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Is there a Link Between Non-HDL Cholesterol and Blood Pressure? An Age and Gender Directed Analysis of 7500 Hypertensive Outpatients

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Abstract: *Background:* Both elevated non-HDL cholesterol (C) and elevated blood pressure (BP) are major risk factors for the development and targets for the prevention of atherothrombotic disease. Possible interactions between these two parameters have not been studied in detail. This investigation aims at looking at values of blood serum cholesterol, blood pressure and possible correlations therein in men and women, with a special focus on the impact of age. *Methods:* A total of 7496 outpatients with arterial hypertension in Austria were recruited. Four age groups were analyzed (≤ 49 years, 50–59 years, 60–69 years and ≥ 70 years). *Results:* Overall, men and women had similar systolic and diastolic blood pressure (158 ± 91 vs 159 ± 19 mmHg and 91 ± 10 vs 91 ± 11 mmHg, respectively; $p < 0.01$) and similar non-HDL C values (165 ± 45 mg/dl; 164 ± 43 mg/dl, resp.; $p < 0.001$). Both, systolic (SBP) and diastolic (DBP) blood pressure correlated highly significantly with non-HDL ($p < 0.001$) in men (mean $r = 0.15$ for SBP and $r = 0.18$ for DBP, both $p < 0.001$). Women showed a weaker correlation with a mean $r = 0.10$ for SBP and $r = 0.09$ for DBP (both $p < 0.001$). With respect to the four age groups, non-HDL C was similar in men between 172 ± 47 and 173 ± 44 mg/dl up to the age of 60 years and declined to 157 ± 43 mg/dl in the group above 70 years of age. In women we detected a rise of non-HDL C by 10 mg/dl around menopause (mean 158 ± 42 mg/dl < 50 years of age, mean 168 ± 42 mg/dl with 50–59 years, afterwards declining to 163 ± 43 mg/dl; $p < 0.01$). SBP was similar in men and women over all age groups (between 157 ± 19 and 159 ± 19 mmHg), whereas DBP declined with increasing age (95 ± 10 to 88 ± 11 mmHg in men; 94 ± 11 to 88 ± 11 mmHg, resp.). Correlations between SBP, DBP and non-HDL cholesterol were greater for men. Women showed an increase around menopause. *Conclusion:* Our results demonstrate a correlation of non-HDL C and blood pressure in hypertensive men of all age groups. In women, menopause has a significant influence not only on lipid levels, but also on correlations to blood pressure.

Key words: non-HDL cholesterol, blood pressure, gender, atherothrombotic disease

Kurzfassung: Gibt es eine Verbindung von Non-HDL Cholesterin zu Blutdruck? Eine auf Alter und Gender fokussierte Analyse von 7500 Patienten mit arterieller Hypertonie. *Hintergrund:* Erhöhtes Non-HDL Cholesterin (C) und erhöhter arterieller Blutdruck (BP) sind maßgebliche Risikofaktoren in der Entwicklung und Ziele der Prävention der atherothrombotischen Krankheit. Mögliche Interaktionen zwischen diesen beiden Parametern wurden bisher nicht im Detail untersucht. Diese Studie setzt Serum-Cholesterinwerte, arteriellen Blutdruck und mögliche Korrelationen bei Männern und Frauen in Bezug, wobei ein spezieller Fokus auf das Alter gelegt wurde. *Methoden:* 7496 hypertensive Patienten wurden in Ordinationen niedergelassener Ärzte in Österreich rekrutiert. Es erfolgte eine Stratifizierung nach 4 Altersgruppen, die analysiert und verglichen wurden: ≤ 49 Jahre, 50–59 Jahre, 60–69 Jahre und ≥ 70 Jahre. *Ergebnisse:* In der Gesamtgruppe hatten Männer und Frauen vergleichbare Blutdruckwerte (158 ± 91 vs. 159 ± 19 mmHg, bzw. 91 ± 10 vs. 91 ± 11 mmHg; $p < 0,01$) und vergleichbare Non-HDL-C-Werte (165 ± 45 mg/dl bzw. 164 ± 43 mg/dl; $p < 0,001$). Sowohl systolischer (SBP) als auch diastolischer (DBP) Blutdruck korrelierten hoch-signifikant zu Non-HDL-C ($p < 0,001$) bei Männern (im Mittel: $r = 0,15$ für SBP und $r = 0,18$ für DBP, beide $p < 0,001$). Frauen zeigten eine schwächere Korrelation von im Mittel $r = 0,10$ für SBP und $r = 0,09$ für DBP (beide $p < 0,001$). Unter Bezugnahme auf die 4 Altersgruppen lagen die Werte für Non-HDL-C bei Männern bis 60 Jahre stabil zwischen 172 ± 47 und 173 ± 44 mg/dl und sanken dann auf 157 ± 43 mg/dl in der Gruppe > 70 Jahre. Bei Frauen konnten wir einen Anstieg des Non-HDL-C um 10 mg/dl um die Menopause feststellen (im Mittel 158 ± 42 mg/dl < 50 Jahren; 168 ± 42 mg/dl zwischen 50 und 59 Jahren, danach Senkung auf 163 ± 43 mg/dl; $p < 0,01$). SBP war ähnlich bei Männern und Frauen aller Altersgruppen (zwischen 157 ± 19 und 159 ± 19 mmHg), wobei der DBP mit zunehmendem Alter sank (95 ± 10 bis 88 ± 11 mmHg bei Männern; 94 ± 11 bis 88 ± 11 mmHg bei Frauen). Korrelationen zwischen SBP, DBP und Non-HDL-C waren größer bei Männern. Frauen zeigten einen Anstieg um die Menopause. *Schlussfolgerung:* Unsere Resultate zeigen die Korrelation von Non-HDL-Cholesterin und Blutdruck bei hypertensiven Männern aller Altersgruppen. Bei Frauen hat die Menopause einen signifikanten Einfluss auf die Lipidwerte und auch die Korrelation von Non-HDL-Cholesterin zu arteriellem Blutdruck. **J Kardiol 2012; 19 (1–2): 11–6.**

Schlüsselwörter: Non-HDL-Cholesterin, Blutdruck, Geschlecht, Atherothrombose

Introduction

In the western world, cardiovascular diseases are the by far leading causes of morbidity and mortality. Even worldwide, ischemic cardiovascular and cerebrovascular diseases account for one fifth of all deaths [1].

The total cardiovascular risk burden in patients is determined by gender, age, genetic predisposition and modifiable risk factors, such as smoking, elevated cholesterol levels, hypertension, visceral obesity and diabetes [2, 3]. A high risk diet and psychosocial stress probably underlie many risk factors, whereas physical activity and moderate alcohol consumption appear to have a preventive effect [2]. The dominant risk factor seems to be an impaired lipid metabolism [2], resulting in high low-density lipoprotein cholesterol (LDL), low high-density lipoprotein cholesterol (HDL) and high triglyceride levels [4]. Non-HDL cholesterol comprises the cholesterol carried by all ApoB100 carrying lipoproteins and is of higher prognostic value for ischemic cardiovascular diseases then low-density lipoprotein (LDL) alone [5]. Morphological studies have shown that these lipoproteins accumulate in the atherosclerotic plaque, thereby explaining the basis for the total cardiovascular risk [6]. As non-HDL comprises all athero-

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genic cholesterol fractions and is an emerging new target for prevention, we selected it as the main parameter of our study.

Long lasting hypertension causes end organ damage, lesions in the artery wall and reduced vascular compliance. With increasing age, human arteries show increasing stiffness. These vessel changes lead to a rise in systolic and a decrease in diastolic blood pressure [7].

Coexistence of multiple cardiovascular risk factors increase an individuals risk which has been demonstrated in large databases, such as INTERHEART, EUROASPIRE or FRAMINGHAM [2, 3, 8].

In the Framingham heart study, a positive correlation (r = 0.12) between LDL cholesterol and blood pressure was shown [9]. In a small scale study similar results were observed [10]. The Oslo study confirmed such a correlation for men under the age of 49 years [11] and the Tromso study [12] detected differences concerning this correlation between men < 55 years of age and women < 50 years of age.

The aim of the present investigation was to evaluate the correlation of non-HDL to blood pressure with respect to differences in gender at all age groups representing in a representative western world population at high risk.

Patients and Methods

Patients

7496 men and women with hypertension were included. Exclusion criteria were age < 18 years and possible pregnancy.

Study Design

A population based scientific project was performed at 965 offices of cardiologists, internists and general practitioners in Austria from March 2004 to September 2008. The present investigation was a cross sectional examination of outpatients with hypertension. In the 7496 patients analyzed, epidemiological data and the main risk factors were assessed. Thus, this study reflects well real world conditions.

Data were collected on the base of regular physician visits. No examinations were performed additionally at patients that would not have been made on a regular base, nor were patients confronted with doctor visits apart from routine visits. The study was performed in accordance to the declaration of Helsinki. Patients have been informed about and gave informed consent to anonymized collection of the data of the respective visit and examination.

Blood pressure was measured at the arteria brachialis using sphygmomanometers in a sitting position with the forearm resting on a table in front of the patient. Pulse pressure was then calculated as the difference of Sitting Diastolic Blood Pressure (SiDBP) to Sitting Systolic Blood Pressure (SiSBP).

Total cholesterol and HDL cholesterol levels were measured by standard laboratory procedures in validated quality controlled laboratories. Non-HDL C was calculated by subtracting HDL C from the total cholesterol value. LDL C was calculated using the Friedewald formula.

Table 1. Patients' Characteristics

	Men		Women		
	Mean	SD	Mean	SD	p
n	3544		3952		
Age (years)	63	12	68	12	< 0.001
BMI (kg/m²)	28	4	28	5	n.s.
Total cholesterol (mg/dl)	217	44	224	41	< 0.001
LDL cholesterol (mg/dl)	132	38	136	38	< 0.001
HDL cholesterol (mg/dl)	52	18	59	18	< 0.001
Triglycerides (mg/dl)	165	71	151	64	< 0.001
Fasting glucose (mg/dl)	109	32	107	31	< 0.001
Uric acid (µmol/l)	6	1	5	1	< 0.001

BMI: body mass index; LDL: low density lipoprotein; HDL: high density lipoprotein; p-values given for differences between men and women

Data Analysis

SPSS Version 17.0 was used for all statistical analyses. Descriptive statistics were performed for mean values and standard deviations as reported parameters were continuous variables and their distribution was close to normal. To assess correlation between blood pressure and non-HDL cholesterol, the Spearman Rho test on rank correlation was used and r^2 was obtained by arithmetic squaring. Levels of significance are reported as p values.

Data were analyzed separately in men and women who were stratified into four distinct age groups (< 49 years, 50–59 years, 60–69 years, and > 70 years of age).

Results

Patient Characteristics

Data of 7496 patients were analyzed. Men represented 47% of the sample (n = 3544) and women 53% (n = 3952), respectively. Hypertension was known for an average of 8 ± 6 years in both groups. 15,5% of men and 9,8% of women had a history of a major atherothrombotic event (p < 0.01). The prevalence of diabetes mellitus was 23.6% for men and 24.0% for women (n. s.).

Table 1 summarizes patients' characteristics separately for men and women.

Non-HDL Cholesterol and Blood Pressure

Sitting systolic blood pressure (SiSBP) was 158 ± 19 mmHg for men and 159 ± 19 mmHg for women. Due to the large sample size, these small differences between the sexes were significant (p < 0,01). Sitting diastolic blood pressure (SiDBP) was equally similar for both sexes at 91 ± 10 vs 91 ± 11 mmHg, respectively (p < 0.01).

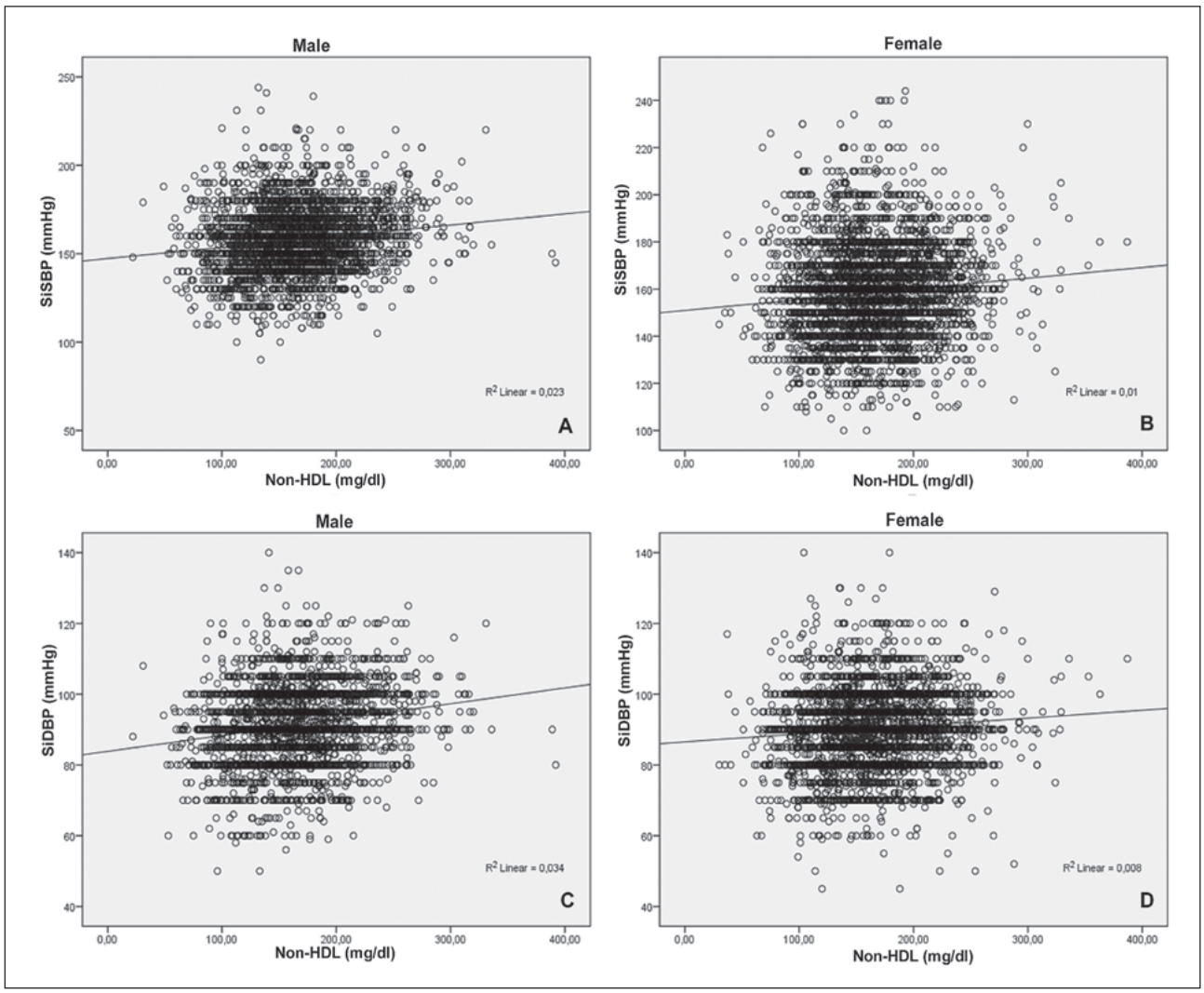

Figure 1. Scatter Plots of Correlation Between Blood Pressure and Non-HDL C in Men and Women. **(A):** SiSBP & Non-HDL, men; **(B):** SiSBP & Non-HDL, women; **(C):** SiDBP & Non-HDL, men; **(D):** SiDBP & Non-HDL

Non-HDL C values were 165 ± 45 mg/dl for men and 164 ± 43 mg/dl for women ($p < 0.001$).

A difference between the 2 genders could be shown for the correlation of these parameters. In men, systolic and diastolic blood pressure correlated highly significantly with non-HDL (mean $r = 0.15$ for SBP and $r = 0.18$ for DBP; both $p < 0.001$), whereas in women the correlation was much weaker, but still significant (mean $r = 0.10$ for SBP and $r = 0.09$ for DBP; both $p < 0.001$). Scatter plots of these correlations are provided in Figure 1.

Impact of Age

The 4 distinct age groups were distributed differently between the genders. Of all men included, 10% were < 50 years of age, 23% between 50 and 59 years; 39% between 60 and 69 years and 28% > 70 years. In women, the distribution was 7%, 17%, 29%, and 47%, respectively; see also Figure 2.

Men demonstrated a stable non-HDL between 172 ± 47 and 173 ± 44 mg/dl up to the age of 60 years, followed by a decline to 157 ± 43 mg/dl in the elderly > 70 years of age. In women, non-HDL rises in the postmenopausal age group > 50 years from a mean 158 ± 42 mg/dl to a mean of 168 ± 42 mg/dl and declined thereafter to 163 ± 43 mg/dl. Significant differences of non-HDL-C between the 2 sexes ($p < 0.05$) were observed for all age groups besides 60–69 years where non-HDL was comparable. Figure 3A summarizes the development of non-HDL C with increasing age.

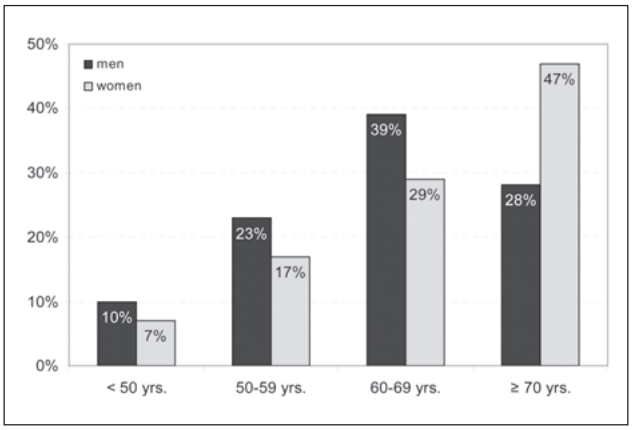

Figure 2. Distribution of Age Groups in the Sample Studied. percentages (%): respective gender; yrs: years of age.

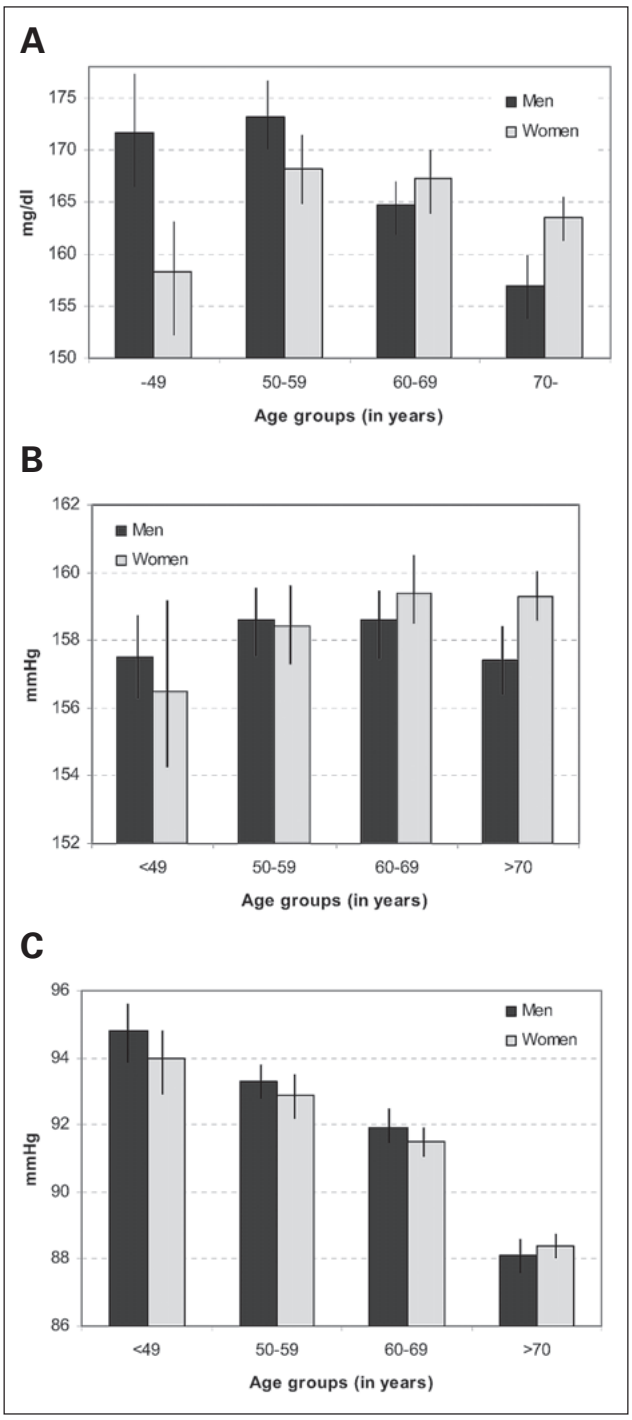

Figure 3. (A): Non-HDL Cholesterol; **(B):** Sitting Systolic; **(C):** Sitting Diastolic Blood Pressure in Men and Women, Distribution over the age groups. Values are means, bars represent two-sided 95% CIs.

Systolic blood pressure was similar in men and women over all age groups (between 157 ± 19 and 159 ± 19 mmHg). With increasing age, diastolic blood pressure levels decreased significantly ($p < 0.01$), from 95 ± 10 to 88 ± 11 mmHg in men, and from 94 ± 11 to 88 ± 11 mmHg in women, resp.). The development of blood pressure within the age groups is illustrated in Figure 3B–C.

Correlations between SBP, DBP and non-HDL cholesterol as for the whole sample studied are greater for men. Women show an increase around menopause before which, no significant correlation could be detected. The correlation coefficients between non-HDL C and SBP, as well as non-HDL C and DBP are shown in Table 2.

Both systolic and diastolic blood pressure correlated highly significantly to non-HDL ($p < 0.001$). In men the range over the age strata is from $r = 0.09$ to $r = 0.20$ for systolic and $r = 0.07$ to $r = 0.18$ for diastolic blood pressure (mean $r = 0.15$ for SBP and $r = 0.18$ for SiDBP, both $p < 0.001$). For women, the correlation is weaker, but increases with age (Tab. 3, Fig. 2). The correlation coefficients range between $r = 0.07$ to $r = 0.14$ for systolic and diastolic blood pressure (mean $r = 0.11$ for SiSBP and $r = 0.09$ for SiDBP, both $p < 0.001$), in the age group < 50 years, correlations are not significant for women (SiSBP: $r = 0.07$, $p = 0.28$; SiDBP: $r = 0.02$, $p = 0.70$), but highly significant for men (SiSBP: $r = 0.13$, $p = 0.01$; SiDBP: $r = 0.20$; $p < 0.01$).

Discussion

In this large scale, cross-sectional study we detected important, significant correlations between systolic as well as diastolic blood pressure and non-HDL-C levels. We found for the first time significant changes in the correlation for women around the age of menopause. In men, such differences could not be detected.

Considering the relevance of ties of blood pressure to serum cholesterol levels and their atherogenic potential, only few attempts have been made so far to evaluate them in a sample comprising different age groups and the two genders. For the Framingham study population an overall correlation coefficient of $r = 0.12$ was detected [9], but never been further evaluated or looked at in more detail. A small scale study performed in Persia is in line with the Framingham data, but due to its overall sample size it does not give further information [10]. In both the Physicians Health Study [26] and the Oslo study [11] only men were studied. The Tromso study aimed at investigating this association in a broader sample, but stopped for women at the age of 49 years, thus before menopause [12]. The lack of data for men and women at all age groups is therefore filled by our cross sectional analysis of 7500 western world hypertensive subjects.

The relevance of our findings is given by the fact that cut off levels for initiation of both cholesterol reducing as well as blood pressure reducing therapy depend on concomitant diseases and the prevalence of cardiovascular risk factors. Therefore, patients with newly diagnosed hypertension should also be screened for hyperlipidemia. *Vice versa*, blood pressure should be assessed in all hypercholesterolemic patients.

Our findings indicate the rise of LDL-cholesterol around menopause, whereas no change in HDL levels could be detected. No such change could be found in men. To put this gender specific outcome in context, epidemiological data suggest that the first myocardial infarction occurs in men nine to ten years earlier than in women [2, 25]. Furthermore, the more favorable lipid profile for women in younger ages is expressed in all risk assessments as the Framingham risk score [8] and the European SCORE system [27]. Major relevance

Table 2. Distribution of Lipid Parameters over the Age Groups

Age (years)	Sex	n	Total cholesterol (mg/dl)		Non-HDL-C (mg/dl)		LDL-C (mg/dl)		HDL-C (mg/dl)	
			Mean	SD	Mean	SD	Mean	SD	Mean	SD
≤ 49	M	364	223	45	172	47	135	40	51	20
	W	278	217	40	158	42	130	34	57	18
50–59	M	810	223	44	173	44	136	39	49	16
	W	671	225	40	168	42	141	39	58	18
60–69	M	1380	218	43	165	44	132	36	52	17
	W	1153	227	41	167	43	137	38	60	20
≥ 70	M	990	211	42	157	43	127	36	54	18
	W	1850	222	41	163	43	134	38	60	17

M: men; W: women; HDL: high density lipoprotein; LDL: low density lipoprotein

Table 3. Spearman Rank Correlation of Blood Pressure and Non-HDL in the Stratified Age Groups

Age (years)	Sex	n	SiSBP to Non-HDL C			SiDBP to Non-HDL C		
			r	r^2	p	r	r^2	p
≤ 49	M	364	0.13	0.02	0.01	0.20	0.04	< 0.01
	W	278	0.07	0.00	0.28	0.02	0.00	0.70
50–59	M	810	0.09	0.01	0.01	0.07	0.00	0.05
	W	671	0.13	0.02	< 0.01	0.07	0.00	0.07
60–69	M	1380	0.20	0.04	< 0.01	0.18	0.03	< 0.01
	W	1153	0.08	0.01	0.01	0.07	0.00	0.02
≥ 70	M	990	0.15	0.02	< 0.01	0.18	0.03	< 0.01
	W	1850	0.14	0.02	< 0.01	0.13	0.02	< 0.01

M: men; W: women; SiSBP: sitting systolic blood pressure; SiDBP: sitting diastolic blood pressure; Non-HDL C: non high density lipoprotein cholesterol; p-values given for significance of rank correlation

could be given to estrogen protection in premenopausal women [28,29], but further reasons are still to be evaluated.

As to our findings, the prevalence of elevated non-HDL-cholesterol accounts for around 3% of the risk of developing hypertension for men at all ages and for women after menopause. This could be explained by a biological model of impaired endothelial function or of an upregulation of the AT1 receptor by low density lipoprotein cholesterol [31–33], leading to an increase in both systolic and diastolic blood pressure. This enhanced gene expression is also reflected by increased sensitivity for angiotensin II in patients with hypercholesterolemia [34]. Moreover, data from the ARIC study suggest that carriers of a certain allele in the PCSK9 gene show reduced LDL levels which are associated with decreased blood pressure compared to non carriers [36, 37].

A further relevant aspect is that reduction in blood pressure was reported as one of the pleiotropic effects of statin therapy [13–19]. Several studies and meta-analyses addressed this effect, results were not entirely consistent, but there is a trend towards a reduction of up to 4–5 mmHg for systolic and about 1 mmHg for diastolic values, depending on the level of LDL reduction, which is a long-term effect. No acute drug effects of statins on blood pressure could be shown [30]. Niacin, another type of cholesterol altering drugs, has also shown to have blood pressure lowering effects [20–22]. In contrast to statin therapy, also an acute blood pressure lowering effect was reported [20]. One possible mechanism is an interaction of niacin with dermal Langerhans cells leading to an increase in dermal vasodilatation. A recent evaluation, though, revealed no difference in the reduction of blood pressure regardless of whether extended release niacin is combined with placebo or laropiprant, a prostaglandin PG_2 receptor antagonist reducing the described vasodilatation [20].

Improved endothelial function seems to be one explanation for the described effect. Yet, the significant correlation of non-HDL cholesterol with blood pressure is of major interest. Therefore, any cholesterol lowering measure should also be accompanied by a close look at blood pressure.

The limitations of our study include the fact that only hypertensive subjects were included. Yet the sample reflects a representative hypertensive population at high risk [4].

In conclusion, our results suggest a correlation of non-HDL and blood pressure in hypertensive men at all age groups. In women, menopause has a significant influence on not only lipid levels, but also on correlations to blood pressure and determines the individual cardiovascular risk.

Appendix

The LIIFE-in-LIFE study board: H. Drexel, Feldkirch; B. Eber, Wels; F. Hoppichler, Salzburg; K. Huber, Vienna; W. Lang, Vienna; B. Ludvik, Vienna; G. Mayer, Innsbruck; K.P. Pfeiffer, Graz; M. Pichler, Salzburg; E. Rebhandl, Haslach; A. Rieder, Vienna; K. Silberbauer, Eisenstadt; J. Slany, Vienna; G. Stark, Deutschlandsberg; K. Stoschitzky, Graz; O. Traindl,

Mistelbach; H. Wimmer, Villach; G. Zenker, Bruck/Mur; all Austria.

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Conflict of Interest

The authors report no conflict of interest. DL is an employee of MSD Austria. No influence on this study question, writing or search for literature was executed by the company.

Fragen zum Artikel

1) Wie kann man den blutdrucksenkenden Effekt von Lipidsenkern erklären?
2) Beeinflusst Menopause das kardiovaskuläre Risikoprofil von Frauen?
3) Korreliert Non-HDL-Cholesterin zu Blutdruck?

Lösung

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Richtige Lösung von S. 16:

1. Prinzipiell ist der Effekt schwach ausgeprägt, postuliert wird eine direkte Wirkung auf den AT1-Rezeptor im Umkehrschluss der Up-Regulation der Expression der Rezeptoren durch erhöhte LDL-C-Last.
2. Um die Menopause kommt es zum Non-HDL-Cholesterinanstieg um ca 10 mg/dl. Frauen haben auch danach noch ein geringeres kardiovaskuläres Risiko als gleichaltrige Männer, was über den (gegenüber Männern) reduzierten subendothelialen Stress und damit verbesserte Gefäßfunktion erklärt werden kann. Schwerwiegende kardiovaskuläre Ereignisse treten bei Frauen ca. 10 Jahre später auf, dies ist in den gängigen „risk assesments“ abgebildet.
3. Ja, vor allem bei hypertensiven Männern aller Altersgruppen und hypertensiven Frauen nach der Menopause. Die Korrelation schwankt zwischen 0,0 (prämenopausale Frauen) und 0,2 (Männer).

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