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Ecological Correlations between Dietary Fat and Carbohydrates and Mortality from CVD considering Trends in Time

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Vorwort

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Auch Mag. Manfred Ditto danke ich für seine Unterstützung.

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[1]EEC: Czech Republic, Estonia, Hungary, Latvia, Lithuania Poland, Romania, Slovenia; WEC: Austria, Belgium, Denmark, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Norway, Portugal, Spain, Sweden, The Netherlands, United Kingdom; I00-99: deaths from diseases from the circulatory system (CVD); I60-69: deaths from ischemic heart diseases; I20-25: deaths from cerebrovascular diseases; energy (kcal/day); carbohydrate intake (% of energy); fat intake (% of energy); SFA: saturated fatty acids (% of energy); MUFA: monounsatruated fatty acids (% of energy); PUFA: polyunsaturated

fatty acids (% of energy); BMI: body mass index (kg/m^2); smoking (current smokers in % of the population, alcohol consumption in g/day); PAL: physical activity level (% of population which never exercises)

Chapter 1

Literature review

1.1 Introduction

There has been a growth in the elderly population as well in developed as in developing countries [Rorrey *et al.*, 1987]. Elderly people are defined by the WHO as people over 65 years of age. In 1994, Sweden showed the largest proportion of over 65 years olds (15 %). Sweden was the "oldest" nation with a percentage of 18 % of over 65 year old people [WHO, 1995]. Life expectancy still increases steadily in developed countries. For example, life expectancy in Sweden increased from 78 years in 1990 to 81 years in 2009 [WHO, 2011b].
In most industrialised countries ratio between men and women is 65:100 due to differences between life expectation of male and female. Further, this ratio is likely to decrease in industrialised countries [WHO, 1995].
Cardiovascular diseases act as death cause number one in industrialised countries [Ali *et al.*, 2009]. 80 % of CVDs occur in low and middle income countries. Men and women are affected almost equivalent. It is expected that CVD will contribute to about 23,6 million deaths in 2030, thus CVD will continue to be the worlds leading death cause [WHO, 2011a].

CVD does not nessesarily lead to death. After an cardiovascular incident physical disability, lifelong dependence and medications are possible consequences. Thus, in prospective to the financial burden of the health system, the social and physical burden of the patients and their relatives need to be considered [Panico and Mattiello, 2010]. With regard to the public health burden of CVD it is important to operate with well implemented and planned programs of prevention [EUROCISS, 2007]. Therefore, it is essential to give accurate dietary recommendations.

For the last five decades research payed attention to the role of dietary fat in CVD. When until recently it was recommended to decrease the fat intake, it is now discussed whether or not it is more beneficial to lay focus on fat quality to lower the CVD risk. The food industry recognised the marketing potential of health products. Thus customers sight was changing. The word dietary fat adopted a bad image for the bodies health, while "fat-free" or "low-fat" equaled health. However sugars and refined carbohydrates were the substitute for fat [Hu *et al.*, 2001].

The European nutrition and health report 2009 collected data of dietary intake from 25 European countries. These data were used to observe a link at an ecological level between dietary habits and mortality from CVD of the European population. Further it was to ascertain differences among regions in energy providing nutrients and CVD mortality.
It was to evaluate whether a population with a high fat intake or a high carbohydrate intake is troubled with a higher CVD mortality. Furthermore it was to identify the role of quality of fat in mortality from CVD.
In addition, trends in time of nutrient uptake and mortality from CVD in the various European populations should have been detected.

1.2 Cardiovascular Disease (CVD)

CVD's are diseases of heart and blood vessels which comprise [WHO, 2011a]:

- coronary heart disease: disease of the blood vessels supplying the heart muscle
- cerebrovascular disease: disease of the blood vessels supplying the brain
- peripheral arterial disease: disease of blood vessels supplying the arms and legs
- rheumatic heart disease: damage to the heart muscle and heart valves from rheumatic fever caused by streptococcal bacteria
- congenital heart disease: malformations of heart structure existing at birth
- deep vein thrombosis and pulmonary embolism blood clots in the leg veins, which can dislodge and move to the heart and lungs

1.2.1 Risk factors

Blood pressure

A high blood pressure is linked directly to coronary heart diseases (CHD) [Stamler *et al.*, 1993]. The WHO MONICA project evaluated within 38 populations in 21 countries that systolic and diastolic blood pressures varies between the populations [op Reimer *et al.*, n.d.] Further, the Seven Countries Study has demonstrated that the relative mortality increase of CHD is similar to a given increase in blood pressure between varying populations [Menotti *et al.*, 1996].

Table 1.1: Reference values for lipoproteins in blood serum [Graham *et al.*, 2007]

Total cholesterol	< 5.0 mmol/l
LDL-cholesterol	< 3.0 mmol/l
HDL-cholesterol	>1.2 mmol/l
TG	< 1.7 mmol/l

Blood lipids

The fat-like substance cholesterol which travels in blood is part of the cell-membranes and precursor of bile acids and steroid hormones. It contains lipoproteins like the low density lipoproteins (LDL), the high density lipoproteins (HDL) and the very low density lipoproteins (VLDL). Another lipoprotein of the cholesterol appears as intermediate density lipoprotein (IDL) which exists between VLDL and HDL, therefore, is included in the LDL measurement in clinical practice [NCEP, 2002].

Cholesterol levels are linked to the CVD risk. According to Graham et al., 2007, LDL makes usually up 60-70 % of the total serum cholesterol and represents the most artherogenic lipoprotein. HDL is with a content of 20-30 % inversely correlated with CHD risk. The chylomicrons are built in the intestine and are TG rich lipoproteins, which are probably artherogenic [NCEP, 2002].

The European society of Cardiology published guidelines for reference values to see in Table 1.1 [Graham *et al.*, 2007].

Plasma lipid levels are as well affected by dietary factors like saturated fat, polyunsaturated fat, monounsaturated fat, total fat, dietary cholesterol, dietary carbohydrates, dietary fibre, alcohol, and caloric intake as by body weight [Shils *et al.*, 1994].

Diabetes

Diabetics show higher serum lipids, remarkably in triglycerides. Therefore diabetics are exposed to a higher risk of CVD [Watsen, 2000].The San Antonio Heart Study found higher parameters of CVD risk factors (Table 1.2.1).

Table 1.2: Parameter of CVD risk factors in diabetic and non-diabetic persons [Haffner *et al.*, 1990]

	Diabetics (n18)	Nonconverter (n490)	P value
BMI (kg/m^2)	30,2	27.2	0.472
Triglycerides (mmol/l)	1.83	1.28	0.006
HDL-cholesterol (mmol/l)	1.14	1.28	0.045
Systolic blood pressure (mmHg)	116.8	108.8	0.004

Obesity

Obesity is an independent risk factor for CVD [Poirier *et al.*, 2006]. It is caused by positive caloric balance, unhealthy lifestyle, genetic predisposition, and environmental causes [Bays, 2011]. Further, obesity (BMI > 30 kg/m^2) is a risk factor for metabolic diseases such as Typ II Diabetes Mellitus, high blood pressure, and dyslipidemia which indirectly rise the CVD risk [Bays, 2009]. The WHO suggested a median BMI of 21-23 kg/m^2 as most favourable for a population <65 years to reduce the risk of chronic diseases and obesity. A BMI of 21 kg/m^2 is considered to be more advantageous for wealthy societies with a more inactive lifestyle and a BMI of 23 kg/m^2 for developing countries [(WHO) *et al.*, 2003].

Despite the fact that visceral fat tissue plays a major part in the development of metabolic diseases, circulating free fatty acids coming from subcutaneous fat tissue (80 % of body fat) contribute to lipotoxic effects on the liver. Thus, CVD risk factors like hyperglyceremia and dyslipidemia occur [Bays and Ballantyne, 2006]

Cardiovascular and adipose cells have the same origins of steam cell, therefore, once adipocyte formations of the stem cell preceeded, adipogenesis is relevant for development of CVD [Tchoukalova *et al.*, 2010]. Pericardial and perivascular adipocytes may have direct pathogenic effects on myocardium, coronary arteries and peripheral vessels. Disregulated local secretion of vasoactive and inflammatory factors may be responsible for unfavourable cardiovascular pathophysiologic effects [Higuchi *et al.*, 2002]. Furthermore, adipose tissue may directely contribute to CVD risk supported by the strong link between pericardial adipose tissue and coronary calcification [Liu *et al.*, 2010].

Smoking

Tobacco use is a further independent risk factor for CVD. A cross sectional study investigated the impact of cigarette smoking and novel risk factors such as serum-C-reactive-protein, fibrinogen and homocystein levels. With a significant dose-response relationship between smoking and these three novel risk factors, it is likely that inflammation and hyperhomocysteinaemia are a pathway of CVD development [Bazzano *et al.*, 2003]. Tobacco use has also been associated with traditional risk factors like serum cholesterol levels and hypertension [Muscat *et al.*, 1991].

Physical activity

Physical activity reduces clearly the risk of a CVD incidents. Although mechanisms are not well understood, it is inversely correlated with blood pressure, blood lipids, and diabetes mellitus (traditional risk factors) [Hu *et al.*, 2004].

Mora et al. took data from the Women's Health Study to evaluate the impact of physical activity on the CVD risk. The mean follow up of 10.9 ± 1.6 years of 27,055 apparently healthy women showed a linear decrease of the CVD risk. Subjects with a physical activity lower than 200 kcal/week presented the reference group. Observed risk reduction was 27 % (PA 200-599 kcal/week), 31 % (600- 1499 kcal/week) and 41 % (>1500 kcal/week). They examined the risk factors and noticed inflammatory and homeostatic biomarkers such as high-sensitivity C-reactive-protein made the largest contribution to lower the CVD risk (32.6 %) followed by blood pressure (27.1 %). The change of risk factors is to see in Table 1.2.1 [Mora *et al.*, 2007].

Table 1.3: Classification of the amount of physical activity and CVD risk reduction ($P < 0.001$) regarding risk parameters (mean (SD)) hypertension, body mass index (BMI), total cholesterol (TC), low density cholesterol (LDL), high density cholesterol (HDL), and high sensitivity C-reactive-protein (CRP) [Mora *et al.*, 2007].

	Physical Activity (kcal/week)			
	< 200	200-599	600-1499	>1500
Hypertension %	28.6	24.7	23.0	23.8
BMI, kg/m^2	26.9 (5.6)	25.9 (4.9)	25.3 (4.5)	25.6 (4.7)
TC, mg/dL	210 (186-237)	209 (184-236)	207 (183-234)	207 (182-234)
LDL, mg/dL	124 (103-147)	122 (102-145)	120 (99-143)	119 (98-142)
HDL, mg/ dL	49.5 (41.3-60.2)	51.6 (43.4-61.9)	53.1 (44.1-63.1)	53.5 (44.1-64.3)
High-sensitivity CRP, mg/L	2.5 (1.0-5.1)	1.0 (0.8-4.4)	1.8 (0.7-4.1)	1.8 (1.7-3.8)

Adults should exercise at least thirty minutes five days a week in moderate intensity or with a powerful physical activity for twenty minutes 3 days a week [Haskell *et al.*, 2007]. In terms of physical activity, in Europe a North-South was gradient observed . People in the North of Europe tend to exercise more in there leisure time than people in the south [Elmadfa, 2009].

Alcohol

Consumption of alcoholic beverages is a common habit in most populations of the world. Alcohol may be protective in lower dose while excessive alcohol consumption has harmful effects on cardiovascular health. Alcohol has unfavourable effects on mediating pathways of the vascular endothelium, such as oxidative stress, lipoproteins and insulin resistance. Hence immoderate alcohol consumption has an effect on classical CVD risk factors. Therefore, an excessive alcohol consumption is unfavourable for CVD risk [Baua *et al.*, 2007].

Social and environmental influences

In the past decades mortality rate of CVD dropped rapidly in western countries, whereas in some eastern countries mortality rate rose. A review of Chow et al. of environmental and societal influences on CVD risk factors showed dramatic falls due to risk factor changes on the population level, combined with consequences of improved interventions and better curative care [Chow *et al.*, 2009].

It is hardly to believe that the rapid fall is caused only by individually targeted interventions and differences in medical treatment. Behaviour and choice making of human beings is determined by social norms, culture, geographics, as well as the political, economic and legal factor [Chow *et al.*, 2009].

There are further environmental influences such as the tobacco environment, physical activity environment, and environmental influences on diet. When smoking is easier to determine through price regulations, public information or policies such as smoke free workplaces or restaurants and bars, the diet is a more complex field [Chow *et al.*, 2009].
The price of cigarettes rose in developed countries and on the contrary decreased in developing countries [Guindon *et al.*, 2002]. It is more likely to

improve smoking habits when social norms are suitable. For instance in parts of South Asia, the social acceptance of smoking among women is modest, hence smoking rate is low [Rani *et al.*, 2003]

Physical activity environment contributes to the rates of obesity [Boehmer *et al.*, 2006]. Further, neighbourhood safety (crime, traffic, stray dogs) and neighbourhood attractiveness are linked to higher physical activity [Saelens *et al.*, 2003], as well as societal and cultural norms (car or TV ownership, cultural influences) and policy and legislation [Chow *et al.*, 2009].

Dietary and nutrition environment is influenced by the macro-level (legislation and policy), the physical environment level (access and availability), and the social environment level (social norms, culture) [Chow *et al.*, 2009]. There have been associations between the local food environment (density of fast-food-restaurants and supermarkets) and prevalence of CVD risk factors in North America [Morland *et al.*, 2006]. In European studies this link could not be confirmed significantly [Cummins and Macintyre, 2006].

1.3 Dietary fat and CVD

In earlier times, it was believed that the human body could survive with enough energy whether it came from fat or any other dietary compound. Nowadays science is enlighten of the knowledge that unsaturated fatty acids are an essential component in human nutrition to satisfy requirements of the human body. An important reason for this is for instance the occurrence of those unsaturated fatty acids in membranes [Elmadfa and Leitzmann, 2004]

Most natural dietary fats comprise 98-99 % triglycerides. Triglycerides consist of glycerol esterified with three mainly long-chain fatty acids. The remaining 1-2 % take in by diglycerides, monoglycerides, free fatty acids unsaponifiable compounds, phospholipids, and sterols [Elmadfa and Leitzmann, 2004].
Most important characteristics of fatty acids are the length of the chain

(number of C-Atoms) and the degree of saturation (number of H-Atoms). Therefore, they have an enormous influence on physical properties and biochemical functions of fatty acids [Elmadfa and Leitzmann, 2004]

Dietary fat has an impact on plasma cholesterol which is known as a classical risk factor of CVD. The amount of energy taken in by saturated and unsaturated fatty acids is a crucial point regarding plasma cholesterol [Shils *et al.*, 1994]. Further, plasma cholesterol is affected by dietary cholesterol intake. It raises levels of total and LDL cholesterol [Hengsted *et al.*, 1965]. However, compared to the effects of saturated fatty acids and trans fatty acids the impact of dietary cholesterol on plasma cholesterol is relatively small [Howell *et al.*, 1997]. These equations as basis lead to calculations of the effects of dietary modification on plasma cholesterol. Moreover, increase or decrease of the CVD risk could be estimated [Shils *et al.*, 1994].

Between fatty acids, triglycerides, cholesterol, and hyperlipidemia there is a strong link, in the same manner as hyperlipidemia and risk of CVD [Watsen, 2000].

1.3.1 Saturated fatty acids (SFA)

It is widely believed that a higher intake of total and saturated fat encourages the development of CVD. Ecological studies are mainly the basis for this belief.
A recent meta-analysis of Siri-Tarino et al. conducted well-designed prospective epidemiological studies to evaluate the risk of CHD stroke and total CVD associated with a higher SFA intake. Within twenty-one studies and a follow-up from five to twenty-three years of 347,747 subjects 11,006 developed CHD or stroke. However, intake of saturated fat was not linked to a higher risk of CHD, stroke or CVD. Pooled RR for the highest quantiles of saturated fat intake was 1.07 (95 % CI: 0.96, 1.19; P = 0.22) for CHD, 0.81 (95 % CI: 0.92, 1.05; P = 0.11) for stroke and 1.00 for overall CVD(95 % CI: 0.89, 1.11; P = 0.95) [Siri-Tarino *et al.*, 2010].

The association observed between SFA and CHD in the Nurses' Health Study was much weaker than predicted by international comparisons [Keys, 1980]. However, in metabolic studies effects on plama lipid and lipoprotein levels were different for different SFA. SFA with 12 to 16 carbon atoms are likely to increase total and LDL cholesterol levels. In contrast, stearic acid (18:0) has no cholesterol-raising effect when compared with oleic acid (18:1). Myristic acid (14:0) seems to rise cholesterol levels more than lauric acid (12:0) or palmitic acid (16:0) [Kris-Etherton and Yu, 1997].

SFA demonstrated in early animal studies an increase of plasma cholesterol as well as atherosclerotic lesion which are risk factors in development of CVD [Anitschkow, 1967]. Further, in humans dietary saturated fat correlated with the risk of CHD [Kato *et al.*, 1973]. Randomized clinical interventions observed protective [Ascherio *et al.*, 1996], inverse [Gillman *et al.*, 1997], and no effects [Ascherio *et al.*, 1996] when reducing SFA.

Hu et al. conducted a detailed prospective analyses of dietary fat and CHD. They used data from the Nurses' Health Study when 80,082 women aged 34 to 59 years participated a 14-year follow up. Due to the large sample size and repeated assessments of diets, this study was powerful. A weak positive association between saturated fat intake and the CHD risk was found. 5 % of energy of saturated fat in contrast to equivalent energy from CHO, was linked with a 17 % higher risk of CHD (RR = 1.17, 95 % CI: 0.97-1.41; P = 0.10) [Hu *et al.*, 1997].

Even if studies are not consistent, in case of a higher intake of saturated fat and a increase of CVD risk, it is to see in the following chapters that a replacement and, more important, which replacement of SFA could be beneficial to reduce risk of CVD.

1.3.2 Monounsaturated fatty acids (MUFA)

MUFA have one double bound and a higher melting point than SFA. They are liquid at room temperature. Representatives are palmitoleic acid (16:1 n-7) and oleic acid (OLA) (16:1 n-9). The most common MUFA in human nutrition is OLA which contributes to 92% of total MUFA intake [Kris-Etherton, 1990]. Olive oil and canola oil represent the most frequent used oils [Gillingham *et al.*, 2011].

Replacement of SFA with MUFA has impact on the total high-density lipoprotein cholesterol (TC:HDL-C) ratio due to reduction of LDL-C levels and sustaining HDL-C levels [Gillingham *et al.*, 2011].

In the Nurses' Health Study, after adjustment of fats, MUFA intake was inversely linked with risk of CHD. Exchanging 5 % of the energy from SFA with MUFA the estimated RR was 0.81 (95 % CI, 0.65-1.00; P = 0.05) [Hu *et al.*, 1997].

A Meta-analysis of Cao et al. investigated 30 controlled feeding studies with subjects with and without diabetes to examine effects of MUFA and CHO to bloodlipids and lipoproteins. They compared a moderate fat diet (30.2-50 % of energy; mean MUFA intake 23.6 % of energy) with a lower fat diet (18.3-30.2 % of Energy, mean MUFA intake 11.4 %). LDL-C decreased in both groups similar. Moderate fat diets increased HDL-C by 2.28 mg/dl (95 % confidence interval 1.66 to 2.90 mg/dL, P, .0001) more than low fat diets. TAG went down in moderate fat diets (-24.79 mg/dL, P 0.00001), whereas with low fat diets TG increased. Further, a decline of TC:HDL-C (-0.62, P 0,0001) and non-LDL-C (-5.39 %, P 0.06) was observed in MF diet. Conclusion of the authors was that there is a reduction of CHD risk by 6.37 % in men and 9.34 % in women (subjects with diabetes included). It may be even more beneficial for subjects predisposed with diabetes mellitus or metabolic syndrome to replace SFA with MUFA instead of CHO [Cao *et al.*, 2009].

A landmark epidemiological trial of 11,579 men (40-59 years) in the Seven Countries study, presented important data revealing that areas consuming a Mediterranean diet (rich in MUFA from olive oil), even though higher in total fat (33-40 % of energy), exhibited lower incidence of CHD mortality [Keys and et al., 1986]. In contrast, Hegsted and Ausman observed a positive correlation between MUFA and CHD mortality in men from 18 countries [Hegsted and Ausman, 1988].

1.3.3 Polyunsaturated fatty acids (PUFA)

PUFA have at least two double bounds and an even higher melting point than MUFA. Most important PUFA are linoleic acid (18:2) and α-linolenic acid (18:3) [Gillingham *et al.*, 2011]. A higher consumption of PUFA reduces the risk of CHD due to a decrease of the TC:HDL-C [Lewington *et al.*, 2007]. Further, improvement of insulin resistance is given [Summers *et al.*, 2002] and the reduction of systemic inflammation [Pischon *et al.*, 2003]

As per Hu et al. due to the Nurses' Health Study trading SFA with PUFA it results a RR of 0.62 (95 % CI, 0.46-0.85; P = 0.002) [Hu *et al.*, 1997].

Mozaffarian et al. observed a reduction of 19 % of CHD occurrence when SFA are replaced with PUFA. The Meta-analysis included randomized controlled trials with adults. The intervention group replaced SFA with total PUFA of 14.9 % of total energy intake (n-3 and n-6 PUFA). The control group ingested 5 % PUFA of total energy intake. The estimated risks had to be quoted with standard errors. The eight included trials had restricted statistical power, nevertheless they were homogen in their study design [Mozaffarian *et al.*, 2010].

However, some scientists do not share the protective opinion of PUFAs in regard to the CVD risk. There are studies which observed an increasing risk of CVD occurrence with a high PUFA intake [Hamazaki and Okuyama, 2003]. Linoleic acid plays a key role in the inflammatory process which is the starting point of this hypothesis [Kris-Etherton *et al.*, 2010].

A higher linolenic acid intake increases tissue levels of arachidonic acid which further raises inflammatory eicosanoids. Inflammation is a main reason to the onset and progression of CHD. Therefore CHD risk must be higher. Additionally lowering linolenic acid leads to an increase of tissue levels of the cardioprotective eicosapentaenoic acid (n-3 fatty acid) [Kris-Etherton *et al.*, 2010].
Therefore, despite the cardiovascular benefits the ADA (American Dietetic Association) restricts intakes to <10 % due to adverse effects [Moreno and Mitjavila, 2003]. EFSA (European Food Safety Authority) does not restrict the intake of PUFA, instead they want to set an adequate intake, 4 % of energy for linolenic acid and 0.5 % of energy for α-linilenic acid [EFSA, 2010].

1.3.4 Trans-fatty acids (t-FA)

In the Nurses' Health Study a positive relationship between trans fat intake and risk of CHD was observed. This is gernerally consistent with the prospective Health Professionals' Follow-up Study conducted in men [Ascherio *et al.*, 1996].

Judd et al. investigated the impact of cis- and trans-fatty acids on blood lipids. Trans fatty acids (t-18:1) change the apolipoprotein(a) and (b) profile which leads to an increase of the LDL-cholesterol. The lipogenic effect of trans-fatty acids is slightly smaller than the lipogenic effect of saturated fatty acids [Judd *et al.*, 1994]. Figure 1.1 gives an overview of the impact of various fatty acids on plasma total cholesterol. Although this review of effects of fatty acids on plasma cholesterol by Kris-Etherton and Yu derived in 1997, it already shows the scope of consequences [Kris-Etherton and Yu, 1997].

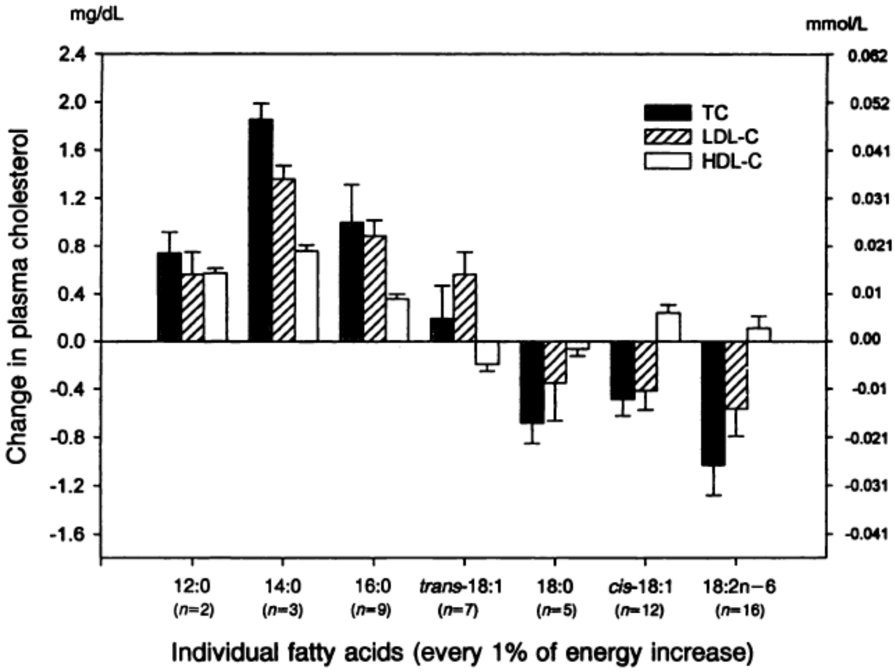

Figure 1.1: Effects of lauric (12:0), myristic (14:0), palmitic (16:0), elaidic (trans 18:1), stearic (18:0), oleic (cis 28:1) and linolenic (18:2n-6) acids differentiated to 18:1 - TC, plasma total cholesterol [Kris-Etherton and Yu, 1997]

	CHO	MUFA
LDL	-7.1 %	-6.3 %
HDL	-7.2 %	4.3 %
TG	↑	↓

Table 1.4: Change in metabolic risk factors replacing SF with CHO or MUFA [Berglund *et al.*, 2007]

1.4 Carbohydrates and CVD

Studies evaluated the change in cholesterol levels in response to a relatively high total fat diet (33-50% of energy) rich in MUFA, compared to low fat diets (18-32% of energy) with a big fraction of carbohydrates [Kris-Etherton and Yu, 1997]. An increase of 6-22% in HDL-cholesterol and a decrease of 2-24% in plasma triglycerides was reported in favour of fat rich diets. In both diets, LDL-cholesterol decreased [Berry *et al.*, 1992].

Berglund et al. conducted a 3 period 7-week randomized crossover study with fifty-two men and thirty-three women. An average american diet with 36% fat of energy intake was modified. Of those 36%, 7% had been replaced with CHO or MUFA. The change in LDL, HDL and TG is to see in Table 1.4. LDL-cholesterol had a higher decrease with a CHO-diet. Further with a CHO-diet TG rose, when with a MUFA rich diet TG decreased. Further, with a MUFA rich diet HDL-cholesterol increased. All changes of the metabolic risk factors were significantly different to the average american diet. Further, changes in HDL and TG with the CHO-diet were significantly different to changes evoked by the MUFA-diet.

Chapter 2

Material and Methods

2.1 Trends in time

Data of mortality for 25 European countries was given by the WHO database on May 5th 2011 (http://data.euro.who.int/hfadb/). Diseases are labeled by the International Classification of diseases (ICD10). Data for the supply of the items, like energy, fat, carbohydrates and sugar and sweeteners provided from Food Balance Sheets (FBS) collected by the FAO (Food and Agriculture Organisation) database, downloaded on May 12th 2011(http://faostat.fao.org/).

FBS are assembled by the FAO and provide data on food supply at the population level. The information is estimated on the basis of the annual food production, imports and exports, changes in stocks, agricultural and industrial uses within a country, as well as losses during storage and transportation. FBS allow comparisons of current and developing structure of dietary patterns at national or regional level [FAO, 2008].

Further, the 25 countries were split into four regions, due to their dietary habits and similarities in order to receive a better picture about the trend in time. These four regions are shown in Table 2.1.

North	Central East	West	South
Denmark	Austria	Belgium	Cyprus
Finland	Germany	Ireland	Italy
Lithuania	Poland	United Kingdom	Spain
Sweden	Slovenia	France	Greece
Estonia	Czech Republic	Netherlands	Portugal
Latvia	Hungary	Luxembourg	
Norway	Romania		

Table 2.1: Division of the 25 european countries

FAO database provides supply data from 1961 till 2007, however, WHO database covers only the time form 1970 to 2007 with its mortality data [Elmadfa, 2009].

For some countries, data were not available for all 40 years. In the case of mortality, Cyprus collected data since 2004. Estonia, Lithuania, Latvia and Slovenia started arising mortality data in the eighties and Germany in 1991. For macronutrients, supply data are available since 1961 except for the Czech Republic, Estonia, Latvia, Lithuania and Slovenia which contributed in the nineties. Further, data of Belgium and Luxembourg were summed until the year 2000.

Data was imported into Stata 10.0 and analysed in this statistical software.

2.1.1 Energy

FAO FBS data provide data on energy supply as kcal/capita/day and kg/capita/day which allegorise the food supply quantity. Here data in kcal/capita/day were used. Energy supply was calculated by the FAO out of the grand total food supply.

2.1.2 Fat

Fat consumption was separated in the trend by animal fats, vegetable oils, and oil crops. The FAOStat Database gave values in gram/capita/day for all three parts. Total fat supply was calculated by the FAO out of the grand total. Data of vegetable oils comprise rape and mustard seed oil, sunflower seed oil, cottonseed oil, linseed oil, hempseed oil, sesame seed oil, copra and coconut oil, palm kernel oil, palm oil, soybean oil, olive oil and maize oil. Data of animal fats comprise butter, ghee, fish liver oil, other animal fats and whale oil [FAO, 2008].

2.1.3 Carbohydrates

The FAO database did not include information about a carbohydrate supply. Therefore, the protein supply quantity (gram/capita/day) of the grand total was multiplied by 4 to receive the protein supply in kcal. Fat supply quantity (gram/capita/day) was multiplied by 9.3. After subtraction of the fat and protein supply in kcal from the energy supply, the carbohydrate supply in kcal/capita/day results. To get carbohydrate supply in gram/capita/day numbers were divided by 4.

2.1.4 Sugar and Sweeteners

Supply of sugar and sweeteners is specified in kilogram/capita/year. Thus, it was divided by 365 multiplied by 1000 to compute the supply in gram/capita/day and

2.1.5 Mortality

To give an expression about the burden of CVD in an economic level, mortality seemed to be the most reliable parameter [EUROCISS, 2007]. Data about mortality from CVD was downloaded on 5th May 2011 and updated by the WHO in June 2010. Mortality was stated in SDR (standardised death rate) per 100,000 inhabitants for the population till 65 years and further subclassified in:

- death from diseases of the circulatory system (ICD 10 codes: I00-99; all cardiovascular diseases)
- death from cerebrovascular diseases (ICD 10 codes: I20-25)
- death from ischemic heart diseases (ICD 10 codes: I60-69)
- all deaths.

"SDR are age-standardized mortality rates, which are weighted average of the age-specific mortality rates per 100,000 persons, where the weights are the proportions of persons in the corresponding age groups of the WHO standard population." [WHO, 2008]. The standard population is based on the average world population structure from the year 2000 to 2025. The United Nations Population Division assesses this every two years for each country (age and sex). Death rates of a country are multiplied with the factor of worlds average, hence, SDR results [WHO, 2008].

2.2 Ecological Correlation

Data for nutrient intake of adults (< 65 years) and other risk factors were taken from the European Health and Nutrition Report 2009. Objectives were energy (MJ), total carbohydrates (% of Energy), sucrose (% of Energy), fat

(% of Energy), saturated fatty acids (% of Energy), monounsatruated fatty acids (% of Energy), polyunsaturated acids (% of Energy), and cholesterol (mg).

Further risk factors included in the European Health and Nutrition Report 2009 were mean body mass index (BMI, kg/m^2), mean alcohol consumption (g/d/capita), smoking behaviour (percentage of current smokers in the population) and physical activity level (% of population which never does sports or exercises). With exception of the physical inactivity, all data was available for male and female.

Age group for BMI was 19-64 years. BMI data was self reported as well as measured. If both was given, measured data was used for calculation of spearman's rho. Age group of alcohol consumption was mainly 19-64 years with exception of Germany (15-80 years), France (19-74 years) and Italy, Portugal, and Spain which included all ages. Current smokers were aged 45-64 years.

For mortality the same data source as for the trend evaluation (SDR per 100,000 inhabitants) was used. Data was downloaded from the WHO health for all database (5th May 2011). Each objective was correlated with mortality from circulatory diseases, ischemic diseases, cerebrovascular diseases and overall death rate. It was operated with mortality data from the same year the survey or the study was conducted. If there was no data available, data of the following or previous year was used.

To evaluate the extent to which the dietary survey-derived data and mortality correlate, Spearman coefficients between nutrient consumption and modifiable risk factors (BMI, alcohol consumption, smoking behaviour and PAL) and mortality was estimated. Statistical software was Stata 10.0.

After evaluating data with scatter plots a new division was made. Due to higher mortality rates of Eastern European Countries (Czech Republic, Estonia, Hungary, Latvia, Lithuania Poland, Romania, Slovenia) they were split from Western European Countries (Austria, Belgium, Denmark, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Norway, Portugal, Spain, Sweden, The Netherlands, United Kingdom)

In addition spearman's rho was estimated between all objectives and the different mortality rates, for eastern and western european countries.

Chapter 3

Results

3.1 Time Trends in food supply and mortality from CVD

Present study was conducted to describe a trend in time for supply of different nutrients and the mortality from CVD. A further aim was to compare trends of European countries. Therefore, the mean, the minimum and the maximum of the intake data (energy, total fat, animal fat, vegetable oil, oil crops, carbohydrates and sugar and sweetener) were calculated for the four regions (South, Central East, West and North) and the development was presented in graphs.

When looking at the results, it is to keep in mind that data were received from FBS, which calculate the food supply at the population level. Thus information for each parameter were higher than in individual based surveys. Further, FAO received data from national statistic institutes. Data evaluation as well as cultural habits differ within countries. Further reliability and precision are questionable. Nevertheless FBS are adequate sources to compare dietary habits among countries [Schwartz, 2003].

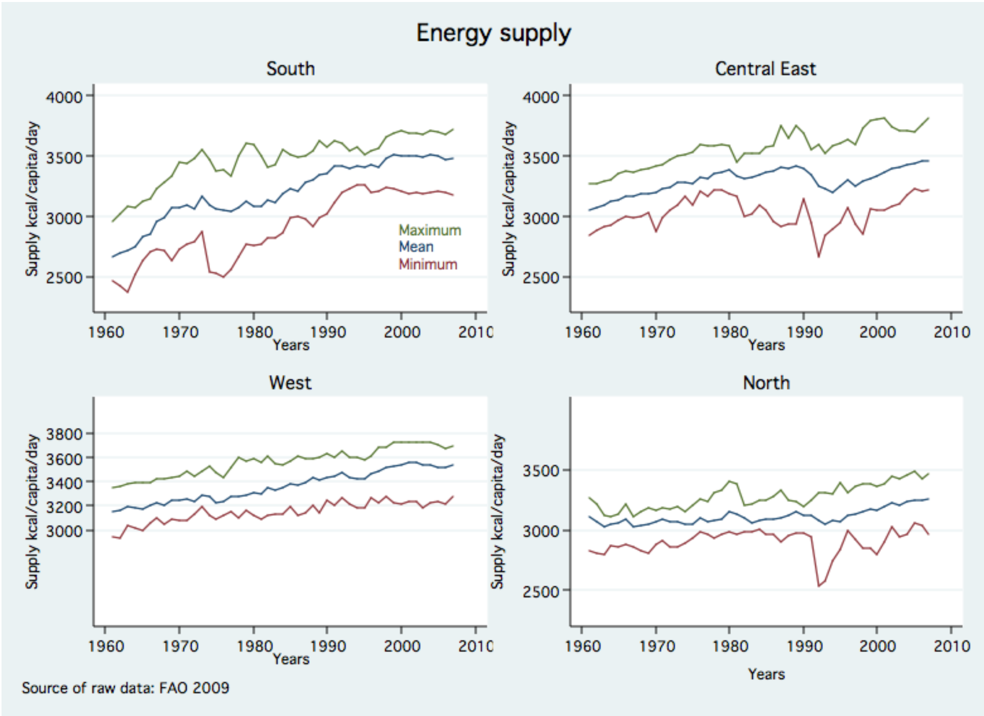

Figure 3.1: Average energy supply in the four regions from 1961 to 2007. Source of raw data: FAO, 2009

3.1.1 Energy

The mean energy supply in the **South** of Europe was rising from 2,671 kcal in 1961 to 3,482 kcal/capita/day in 2007, graphically presented in figure 3.1 Italy started with the highest supply of 2,956 kcal/capita/day in 1961 which increased to 3,646 kcal/capita/day till 2007. Cyprus represented the country with the lowest energy supply with 2,473 kcal/capita/day in 1961 and by 3,181 kcal/capita/day in 2007.

Mean energy supply of the **Central East** region increased over the last decades from 3,053 kcal/capita/day in 1961 to 3,456 kcal/capita/day in 2007. The maximum supply developed from 3,260 kcal/capita/day to 3,819 kcal/capita/day in 2007. Energy supply of Austria was the highest in 2007 in the Central Eastern region. Poland started with the highest energy supply, curve rise till the eighties when it had a slight decrease. Supply is stable since then. Romania had the lowest energy supply of 2,841 kcal/capita/day in 1961. Since the nineties, there is a rising tendency which reached its peak in 2006 with 2,562 kcal/capita/day. Czech Republic and Slovenia started to collect data in 1993 (CZ) and 1992 (SL). Despite a strong increase till 2007, they representing the countries with the lowest energy supply in the Central East.

The progression of the **West** had a steady increase of the mean energy supply from 3,208 kcal/capita/day in 1961 to 3,543 kcal/capita/day in 2007. The minimum of 3,042 kcal/capita/day in 1961 went up to 3,278 kcal/capita/day in 2007. The maximum energy supply of the West rise was from 3,353 kcal/capita/day in 1961 to 3,694 kcal/capita/day in 2007.
While Ireland was in the maximum area in 1961 with 3,353 kcal/capita/day, the Netherlands lay in the minimum area with 3,042 kcal/capita/day. Further, energy supply of Belgium and Luxembourg, which was counted together back then, was the lowest in 1961 with 2,942 kcal/capita/day. This changed over the years with a steady increase to 3,667 kcal/capita/day in the year 2000. Hence, Belgium and Luxembourg represented with 3,694 kcal/capita/day (BE) and 3,681 kcal/capita/day (LU) as well as Ireland (3,612 kcal/capita/day) the maximum area.

Mean energy supply in the **North** was 3,106 kcal/capita/day in 1961 and stayed stable until the middle of the nineties when it started to increase slightly to 3,253. kcal/capita/day. In 1961, the minimum of 2,829 kcal/capita/day increased slightly to 2,962 kcal/capita/day in 2007. The maximum had a slight rise from 3,265 kcal/capita/day in 1961 to 3,464 kcal/capita/day in 2007. Finland, Denmark, and Norway represented the maximum area in 1961. Norway and Denmark remained in the maximum area till 2007 with 3,464 and 3,416 kcal/capita/day.
Sweden had the minimum energy supply of 2,829 kcal/capita/day in 1961 which it runs more or less constant to 3,110 kcal/capita/day in 2007.
The Scandinavian development was stable in opposite to the Eastern Coun-

tries of the Northern region. Estonia started with a low energy supply of 2,523 kcal/capita/day, nevertheless went up to 3,154 kcal/capita/day in 2007. Furthermore, Lithuania showed an increase from 2,970 kcal/capita/day in 1992 to 3,436 kcal/capita/day in 2007 which represents nearly the maximum. On the contrary, energy supply in Latvia decreased from 3,272 kcal/capita/d in 1992 to 2,962 kcal/capita/day in 2007 with its lowest of 2,799 kcal/capita/day in the year 2000.

3.1.2 Fat

Total Fat

Total fat supply increased from 1961 to 2007 in all regions except the North where supply stayed stable. From 1961 to 2007 the South had an increase of 75.3 the West 32.4 and the Central East region 40.8 g/capita/day (Figure 3.2).

The mean of total fat supply in the **South** increased steadily from 73.5 g/capita/day in 1961 to 148.8 g/capita/day in 2007. The minimum ranged from 55.7 g/capita/day in 1961 to 138.7 g/capita/day. Supply maximum climbed from 87.2 g/capita/day in 1961 to 158.6 g/capita/day.
All countries of the South had a steady and notable increase over the last decades. Portugal represented the minimum in 1961 with 55.8 g/capita/day however went up to 141.3 g/capita/year. The maximum reflected by Greece with 87.2 g/capita/day in 1961 went up to 152.0 g/capita/day in 2007. Italy pointed out the maximum of 158.6 g/capita/day in 2007 when started with 77.2 g/capita/day in 1961.

The mean fat uptake of the **Central East** rose up from 93.2 g/capita/day in 1961 to 134 g/capita/day in 2007. The gap between the minimum and the maximum is wider compared to the South. The minimum went up from 55.8 g/capita/day in 1961 to 107.6 g/capita/day in 2007. The maximum supply

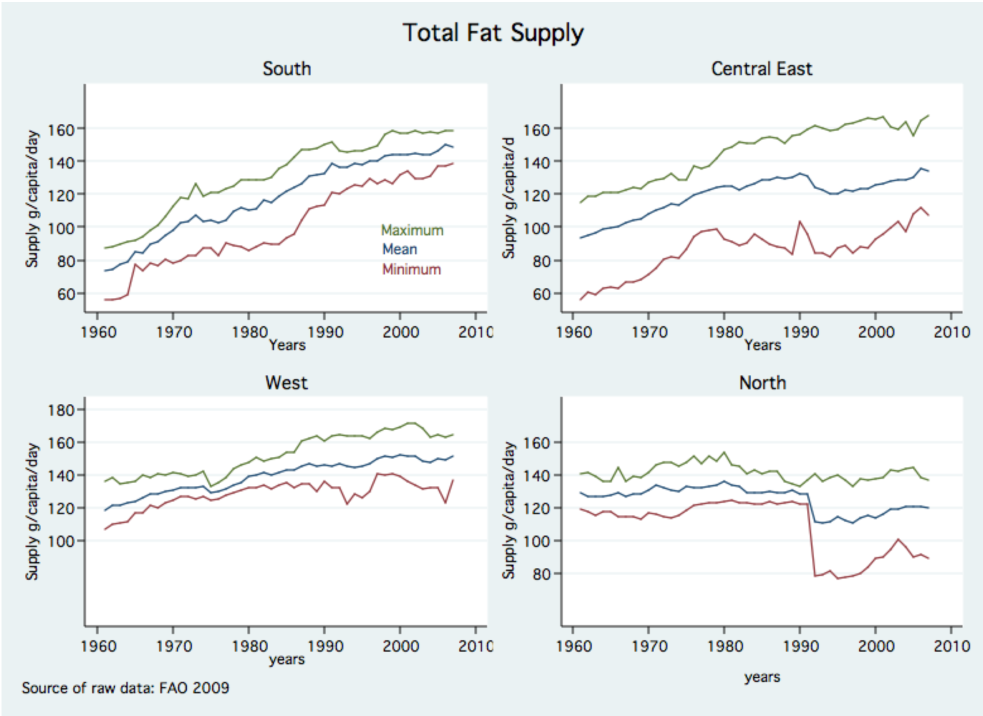

Figure 3.2: Average fat supply in the four regions from 1961 to 2007. Source of raw data: FAO, 2009

increased from 115 g/capita/day in 1961 to 167.9 g/capita/day in 2007. Austria lay in the maximum area with a growth from 111.5 g/capita/day in 1961 to 167.9 g/capita/day in 2007. The minimum was constantly represented by Romania.

The mean supply of the **West** evolved from 119.2 g/capita/day in 1961 to 151.6 g/capita/day in 2007. The minimum in 1961 accounted for 106.9 g/capita/day which rose to 136.6 g/capita/day in 2007. The maximum increased from 136.4 g/capita/day in 1961 to 164.7 g/capita/day in 2007. France showed the minimum supply of 106.9 g/capita/day, despite that climbed up to 164.7 g/capita/day in 2007. Belgium and Luxembourg started with 117 g/capita/day in 1961 and increased their fat supply to 159.3 g/capita/day in 1999. These three countries represented the maximum area in 2007.

In the **North** region, mean and maximum supply stayed about constant. The mean went from 128.9 g/capita/day in 1961 to 119.6 g/capita/day in 2007. The maximum was at 140.5 g/capita/day in 1961 and 136.8 g/capita/day in 2007. The minimum was constant to 1992 when fat supply dropped to 78.8 g/capita/day.

Denmark, Norway, Sweden, and Finland had a constant fat supply, thus Norway had the maximum supply most of the past forty-seven years. Lithuania, Estonia, and Latvia added the minimum supply in 1992. However, all of them had an increase until 2007. The fat supply of Latvia increased from 94.4 g/capita/day in 1992 to 118.5 g/capita/day in 2007.

Animal Fat

The mean animal fat supply of the South had an increase of 7.4 g/capita/day from 1961 to 2007. Animal fat supply of the Central East decreased by 6.6 g/capita/day, of the West by 9.1 g/capita/day and of the North by 25 g/capita/day.

The mean of animal fat supply in the **South** rose from 4.8 g/capita/day in 1961 to 12.2 g/capita/day in 2007. The maximum had a higher increase from 7.3 g/capita/day in 1961 to 25.8 g/capita/day in 2007. The minimum of the animal fat supply stayed almost constant with 2.1 g/capita/day in 1961 and 4.4 g/capita/day in 2007. Italy represented the maximum in 1961 and increased the supply up to 17.6 g/capita/day in 2007. Portugal had a animal fat supply of 7.2 g/capita/day in 1961. It increased to the maximum of 25.8 g/capita/day in 2007. Cyprus, Spain and Greece stayed about constantly in the minimum area.

The gap between the minimum and the maximum of the **Central East** Region, was rather high in 1961. The mean decreased from 33.4 g/capita/day

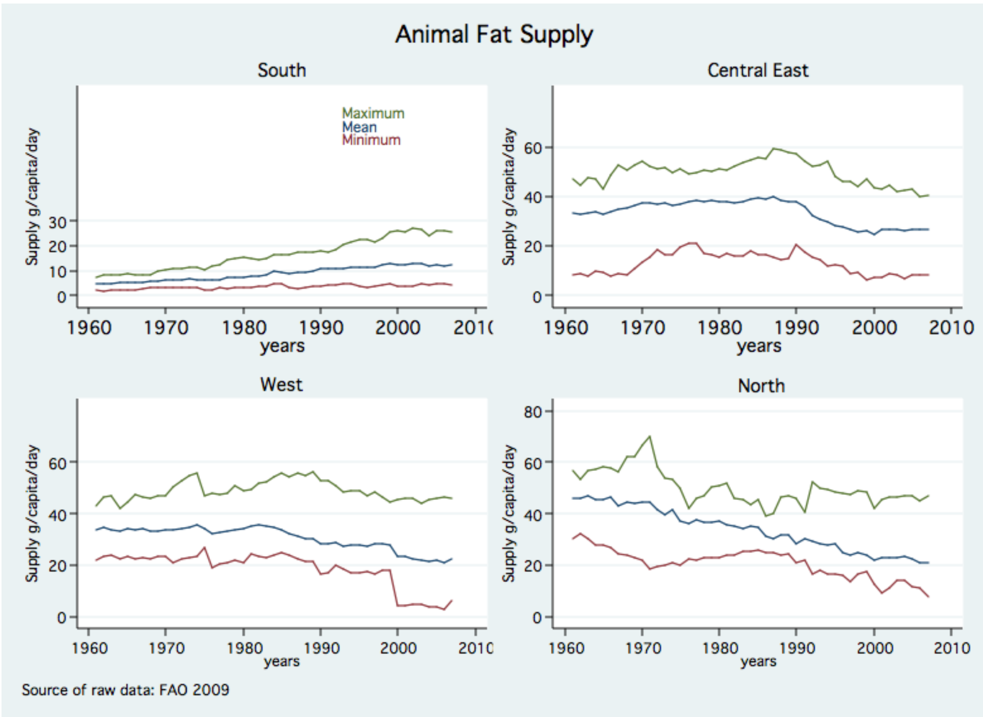

Figure 3.3: Average fat supply in the four regions from 1961 to 2007. Source of raw data: FAO, 2009

in 1961 to 26.8 g/capita/day in 2007. The maximum started with a supply of 46.8 g/capita/day in 1961 and dropped to 40.4 g/capita/day in 2007. The minimum went from 8.1 g/capita/day in 1961 to 8 g/capita/day in 2007. While Hungary posed as the maximum in the last five decades, Romania was the minimum. Thus they showed the same trend like the minimum and the maximum in figure 3.3. Poland had a slight drop down from 40.3 g/capita/day in 1987 to 24.5 g/capita/day in 1994, then supply remained stable. Animal fat supply of Austria and Germany stayed constant nevertheless high in the maximum area. Further, the minimum area is represented by the Czech Republic and Slovenia.

In the **West** the mean decreased from 31.3 g/capita/day in 1961 to 22.2 g/capita/day in 2007. The minimum supply went from 21.7 g/capita/day in 1961 to 6.1 g/capita/day in 2007. The maximum, otherwise, rose from 40.6 g/capita/day in 1961 to 45.8 g/capita/day in 2007.
Belgium and Luxembourg showed the maximum until 1999. While Belgium (45.5 g/capita/day) remained at the maximum level, Luxembourg (4.4 g/capita/day) remained at the minimum. In Ireland and the United Kingdom the animal fat supply decreased. France featured the only country in the West with a slight increase from 21.7 g/capita/day in 1961 to 30.8 g/capita/day in 2007.

The mean of the **North** decreased from 45.8 g/capita/day in 1961 to 20.8 g/capita/day in 2007. Accordingly the maximum went from 56.7 g/capita/day in 1961 to 46.9 g/capita/day in 2007. The minimum dropped from 30.3 g/capita/day in 1961 to 7.7 g/capita/day in 2007.
While Denmark showed the maximum supply in most of the years from 1961 to 2007, Estonia presented the minimum supply from 1992 to 2007.

Vegetable Oil

Vegetable oil trend is ascending in the South and amounts to 37,6 g/capita/day. Iin the Central East vegetable supply increased by 29.9 g/capita/day and in

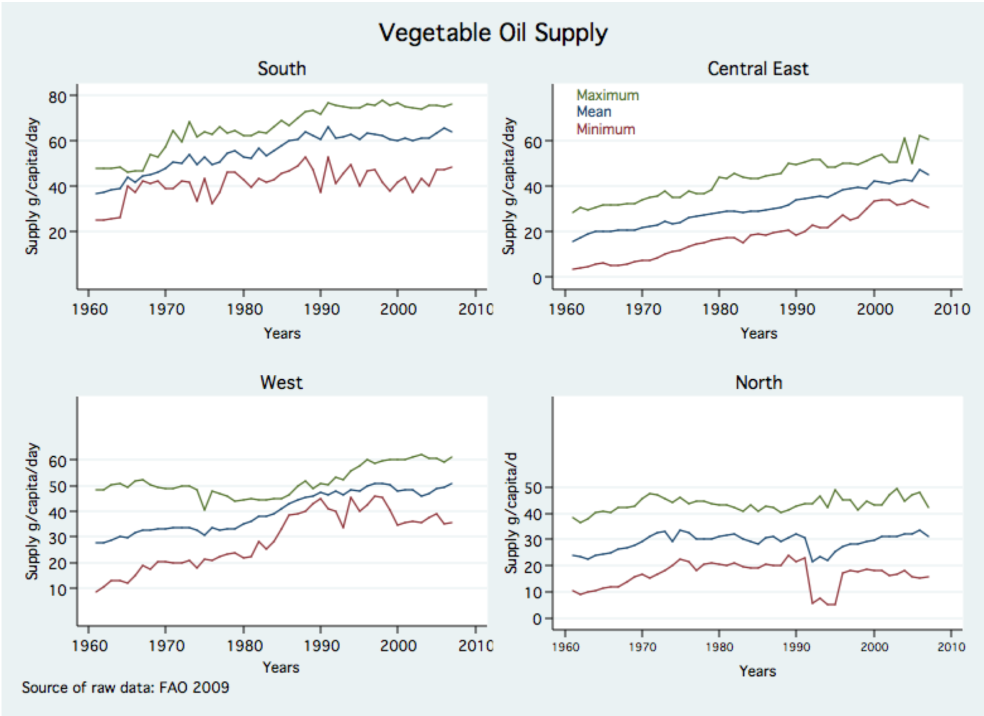

Figure 3.4: Average vegetable oil supply in the four regions from 1961 to 2007. Source of raw data: FAO, 2009

the West by 23.1 g/capita/day. Supply of the North stayed rather stable (Figure 3.4).

The mean of the **South** increased steadily from 26.6 g/capita/day in 1961 to 64.2 g/capita/day in 2007. While the maximum grew more constantly from 47.8 g/capita/day in 1961 to 76.1 g/capita/day in 2007, the minimum fluctuated from 25.2 g/capita/day in 1961 to 48.3 g/capita/day in 2007. Greece represented the maximum in 1961 (47.8 g/capita/day) and further increased the vegetable oil supply to 72 g/capita/day. Portugal represented the minimum in 1961 and in 2007 with a constant development.

The mean of the **Central East** rose from 15.5 g/capita/day in 1961 to 45.4 g/capita/day in 2007. The maximum went constantly up from 28.5 g/capita/day in 1961 to 60.7 g/capita/day in 2007. The minimum increased steadily from 3.6 g/capita/day in 1961 to 30.9 g/capita/day in 2007. Germany showed the maximum in 1961 with 28.5 g/capita/day and rose to 47.3 g/capita/day. Since 1966 Austria represented the maximum with a supply of 60.7 g/capita/day in 2007. Hungary started as the minimum in 1961 with 3.6 g/capita/day thus increased to 54.3 g/capita/day in 2007. In 2007 Poland showed the minimum with 30.9 g/capita/day which rose from 8.9 g/capita/day in 1961.

The **West** developed the mean from 27.7 g/capita/day in 1961 to 50.8 g/capita/day in 2007. In 1961, minimum supply provided Ireland with 8.4 g/capita/day consequently increased to 54.3 g/capita/day in 2007. Maximum in 1961 were the Netherlands with 48.5 g/capita/day. This vegetable oil supply was about constant the last 47 years. While Belgium with a supply of 61.4 g/capita/day was the maximum in 2007, Luxembourg represented the minimum by 35.8. g/capita/day.

The mean of the **North** developed from 24.1 g/capita/day in 1961 to 31.2 g/capita/day in 2007. Sweden was constant in the maximum area with a supply of 42.1 g/capita/day in 2007. Supply of Lithuania, Latvia, and Estonia was in the minimum area in 1992, however, supply of Lithuania rose up to 28.1 g/capita/day in 2007. Supply of Latvia ended with 41 g/capita/day in 2007 and Estonia increased to 22.6 g/capita/day in 2007. Denmark is the only country in the North decreasing vegetable oil supply from 32.2 g/capita/day in 1961 to 15.8 g/capita/day in 2007.

Oil crops

The mean supply of oil crops had a slight increase in all four regions. In the South supply went up by 1.26 g/capita/day, in the Central East 1.32 g/capita/day, in the West 1.39 g/capita/day and in the North the slightest with 0.99 g/capita/day (Figure 3.5).

In the **South** Cyprus presented the maximum in all the 47 past years.

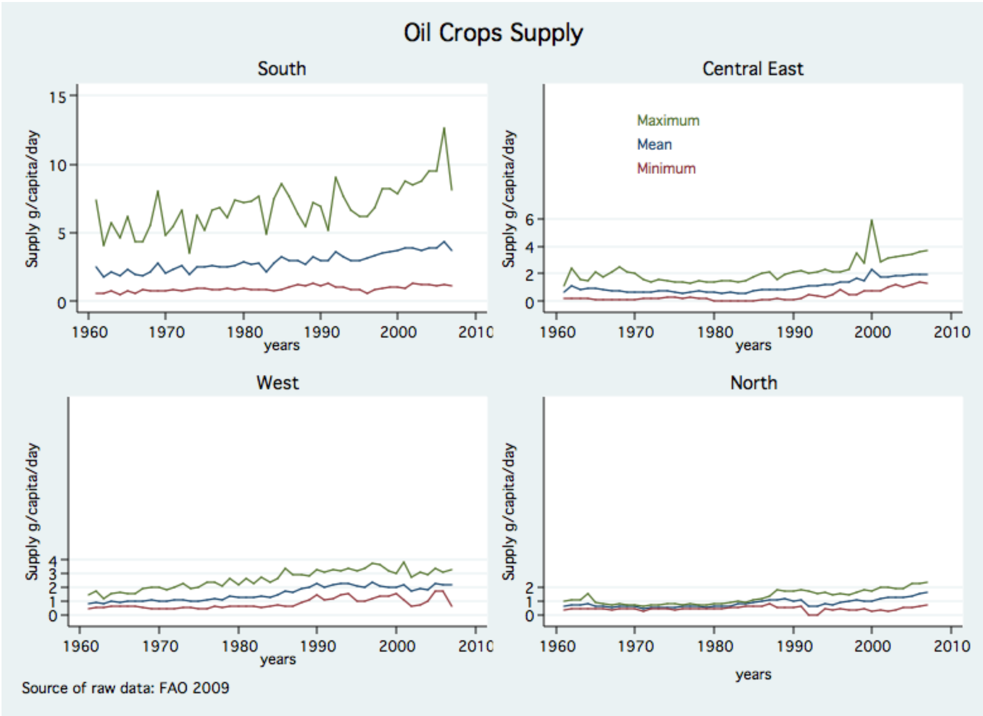

Figure 3.5: Average oil crops supply in the four regions from 1961 to 2007. Source of raw data: FAO, 2009

Supply fluctuated from 7.4 g/capita/day in 1961 to 8.1 g/capita/day in 2007. Italy represented the minimum in 1961 with 0,6 g/capita/day which went to 1.2 g/capita/day in 2007. Supply of Portugal was constant, and represented the minimum in 2007 with 1.1 g/capita/day.

In the **Central East** Germany with 1.1 g/capita/day had the maximum supply which went to 2.2 g/capita/day in 2007. However, Austria's supply increased from 0.6 g/capita/day in 1961 to 3.7 g/capita/day in 2007 when it reached the maximum supply. In 1961 minimum supply was represented by Romania with 0.2 g/capita/day which rose to 1.1 g/capita/day in 2007.

Maximum of the **West** was represented by the United Kingdom in 1961 (1.4 g/capita/day) as well as in 2007 (3.3 g/capita/day). Ireland started with the minimum of the West in 1961 with 0.4 g/capita/day rose since the end of the eighties to 1.5 g/capita/day in 2007. Belgium and Luxembourg lay also in the minimum area, however, in 2000 Luxembourg had the minimum supply of 1.5 g/capita/day which decreased to 0.6 g/capita/day with and reached a peak of 2.3 g/capita/day in 2005.

Sweden demonstrated the highest supply of oil crops of the **North** most of the past 47 years, thus had an increase from 1 g in 1961 to 2.4 g/capita/year in 2007. Norway and Finland presented the minimum of 0.3 g/capita/year in 1961 which went up to 1.4 g (Norway) and 1.6 g/capita/day (Finland) in 2007. Estonia, the minimum, in 2007 had the slightest increase of 0.6 g/capita/day of the eastern countries of the northern region. Supply was 0.7 g/capita/day in 2007.

3.1.3 Carbohydrates

Carbohydrates supply stayed rather stable, some fluctuations were observed but no trends (Figure 3.6). Mean supply of carbohydrates in the **South** went from 420.62 g/capita/day in 1961 to 414 g/capita/day in 2007. Cyprus represented the minimum in 1961 by 366.71 and rose to 375.67 g/capita/day in 2007. Carbohydrates supply of Spain (421.13 g/capita/day, 1961) decreased to the minimum in 2007 (351.88 g/capita/day) of the Southern region. The highest supply in 1961 had Italy (477.01 g/capita/day) which fell to 431.36 g/capita/day in 2007. Supply of Greece increased from 419.56 g/capita/day in 1961 to the maximum supply in 2007 (458.75 g/capita/day).

Germany had the lowest supply in the **Central East** in 1961 (374.8 g/capita/day) developing to 450.3 g/capita/day in 2007, whereas Czech Republic had the maximum supply of 416.4 g/capita/day in 1961. In 1961 Maximum was hold by Poland with 515.5 g/capita/day which slightly decreased to 487.7 g/capita/day in 2007. Romania had a supply of 492.7 g/capita/day in 1961 which fell till the nineties, however the supply returned to 503.68 g/capita/day in 2007, representing the maximum. The mean from 458.8 g/capita/day in 1961 to 451.6 g/capita/day in 2007 stayed stable.

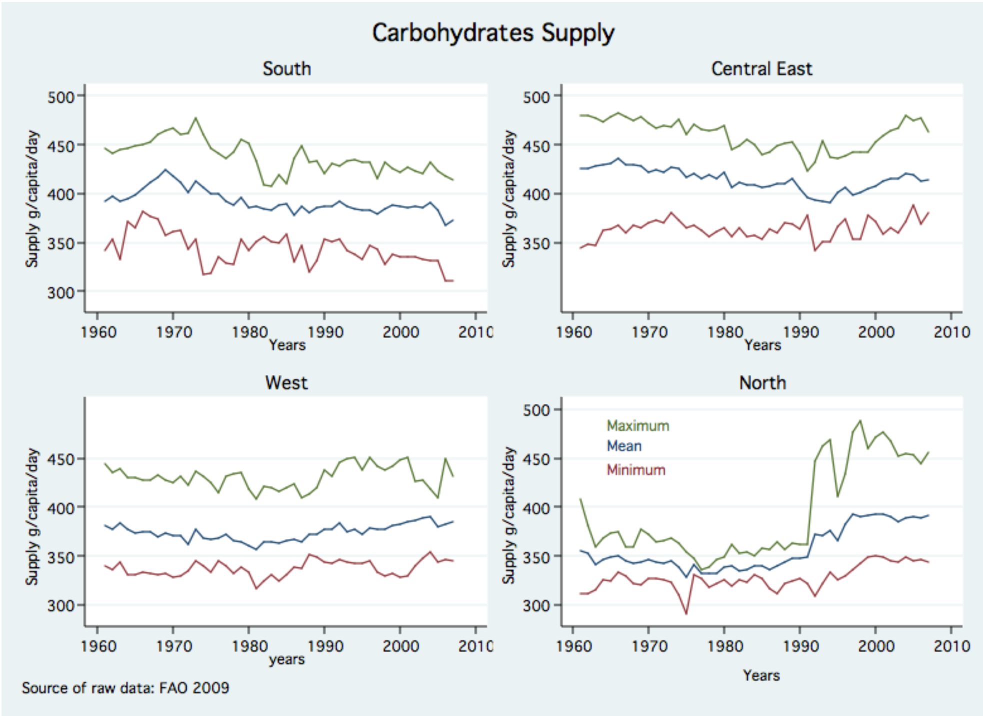

Figure 3.6: Average supply of carbohydrates in the four regions from 1961 to 2007. Source of raw data: FAO, 2009

Carbohydrate supply in the **West** stayed as well constant with a mean of 417.3 g/capita/day in 1961 and 425.1 g/capita/day in 2007. Minimum supply of 373.78 g/capita/day had Belgium and Luxembourg in 1961, later in 2007 France with 387.17 g/capita/day. Ireland held the maximum most of the past decades with a supply of 481.86 g/capita/day in 1961 and 472.03 g/capita/day in 2007.

The mean of the **North** went from 388.3 (1961) to 430.4 g/capita/day (2007), however, supply of the countries remained stabile. Slight increase was due to additional countries since 1993, which had a higher carbohydrate supply. For instance Lithuania (499.7 g/captia/day, 2007) which presented the maximum in 2007 and Latvia which dropped to the minimum in 2007 (376.69 g/capita/day).

3.1.4 Sugar and Sweeteners

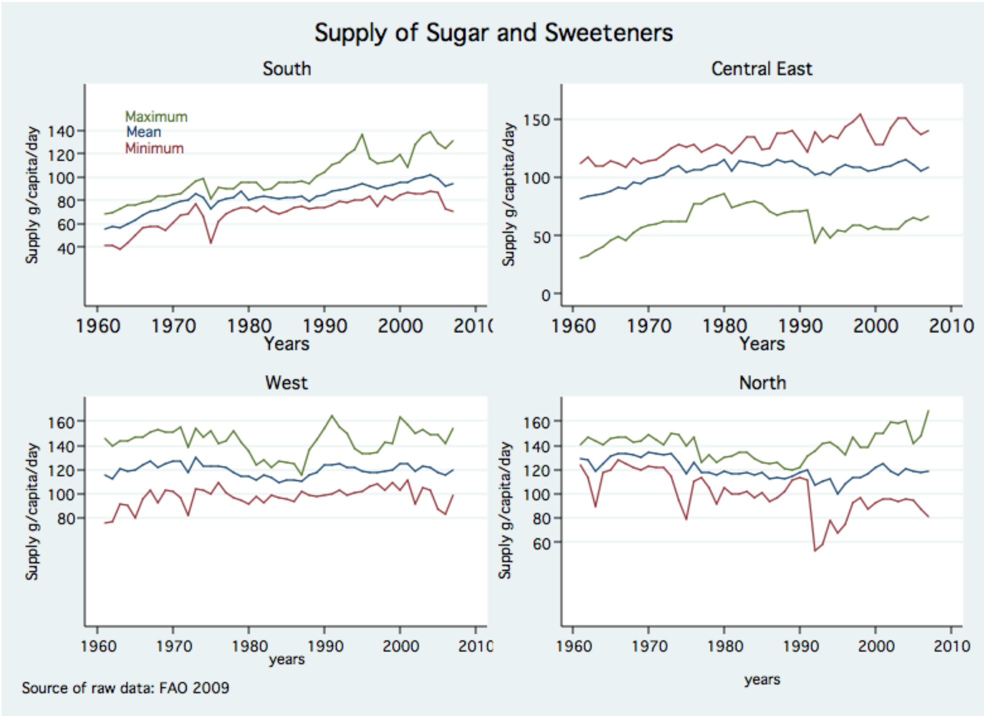

Figure 3.7: Average supply of sugar and sweeteners in the four regions from 1961 to 2007. Source of raw data: FAO, 2009

There was no trend observed of the mean of sugar and sweeteners supply for the North and the West. The South showed an increase of 38.3 g/capita/day and the Central East rose by 26,9 g/capita/day from 1961 to 2007 (Figure 3.7)

The mean of the **South** started with 55.9 g/capita/day in 1961 and rose consequently to 94.2 g/capita/day with a minimum of 41.9 g/capita/day in 1961 developing to 70 g/capita/day in 2007. The maximum supply at 68.8 g/capita/day in 1961 increased to 131.5 g/capita/day
Cyprus, with a supply of sugar and sweeteners of 61.4 g/capita/day in 1961 had an increase to the maximum of 131.5 g/capita/day in 2007. Greece had the lowest supply of 41.9 g/capita/day in 1961 which rose to 90.9 in 2007. Greece reached the peak in 1989 with 100.8 g/capita/day. The supply of

Spain rose from 56.2 g/capita/day in 1961 to 70.4 g/capita/day in 2007, hence Spain represented the minimum supply in 2007.

In the **Central East** the mean ranged between 81.8 g/capita/day in 1961, and 108.7 g/capita/day in 2007. The maximum supply went from 112.1 g/capita/day in 1961 to 140.3 g/capita/day in 2007. While Austria, Poland, Hungary, and Germany had an approximate stable development over the last fourty years, the Czech Republic had a decrease from 112.9 g/capita/day in 1993 to 101.9 g/capita/day in 2007 with a peak of 150.7 g/capita/day in 2004.
Romania presented the minimum with 30.1 g/capita/day in 1961 and rose to 78.63 g/capita/day in 2007. Slovenia reflected the minimum of 43.3 g/capita/day in 1993 and increased to 66 g/capita/day in 2007.

Countries of the **West** had a diverse development. The mean with 125.1 g/capita/day in 1961 and 120.1 g/capita/day in 2007 stayed constant, respectively. The maximum went from 145,8 g/capita/day in 1961 to 154 g/capita/day in 2007 however maximum supply had a peak of 164.9 g/capita/day in 1991 and its lowest of 115,9 g/capita/day in 1987. The minimum ranged from 83.6 g/capita/day in 1961 to 99.2 g/capita/day in 2007. Ireland represented the maximum of 145.8 g/capita/day in 1961. It reduced the supply to 114.5 g/capita/day with the lowest of 86.6 g/capita/day in 2005. The Netherlands had a similar supply in 1961 and 2007 with 128.8 and 128.5 g/capita/day respectively, nevertheless showed the peak of 164.9 g/capita/day in 1991. United Kingdom had a decreasing trend. The supply of sugar and sweeteners declined from 142.2 g/capita/day in 1961 to 99.1 g/capita/day in 2007. In contrast, supply in Belgium and Luxembourg increased from 75.9 g/capita/day in 1961, which was the minimum at that time, to 141.9 g/capita/day in 1999. In the year of the millennium Belgium had a higher supply than Luxembourg. Although it decreased from 163.6 g/capita/day to 154 g/capita/day in 200, it still represented the maximum.

The mean of the **North** decreased from 128.9 g/capita/day in 1961 to 119.1 g/capita/day in 2007. While the minimum supply increased from 123.8

g/capita/day in 1961 to 81.1 g/capita/day, maximum rise up from 140.8 g/capita/day in 1961 to 168.5 g/capita/day in 2007.
Denmark represented the maximum with 140.8 g/capita/day in 1961. The supply of sugar and sweeteners went up to 168.5 g/capita/day in 2007. Finland started with the highest supply of 123.8 g/capita/day in 1961. After a irregular development it declined to 94.3 g/capita/day in 2007.
Estonia contributed with the lowest supply of 52.2 g/capita/day in 1992, however went up to 127 g/capita/day in 2007. Latvia reduced its supply form 110.1 g/capita/day in 1992 to 81.1 g/capita/day in 2007, and therefore represented the minimum supply.

3.1.5 Trends of mortality from CVD

Mortality of male and female dropped from 1970 to 2009 in all four mortality causes (mortality from diseases of the circulatory system, mortality from ischemic diseases, mortality from cerebrovascular diseases and mortality from all causes). In general standardised (and non standardised) death rates for men are higher than for women in all regions and in all 4 categories. Further, a stronger decline was observed in men than in women from 1970 to 2009 with exception of the North regarding the SDR of cerebrovascular diseases. In that case they declined about the same amount.

The **South** had the lowest mean SDR for deaths caused by diseases of the circulatory system in female in 1970 and for deaths from ischemic heart diseases in both sexes in 1970 and 2009. In 2009, the South had the lowest SDR for male which died of diseases of the circulatory system. Overall mortality was the lowest in 1970 for male and in 2009 for female.

The **Central East** had the highest mortality rate over the years for all causes and all years except of the mortality from ischemic diseases of men. The North contributed the highest mortality rate in 1970 and 2009. However, it was the region with the lowest mortality decrease of deaths from ischemic diseases in male and female and for the SDR of cerebrovascular diseases in female.

The lowest SDR for deaths of the circulatory system in male in 1970 and in female in 2009 had the **West**. Further in 2009 for the deaths of cerebrovascular diseases in male and female and mortality from all causes in male. The highest decrease of the West was for the deaths of the diseases of the circulatory system (female), of ischemic diseases (male and female), of cerebrovascular diseases (male and female) and the mortality from all causes (male). The lowest decline of deaths of the circulatory system in male was also presented by the West.

The **North** had the lowest SDR in 1961 regarding cerebrovascular diseases of male and female as well as the total mortality of female. It had the highest SDR of ischemic diseases of male in 1970 and 2009. The lowest decline of overall mortality and the SDR of cerebrovascular diseases in male was provided by the North.

Mortality of diseases of the circulatory system (Figure 3.8)

In 1970 highest SDR (deaths per 100,000 inhabitants) in the **South** was seen in Portugal with 416.79 in female and 599.11 in male which decreased constantly to 152.35 in female and 208.3 in male in 2009. In 1970 Greece showed the minimum SDR of 278.58 (female) and 326.53 (male) which went to 219.24 (female) and 277.03 (male) in 2009, therefore represented the maximum in the southern area in 2009. Further, Greece had the lowest SDR in male in comparison with all 24 countries in 1970. In 2008 Spain reached the lowest SDR of 122.43 (female) and 183.72 (male).

Women of Romania reflected the highest SDR in the **Central East** over the past 39 years as well as in comparison with all the 24 countries. There was still a decrease from 638.89 (female) in 1970 to 461.9 in 2009. According to men Romania was the central eastern country with the highest SDR in 2009. SDR developed from 711.19 in 1970 to 658.06 in 2009. In male,

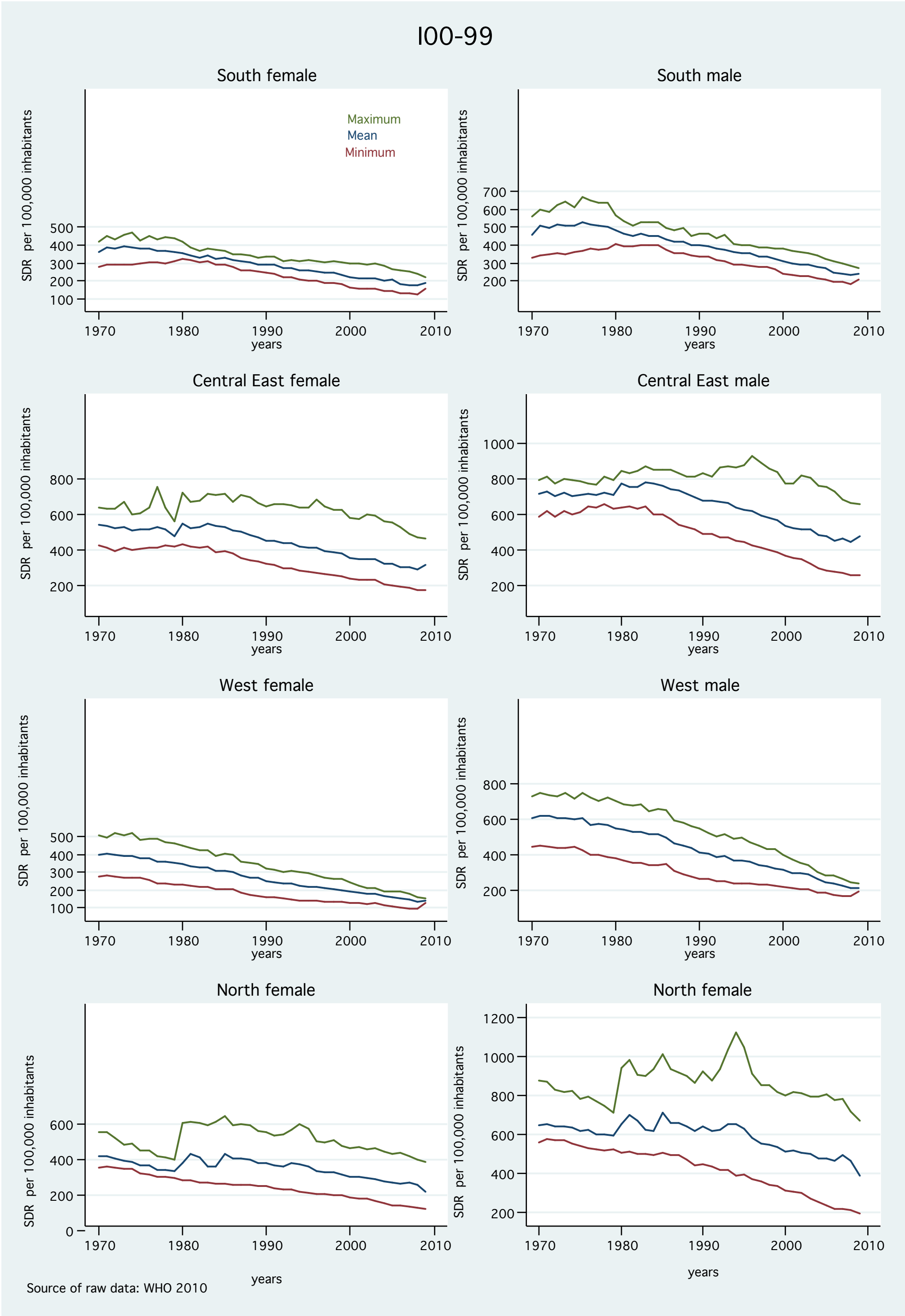

Figure 3.8: Standardised death rate per 100,000 inhabitants for mortality from diseases of the circulatory system for male and female from 1970 to 2009. Source of raw data: WHO, 2010

Hungary had the highest SDR in 1970 with 796.21 falling to 548.41 in 2009. In 1970 the lowest SDR was observed in gave Poland for female (427.02) and male (589.66) decreasing after a slight increase to 277.17 (female) and 463.74 (male) in 2008. SDR of Austria declined from 476.58 (for female and 684.03 for male in 1970, to the bottom of 177.23 (female) and 258.67 (male) in 2009.

In the **West**, Ireland had the highest SDR most of the years since 1970 when SDR was 505.93 for female and 727.6 for male. SDR dropped to 151.39 (female) and 237.85 (male) in 2009 still representing the maximum in the West. France had the lowest SDR from 1970 to 2008, starting with 276.88 (female) and 444.05 (male) in 1970, and decreased constantly to 123.98 (female) and 193.19 (male) in 2008. Among all 25 countries France possessed the lowest SDR for deaths of diseases of the circulatory system in female in 1961 and 2008 of all 25 countries.

Finlands SDR was the highest in the **North** with 554.52 (female) and 875.71 (male) in 1970. However, SDR dropped to 157.86 (female) and 294.9 (male) in 2009. Furthermore, Finland had the highest SDR in male among all countries in 1961. In females, Lithuania demonstrated the highest SDR in the northern region as well as in all other 25 countries in 2009. SDR decreased from 492.12 in 1981 to 384.92 in 2009. Norway had the lowest SDR most of the years, it dropped from 358.86 in 1970 to 124.22 in 2009. In male Latvia had the highest SDR in 2009 which increased from 940.45 in 1980 to 671.56 in 2008. In 1970 Sweden reflected the minimum with 558.21 which fell to 245.29 in 2009. Norway's SDR was the lowest in the northern region and in regard to all countries. SDR fell from 581.58 in 2970 to 198.11 in 2009.

Mortality of cerebrovascular diseases (Figure 3.9)

Portugal was representing the maximum with 238.43 (female) and 292.54 (male) in 1970 for the **South** and for all other countries. It decreased steadily to 66.57 in female and 84.55 in male in 2009. While in females Portugal had the lowest SDR of the south in 2009 whereas in male it was still the highest. Nevertheless in 2008 Spain reflected the lowest SDR of 33.8 in female in contrary to male where Cyprus with a SDR of 39.85 was the lowest. Greece supplied the minimum SDR of 124.07 in 1970 in female. SDR decreased slightly to 74.93, however presented the maximum in this area. In male Greece represented the lowest SDR of 118.24 in 1970 which decreased to 73.64 in 2009.

The Czech Republic had the highest SDR in the **Central East**. It dropped from 187.01 (female) and 229.46 (male) in 1970 to 71.62 (female) and 88.52 (male) in 2009. Romania represented the maximum with a SDR of 194.3 (female) and 195.3 (male) in 2009 which increased slightly form 185.73 (female) and 187.26 (male) in 1970. Overall the trend of Romania is decreasing since the middle of the nineties. However, Romania had the highest SDR among all countries in 2009. The minimum SDR presented Poland with 53.76 (female) and 57.93 (male) in 1970 for the South as well as for all countries. Mortality slightly increased to 64.09 (female) and 91.42 (male) in 2008. In 2009, Austria showed the minimum SDR. There was a strong decrease observed from 160.41 to 30.23 (female) and 201.88 to 37.96 (male).

In the **West**, Ireland had the highest SDR in female with 159.94 falling to 38.74 till 2009. In male, the highest SDR hold Belgium with 170.83 in 1970 which fell to 47.24 in 2005. United Kingdom presented the maximum SDR in 2009 with 41.72 (female) and 44.11 (male) falling from 143.39 (female) and 161.3 (male). Minimum in the West showed the Netherlands in 1970 and 2009 in male as well as in female. SDR decreased from 108.86 (female) and 116.34 (male) to 33.52 (female) and 35.93 (male). Furthermore, the Netherlands represented the lowest SDR in male.

In 1970, the maximum SDR in the **North** showed Finland with 170.68 (female) and 195.31 (male) which went down to 40.33 (female) and 52.1 (male). In 2009, Lithuania had the highest SDR in the North with 105.86 in female and 138.61 in male. SDR of Lithuania stayed constant since 1981. Sweden represented the minimum SDR in 1970. Starting with a SDR of 88

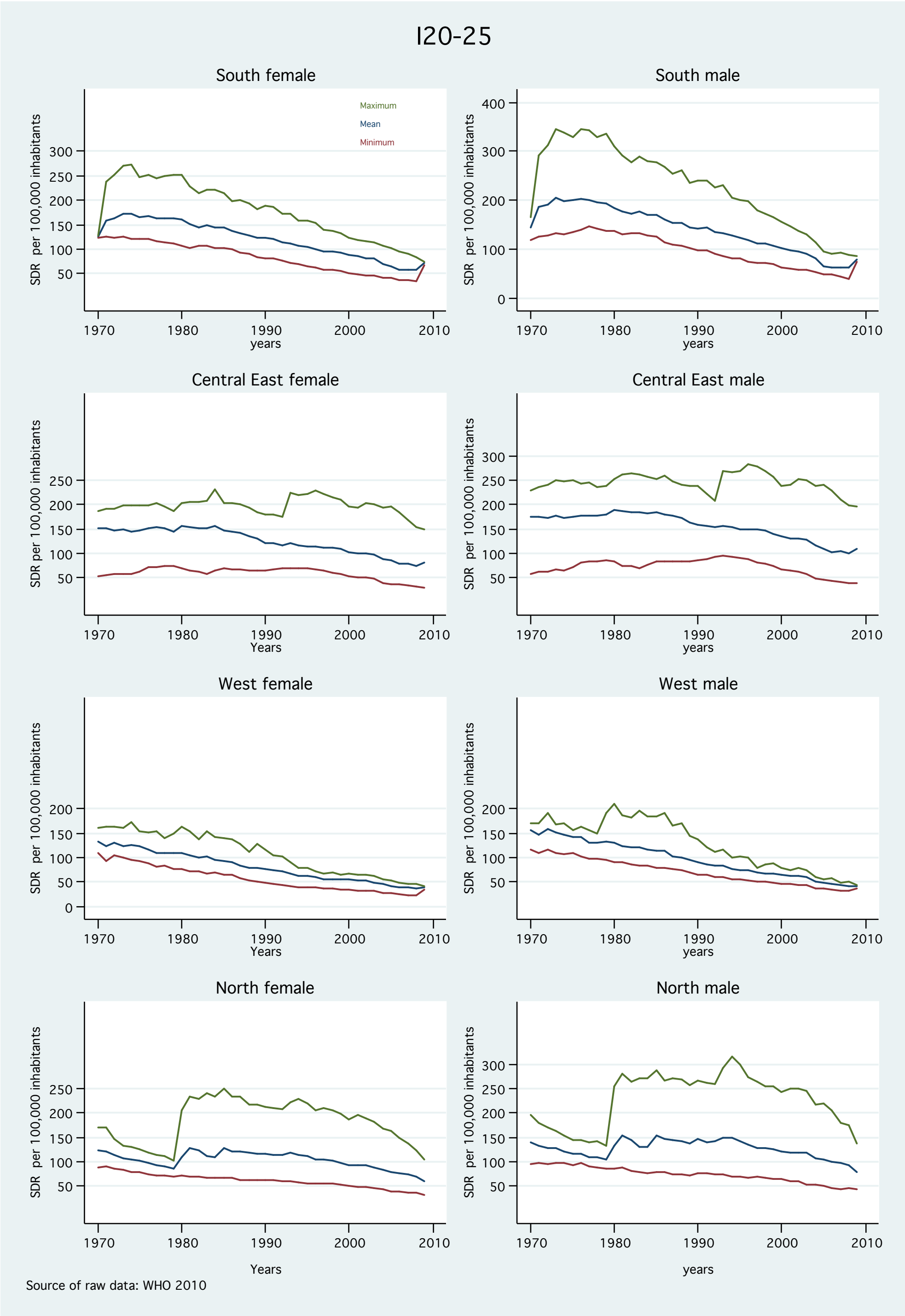

Figure 3.9: Standardised death rate per 100,000 inhabitants for mortality from cerebrovascular diseases for male and female from 1970 to 2009. Source of raw data: WHO, 2010

(female) and 95.58 (male) in 1970 it declined to 38.83 (female) and 46.96 in 2008. In 2009, Norway submitted the lowest SDR of 33.3 (female) and 43.38 (male) which decreased from 132.62 (female) and 155.87 (male) in 1970. Norway further performed as minimum of all countries in 2009.

Mortality of ischemic diseases (Figure 3.10)

In 1970 the **South** had the maximum SDR represented by Italy in female and male. SDR fell from 110.89 (female) and 189.08 (male) to 44.13 (female) and 85.55 (male) in 2007. In 2009, Greece represented the maximum SDR which stayed constant over the past 39 years. In 2009 SDR for deaths of ischemic diseases was 41.13 in female and 96.63 in male. Spain had the lowest SDR in 1970 of 38.25 (female) and 77.55 (male) which decreased slightly to 29.47 (female) and 69.32 (male). In 2009 Portugal pictured the minimum SDR by falling from 70.11 (female) and 140.63 (male) in 1971 to 30.55 (female) and 56.7 (male) in 2009. Moreover Portugal represented the smallest SDR for all countries.

The **Central East** had a maximum of 226.51 (female) and 406.7 (male) presented by Czech Republic. SDR decreased to 133.58 (female) and 218.29 (male) in 2009. Further among all countries, the Czech Republic was the highest in 1970. Hungary had the highest SDR in 2009 with 163.04 (female) and 288.98 (male) in 2009, but the decrease was small because of the low initiating SDRs, 198.2 for female and 313.94 for male. In Poland SDR increased from the minimum of 51.72 in female and 121.42 in male in 1970, to 68.29 (female) and 147.53 (male) in 2008. Slovenia had the lowest SDR in 2009. SDR went from 89.14 (female) and 185.88 (male) in 1985 to 72.56 (female) and 131.48 (male).

In women Ireland showed the maximum SDR in the **West** in 1970 as well as in 2009. SDR decreased from 195.14 in 1970 to 69.2 in 2009. In male Ireland hold the maximum SDR most of the years and had an SDR of 143.74 in 2009 falling from 375.3 in 1979. However, in 1970, the United

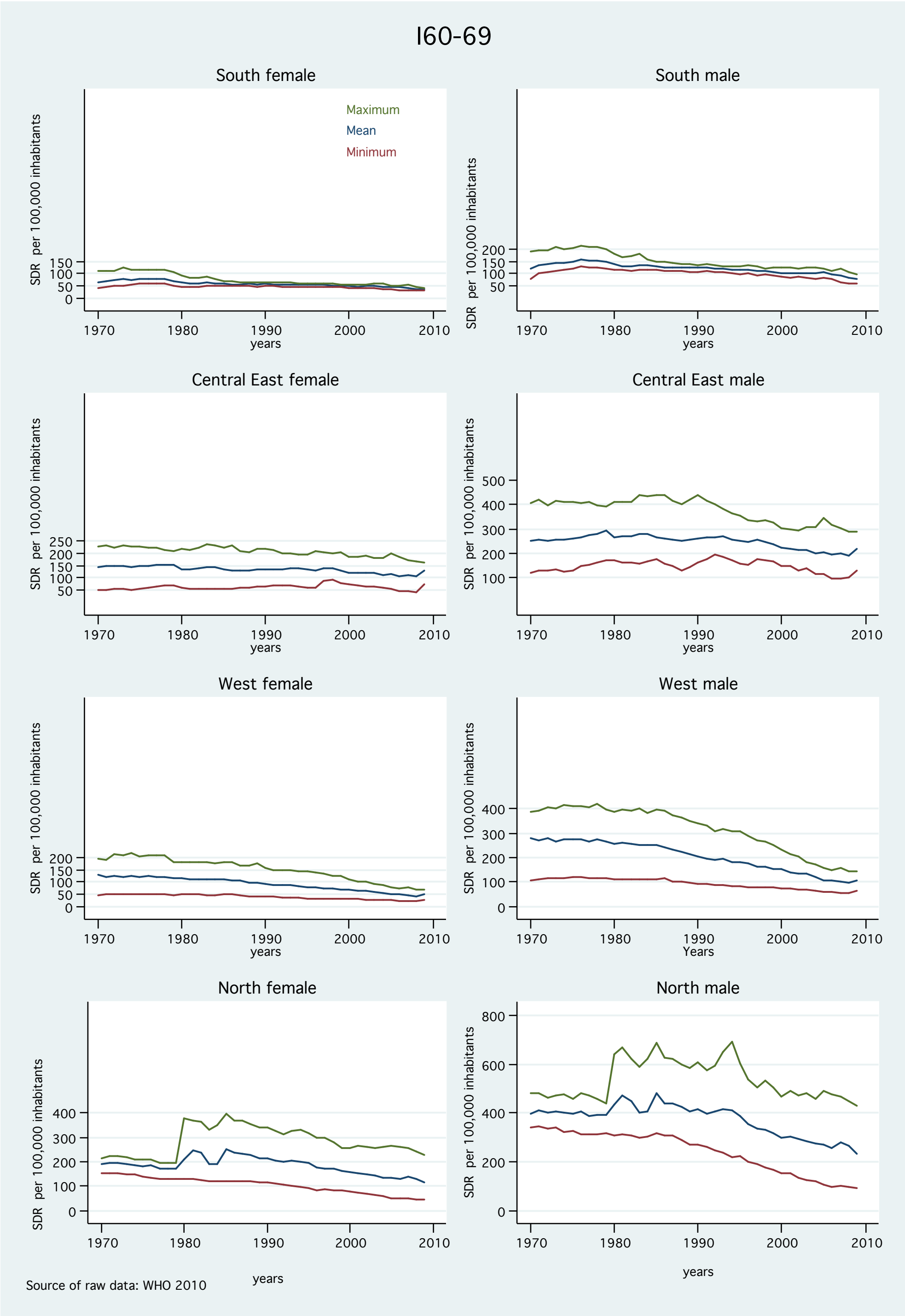

Figure 3.10: Standardised death rate per 100,000 inhabitants for mortality from ischemic diseases for male and female from 1970 to 2009. Source of raw data: WHO, 2010

Kingdom had a higher SDR of 387.46 decreasing to 115.6 in 2009. SDR of France reflected the minimum from 46.6 (female) and 106.24 (male)in 1970 to 20.31 (female) and 53.28 (male) in 2008. In 2009, minimum SDR found in the Netherlands with 28.16 (female) and 64.64 (male) decreasing from 134.86 (female) and 294.36 (male) in 1970. While the Netherlands had the lowest SDR in male in 2009, Spain possessed the minimum in female and male in 1961 among all countries.

While Norway had the lowest SDR in the **North**, declining from 135.77 (female) and 340 (male) in 1970 to 44.85 (female) and 92.35 (male) in 2009, Sweden showed the maximum SDR in female and Finland in male in 1970. Sweden went from 214.93 (female) in 1970 to 62.14 in 2008. Finland reduced the SDR from 480.22 (male) in 1970 to 92.35 in 2009. However Lithuania presented the maximum in 2009 in female and male. It decreased from 351.44 (female) and 516.07 (male) in 1985 to 229.46 (female) and 428.55 (male) in 2009.

Mortality of all causes (Figure 3.11)

The **South** presented the maximum by Portugal in 1970 (male and female). SDR of total mortality declined from 1117.45 in 1970 to 470.69 in 2009 (female). In male SDR declined from 1682.88 to 790 in 2009. Additionally, in female and in male Portugal demonstrated the maximum SDR in 1970. Portugal decreased to the minimum SDR in female in 2009. In 2009 the highest SDR in female was found in Greece with 473.26.
In 1970 and in 2009 the lowest SDR in male hold as well Greece (753.96) which represented the minimum over all countries in male in 1970. The greek SDR went from 1016.25 in 1970 to 693.18 in 2009.

While in the **Central East** Romania had the highest SDR from 1970 (1085.77) to the year 2009 (733.01) in female, in 1970 Romania presented the minimum SDR by 1422.98 in male. Mortality stayed rather constant. In 2009 SDR was 1236.34. Furthermore Romania had the highest SDR in all

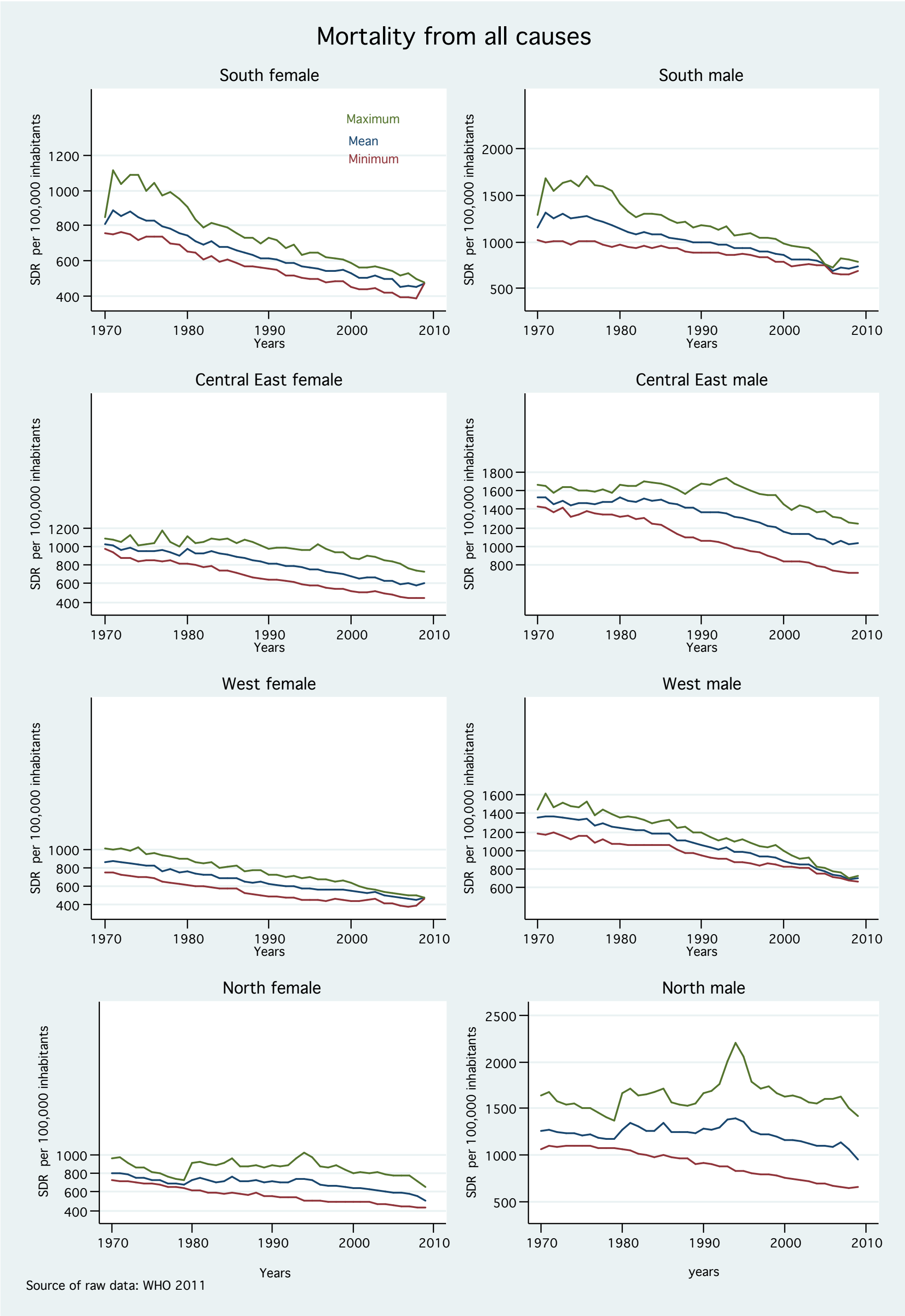

Figure 3.11: Standardised death rate per 100,000 inhabitants for mortality from all causes for male and female from 1970 to 2009. Source of raw data: WHO, 2010

countries in female in 1970. However in male the Czech Republic hold the maximum SDR with 1656.24 in 1970 decreasing to 962.54 in 2009. Hungary declined the SDR from 1970 (1513.96) to the maximum of 2009 (1244,83) In 1970 the lowest SDR in female had Poland declining from 975,32 to 588,93 in 2008. In 2009 Austria showed the minimum in female and male. In female SDR went from 733.01 (1970) to 422.06 (2009). In male there was a stronger decrease from 1548.2 to (1970) to 718.57 (2009).

In the **West** Ireland presented the highest SDR in female from 1970 to 2009. SDR dropped from 1009.24 to 476.50. Further in male Ireland hold the highest SDR in 2009 which went from 1432,6 in 1970 to 729.79 in 2009. In 1970 it was Belgium which had the highest SDR of 1441.37 declining to 811.42 in 2005. Netherlands showed the minimum SDR in male in 1970 and in female in 2009. In male SDR decreased from1183.44 to 693,11 in 2009. In female SDR went from 755.78 to 465.12 in 2009. France hold the minimum for female in 1970 with a SDR of 743.84 going down to 385,57 in 2009. United Kingdom had the lowest SDR in 2009 (male) developing from1411.79 in 1970 to 670.69 in 2009.

Finland had the highest SDR in the **North** in 1970 (female and male). While in male SDR decreased from 1644.29 (1970) to 768,29 (2009), in female SDR went from 966.4 (1970) to the lowest SDR of 431.92. In 2009 Lithuania presented the maximum which stayed more stable in male than in female. In female SDR decreased from 802.24 in 1981 to 659.24 in 2009. In male SDR went from 1416.99 (1981) to 1416.99 (2009). Further Lithuania hold the maximum SDR for all countries in 2009. Sweden had the lowest SDR in both sexes in 1970 and the minimum SDR for all countries in male in 2009. In female SDR declined from 728.1 (1970) to 431.92 (2009) and in male from 1065.45 (1970) to 642.93 (2008). In 2009 Norway represented the minimum for the northern region with a SDR going from 1155.03 (1970) to 657.05 (2009) which additionally is the minimum for all countries in 2009.

However, examining the development of the gaps between the mean, the minimum, and the maximum of the North and the Central East, it has been obvious that there was a difference in mortality from CVD in some countries. When the gaps of the South and West heading towards each other, gaps of the North and Central East drifted apart. Due to the fact that the division of these regions were made in respect of the common dietary habits, there had to be further reasons for mortality from CVD. Taking a closer look at the countries, it appeared that EEC had a significantly higher mortality from diseases of the circulatory system (Figure 3.12).

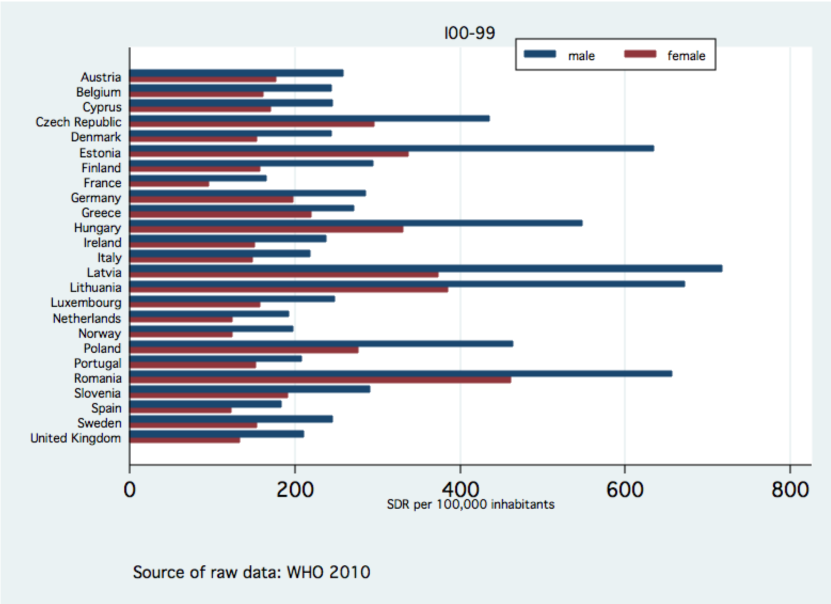

Figure 3.12: Mortality from diseases of the circulatory system for all countries in 2009

3.2 Dietary Fats and Carbohydrates and Mortality from CVD

Table 3.2 and Table 3.1 show the Spearman rank correlation's rho for all objectives as well as other risk factors (alcohol, smoking behaviour, PAL) for women and men.

Table 3.1: Spearman rank correlation coefficient for all objectives for male[a]

		All Countries		EEC		WEC	
		r	P	r	P	r	P
Energy		n 22		n 7		n 15	
	I00-99	0.1992	0.3743	-0.6071	0.1482	-0.0215	0.9394
	I60-69	0.1149	0.6108	-0.6786	0.0938	-0.222	0.4264
	I20-25	0.34	0.1215	-0.1429	0.7599	0.1629	0.5618
	total mortality	0.3078	0.1635	-0.6786	0.0938	0.188	0.5022
CHO total		n 21		n 7		n 14	
	I00-99	-0.0182	0.9376	-0.9643	0.0005	0.3451	0.2269
	I60-69	-0.0234	0.9199	-0.9286	0.0025	0.3143	0.2738
	I20-25	-0.0734	0.7519	-0.6071	0.1482	0.1648	0.5733
	total mortality	-0.1715	0.4573	-0.9286	0.0025	-0.0637	0.8286
Fat		n 22		n 7		n 15	
	I00-99	0.4167	0.0537	0.75	0.0522	-0.0036	0.9899
	I60-69	0.4676	0.0282	0.7143	0.0713	0.127	0.6519
	I20-25	0.3042	0.1687	0.5	0.2532	-0.3292	0.2309
	total mortality	0.3472	0.1134	0.7143	0.0713	-0.1932	0.4903
SFA		n 18		n 5		n 13	
	I00-99	-0.0072	0.9773	0.5	0.391	-0.0496	0.8722
	I60-69	-0.0021	0.9935	0.1	0.8729	0.0275	0.9288
	I20-25	-0.215	0.3916	0.7	0.1881	-0.4821	0.0952
	total mortality	-0.1034	0.6832	0.1	0.8729	-0.1818	0.5522
MUFA		n 15		n 4		n 11	
	I00-99	0.2288	0.4121	0.4	0.6	-0.1185	0.7278
	I60-69	0.3253	0.2368	0.4	0.6	0.0729	0.8313
	I20-25	0.1001	0.7227	-0.6	0.4	-0.3098	0.3539
	total mortality	0.1573	0.5756	0.4	0.6	-0.2096	0.5363
PUFA		n 15		n 4		n 11	
	I00-99	0.3211	0.2433	0.4	0.6	0.0966	0.7776
	I60-69	0.4646	0.081	0.4	0.6	0.3724	0.2593
	I20-25	-0.0646	0.8192	-0.4	0.6	-0.5702	0.067
	total mortality	0.2493	0.3702	0.5	0.6	-0.0322	0.9252
Cholesterol		n 17		n 6		n 11	
	I00-99	0.451	0.0692	0.0286	0.9572	-0.4909	0.1252
	I60-69	0.4559	0.0659	-0.1429	0.7872	-0.4273	0.1899
	I20-25	0.5245	0.0307	0.2	0.704	-0.2273	0.5015
	total mortality	0.4926	0.0445	-0.1429	0.7872	-0.3455	0.2981
BMI		n 22		n 7		n 15	
	I00-99	0.3265	0.138	-0.7857	0.0362	0.5627	0.029
	I60-69	0.3622	0.0976	-0.75	0.0522	0.6308	0.0117
	I20-25	0.253	0.256	-0.4286	0.3374	0.3369	0.2195
	total mortality	0.1834	0.414	-0.75	0.0522	0.2903	0.2939
Somking		n 16		n 3		n 13	
	I00-99	0.6814	0.0037	-0.5	0.6667	0.5695	0.0422
	I60-69	0.4812	0.0591	-0.5	0.6667	0.2613	0.3884
	I20-25	0.6137	0.0115	-0.5	0.6667	0.4484	0.1243
	total mortality	0.6313	0.0087	-0.5	0.6667	0.4924	0.0873
Alkohol		n 17		n 5		n 12	
	I00-99	-0.0392	0.8812	-0.2	0.7471	-0.0629	0.8549
	I60-69	-0.0809	0.7576	-0.2	0.7471	-0.1259	0.6967
	I20-25	0.098	0.7982	-0.3	0.6238	0.2238	0.4845
	total mortality	0.2157	0.4057	-0.2	0.7471	0.4056	0.1908
PAL		n 20		n 6		n 14	
	I00-99	0.281	0.2301	0.058	0.9131	0.0838	0.7758
	I60-69	0.2719	0.2461	0.058	0.9131	-0.0419	0.8869
	I20-25	0.3028	0.1944	0.116	0.8268	0.1323	0.6521
	total mortality	0.116	0.6262	0.058	0.9131	-0.2823	0.3283

[a] EEC: Czech Republic, Estonia, Hungary, Latvia, Lithuania Poland, Romania, Slovenia; WEC: Austria, Belgium, Denmark, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Norway, Portugal, Spain, Sweden, The Netherlands, United Kingdom; I00-99: deaths from diseases from the circulatory system (CVD); I60-69: deaths from ischemic heart diseases; I20-25: deaths from cerebrovascular diseases; energy (kcal/day); carbohydrate intake (% of energy); fat intake (% of energy); SFA: saturated fatty acids (% of energy); MUFA: monounsatruated fatty acids (% of energy); PUFA: polyunsaturated fatty acids (% of energy); BMI: body mass index (kg/m^2); smoking (current smokers in % of the population, alcohol consumption in g/day); PAL: physical activity level (% of population which never exercises)

There was no correlation observed for all countries for EEC or WEC. Overall mortality and energy intake showed as well no link for all countries in women and men. Further, in EEC an inverse not significant correlation for men and was calculated.

CHO intake was in men and in women about the same, however, men had a higher mortality rate in all four mortality causes (Figure 3.13). There was no clear correlation, of mortality from diseases of the circulatory system and carbohydrate intake, whereas for EEC (n 7) not significant, inverse correlation coefficient for male and female was found.

Mortality from ischemic heart diseases had no interdependence with the CHO intake in all European Countries, however, for EEC a strong, statistically significant, inverse rank correlation of -0.9268 ($P<0.05$) for male was calculated.

In all countries deaths of all causes had a high significant inverse correlation of -0.9286 ($P<0.05$) for male. However not significant correlations have been estimated for female.

When correlating fat intake with mortality diseases of the circulatory system EEC (n 7) showed a relatively high correlation coefficient with 0.75 ($P>0.05$) for male. WEC (n 15) showed no correlation. Other causes of

Table 3.2: Spearman rank correlation coefficient for all objectives for female[a]

		All Countries		EEC		WEC	
		r	P	r	P	r	P
		n 22		n 7		n 15	
Energy	I00-99	0.1992	0.3743	-0.6071	0.1482	-0.0215	0.9394
	I60-69	0.1149	0.6108	-0.6786	0.0938	-0.222	0.4264
	I20-25	0.34	0.1215	-0.1429	0.7599	0.1629	0.5618
	total mortality	0.3078	0.1635	-0.6786	0.0938	0.188	0.5022
CHO total		n 21		n 7		n 14	
	I00-99	-0.0182	0.9376	-0.9643	0.0005	0.3451	0.2269
	I60-69	-0.0234	0.9199	-0.9286	0.0025	0.3143	0.2738
	I20-25	-0.0734	0.7519	-0.6071	0.1482	0.1648	0.5733
	total mortality	-0.1715	0.4573	-0.9286	0.0025	-0.0637	0.8286
Fat		n 22		n 7		n 15	
	I00-99	0.4167	0.0537	0.75	0.0522	-0.0036	0.9899
	I60-69	0.4676	0.0282	0.7143	0.0713	0.127	0.6519
	I20-25	0.3042	0.1687	0.5	0.2532	-0.3292	0.2309
	total mortality	0.3472	0.1134	0.7143	0.0713	-0.1932	0.4903
SFA		n 18		n 5		n 13	
	I00-99	-0.0072	0.9773	0.5	0.391	-0.0496	0.8722
	I60-69	-0.0021	0.9935	0.1	0.8729	0.0275	0.9288
	I20-25	-0.215	0.3916	0.7	0.1881	-0.4821	0.0952
	total mortality	-0.1034	0.6832	0.1	0.8729	-0.1818	0.5522
MUFA		n 15		n 4		n 11	
	I00-99	0.2288	0.4121	0.4	0.6	-0.1185	0.7278
	I60-69	0.3253	0.2368	0.4	0.6	0.0729	0.8313
	I20-25	0.1001	0.7227	-0.6	0.4	-0.3098	0.3539
	total mortality	0.1573	0.5756	0.4	0.6	-0.2096	0.5363
PUFA		n 15		n 4		n 11	
	I00-99	0.3211	0.2433	0.4	0.6	0.0966	0.7776
	I60-69	0.4646	0.081	0.4	0.6	0.3724	0.2593
	I20-25	-0.0646	0.8192	-0.4	0.6	-0.5702	0.067
	total mortality	0.2493	0.3702	0.5	0.6	-0.0322	0.9252
Cholesterol		n 17		n 6		n 11	
	I00-99	0.451	0.0692	0.0286	0.9572	-0.4909	0.1252
	I60-69	0.4559	0.0659	-0.1429	0.7872	-0.4273	0.1899
	I20-25	0.5245	0.0307	0.2	0.704	-0.2273	0.5015
	total mortality	0.4926	0.0445	-0.1429	0.7872	-0.3455	0.2981
BMI		n 22		n 7		n 15	
	I00-99	0.3265	0.138	-0.7857	0.0362	0.5627	0.029
	I60-69	0.3622	0.0976	-0.75	0.0522	0.6308	0.0117
	I20-25	0.253	0.256	-0.4286	0.3374	0.3369	0.2195
	total mortality	0.1834	0.414	-0.75	0.0522	0.2903	0.2939
Somking		n 16		n 3		n 13	
	I00-99	0.6814	0.0037	-0.5	0.6667	0.5695	0.0422
	I60-69	0.4812	0.0591	-0.5	0.6667	0.2613	0.3884
	I20-25	0.6137	0.0115	-0.5	0.6667	0.4484	0.1243
	total mortality	0.6313	0.0087	-0.5	0.6667	0.4924	0.0873
Alkohol		n 17		n 5		n 12	
	I00-99	-0.0392	0.8812	-0.2	0.7471	-0.0629	0.8549
	I60-69	-0.0809	0.7576	-0.2	0.7471	-0.1259	0.6967
	I20-25	0.098	0.7982	-0.3	0.6238	0.2238	0.4845
	total mortality	0.2157	0.4057	-0.2	0.7471	0.4056	0.1908
PAL		n 20		n 6		n 14	
	I00-99	0.281	0.2301	0.058	0.9131	0.0838	0.7758
	I60-69	0.2719	0.2461	0.058	0.9131	-0.0419	0.8869
	I20-25	0.3028	0.1944	0.116	0.8268	0.1323	0.6521
	total mortality	0.116	0.6262	0.058	0.9131	-0.2823	0.3283

deaths pictured the same scheme. Mortality of men was higher. Although the difference in fat intake was not that much higher in men (Table 3.2).

Further, Figure 3.14 shows the correlation between saturated fatty acids (SFA) and CVD. Spearman's rho for correlation between mortality from diseases of the circulatory system and SFA was for all 18 countries 0.2572 for women. For men no correlation was observed. EEC showed with 0.9 ($P<0.05$) for female a strong link between mortality and SFA intake. Western European Countries seemed to have no association.

No correlation has been observed between MUFA intake and CVD mortality. MUFA intake was higher for Lithuania, Poland Spain and Greece (<14 % of Energy). Mortality in all four division was slightly higher in men, nevertheless with the same correlation pattern (Figure 3.14)

PUFA intake was below the recommended 10 % of energy in all european countries. The difference in mortality rates of Hungary in male and female led to a not significant inverse correlation coefficient in EEC in all four mortality divisions (Table 3.2).

Risk factors for CVD (BMI, smoking behaviour, alcohol consumption, PAL) showed a link with mortality rates for male (Figure 3.15). Interestingly, a higher BMI correlates with higher mortality of diseases of the circulatory system in WEC in men (r= 0.5627, $P<0.05$; n 15). In contrast, in EEC an inverse correlation was estimated in men (r= -0.7857, $P<0.05$; n 7) and no correlation for female population existed.
For correlation of the BMI with mortality from ischemic heart diseases, a Spearman's rho of 0.4969 ($P<0.05$) was estimated. In WEC a stronger correlation for male (r= 0.6308; $P<0.05$) was detected (Table 3.1).

Smoking behaviour of all observed countries was linked to mortality from CVD in male (r= 0.6814, $P<0.05$; n 16). In WEC a correlation coefficient of

0.5696 ($P<0.05$; n13) was estimated for male (Table 3.1.

For alcohol consumption no correlation was observed in male for all countries and WEC.

Correlating all countries PAL, when comparing with CVD mortality, for EEC and WEC no correlation was observed. All correlations are shown graphically in Figure 3.14 and Figure 3.15.

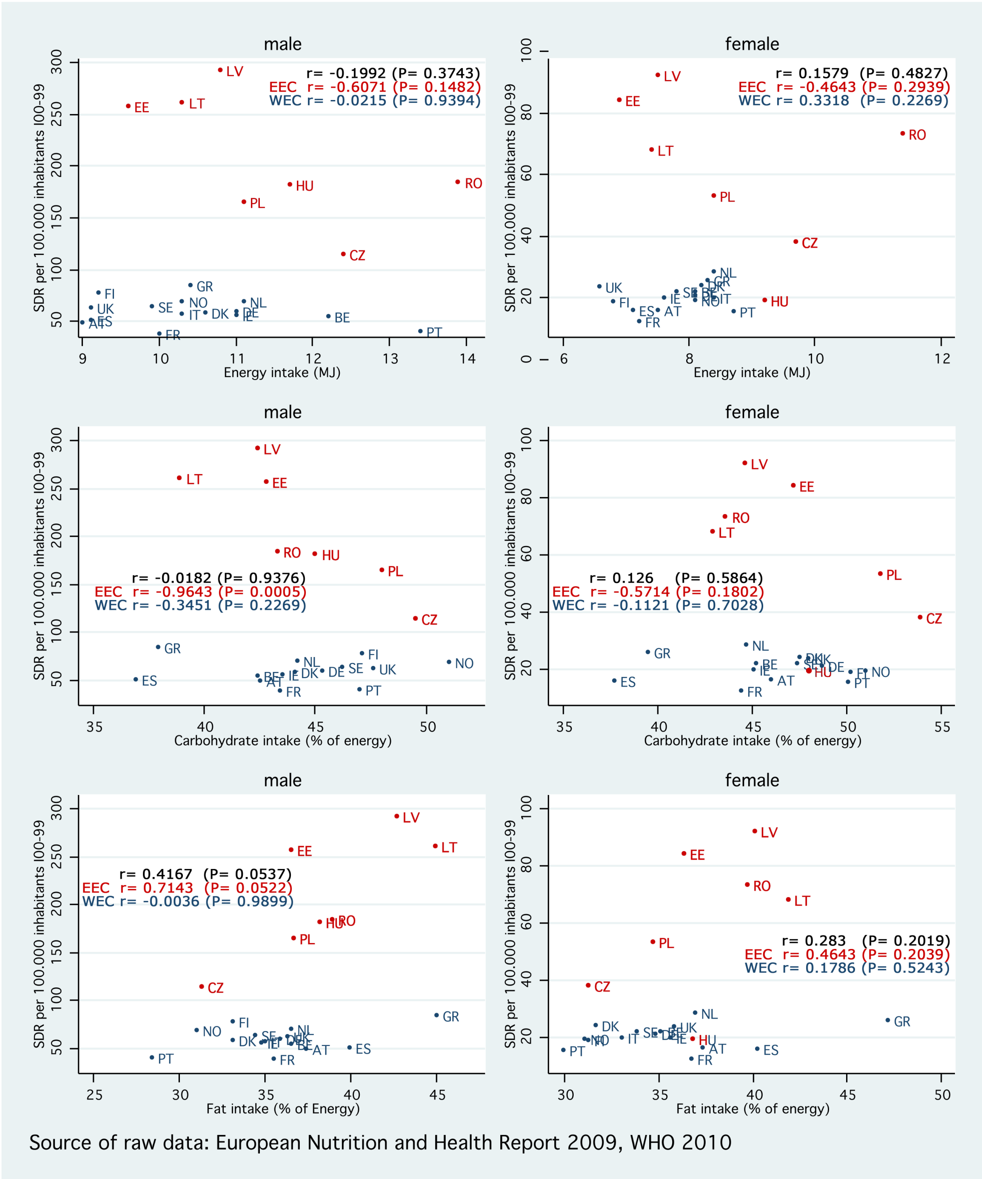

Figure 3.13: Correlation between mortality of diseases of the circulatory system (SDR per 100.000 inhabitants) and Energy intake (MJ) in male and female. EEC: Czech Republic, Estonia, Hungary, Latvia, Lithuania Poland, Romania, Slovenia; WEC: Austria, Belgium, Denmark, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Norway, Portugal, Spain, Sweden, The Netherlands, United Kingdom. Source of raw data: European Health and Nutrition Report 2009; WHO, 2010

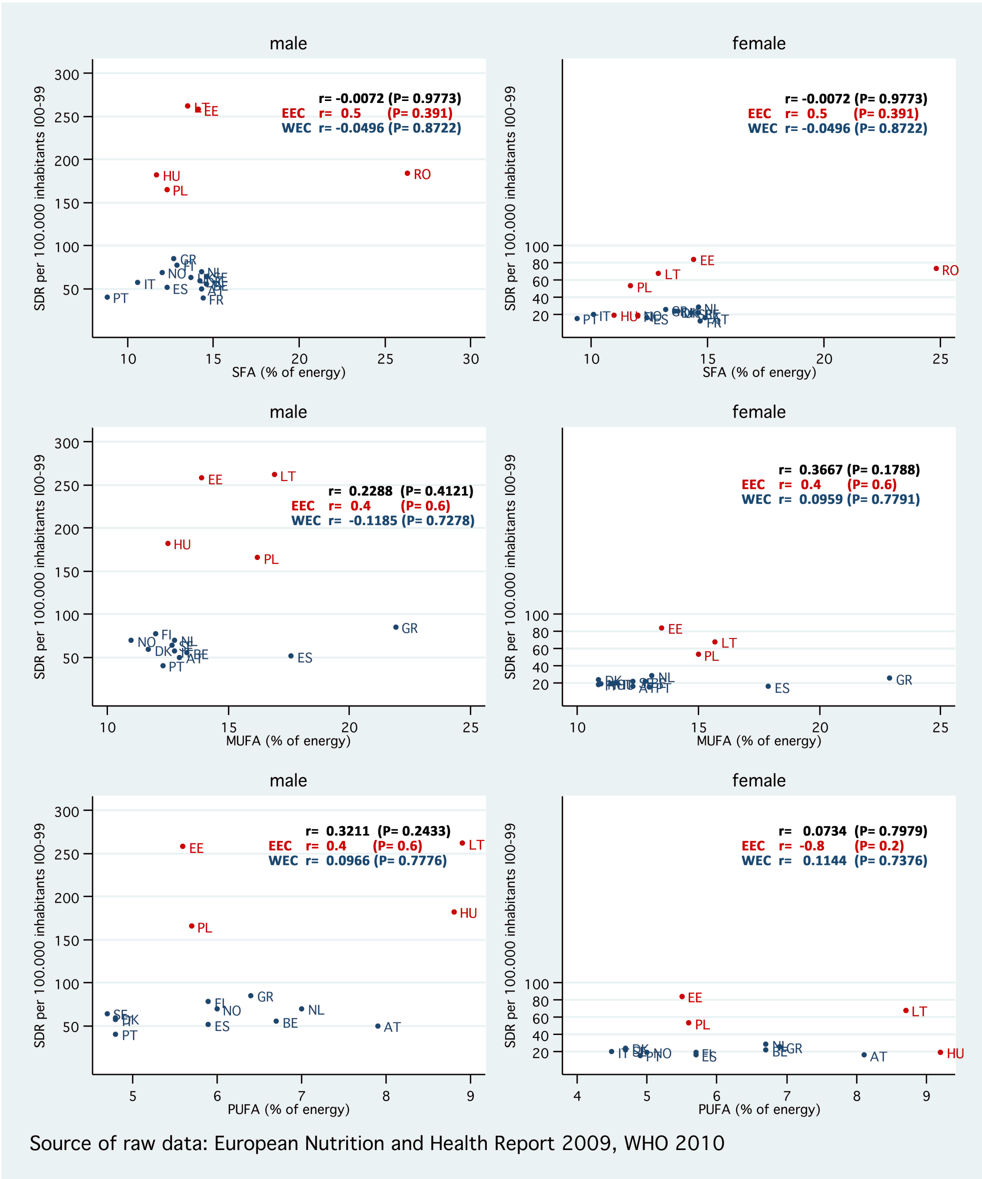

Figure 3.14: Correlation between mortality of diseases of the circulatory system (SDR per 100.000 inhabitants) and SFA intake (% of energy) MUFA (% of energy) and PUFA (% of energy) in male and female. Source of raw data: European Health and Nutrition Report 2009; WHO, 2010

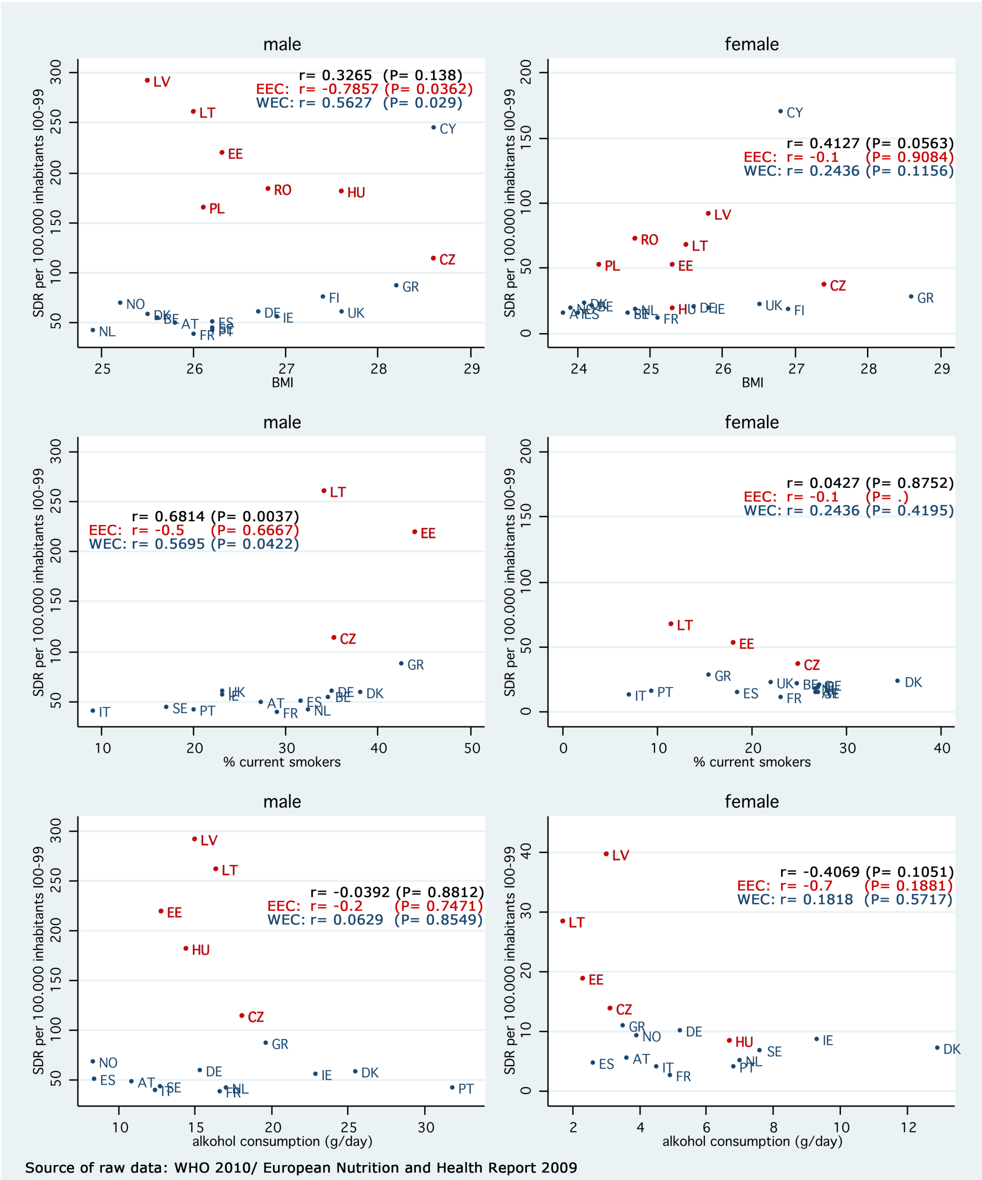

Figure 3.15: Correlation between risk factors and mortality of diseases from the circulatory system. BMI (kg/m^2), smoking behaviour (% of current smokers in a population), alcohol consumption (g/day) in male and female. Source of raw data: European Health and Nutrition Report 2009; WHO, 2010

Chapter 4

Discussion

There has been observed a decreasing trend in mortality from CVD in all four regions. Mortality rates for male declined more than for female. This could have been explained with the gender ratio. Data from WHO was for the whole population of the country, which means that 100,000 inhabitants were male and female counted together. In most industrialised countries this ratio was 65:100 (female:male), due to differences between life expectation [WHO, 1995]. However, this did not implicate that CVD was no problem in the worlds population. Better medical health systems and interventions could have prevented CVD deaths. Nevertheless, with respect to the death causes, the CVD trend was rising [WHO, 2011a].

Men showed a higher consumption of alcohol. Furthermore, more men tended to be smokers. These risk factors could have lead to a higher mortality of CVD in male than in female. In this examination, no link between mortality from CVD and alcohol consumption was found for men. For women, a non significant inverse correlation between mortality from diseases of the circulatory system and alcohol consumption was estimated. Correlation coefficient was significant for mortality from ischemic diseases and alcohol consumption ($r = -0.4877$, $P < 0.05$; n17). In EEC, correlation coefficients were even higher, although the number of objectives was low and the estimates were not significant. Alcohol intake could have had protective effects in lower dose, otherwise excessive alcohol consumption had unfavourable effects on cardiovascular health [Baua *et al.*, 2007]. Intake of female, with exception of danish women, was below the recommendations. Consequently the estimates were conform to the literature. Alcohol consumption of male was slightly over the recommended amount, however, there was no correlation in respect to cardiovascular mortality in Western European Countries for men and women.

Examining the development of the gaps between the mean, the minimum, and the maximum of the North and the Central East, it has been obvious that there was a difference in mortality from CVD in some countries. When the gaps of the South and West heading towards each other, gaps of the North and Central East drifted apart. Due to the fact that the division of these regions were made in respect of the common dietary habits, there had to be further reasons for mortality from CVD. Taking a closer look at the countries, it appeared that EEC had a significantly higher mortality from diseases of the circulatory system (Figure 3.12).

This was a result of differences in medical treatments and individually targeted interventions. Furthermore, disparity between social and economic risk factors might have been a reason for higher mortality from CVD in eastern european countries [Chow *et al.*, 2009]. For instance, the price of cigarettes rose in most developed countries, while in contrast they were affordable easier in developing countries [Guindon *et al.*, 2002]. Examining the smoking habits of eastern european countries, the proportion of current smokers as percentage of the population was not higher than WEC, even lower. Estimated Spearman's rho for mortality from diseases of the circulatory system and smoking of men for 16 countries over europe was significant with 0.6814 ($P<0.05$). For cerebrovascular diseases, calculated spearmans rho of 0.6137 ($P<0.05$; $n= 16$) for men was similar. The not significant correlation for mortality of ischemic diseases was slightly lower. In EEC, no correlation was found. Smoking habits of men in WEC correlated with the CVD mortality ($r= 0.5695$, $P<0.05$; $n= 15$). These were quite strong correlations and consistent with the literature that smoking is an independent risk factor for CVD [Bazzano *et al.*, 2003].

Another independent risk factor was the BMI. Mean was beyond the recommendations for a population by every country with exception of females of Austria and Poland [Elmadfa, 2009]. Non significant correlations between mortality from diseases of the circulatory system and BMI for men and for women were estimated. BMI of men from EEC correlated inversely with the mortality from CVD ($r= -0.7857$, $P<0.05$; $n= 7$). Social and economic reasons might have been responsible for this [Chow *et al.*, 2009]. It is likely that people which had a purchasing power in EEC had a higher BMI [Ulijanszek and Koziel, 2007], hence, could have had access to better medical treatment in EEC.

The trend of energy supply in Europe showed a clear rising tendency, with exception of the North (dietary devision), which only had a slightly rising trend. It needs to be asked what the nutrient is that was consumed by the population. When the nutrition habits would have been equal to those 47 years ago, the trend in all commodities would have risen equal the increase of energy.

In the South, energy supply had the strongest increase in the past decades. While mean total fat supply rose, mean carbohydrate supply decreased slightly in the South. In the Northern region, a rising trend in energy supply was observed as well as in the western region. Although the increase was stronger in the North than in the West. However, both had a constant mean carbohydrate supply and a rising tendency in mean total fat supply. The North showed the slightest rising mean energy supply. While the mean fat supply remained constant, mean carbohydrate supply rose slightly in the North since the nineties. Further, the North had the strongest decline in animal fat, however the south had a lower animal fat supply in 2007. Furthermore, the North had the slightest increase in vegetable oil.
Finland, for instance, had the highest international decline in coronary heart

diseases since 1960. After 1977 national preventive activities were conducted [Puska *et al.*, 1983]. Due to Vartiainen et al. the 80 % decline in coronary mortality in Finland was achieved through a great reduction of the metabolic risk factor levels [Vartiainen *et al.*, 2010]. The observed trends of nutrient intake of the North pointed out the findings of Vartiainen et al.

Further, calculations in this study estimated a link of mortality from CVD and energy intake. For women of the EEC, a not significant inverse correlation between energy intake and mortality from ischemic diseases was found. This could have also been a result of socioeconomical differences. In most of the EEC, economic inequality rose after the collapse of communism [Rosser *et al.*, 2000]. Therefore, people with a high purchasing power could afford a high energy intake, and further a better medical treatment.
However, a positive caloric balance leads to obesity [Bays, 2011] which is an independent risk factor for CVD [Poirier *et al.*, 2006]. Further, a BMI over 30 kg/m^2 is a risk factor for Type II Diabetes Mellitus, high blood pressure and dyslipidemia which indirectly increases CVD risks well [Bays, 2009].
Considering the trend of energy intake which was rising especially in the South and the West, it was obvious that BMI was increasing as well.

An important finding were the correlation coefficients between carbohydrate intake and CVD mortality, and between fat intake and CVD mortality. In EEC, an inverse correlation was observed for male between carbohydrate intake and total CVD mortality ($r= -0.9643$, $P<0.05$; $n= 7$), and mortality from ischemic diseases ($r= -0.9286$, $P<0.05$). Further, in women of the EEC, correlation was inverse, however, no significance was given. This could have been due to the differences of independent risk factors such as smoking or alcohol. In this study these risk factors were not implemented. More men tended to be smokers or drinking more alcohol which lead to a higher mortality from CVD [Baua *et al.*, 2007]. Therefore, CVD mortality of men was higher than of women.
In men, a not significant correlation between fat intake and CVD mortality was observed within all countries. In EEC, the correlation was even stronger, nevertheless not significant. However, in WEC no correlation was

found, which could have been due to the differences in the medical treatment [Chow *et al.*, 2009].
The study of Berglund et al. estimated changes in metabolic risk factors. Exchanging fat of the diet with carbohydrates lowers LDL and HDL but rises TG levels [Berglund *et al.*, 2007].
The amount of kcal per gram carbohydrate was about the half of the kcal per gram fat. Hence, caloric balance might have been more likely to be achieved in order to avoid obesity. The strong, not significant correlation between total fat intake and mortality from CVD for male and female was supporting this thought, on the basis of the fact that obesity an independent risk factor for CVD mortality [Poirier *et al.*, 2006].

However, the importance of the quality of fat was beyond controversy, when thinking about the functions such as being part of membranes. Saturated fatty acids were not linked with cardiovascular mortality in male (all countries). In EEC, a strong significant correlation was found in women ($r = 0.9$, $P<0.05$). In the meta-analysis of Siri-Tariano et al., who evaluated epidemiological studies, saturated fatty acids were not significantly linked to a higher CVD risk [Siri-Tarino *et al.*, 2010]. However in metabolic studies saturated fatty acids had a cholesterol rising effect [Kris-Etherton and Yu, 1997] which increased the CVD risk [NCEP, 2002]. Most of the estimated correlations were not significant regarding SFA, however there was a correlation between CVD mortality and SFA intake in EEC.

No correlation between MUFA and CVD mortality was observed. Authors of prior ecological studies emphasised a rather high correlation between MUFA and SFA intakes and specified SFA as a confounding variable compromising conclusions, when linking MUFA with risk of CHD [Gillingham *et al.*, 2011]. Further studies regarding an exchange of SFA with MUFA advocated a higher MUFA intake, however, these studies observed risk factors such as plasma cholesterol to estimate the CHD risk [Cao *et al.*, 2009].

In all European countries a not significant correlation between PUFA intake and mortality from ischemic diseases was observed for male. Contrarily, in WEC an inverse not significant correlation was observed between mortality from cerebrovascular diseases and PUFA intake. All countries stayed below the reference value (6-11% of energy), some even lower [Elmadfa, 2009]. However, estimates of EEC resulted in no significance, due to a small number of observations. Countries of EEC could have confounded the estimates for all european countries. Nevertheless the inverse relationship of the PUFA intake and mortality from cerebrovascular diseases agreed with the findings of Mozzafarin et al., that a higher PUFA intake could have decreased the risk of mortality from CVD [Mozaffarian *et al.*, 2010].

In EEC, it seemed nutrient uptake was correlating higher with mortality than in WEC. On the contrary, in WEC independent risk factors gave the impression to be rather linked to CVD mortality. CVD is a multifactorial disease. The various risk factors (dietary and individual) changed the metabolic risk factors, such as plasma cholesterol and hypertension. Medical treatment would have been able to change these risk factors by medical interventions, which differed between WEC and EEC. The objective of this study, though, was mortality from CVD, not the incidents. Furthermore, it is suspected that the inverse relationship, between CVD mortality and the BMI in EEC indicates the possibility of a link between purchasing power for food and further, for medical treatment in EEC. Further energy intake of EEC correlated, although not significant with the CVD mortality in EEC.

Nevertheless, independent risk factors correlated, with exception of alcohol consumption and physical activity level, with CVD mortality in male within all countries. Therefore, these risk factors should be taken into account in further ecological studies to estimate the impact of total fat uptake and the quality of fat on mortality from CVD.

A caveat of this study was the small number of countries. Only some correlations had a P value lower than 0.05. Further, the questionable reliance on the accuracy of the dietary assessments of the nutrient assessment needed to be kept in mind. Underreporting of food intake was likely. Regarding the high prevalence of overweight and obesity in european countries, most adults did not meet the recommended balance of energy intake and energy requirement, especially when considering the low physical activity in all european countries [Elmadfa, 2009]. Nevertheless, the collected data are a useful source to compare the countries and to estimate correlations to indicate the risk of CVD.

However, this study showed that excessive energy intake, hence obesity, had unfavourable influences on CVD mortality. Reducing energy intake with replacement of fat with carbohydrates had a positive impact on obesity prevalence, thus, a cardiovascular protective effect. Regarding the fat quality there was no clear indication, that it would have been favourable to replace SFA with PUFA, when energy intake was not over requirement.

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Abstract

The science of nutrition is discussing whether it is more beneficial for the cardiovascular health to replace fat with carbohydrates, or replace saturated fatty acids with fats of higher quality. Metabolic studies showed that CVD risk factors such as hypertension and blood lipids can be influenced positively when exchanging saturated fatty acids with monounsaturated fatty acids, polyunsaturated fatty acids or carbohydrates. However, excessive energy intake is a further risk factor for CVD. The trend of the nutrient intake was observed in four regions of Europe, which were classified according to dietary habits of each country.
The main part of this study estimated the extent to which dietary nutrient intake, such as fat and carbohydrates, and the different fatty acids correlate with CVD mortality in 25 european countries. Further, correlations of independent risk factors, such as smoking, alcohol consumption, and BMI were estimated.

After comparing the mortality from CVD with fat and carbohydrate intake there is a coherence between fat intake and CVD mortality. Further, the carbohydrate intake correlated inversely with the CVD mortality. Nevertheless, no clear indication for the mortality risk from CVD and the fat quality could be made. However, the increasing trend in energy intake is leading to the conclusion of this study, that exchanging fat with carbohydrates could decrease the energy intake and therefore the CVD risk factor obesity.

Zusammenfassung

In der Ernährungswissenschaft wird diskutiert, ob es für die kardiovaskuläre Gesundheit vorteilhafter ist, diätetisches Fett durch Kohlehydrate, oder gesättigte Fettsäuren durch qualitativ hochwertigere Fettsäuren zu ersetzen.

Aus metabolischen Studien geht hervor, dass Risikofaktoren für kardiovaskuläre Erkrankungen, wie Hypertonie und schlechte Blutfettwerte, positiv beeinflusst werden können, indem gesättigte Fettsäuren durch einfach ungesättigte, mehrfach ungesättigten Fettsäuren, oder aber auch mit Sacchariden in der Diät ersetzt werden. Eine übermä§ige Energieaufnahme zeigte ebenso eine Beeinflussung der Risikofaktoren. Der Trend für die Nährstoffaufnahme und die Mortalität wurde für vier länderübergreifenden Regionen in Europa ermittelt. Die Länder, die den Regionen zugeteilt wurden, glichen sich in ihren Ernährungsgewohnheiten. Es wurde der Zusammenhang zwischen der Aufnahme von Fett und Kohlehydraten, mit der Mortalität, die durch Herz-Kreislauf-Erkrankungen hervorgerufen wurde, in 25 europäischen Ländern ermittelt. Des weiteren wurden Korrelationen zwischen unabhängigen Risikofaktoren, wie Rauchen, Alkoholkonsum und BMI und der Mortalität von Herz-Kreislauf-Erkrankungen erhalten.

Es stellte sich eine positive Korrelation zwischen der Fettaufnahme, und eine inverse Korrelation zwischen der Kohlehydrataufnahme und der Mortalität heraus. Jedoch konnte keine eindeutige Erkenntnis über das Mortalitätsrisiko in Bezug auf die Fettqualität gewonnen werden. Nichts desto trotz könnte eine Senkung der Energieaufnahme durch den Austausch von

Fetten durch Kohlehydrate das Übergewicht und in weiterer Folge das Herzkreislauferkrankungsrisiko senken.

Curriculum Vitae

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Ausbildung:

Seit 2003	Universität Wien, Österreich Studiengang: Diplomstudium der Ernährungswissenschaften Spezialisierung: Ernährung und Ökonomie
1998-2003	Sacré Coeur Riedenburg, Bregenz, Österreich Höhere Lehranstalt für wirtschaftliche Berufe

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WS 2011/2012	Tutorium der chemischen Übungen für Ernährungs- wissenschafter und Biologen an der Universität Wien
08/2011	Volontariat im Bundesministerium für Gesundheit Abteilung für Ernährung, besondere Waren und internationale Lebensmittelangelegenheiten
07-08/2007 07-08/2008 07-09/2009	Hilcona/Schaan, Fürstentum Lichtenstein Qualitätssicherung und Backofficemanagement im Bereich des Rohwahreneinganes
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Sprachkenntnisse

Englisch	fließend in Wort und Schrift
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Zusätzliche Qualifikationen

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Fotografie:	fotoschule.at Einfühung in die digitale Fotografie Universität für angewandte Kunst Licht und Belichtung

Interessen

Sport (Wasserball, Schi fahren, Yoga)
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Fotografie
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Wien, 10 April 2012