

Research Article

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Replacing Potassium by Phosphorus in Finnish CHD and Non-CHD Mortality Regressions by Fertilization Rates in 1961-2000 increased the Strength of Associations



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Received:  August 10, 2018; Published:  September 05, 2018

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Abstract

Aim: Changes in human non-CHD (nCHD) mortality has not thoroughly been explained. Regressions by fertilization parameters, e.g. Mg/Ca and K/Mg, have been given and discussed. Daily allowance of phosphorus (P) is about double to the RDA. The possible role of excess phosphorus fertilization on human general and vascular health has not got much attention. This study shows regressions of human CHD and nCHD (death-rates) in 1961-2000 by single and multiple fertilization parameters, assessing especially the difference caused by replacement of potassium with phosphorus in regressions by two parameters or parameter ratios. Mortality and fertilization (Eq/ha) data are from the same sources as earlier published. In the results parameters x are Nitrogen (N), phosphorus (P) and potassium (K).

Results: Associations of [P/Mg], [P;Mg], [P/NPK] and [P;NPK] were stronger with CHD and nCHD than respective associations before replacement of K with P. [x/Mg], explained stronger nCHD than CHD, [x/NPK] explained them similarly and [x;NPK] explained stronger CHD. [P;Mg] explained stronger CHD, [N;Mg] explained stronger nCHD, [K;Mg] explained them similarly. Regression coefficients were positive with P and K, except single K with nCHD. Regression coefficient was positive with [N/Mg], but in other N regressions negative. Associations for given (N 32) regressions were highly significant ($p < 0.001$) in all but three cases.

Conclusion: Many changes in fertilization are statistically associated with human mortality. Replacing K by P in CHD and nCHD regressions increased the strength of associations. Causes of associations are discussed.

Keywords: CHD; Non-CHD; Potassium; Phosphorus; Nitrogen; Magnesium; Fertilization; Time Related

Abbreviations: CHD: Age Adjusted Coronary Mortality (I20-I25) of 35-64yrs Old Humans (1/100,000); (here Calculated as Mean of Female and Male CHD), K: Potassium; Mg: Magnesium; N: Nitrogen; nCHD (mortality) – difference between Total and CHD Mortality; P: Phosphorus

Introduction

Associations of changes in fertilization with mortality have been represented earlier from different periods and different population groups [1-3] even including P [1]. Low serum phosphorus concentration has been associated with lesser coronary calcification [4]. Excessive P intake has been associated with carotid intima thickening [5] and to impair endothelial function [6]. In 1975-78 Finnish daily diet contained P 2000 mg/d [7], per 10 MJ [8]. Intake of phosphorus was more than two-fold to RDA [9]. During the reduction of CHD mortality dietary intake of P seems to have been decreased, because 2012 it was ca 1880/ 10 MJ by middle aged people (men 1738 mg/9.4 MJ, women 1402 mg/7.3 MJ) [9]. P-excess with low Ca and Mg in plasma are reported to be associated with spastic paresis and nervous excitation ("tetany" in cattle)[10], and so obviously with increased risk of fatal CVD with dynamic mechanisms [11]. On the other hand, harmful associations of P fertilizers with death-

rates could be explained by Mg precipitation in soil and cattle rumen as "very slightly soluble" MgNH_4PO_3 [12]. Sulfur compound (gypsum) formed in superphosphate synthesis [12] is not included into the commercial superphosphate (error in [1]). The special aim of this mainly statistical study was to assess the difference caused by replacement of potassium with phosphorus in regressions by two parameters or parameter ratios.

Materials and Methods

Age adjusted total mortality of 35-64-y. females and males are from Valkonen and Niemi [13] and CHD death-rates from Valkonen and Martikainen 1990, for period 1951-68 [14]. Respective total and CHD mortality in 1969-2000 are from Statistics Finland [15]. Female (F) and male (M) total (TOT) and CHD mortality statistics (1/100,000) are represented in [2]. Approximate for human CHD

comes from equation: $(FM.)CHD = \frac{1}{2}*(F.CHD+M.CHD)$. $(FM.)_nCHD = \frac{1}{2}*(F.TOT+M.TOT) - (FM.)CHD$. CHD and nCHD without prefix "FM" represent here "human" values. Approximated Mg rates per hectare are from [3]. Annual consumption of N, P₂O and K₂O fertilizers [16] and arable land [17] are from FAOSTAT. By them are calculated values for N, P and K kg/ha. They are changed to equivalents (Eq/ha) to help comparison. Values for K are totally represented in [3], P and N partially in [1].

Results

Table 1 In the group of single fertilizers Mg associated strongest with CHD and nCHD with negative coefficient. Next strongest

association CHD had with P(positive) and nCHD with N (negative). Associations of [P/Mg], [P;Mg], [P/NPK] and [P;NPK] were stronger with CHD and nCHD than with respective [K/Mg], [K;Mg], [K/NPK] and [K;NPK]. [x/Mg], explained stronger nCHD than CHD, [x/NPK] explained them similarly and [x;NPK] explained stronger CHD. [P;Mg] explained stronger CHD, [N;Mg] explained stronger nCHD, [K;Mg] explained them similarly. (Tables 1 & 2) Regression coefficients of CHD and nCHD were positive with P and K, excluding only nCHD with single K. Coefficients were positive with [N/Mg], but in other N regressions negative. Associations for given regressions were highly significant ($p < 0.001$) in all but three cases of 32 (Tables 1 & 2).

Table 1: Associations of mineral fertilizers or their combinations or ratios with CHD and non-CHD mortality.

Associations of mineral fertilizers or their combinations or ratios with CHD and non-CHD mortality				
	CHD		FM.nCHD	
	R square	coefficients	R square	coefficients
K	0.04	(+)	0.028	(-)
P	0.409	(+)	0.112	(+)
N	0.327	(-)	0.635	(-)
Mg	0.537	(-)	0.737	(-)
K/Mg	0.675	(+)	0.778	(+)
P/Mg	0.693	(+)	0.806	(+)
N/Mg	0.439	(+)	0.474	(+)
[K;Mg]	0.766	(+.)	0.756	(+.)
[P;Mg]	0.844	(+.)	0.791	(+.)
[N;Mg]	0.537	(-.)	0.789	(-.)
[K/NPK]	0.732	(+)	0.726	(+)
[P/NPK]	0.872	(+)	0.873	(+)
[N/NPK]	0.86	(-)	0.859	(-)
[K;NPK]	0.811	(+.)	0.734	(+.)
[P;NPK]	0.953	(+.)	0.924	(+.)
[N;NPK]	0.954	(-.)	0.906	(-.)

Table 2: Summary of CHD and nCHD regressions, relative strength of associations, direction of coefficients. Mg coefficient was always negative. (x = K, P or N).

Table 2. Summary of CHD and nCHD regressions, relative strength of associations, direction of coefficients. Mg coefficient was always negative. (x = K, P or N)				
CHD and nCHD regression by	Strength of association with	K	x	P
x/Mg	CHD <nCHD	(+)	(+)	(+)
x/NPK	CHD $\approx$ nCHD	(+)	(+)	(-)
x;NPK	CHD >nCHD	(+)	(+)	(-)
K;Mg	CHD $\approx$ nCHD	(+)		
P;Mg	CHD >nCHD		(+)	
N;Mg	CHD <nCHD			(-)

Discussion

In the statistical replacement K by P increased the strength of associations with CHD and nCHD. Strong Mg deficiency is known to cause in man semicoma, tetany-like neuromuscular irritability in humans, which can be promptly cured by Mg administration [18]. Haaranen reported in 1968 that cattle hypomagnesemic tetany was associated with hyperphosphatemia [10]. On the other hand, parenteral administration of large amounts of Mg can cause anesthesia, which can be stopped in a few seconds by Ca [18]. Haaranen reported on somnolent paretic cows (flaccid paresis) on forest pasture with poor P content supplemented with excessive amounts of Mg (not on field pastures, with higher P content). They had high serum Mg and low Ca and especially low serum P [19] (Table 3). Possibly dietary P can regulate serum Mg and Ca levels. Grass (hypomagnesemic) tetany occurs most often in early spring and late autumn [20].

Table 3: Serum values of somnolent paretic cows with dietary Mg excess and P deficiency [19].

Table 3. Serum values of somnolent paretic cows with dietary Mg excess and P deficiency [19]			
	Ca	Mg	P
mmol/L	1.40	1.98	0.13
(ref. mmol/L)	2.17 - 2.74	0.82 - 1.43	1.45 - 2.58

This can be dependent on characteristics of biotite – the main Mg source of Finnish soils (rich in clay soils) [21]: The temperature dependent release of interlayer K is about one week faster than the release (by weathering) of Mg (and Si) [22]. High K installments on clay soils are reported not to decrease Mg herbage content as much as on the sandy soils [23]. Characteristics of biotite could partially explain the different variation of Mg (and Si), K and P in early spring, dry and rainy summers [24]. Possibly depending on cold June 1974 Mg contents of timothy were the same in clay and sandy soils in [25]. (Temperature in May 1974 was 2.7 °C below the average + 7 °C in randomly selected five Finnish towns - Helsinki, Turku, Kouvola, Jyväskylä and Kuopio) [26]. Maybe low Si content could not balance P values [27] in early spring and rainy summer [24]. Of course, the high K and P grass content in the spring can be based on high amount of fertilizers in early spring.

In 1978 was reported on high seasonal variation in grass K content and recommend giving K in multiple installments [28]. Between 1961-74 K/P ratio in fertilizers was quite constant why statistical differences are difficult to distract. So, there are several mechanisms, which could explain covariance of K with P. They can partially explain why grass tetany index $K/(Ca+Mg)$ [20] can reflect availability of P, too. Changes in N-fertilization are associated with reduction of hay production (one yield/year) and increased silage production (tree yields/year). The inverse association of N-fertilizers in NPK-complex on mortality could possibly be explained by harms of excessive P, benefits of silage, benefits of N on Mg uptake [29] and changes in selenium supplementation of

fodder (+food) and fertilization [30]. These possibly exceeded the harms of antagonistic effect on Si content [31]. Amounts of N, P, and K are accurate, but Mg values are approximations [3].

Conclusion

Many changes in fertilization are statistically associated with human mortality. Replacing K by P in CHD and nCHD regressions increased the strength of associations. Causes of associations are discussed.

Acknowledgement

I am grateful to Professor Osmo Hänninen and late veterinary surgeon Seppo Haaranen for several discussions.

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ISSN: 2574-1241

DOI: [10.26717/BJSTR.2018.08.001696](https://doi.org/10.26717/BJSTR.2018.08.001696)

Töysä T. Biomed J Sci & Tech Res



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