang, Livia Thomas, Claudia Zuccarella-Hackl, Cathy Degroote, Angelina Gideon, and Roland Wiest.

Ethical Approval: All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent: It was obtained from all study participants at both assessments

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