

Finnish Dietary Selenium and CHD Mortality of Middle-Aged Females and Urban Males in the 1950's – Associations by Product Moment Correlation (Pearson) and by “Proportional Deviation from Exponential Trendline”

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ABSTRACT

Selenium can have a role in a short and a long-term heart protection via managing oxidation and inflammation, cell injury and atherosclerosis. This is supported by some statistical studies. In the mid-1970's the estimates of Finnish daily intake of selenium [Se] (20-30 µg/d) were about only a half of the minimum recommended amounts. Later was estimated that the range of Finnish intake of Se in the 1950's had been 30-55 µg/d caused by the imported grain. Concurrently with this increase in Se intake occurred a decrease in age-adjusted CHD mortality amongst middle-aged females (F) and amongst urban males (M.CHD.urb), less by rural (rur) males. Values are given by 3-year means (3ym) from 1951-61, in calculations (1952-60). This range is based on that in 1961 F. (CHD/Total) mortality, “F. (CHD/Tot)” increased above its respective value of 1952. This range selection is explained in Introduction. Results are given by Pearson correlations “Pearson (a; b)” and by regressions of their proportional deviations from their own exponential trendlines, between start (α) and end (ω) point, [Dev.%].

Results: Pearson. [Se; F.CHD] = +0.40 (p = 0.280). Pearson. [Se; M.CHD] = 0.00 (ns). [Se.Dev.%] explained [F.CHD. (Dev.%)] by 47.6 % (p = 0.040) and [M.CHD.urb] by 36.6 % (p = 0.084).

Conclusion: Proportional deviations from exponential trendline show inverse significant or trend-like association between CHD mortality and Se intake by females and urban males, but Pearson associations were insignificant (even positive).

Keywords: CHD; Selenium; Grain; Import; (CHD/Tot)

Abbreviations: **3ym:** 3-year mean; CHD: coronary heart disease; CHD.i: age standardized CHD mortality (1/100,000) amongst (selected) population group aged 35-64 years in year i; (i/52): post-suffix for a parameter value from year “i”, when divided by its value of 1952; F: female; M: male; rur: rural, from countryside; Se: selenium; Si: silicon; Tot: total; urb: urban.

Introduction

Selenium can have a role in CHD (coronary heart disease) protection via managing reactive oxygen species (ROS) and their products (e.g. lipid hydroperoxide. LOOH), so protecting against inflammation, cell injury and atherosclerosis [1,2]. This is supported with some statistical studies [3,4]. In the mid-1970's the estimates of Finnish daily intake of selenium (Se) (20-30 µg/d) [4] were about only a half of the minimum recommended amounts [4]. In 1983 it was discovered

that the imported grain increased Se intake (up to 30-55 µg/d) in the 1950's [4]. About the same time was reported that CHD mortality (age-adjusted, per 100,000 amongst females and males aged 35-64 years) decreased in the 1950's, especially by females and urban males [5]. The aim of this study is to describe and evaluate the mutual changes in CHD mortality and selenium intake by Pearson correlations and proportional deviations from exponential trendlines [6] and discuss about possible Finnish Se sources in the 1950's.

Table 1: Finnish selenium intake and age-adjusted CHD mortality of Finnish females and urban males, aged 35-64 years, by 3-year means from 1951-61. Separately are given means of the above Se intake values from 1951-57 and 1958-61.

	1952	1953	1954	1955	1956	1957	1958	1959	1960
	3ym								
Se. intake (µg/d)	44,1	43,2	42,1	46,4	45,0	45,2	36,8	36,3	33,6
F.CHD	113	104	101	97	94	89	88	90	93
M.CHD.urb	489	455	455	450	455	433	446	446	465
Mean.Se. intake	44.3							35.6	

Table 2: Finnish selenium intake and age-adjusted CHD mortality of Finnish females and urban males, aged 35-64 years, by 3-year means from 1951-61. Numbers are relative to their values in 1957, labeled by "(i/57)".

	1952	1953	1954	1955	1956	1957	1958	1959	1960
Se.(µg/d). (i/57)	0,98	0,96	0,93	1,03	1,00	1,00	0,81	0,80	0,74
F.CHD. (i/57)	1,27	1,17	1,14	1,09	1,06	1,00	0,99	1,01	1,05
M.CHD.urb. (i/57)	1,13	1,05	1,05	1,04	1,05	1,00	1,03	1,03	1,07

Table 3: Pearson correlations between F.CHD, M.CHD.urb and Se intake.

	Se.µg/d	F.CHD	M.CHD
Se.µg/d	1,00	0,40	0,00
F.CHD	+0,40	1,00	0,82
M.CHD.urb	-0,00	0,82	1,00

Table 4: Includes Se intake, F.CHD and M.CHD.urb, exponential trendlines between their start (α) and end (ω) points and deviations of original (3ym) values from the trendline in percents.

(Dev.%)									
Trend (etl) equation = F (x.i)									
k = log10(x.ω)- log10(Y.α)									
F (x.i) =POWER (10, k*(i-α)) *Y.α									
		yrs	log10		yrs	log10		yrs	log10
Y.α	44,1	1952	1,64	112,99	1952	2,05	489,32	1952	2,69
Y.ω	33,6	1960	1,53	93,09	1960	1,97	465,29	1960	2,67
Δ. (ω; α)	-10,5	8	-0,118	-20	8	-0,084	-24	8	-0,022
Δ per yr		-0,0147	-6,30		-0,0105	-6,30			-0,0027
	Se.(µg/d)	Se.(µg/d). etl	Se.(µg/d). (Dev.%)	F.CHD	F.CHD.etl	F.CHD. (Dev.%)	M.CHD.urb	M.CHD.urb.etl	M.CHD.urb. (Dev.%)
1952	44,1	44,1	0,0	113,0	113,0	0,0	489	489	0,0
1953	43,2	42,6	1,5	104,0	110,3	-5,7	455	486	-6,4
1954	42,1	41,2	2,2	100,8	107,6	-6,4	455	483	-5,8
1955	46,4	39,8	16,6	96,9	105,1	-7,8	450	480	-6,2
1956	45,0	38,5	16,9	93,8	102,6	-8,6	455	477	-4,6
1957	45,2	37,2	21,5	88,7	100,1	-11,4	433	474	-8,7
1958	36,8	35,9	2,3	88,0	97,7	-9,9	446	471	-5,4
1959	36,3	34,7	4,6	89,8	95,4	-5,9	446	468	-4,8
1960	33,6	33,6	0,0	93,1	93,1	0,0	465	465	0,0

Table 5: Shows relative numbers of factors to 1952. Se µg/d is represented by [Se].

	[Se]. (i/52)	[Se]. (i/52). etl	[Se]. (i/52). (Dev.%)	F.CHD. (i/52)	F.CHD. (i/52). etl	F.CHD. (i/52). DEV.%)	M.CHD.urb. (i/52)	M.CHD.urb. (i/52). etl	M.CHD. (i/52). (DEV.%)
1952	1,00	1,00	0,0	1,00	1,00	0,0	1,00	1,00	0,0
1953	0,98	0,97	1,5	0,92	0,98	-5,7	0,93	0,99	-6,4
1954	0,95	0,93	2,2	0,89	0,95	-6,4	0,93	0,99	-5,8
1955	1,05	0,90	16,6	0,86	0,93	-7,8	0,92	0,98	-6,2
1956	1,02	0,87	16,9	0,83	0,91	-8,6	0,93	0,98	-4,6
1957	1,03	0,84	21,5	0,78	0,89	-11,4	0,88	0,97	-8,7
1958	0,83	0,82	2,3	0,78	0,86	-9,9	0,91	0,96	-5,4
1959	0,82	0,79	4,6	0,79	0,84	-5,9	0,91	0,96	-4,8
1960	0,76	0,76	0,0	0,82	0,82	0,0	0,95	0,95	0,0

Table 6: Pearson correlations between proportional deviations of [Se], F.CHD and M.CHD.urb (from their exponential trendlines).

		[Se]. (DEV.%)	F.CHD. (DEV.%)	M.CHD.urb. (DEV.%)
[Se]. (Dev.%)	1,00	-0,69	-0,60	[Se]. (Dev.%)
F.CHD. (Dev.%)	-0,69	1,00	0,90	F.CHD. (Dev.%)
M.CHD.urb. (Dev.%)	-0,60	0,90	1,00	M.CHD.urb. (Dev.%)

Table 7: Imported oil seed remnants, "oil cakes" (after extraction of oil) for dairy cows as protein feed.

	1951	1952	1953	1954	1955	1956	1957	1958	1959
	(1,000 tn)								
Peanut	7,0	24,7	2,2	12,2	18,2	32,3	15,5	2,5	0
Coconut	0	0	0	0	0	0	0	0,0	0
Palm	0	0	0	0	0	0	0	0	0
Soya	26,1	17,9	7,4	5,0	33,8	42,1	15,3	5,9	14,7
Linseed	26,4	0,1	4,2	14,2	13,4	22,9	14,0	11,7	5,4
Cottonseed	13,4	30,7	25,4	12,5	17,5	22,7	16,8	26,7	14,6
Turnip, rape	0,1	3,4	0,0	0	0	0	0	0	0
Sunflower	2,4	2,1	6,6	8,1	7,0	14,3	10,9	10,7	15,4
Oil_cakes.tot	75	79	46	52	90	134	73	57	50
Oil cakes g/capita/d	52	54	31	36	62	92	50	39	34
Means. (1951-57,58-61)	53,7					37,3			

Material and Methods

General principles: National and regional mortality data are attained from the figures in [5] by measuring with ruler and calculated, because the data given were on logarithmic scale. The F.CHD (measured and calculated data from 1951-87) [5] were harmonized by the official data of Statistics Finland [7] for period 1969-2005 by the bias in their overlapping period. After harmonizing it was formed 3-year means (3ym) of F.CHD. It decreased in 1952-58 from 113 to 88 and reached its maximum (integer) value 103 in 1962 and decreased after 1963. M.CHD 3ym decreased in 1952-57 from 387 to 362 and exceeded its start value in 1960. [Annual ratio of F.CHD to total mortality [F.(CHD/Tot)] was in 1961 about the same as in 1951 (16.8 and 17.0 %, respectively), but its 3-year mean (17.0 %) was higher in 1961 to

its value in 1952 (16.2 %). These calculations are not shown here, but that's why period 1951-61 was selected for study object, in 3ym data 1952-60.] With some exceptions in the text seemingly annual values of year "i" means 3-year mean of years: i-1, i and i+1.

Estimates for human Se intake (µg/d) are attained by measuring by ruler from Figure 1 in [5]. F.CHD in 1951-87 is attained from Figure 2 ("Kuvio 1") in [5], where F.CHD (1/100,000) is represented on logarithmic scale with range from 50 to 150. First was measured value for 150 (1/100,000), "Y.150", as centimeters. Then were measured annual values "Y.i" (cm). $F.CHD.i (1/100,000) = POWER(10; LOG10(50) + Y.i/Y.150 * (LOG10(150) - LOG10(50))) = Power(10; 1.7 + Y/Y.150 * (2.2 - 1.7))$. Additionally so achieved F.CHD values were reduced by 1.5 in order to balance the measured values with val-

ues [5] from those of Statistics Finland in 1969-75 [7] (difference 1.5, SD 0.4). F.CHD values are readily represented in [8]. Data on M.CHD.urb is attained from Figure 3 (“Kuvio 5”) in [5], where the data are on logarithmic scale between 300 and 600. M.CHD.urb is attained re-

spectively as F.CHD by changing Y.150 to Y.600 and Y.50 to Y.300. This data is not “harmonized”, because of missing respective numerical data from Statistics Finland. After forming 3ym data values are added to Table 1.

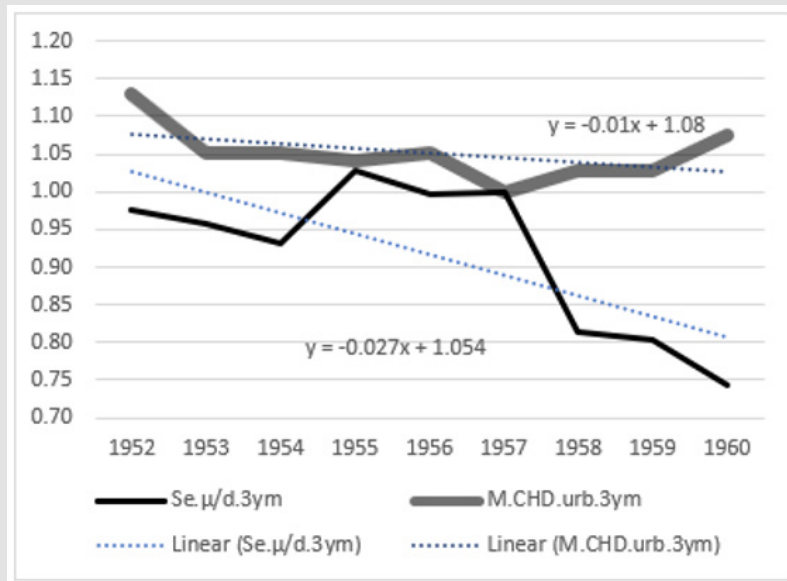


Figure 1: M.CHD.urban and Se intake, values relative to respective values in 1957.

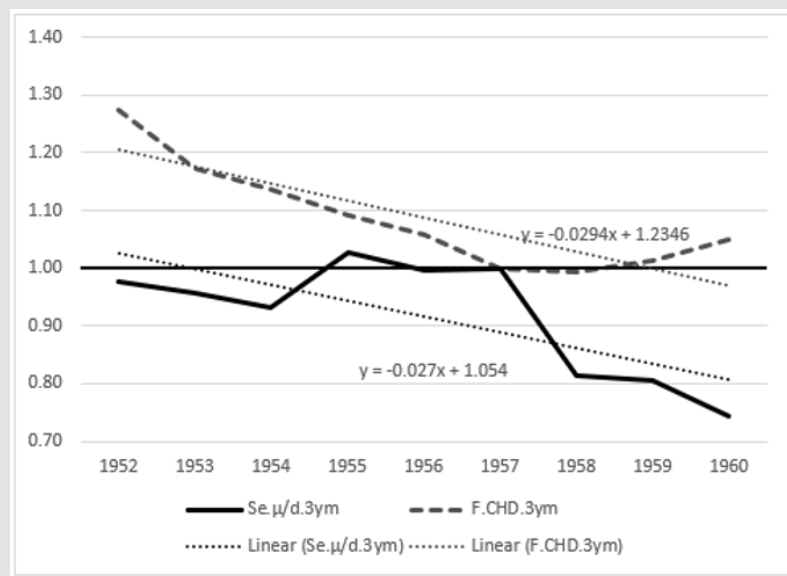


Figure 2: F.CHD and Se intake, values relative to respective values in 1957.

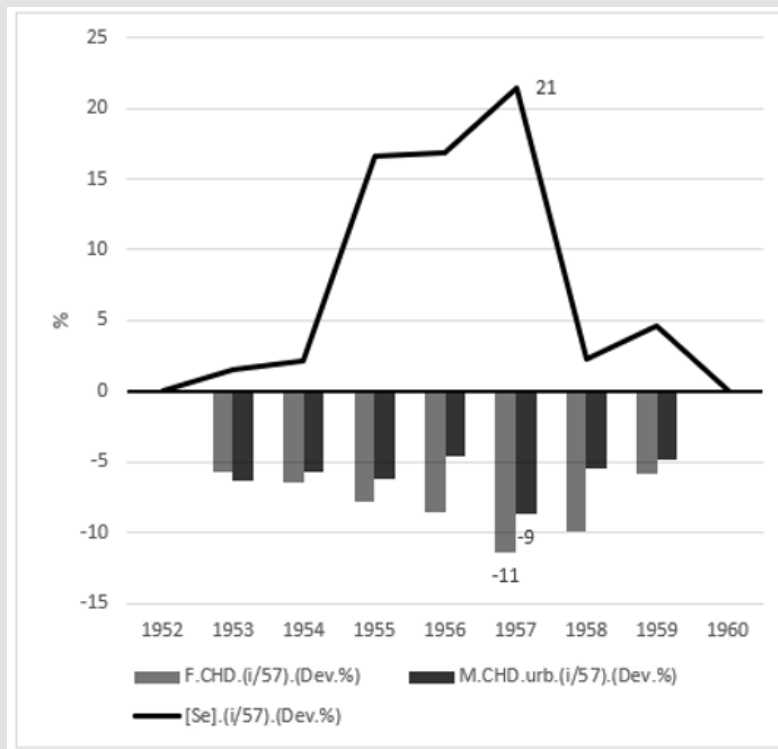


Figure 3: F.CHD and M.CHD.urb deviations from the trendline.

Calculations are made by Microsoft Office Home and Student 2019 and IBM SPSS 29.0.

Table 1 shows that Se intake had been at its highest in 1955-57 [mean 45.5, highest in 1955 (46.4)] during the period 1952-57 with mean 44.3 ($\mu\text{g}/\text{d}$). In period 1958-60 Se intake was 35.5 ($\mu\text{g}/\text{d}$). The lowest CHD values are seen with delay: F.CHD in 1958 (88) and M.CHD.urb in 1957 (433, 1/100,000).

Table 2, Figures (1-3). show the same with relative numbers (annual values divided by their respective values in 1957).

Figure 1 shows development of F.CHD and Se intake. CHD decrease associated with higher Se intake and increase with lower Se intake. Figure 2 shows development of M. CHD. urb and Se intake between 1951-61. CHD decrease associated with higher Se intake and increase with lower Se intake.

Table 3 shows Pearson correlations between Se intake and CHD mortality. Se. intake explained F.CHD by 16 % ($p = 0.280$, i.e. non-significantly). Significance calculated by SPSS. R. (Pearson) was +0.40. R square, ("R²") 0.16, i.e. Se intake associated (insignificantly, "not to mention") positively with F.CHD (Fig.1). Se.intake explained 0.0 % of

M.CHD.urb (Sig.: ns) (Fig.2). Fig. 3 shows development of Se intake, F.CHD and M.CHD.urb, with polynomic trendlines and equations. Values relative to 1957, calculated by 3-year means. Fig.3 shows that Se intake and CHD's associate inversely with the time. Fig.3 shows "the turning point", which is near 1958.

Analysis By Proportional Deviation from Exponential Trendline (etl)

In order to evaluate the inverse behavior of Se intake and CHD was used "proportional deviation from exponential trend-line" method (as a surrogate to obviously better polynomic methods). Exponential trendline is formed as follows: Factor (F) Y (Se; F.CHD or M.CHD.urb). $Y.\alpha$ is the factor value in 1952. $Y.\omega$ is the factor value in 1960. Next are calculated Log₁₀ value for $Y.\alpha$ and $Y.\omega$. These values are divided by 8 (number of years). $k = \log_{10}(Y.\omega) - \log_{10}(Y.\alpha)$. Value of etl in year "i" = F. (Y.i) = POWER (10, $k*(i - \alpha)$) * $Y.\alpha$. F.Dev.% = $(F_i - F_{etl})/F_{etl} * 100$. Figure 4 shows exponential trendlines of Factors ([Se], F.CHD and M.CHD.urb) between start (α , 1952) and end (ω , 1960) points of parameters. Selecting start point to 1952 helps to determine tangent a.

Figure 5 shows that [Se] deviated positively up to 21 %, F.CHD negatively and M.CHD.urb negatively (ad -11 and -9 %, respectively).

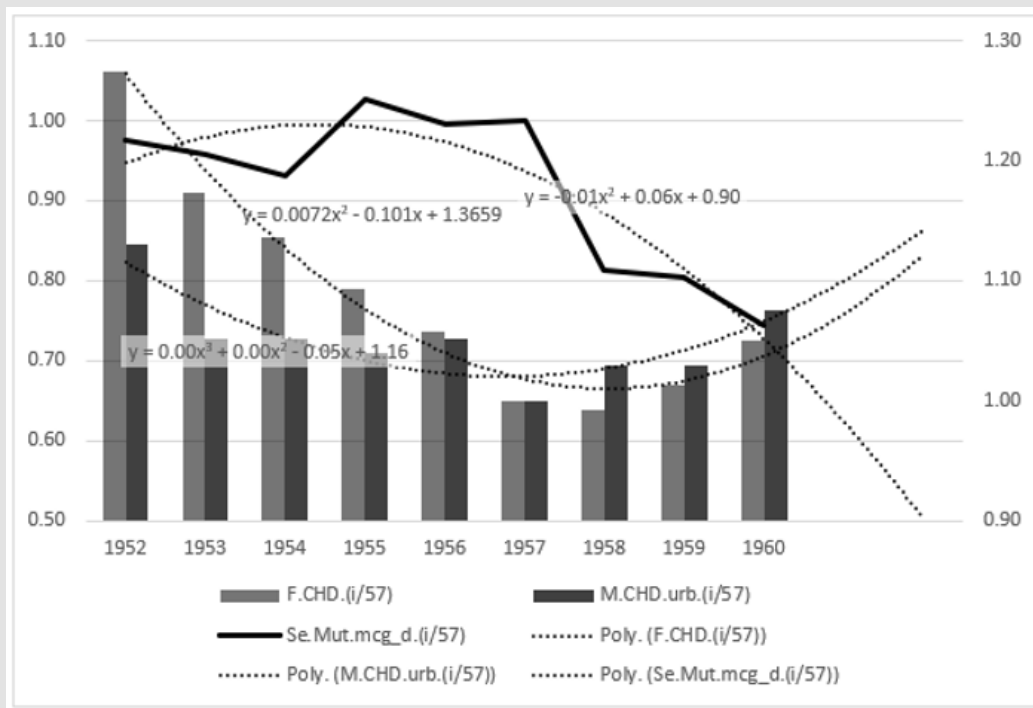


Figure 4: Development of Se intake, F.CHD and M.CHD.urb in 1951-61, with polynomic trendlines and equations. Values relative to 1957, calculated by 3-year means.

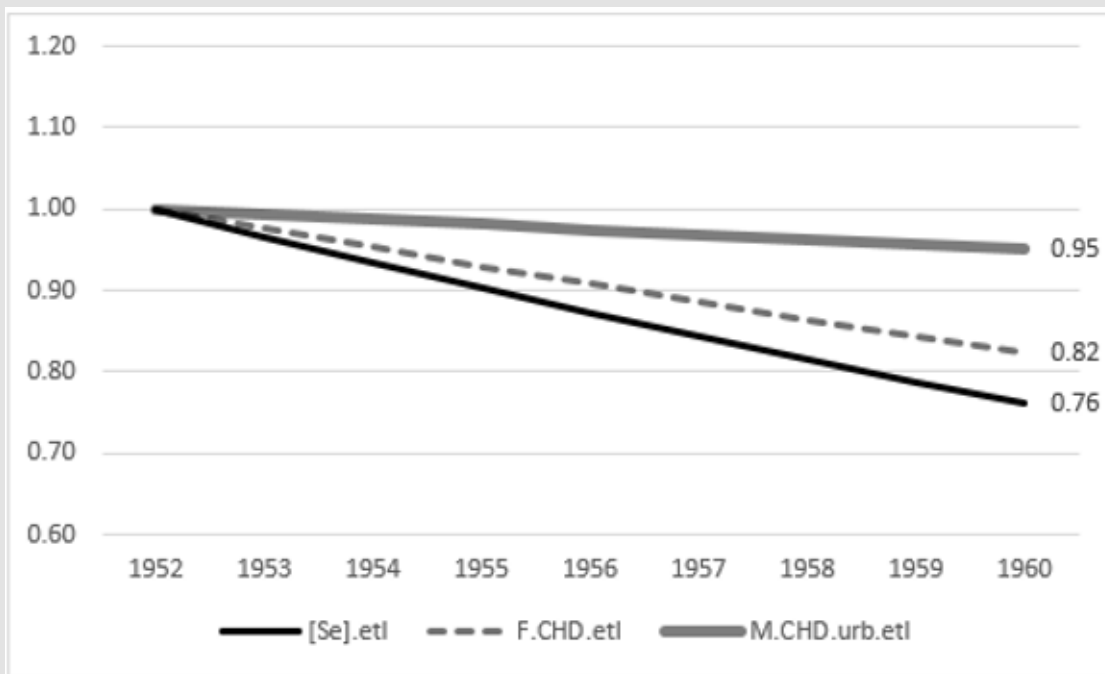
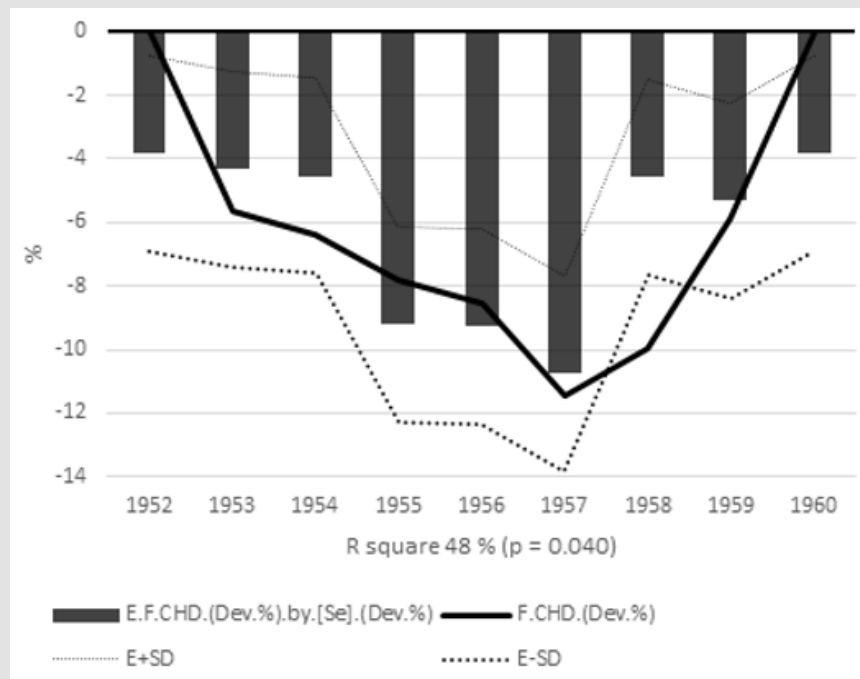


Figure 5: Exponential trendlines of Factors ([Se], F.CHD and M.CHD.urb) between start (α , 1952) and end (ω , 1960) points of parameters.



Note: Regression by [Se]. Dev.% gave prediction equation (E) for F.CHD. Dev.% = $-3.84 + (-0.322) * [Se]. Dev. \%$. It explained F.CHD by 48 % ($p = 0.040$). Regression by [Se]. Dev.% explained 37 % of M.CHD.urb. (Dev.%) ($p = 0.084$).

Figure 6: Shows F.CHD. (Dev.%) and its prediction by [Se]. (Dev.%).

Discussion

The main aim was to explain the disturbance, the downward deviation of CHD mortality in the 1950's, which is visible in female and male CHD in rural as well as in urban areas [5,6] (Fig.7 and Fig.8). The deviation from the exponential trendline (not drawn to Fig.7 nor Fig.8, but could be satisfactorily be replaced by direct line between start and end points) is lesser in rural regions and support the effect of imported grain (Se), which was more consumed in urban areas [4].

Because of the small change in the M.CHD.rur in the 1950's [5,6], the M.CHD.Dev.% is studied statistically only by M.CHD.urb. In order to keep this article compact F.CHD.Dev.% are calculated only by the whole Finland [5,8].

Finnish food chain received even other food and fodder from foreign countries. Remnants of food oil factories, oil cakes, were imported annually ca 50 million kilos to Finland in period 1951-61 [8]. Their protein content was ca 20-40 %, ca 3-fold that of bread grain [9], which (when imported from North America) could contain Se 0.1 ppm [4], about 10x to Finnish grain [10]. Although origin of oil cakes is not announced it is possible only to suggest that they carried annually ca 5-10 $\mu\text{g}/\text{d}$ Se to Finland per capita, but was processed via cattle. Their reduced import since 1957 could have a tiny (1-3 $\mu\text{g}/\text{d}$) reducing Se-effect since 1957.

Additive selenium sources in the 1950's was "soil improvement materials": In 1950 Finnish agricultural soils got ca 5.1 million horse

loads "clay or sand" (their proportion not given) and 5.2 million horse loads (approximated weight a'365 kg) of "peat soil" [12]. Soil improvement materials (together ca 4 billion kg) contained ca 0.10-0.15 ppm Se [13], totaling ca 400-600 kg Se per year, ca 1 – 1.5 kg Se/d together for Finnish people (4 million [14]), i.e. 250-400 μg Se/capita/d to the agricultural soils (supposedly 1/3 – 1/2 of this via peat soil) It is about 1/10 of the amount later given in Finnish fertilizers (2.8 g/ha/a) [15]. Two factors, which could have reduced plant Se content were competition with changing amounts of nitrogen (fertilizers) and sulphur [16] (fertilizers and emissions, which were acting partially via acidity).

Female and male CHD were lower in rural to urban regions until to the first half of the 1970's (in period when it was uncommon to by milk from stores in countryside and the average fat content of the milk was 4.4%, (anyhow consumption of low-fat sour milk was common) and consumption of imported grain was lower. Se consumption seemed to associate mirror-like with CHD in countryside and urban areas.

Lower CHD mortality in rural regions can be associated with higher consumption of whole grain food (with higher availability of Mg and Si with cell and tissue protective properties), when plants took more minerals from soil than given via fertilizers [18]. Effective recycling [18] together with peat soil and clay supplementation seemed to compensate losses of Se (and many other mineral elements, too). Non-regenerative clay could be substituted in the future by stone

meal or commercially available biotite [19,20]. Surprisingly Se content of lake fishes was high, ca 0.2-0.3 ppm as their ratio to S (Sulphur) was similar to fishes in oceans [21]. Possibly this selenium is of atmospheric origin [22]. Supposedly lake fish consumption was higher in countryside, which compensated the lower Se-grain intake, but without similar annual variation. Possibly the comparison of different Deviations (Dev.%) could be more reliable, if the aberrations between their base from the x-axis (Figure 4) were taken in account (by using cosines).

Conclusions

“Proportional deviation of Finnish selenium intake from its exponential trendline” associated mirror-like with CHD deviation from its proportional trendline in the 1950’s. This mathematical association was significant by females and trend-like by urban males and visual by rural males. Pearson associations were insignificant, and even positive.

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