Finnish Human Myocardial Degeneration-Associations with Selenium, P/Mg fertilization Ratio, Total Fertilization, Livestock Fodder, Sugar, Non-CVD and Subtypes of CVD in Finland during 1971 – 2012

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ABSTRACT

Pig heart muscle (Myocardial) Degeneration (MyCD) was efficiently treated since the 1960’s with Selenium (Se), but there are scanty published data on human MyCD. Mineral Fertilization (fm) Parameters, including (P/Mg)fm, have often explained less CHD (Coronary Heart Disease) than Non-CHD (others than CHD), but about similarly CVD (Cardiovascular Diseases) and Non-CVD (others than CVD). This study is treating (mortality from) MyCD, CVD and its subtypes: CHD, “cardiac diseases others than CHD” ([Card.oth]), Cerebrovascular (CbrVasc) and other Vascular Diseases ([Vasc.oth]) of Whole Population (WP) and aged 15-64 (15_64), as well as their associations with (P/Mg).fm, (CaMgNPK).fm, silage production (availability of vitamin E) and human sugar consumption, based on old data. Associations, as concerning Se, are mainly represented by figures, but [Card.oth] and its subtype MyCD are represented by regressions, too. Fertilizer data are slightly smoothened.

Results: CHD. (WP) deviated positively from CVD for the whole period, CHD. (15-64) positively before 1993 and after 1996 negatively. Others deviated mainly negatively. [Card.oth]’s and [Vasc.oth]’s to a lesser amount behaved like mirror-images to CHD. [CbrVasc] behaved like CVD. Non-CVD. (15_64) epidemic (1984-94) associated with peak of CaMgNPK with a small delay. Non-CVD. (WP) caused ca 41,000 extraordinary deaths in 1982-2000. MyCD and [Card.oth] were explained (54.8 – 90.7 %) (p < 0.001) by [P/Mg], [P/Mg;CaMgNPK], [Sugar] and [Silage]. Suspected Se changes are seen after 1985-86.

Conclusion: Fertilization parameters, sugar consumption and silage production explained changes in MyCD and [Card.oth] (15_64) by 55-91 % (p< 0.001). Reduction in [Card.oth], CbrVasc and [Vasc.oth] was faster than in CHD before 1991. Epidemic of Non-CVD associated with maximal total/carbonate fertilization.

Abbreviations: 3ym: 3-Year Sliding Mean, i.e. X.3ym.i = 1/3*[X.(i-1) + Xi + X.(i+1)]; 3ymw: 3-yr Mean Weighted Mean; i.e. X.3ymw.i = ¼*[X.(i-1) + 2*Xi + X.(i+1)]; (Acc.&.Viol): Accidents and Violence; [Card.oth]: Other (than CHD) Heart Diseases excl. Rheumatic and Alcohol-Related (I30-I425, I427-I52); CbrVasc: Cerebrovascular diseases (I60-I69); CVD: Diseases of the Circulatory System Excl. alcohol-related (I00-I425, I427-I99); CHD (as in referred papers): Ischaemic heart diseases (I20-I25); FM: Human (Females and Males); [Vasc.oth]: “Other Diseases of the Circulatory System (I00-I15, I26-I28, I70-I99); WP: Whole Population; (15_64): Population Aged 15-64 (modified abbreviation for ruling spelling in Exel)
Introduction

Heart muscle (Myocardial) Degeneration (MyCD) was efficiently treated by pigs since the 1960’s, but little or nothing has been published on human MyCD [1], if surgical treatments (on an average 16 transplantations annually to adult patients in 1985-2012) [2] are excluded. K/Mg and P/Mg fertilization ratios have associated differently with (mortality from) CHD and Non-CHD [3,4]. Available linear regression (numerical) analyses by fertilizer factors have not satisfactorily explained mortality difference between CVD and Non-CVD [5], although absolute increase in Non-CVD was higher than in Non-CHD (Figures 1 & 2).

Figure 1: CVD, Non-CVD (original and smoothened), Non-CHD, Cerebrovascular and [Vasc.oth] Mortality, aged 15-64, relative to 1971.

Figure 2: Changes in P/Mg and K/Mg fertilizer ratios in 1971-2000.

Historical data shows that fatal pig MyCD was practically ceased before 1971 through improved grain harvesting and post-harvesting methods and fodder Se supplementation [6]. Anyhow Se content of human diet was unsatisfactory (29 mcg/d) still at the mid 1970’s [7]. Se fertilization since autumn 1984 elevated Finnish dietary and body content to satisfactory level [8]. 1986 was the first year, when domestic food and fodder were Se enriched from January to December. It was suspected that the changes in cattle fodder could have had effects on human mortality.
The aim of this study is to clarify associations of selected fertilization factors, sugar consumption and silage production with mortality groups Tot, CVD, Non-CVD and its subtypes of CVD (CHD, [Card.oth], CbrVasc, [Vasc.oth] and the subtype of [Card.oth], MyCD, in the whole population (MyCD.WP) and aged 15-64 (MyCD. (15_64)).

Materials and Methods

Annual mortality data (1/100,000) are from Statistics Finland, as mortality from MyCD [9], (Table 1). Data on total mortality (Tot), CVD and its subtypes {1. CHD, 2. “cardiac diseases other than CHD” [Card.oth], 3. cerebrovascular [CbrVasc] and 4. other vascular diseases [Vasc.oth]} of whole population (WP) [10] and aged 15-64 (15_64) [11] are from Statistics Finland in free access. Mineral Fertilization (fm) factors (P/Mg) and approximate total fertilizer consumption (CaMgNPK), NPK (Eq/ha) and Liming agents (Lim. ag) are the same (and from the same sources) as in [5]. Data on silage production, i.e utilization, are from Statistical Yearbooks of Finland [12]. Human sugar consumption data (as an indicator of dilution of protective nutrients) are provided by Food Balance Sheets of FAOSTAT [13]. Se associations are represented as vertical lines on year 1985 or 1986 on several figures. Population data for calculating the excess Non-CVD mortality for the epidemics is provided by Statistics Finland [14].

|                | Myocardial degeneration | Sugar | Silage |
|----------------|-------------------------|-------|--------|
|                | 35-64 yrs | 65-74 yrs | 35-74 yrs |       |
| 1/100,000      | Total      | kg/cap/yr | 1,000tn |
| 1969           | 16.06      | 176       | 762     |
| 1970           | 12.25      | 161       | 673     |
| 1971           | 9.82       | 94.5      | 449     | 48.3  | 1012 |
| 1972           | 6.17       | 53.5      | 270     | 48.2  | 1394 |
| 1973           | 7.29       | 44.2      | 262     | 49.4  | 1682 |
| 1974           | 4.04       | 34.9      | 184     | 47.0  | 1925 |
| 1975           | 3.36       | 26.9      | 148     | 43.3  | 2025 |
| 1976           | 3.84       | 23.8      | 146     | 42.9  | 2309 |
| 1977           | 3.27       | 17.2      | 115     | 39.3  | 3276 |
| 1978           | 3.45       | 17.2      | 119     | 35.9  | 3891 |
| 1979           | 2.93       | 19.9      | 122     | 31.7  | 4036 |
| 1980           | 3.21       | 11.6      | 96      | 36.8  | 4181 |
| 1981           | 3.74       | 14.8      | 118     | 34.9  | 3861 |
| 1982           | 4.18       | 11.4      | 114     | 35.8  | 4319 |
| 1983           | 3.17       | 12.2      | 101     | 36.6  | 4232 |
| 1984           | 3.71       | 9.7       | 102     | 35.0  | 4563 |
| 1985           | 3.69       | 15.8      | 125     | 36.4  | 4552 |
| 1986           | 4.12       | 13.0      | 124     | 33.2  | 4963 |
| 1987           | 4.22       | 6.7       | 104     | 34.7  | 3580 |
| 1988           | 3.11       | 7.5       | 87      | 36.0  | 4299 |
| 1989           | 2.71       | 5.3       | 72      | 39.5  | 4705 |
| 1990           | 2.37       | 4.1       | 62      | 37.4  | 4318 |
| 1991           | 4.27       | 5.6       | 106     | 37.4  | 4642 |
| 1992           | 1.86       | 5.0       | 57      | 39.9  | 4589 |
| 1993           | 1.79       | 2.0       | 44      | 40.8  | 5337 |
| 1994           | 1.58       | 3.3       | 46      | 37.7  | 4309 |
| 1995           | 2.10       | 5.8       | 68      | 37.2  | 5633 |
| 1996           | 0.34       | 0.5       | 9       | 35.5  | 1012 |
| 1997           | 0.82       | 1.6       | 24      | 35.7  | 1394 |
| 1998           | 0.71       | 1.6       | 22      | 34.5  | 1682 |
| 1999           | 0.61       | 0.2       | 14      | 34.2  | 1925 |
| 2000           | 0.65       | 0.0       | 14      | 36.0  | 2025 |
| 2001           | 16.06      | 0.5       | 15      | 32.6  | 2309 |
| 2002           | 12.25      | 0.9       | 25      | 31.3  | 3276 |
Non-CVD has been attained by subtracting CVD from Tot and Non-CHD by subtracting CHD from Tot, which includes Accidents and Violence-(Acc.&.Viol), too. To avoid the potential risk to mix Non-CVD with pure “disease-Non-CVD” (Tot.nat-CVD), in figures, its label has been kept as (Tot-CVD), in order to keep the database intact. So Non-CVD has been used as synonym for (Tot-CVD) and respectively Non-CHD for (Tot-CHD). Associations are mainly represented by figures, but [Card.oth].(15-64) and its subtype MyCD.(35-64), as well as MyCD(65-74) are represented by regressions, too.

Fertilizer data are slightly smoothened, i.e., utilized as 3-yr mean weighted means (3ymw). X.3ymw,i = ¼ *(X.(i-1) + 2*X.i + X.(i+1)), in order to reduce the lateral shift of oblique parts of curves. Mortality curves are not smoothened with one exception: Non-CVD. (15_64) is given additionally in form of 3-year sliding mean: X.3ym.i = 1/3 *[X.(i-1) + X.i + X.(i+1)] (Figure 3).

Results
Observations

Figure 4 represents consumption of Total and NPK fertilizers (kiloequivalents/ha), as well as Liming agents (Lim.ag), i.e., CO3-fertilizers (3ymw-softened, as represented in Materials and Methods). (Ca and Mg are included in other fertilizers than carbonates, too). In Finland Mg-% has varied between 2 and 7 % in 1950-2000). Amounts of Lim.ag can be changed from kg’s to approximate CO3 (CO3.appr) equivalents by multiplying the amounts by selected fixed coefficient, which is increasing from 20.24 and 20.89, depending on Mg-% (from 2.0 to 7.0 %). 1.5 % change in Mg-% selection changes CO3.appr value only by 1 %. In order to avoid new calculations for different periods Lim.ag’s have been used as such, because variation in it is the same as in CO3.appr. For Figure 1, has been selected period 1971-2000, with biggest changes in fertilization. CaMgNPK and Lim.ag had two elevation periods: in 1977-92 and in 1992-2000. Elevation periods of NPK were in 1969-77 and in 1977-92. CaMgNPK and Lim.ag had highest peaks in 1984, NPK in 1977 (and nearly as high in 1988). By approximated Mg-%’s [2] and of amount of Lim. ag’s [5] calculated CO3 composed in the 1970’s 28 % and in 1980-99 45 % of total fertilization.
Figure 5 presents changes in P/Mg and K/Mg fertilization ratios in 1971-2000, the period with biggest changes in fertilization. P/Mg and K/Mg decreased between 1970-84, made upward deviation between 1984 and 1990-91. After that they were mainly declining until 1996-97 and increasing after that.

Figure 6 shows changes in Total (Tot), Tot natural (Tot.nat), CVD and CHD mortality and mortality caused by Accidents and Violence (Acc.&.Viol) in whole Finnish population in 1971-2017. Between 1984 and 1994 is to be seen elevations in all causes of mortality groups. Difference between Tot and Tot.nat (caused by Accidents and Violence) is rather small.

Figure 7 shows Tot, Tot.nat, CVD, CHD and (Acc.&.Viol) mortality, aged 15-64, in Finland during 1971-2017. Between 1984 and 1994 is to be seen elevations in all mortality groups, stagnation in CVD and CHD begun in 1983. Difference between Tot and Tot.nat, caused by (Acc.&.Viol) is proportionally higher than in (WP).
Figure 6: Changes in CVD, Non-CVD, CHD and [Card.oth] mortality, aged 15-64, relative to 1971.

Figure 7: Changes in CVD, Cerebrovascular, Other vascular, Non-CVD and Non-CHD mortality relative to 1971 in whole population.

Figure 8 shows changes in CVD, Non-CVD, CHD and [Card.oth] mortality of the whole population relative to 1971. [Card.oth] deviated negatively and CHD positively to CVD for the whole period. [CHD] deviation from CVD increased to 1994 (up to 23 %-units) and decreased after that. [CVD] deviation from CVD increased to 1996 (up to 30 %-units) and decreased after that. [Tot-CVD] exceeded its level of 1982 in 1983-2000. Between 1982 and 2001 Non-CVD caused 41,046 extraordinary deaths.

Figure 9 represents changes in original and (3ym) smoothened Non-CVD, CHD, CVD and [Card.oth] mortality, aged 15-64, relative to 1971. Until 1990 [Card.oth] reduction was faster to CHD and slower after 1994 (even stagnation in 1994-2012. [Card.oth] deviated strongly (to negative direction) from CVD in 1971-1978. After that [Card.oth] came horizontally closer and reached CVD in 1986. In 1986-94 it declined alongside to CVD and deviated horizontally from it in 1996-2012. Non-CVD exceeded its 1984 value between 1984 and 1994 and caused 4,688 extraordinary deaths (calculated by original values). CHD deviated positively to CVD before 1993 and negatively after 1996. Smoothened Non-CVD.3ym determines the start point for Non-CVD epidemic to 1983 (the same as the beginning of CHD and CVD stagnation).
Figure 8: Consumption of CaMgNPK, NPK and CO₃ fertilizers in 1969-2000.

Figure 9: Relative Proportions of CVD,(WP) Subtypes in CVD Mortality during 1971-2017.

Figure 10 shows CVD, Non-CVD, Non-CHD, ChrVasc and [Vasc.oth] mortality relative to 1971 in the whole population. Generally, ChrVasc and [Vasc.oth] deviated negatively from CVD, but less than [Card.oth] (Figure 2). [Vasc.oth] is increasing after 2006. ChrVasc and as well as [Vasc.oth] are treated only superficially in this article. 1985 has been marked as year before the whole-year-Sea-enrichment.

Figure 11 represents CVD, Non-CVD (original and smoothened), Non-CHD, ChrVasc and [Vasc.oth] mortality, aged 15-64, relative to 1971. Non-CVD deviation from its 1984 level is lower and shorter than by (Tot-CVD). [Vasc.oth] is seen to deviate negatively to CVD in 1971-1996 and after 2001 positively. Its downward deviation from CVD decreased remarkably between 1980 and 1990. During 1990-2001 [Vasc.oth] declined alongside to CVD and deviated upwards from it after 2001. ChrVasc declined similarly in 1971-2001, but after that alongside to CVD.
Figure 10: Relative Proportions of CVD.(15_64) Subtypes in CVD Mortality during 1971-2017.

Figure 11: MyCD.(35_64) and its regression by fertilization ratio P/Mg.

Figure 12 shows relative proportions of CVD.(WP) subtypes in CVD mortality during 1971-2017. CHD (42-60 %) forms a downwards opening curve with top in 1999, [Vasc.oth] (8-18 %) and [Card.oth] (8-20 %) are like mirror-images of CHD. CbrVasc has been rather stable (mean 23 %). Figure 13 shows relative proportions of CVD. (15_64) subtypes in CVD mortality during 1971-2017 (together 100 %). CHD (47-71 %) forms a downwards opening curve with top in 1980. [Vasc.oth] (6-19 %) and [Card.oth] (6-18 %) are like mirror-images of CHD. CbrVasc stayed rather stable (16-21 %). Proportional CHD reduction seems to accelerate since 1985. Mortality from CVD subtype, in age group 15-64, indicates obviously more closely their incidence than mortality in whole population.
Regressions

Regressions are calculated for the period, when [Card.oth. (15_64)] (since 1971) and MyCD.(35_64) (up to 2007), fertilization and sugar data were available. Regressions for silage are concerning period 1971-2000. R squares were highest with [P/Mg;CaMgNPK] and [P/MgLim.ag] (80 - 91%), lowest with sugar. [P/Mg] explained nearly as well as [P/Mg;CaMgNPK] MyCD.(65_74) and [Card.oth]. All represented results were highly significant (p < 0.001).

Figure 14 represents mortality from Myocardial Degeneration, aged 35-65, and its regression by fertilization ratio P/Mg. Regression explained variation in MyCD by 70.2 %. Positive deviation from the trend-line is seen between 1981 and 1996, during the elevation of CaMgNPK (Figure 4). Average death-rate of MyCD.(35-64) was 2.7, range 0.34-9.8 (1/100,000), which can explain the great variation.
Figure 14: MyCD (65_74) and its combined regression by P/Mg fertilization ratio and total fertilization.

Figure 15 represents mortality from Myocardial Degeneration, aged 65-74, and its regression by fertilization ratio P/Mg. Regression explained variation in MyCD by 89.7%. Between 1984 and 1987 is seen a “mini-epidemic” of MyCD, co-incidentally with the beginning of Non-CVD epidemic (Figure 4). The second MyCD decline (since 1971) started after 1985 earlier than expected by the regression, 1988 – Se effect?

Figure 15: MyCD (35_64) and its regression by sugar consumption in 1971-2007.

Figure 16 shows mortality from Myocardial Degeneration, aged 35-65, and its regression by P/Mg fertilization ratio and total fertilization. Regression explained MyCD variation by 84.3% (p < 0.001 for both beta coefficients, too). Figure 17 represents mortality from Myocardial Degeneration, aged 65-74, and its combined regression by P/Mg fertilization ratio and total fertilization. Regression explained MyCD (65_74) variation by 90.0%. This Fig. explains better than Figure 15 period 1983-87. Figure
17 represents mortality from Myocardial Degeneration, aged 35-65, and its regression by sugar consumption in 1971-2007. Regression explained MyCD variation by 54.8 %. Compliance seems rather good, but during 1986-93 trends go to different directions. Figure 13 represents mortality from Myocardial Degeneration, aged 35-65, and its regression by annual silage production (utilization) in 1971-2000. Regression explained MyCD variation by 67.9 %. Figure 14 Represents [Card.oth] and its regression by MyCD in 1971-2007. They explained variation in each other by 87.8 %.

Figure 16: MyCD.(35_64) and its regression by annual silage production/utilization in Finland in 1971-2000.

Mean mortality from [Card.oth], aged 15-64, was 13.3, range 9.5-25 (1/100,000), ie. on an average ca 5x higher than by MyCD.(35_64) – a subtype of [Card.oth]. Besides of reduction of MyCD, MyCD proportion inside of [Card.oth] was declining: from 27 % in 1971-87 to 20 % in 1988-95 and 6 % in 1996-2007. This and high compliance in Figure 13 shows that other factors inside [Card.oth] comply the variation in MyCD.

Figure 17: [Card.oth.(15_64)] and its regression by MyCD.(35_64) in 1971-2007.
Discussion

The aim of this study was to clarify associations of selected fertilization factors, sugar consumption and silage production with mortality groups, with special interest on MyCD.(35_64), MyCD. (65_74) and [Card.oth].(15-64). The effects of fertilization are not separated from food or feed factors, the changes are assessed as independent phenomena. The main observations are in the figures. This study shows that the decline in the mortality from other subtypes of CVD was higher than from CHD in 1971-1990 (Figures 5-8). Especially fast it was from [Card.oth] in 1971-78, but even faster from MyCD. Effect of cardiac transplantations on MyCD mortality (16/yr; since 1985) [2] was not remarkable. Proportion of CHD(WP) in CVD(WP) began to decrease since 2000, but CHD. (15_64) in CVD.(15_64) as early as in 1980 (Figure 9 & Figure10), i.e. slower than their absolute reduction.

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\frac{[\text{P/Mg}]}{[\text{P/Mg}\text{CaMgNPK}]}\text{, }\frac{[\text{P/Mg}\text{Lim. ag}]}{[\text{P/Mg}]}\text{, sugar consumption and silage utilization}\text{ explained variation in MyCD and [Card.oth] by 55-91\% , (p < 0.001) (Table 2, Figures 11-17). Regression by }\frac{[\text{P/Mg}\text{CaMgNPK}]}{[\text{P/Mg}]}\text{ explains MyCD.(35_64) by 83.4 \% (Figure 13) and MyCD.(65_74) by 90 \% (Figure 14). This difference between R squares can obviously be explained by different number of cases in the groups (Table 1). Figure 13 shows better than Figure 14, the augmentative role of CaMgNPK to [P/Mg] in MyCD regression. The longer stagnation since 1982-84 in Non-CVD [5] than in Non-CHD [3], in working age, can be explained by rather co-incidental stagnation in [Card.oth], CbVasc and [Vasc.oth], which are included in Non-CHD, but not included in Non-CVD (Figure 6 & Figure 8). These changes were obviously more dynamic and could have responded faster to the environmental changes than CHD or the main Non-CVD group. Maybe linear regression was not the best method for comparing associations in [5].

Table 2: Regressions of MyCD and [Card.oth] by fertilization factors [P/Mg], [P/Mg; CaMgNPK] and [(P/Mg); Lim.ag.kg], sugar consumption and silage production.

|                          | MyCD. (35_64) | MyCD. (65_74) | [Card.oth]. (15_64) |
|--------------------------|---------------|---------------|---------------------|
| [(P/Mg)]. [1971-2007]    | 70.2          | 89.7          | 87.0                |
| [P/Mg; CaMgNPK]. [1971-2007] | 83.4     | 90.0          | 90.7                |
| [(P/Mg). Eq; Lim. ag.kg]. [1971-2007] | 80.2   | 90.8          | 89.5                |
| Sugar kg/capita/yr (1971-2007) | 54.8     | 57.1          | 70.7                |
| Silage [1,000 tn/yr] (1971-2000) | 67.9     | 67.3          | 73.2                |

Different changes in mortality of CVD subtypes are not satisfactorily explained. Maybe they are possible to be explained by changes in availability of the mineral elements and principles represented in [3-5] including dualistic action of renin-angiotensin-adrenergic system [3]. Importance of harms of excess phosphorus has been supported by Varo and Koivistoinen [7] and Nuoranne [15]: “The amount of phosphorus may be slightly excessive in the average Finnish (human) diet” (in the 1970’s), when it was 4.5 (2/0.44) (g) [7]. On the other side pig diet, in which P/Mg ratio was 5.3 (5.3/1.0) and which contained Mg more than twice that recommended by international norms, has been reported to produce Mg-deficiency symptoms, which could be prohibited by Mg supplementation [15]. Since 1971 P/Mg ratio has been strongly reduced in fertilization (Figure 2), suggesting on P/Mg reduction in food, even more than calculated with aid of food composition tables from earlier decades.

Stagnation in the reduction of the fatal respiratory and digestive diseases in the 1980’s [5,11] suggests on deficiency in “resistance against biotic and a biotic stress”, a known silicon (Si) effect in plants [16]. Some protective abilities of Si in humans and animals have been observed [17] and suggested [18]. Si content of Finnish wheat in the 1970’s was < 0.1 g/kg [19], but in Australia 10 x higher (0.5-5 g/kg) [20]. The difference can be explained by climatological and soil factors, but possibly by different amounts of fertilizers, too. Through increasing soil pH, liming agents can reduce Si liberation for plants [21]. Possibly Si could explain a part of mortality changes during Non-CVD epidemic as well as the small change in P/Mg ratio (Figure 2). Mg can help in cellular protection: Normal plasma Mg - in contrary to hypomagnesemia (P-Mg 0.48 mmol Mg/L) - has protected cardiac muscle against ATP reduction, lipid peroxidation and LDH (lactate dehydrogenase) release during affection by reactive oxygen species [22]. So, the ability of liming agents [CO3] to fix Mg to insoluble form [23] could cause even some harms, although Mg.fm was increasing to the first half of 1980’s [5]. Big amounts of fertilizers could have caused leaching of Si and some microelements, too.

Silage effects on milk and milk fat quality: The lipid fraction in leaves of herbs and grasses ranges from 30 – 100 g.kg-1 [24]. During hay harvesting hay fat is soft, iodine number ca 100, but the soft fats turns soon in dry hay into resin and lose their butter fat softening effects. Iodine number in milk fat can vary from 25 to 50, but target is 32 –37 [25]. Nobelist AI Virtanen (AIV) developed his rapid silage preservation method (“AIV method”) in the 1920’s, but first at the end of the 1960’s new machinery (field chopper) increased silage utilization. Abundant feeding of AIV silage was found to increase iodine number, as well as carotene, vitamin A and E content in milk and produced butter, which resembled nearly summer butter [26]. These changes in fodder quality obviously changed respectively the quality of dairy products and could explain a part of the MyCD and [Card.oth] mortality reduction. Obviously Se status of humans had been increasing before the mid 1970’s, because at that time Se content of pork was about 4-fold to other meat products and egg yolk 100-fold to milk [19].

Surprisingly rapid decline in MyCD 1969-74 could possibly to be explained by changes in diagnostic methods and in social insurance (legislation on social security occurred mainly in the last half of the 1960’s and at the beginning of the 1970’s) [27].

Conclusion

Fertilization parameters, sugar consumption and silage production explained changes in MyCD and [Card.oth]. (15_64) by
55-91% (p<0.001). Reduction in [Card.oth], ChrVasc and [Vasc. oth] was faster than in CHD before 1991. Epidemic of Non-CVD associated with maximal total/carbonate fertilization.

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