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The Tromsø Family Intervention study

Effects of a family approach to reduce coronary risk factors in children of high-risk men

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This study was done to assess the effect of intervention on coronary heart disease risk factors among children using a family approach. Men at increased risk of coronary heart disease (n=1,373) were randomly allocated to intervention and control groups together with their wives (n=1,143) and children (n=2,838). The intervention families received home visits by a physician and dietician, quarterly newsletters regarding diet, smoking and physical exercise and were invited to 'stop smoking' clinics and meetings on nutrition and exercise. At rescreening 6 years later, 29 of the control children exceeded pre-set risk factor limits compared with 15 in the intervention group ($p<0.05$). Children in the intervention group reported 'better' dietary habits than children in control families, especially for foods commonly eaten at home. At least 7 of the 9 'good' dietary habits were practised by 205 intervention children compared with 156 in the control group ($p<0.01$) and 88 versus 154 reported practising at least 3 of the 9 listed 'bad' dietary habits ($p<0.001$). No significant differences were found between the 12–24 year old children in the 2 groups in mean risk factor levels, the proportion of smokers or in the pattern of physical exercise. It was concluded that coronary heart disease risk reduction in children using the family approach is well received and results in dietary changes and a reduced number exceeding pre-set risk factor limits. The effect on mean risk factor levels, smoking and physical exercise was small. Targeting the intervention more directly to children could possibly improve the results. Also, life-style changes may require a longer follow-up before significant differences can be seen among teenagers.

Key words: CHD, life-style intervention, diet, adolescents

Prevention of coronary heart disease (CHD), commencing with children and adolescents is intriguing because of the prospects of delaying or even preventing the atherosclerotic process before it has really started. On the other hand, motivating children and adolescents to change their life-style when there are no signs of disease, is difficult. Several studies have been done to assess the effect of life-style intervention in children and adolescents.[1–10] Puska et al.[1] reported that a 2 year intensive school-based intervention on health behaviour and CHD risk factors in 13–15 year old children resulted in a significantly lower increase in the proportion of both boy and girl smokers. For total cholesterol, no benefit was seen in boys, but in girls there was a significant decrease as compared to the reference population. In addition, for dietary habits, there was a significantly greater decrease in the intake of dairy fat both among boys and girls as compared to the reference population. No differences were seen in blood pressure. Botvin et al.[2] studied the effect of a school-based intervention programme on being overweight and found a significant short-term effect (10 weeks) in the intervention group. Brownell et al.[3] also studied the effect of a 10 week school-based intervention programme on obesity and found significant decreases in the per cent overweight in the intervention group compared to the control group. Walter et al.[4] studied the effect of a school-based programme aimed at lowering CHD risk factors. At 1 year, diastolic blood pressure was 1.1 mmHg lower in the intervention group and serum thiocyanate was 5.4 μmol/l lower. These authors concluded that "Intervention programs in schools may, after sufficient duration, prove to be effective in lowering CHD risk". Walter et al.[5] reported the 5 year results from the same study. A very small, but significantly larger rate of annual serum cholesterol decrease (1.7 mg/dl/year) was observed in the intervention schools as compared to the control schools in the middle- and upper-class areas. No differences were found between intervention and control schools in the lower-income areas. Neither were there any significant differences in any of the other CHD risk factors. These authors concluded that "educational programs to modify coronary risk factors are feasible and may have a favourable (albeit small) effect on blood levels of cholesterol in children". Nader et al.[6] studied the effects of a family-based CHD risk reduction intervention in Mexican and Anglo-American families and found that after 1 year, diastolic blood pressure in the children was close to 3 mmHg lower than at baseline and the 'salt-score' was also significantly

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lower at 1 year compared to the baseline value. The problem with this study is that it did not have a control group. Another study with only baseline and follow-up measurements was the Heart Healthy Program[8] which reported substantial changes in eating behaviour and in knowledge about heart health and food preferences among children after having been given school-based intervention. Thompson et al.[9] reported significant differences in blood pressure after giving nutrition and physical fitness intervention to school children. Based on these studies, the effect of intervention clearly varies and generally is quite small. It is likely and has also been shown[10] that the eating habits of parents have an impact on the nutrient intake of their pre-school children, thus furnishing indirect support for dietary intervention targeting families for the primary prevention of CHD. The fact that life-styles tend to aggregate in families was the main reason this study used the family approach of intervention. An additional motivating factor for behaviour change among the children and adolescents in the families in the present study was the knowledge that their father had been identified as being at increased risk of CHD. This paper evaluates the effect of life-style intervention among the children of men with increased CHD risk in this context.

MATERIAL

In 1979–1980, all men, 20–54 years and all women, aged 20–49 years, living in the municipality of Tromsø, were invited to the Tromsø Health Survey through a personal letter. A total of 16,621 subjects participated in the screening which was designed mainly to assess coronary risk factors and certain aspects of life-style.[11,12] Based on the findings of this survey, 1,373 healthy men, aged 30–54 years, without known hypertension, diabetes mellitus, myocardial infarction or symptoms of intermittent claudication or angina pectoris, were identified as being at increased risk of developing CHD.[13] High CHD risk was defined as being in the lowest quintile of relative HDL (high density lipoprotein cholesterol/total cholesterol ≤17.6%) and/or the highest decile of total cholesterol (≥7.86 mmol/l). These men, together with their families (those living in the same household), were randomly allocated to an intervention (n=673) or a control group (n=700). In total, the study includes 1,373 men, 1,143 wives and 2,838 children. All children in the study who were 12 years and older and still living in Tromsø were invited to the survey. The effect of intervention was assessed on those who were 12–24 years old at the time of rescreening. Of the 673 men in the intervention group, 608 men and their families were actually given intervention. The control group remained unaware of its risk throughout the study.

Evaluating effect — rescreening in 1986–1987

The intervention and control families were rescreened in the 1986–1987 Tromsø Health Survey,[14,15] a population survey of persons 20–64 years of age living in the municipality of Tromsø. Of the original cohort of 1,373 men and 1,143 wives, 1,060 and 809 respectively attended the 1986–1987 survey. This represents 87.2 and 93.1% of those who had attended the 1979–1980 screening and were still living in the municipality. Of the 2,838 children in these families, 1,749 were 12–24 years old in 1986 and 1,498 of them were still living in the municipality in 1986. Of these, 1,103 (73.6%) attended the 1986–1987 survey.

METHODS

The Family Intervention Study has been described in detail elsewhere.[13,14,16] Briefly, the 673 men in the intervention group were informed in a letter that they had increased CHD risk and that their family was at increased risk due to a shared life-style. The whole family was offered help to reduce this risk. The intervention consisted of 2 home visits, first by a physician and 2–3 weeks later by a dietician. The visits were done during the second year following the 1979–1980 survey. During the home visit, the physician gave information about the risk factors for CHD and how to modify them, with special emphasis on diet. Non-fasting blood samples were collected from all family members 13 years and older and blood pressure was measured of those 7 years and older. The family completed a questionnaire on past and present smoking habits and all smokers were urged to stop smoking. The need for physical exercise was emphasized and an appointment for the dietician's visit was made. The dietician recorded a detailed dietary history of the high-risk man using food models and a short questionnaire on his food habits compared to that of his wife and children. Based on this information, detailed dietary advice was given using food models and slides. Finally, the height (cm) and weight (kg) of all family members were measured. All family members were encouraged to be present during the 2 home visits, taking time off from work and school.

Follow-up

Contact was maintained with the families through quarterly newsletters. In addition, stop-smoking clinics were offered 3 times during follow-up. Also, 2 meetings on nutritional topics with food samples and 1 meeting on exercise with invitations to join an exercise group, were held. A mean of 70 families (mostly adults) were represented at these meetings. The men in the intervention group were offered new blood tests 18 months after the home visit and feedback of the results was done by phone. Another phone call to the high-risk man was made 1 year later by a dietician or medical assistant. The families were free to contact the principal investigator (SFK) for dietary or medical consultation throughout the study, but very few used this opportunity.

Evaluation of effect

Information received at the 1986–1987 Tromsø Health Survey forms the basis for evaluating the effect of intervention. In this survey, blood pressure and pulse rates were measured using a Dinamap automatic blood pressure device. Non-fasting blood was drawn for total cholesterol and HDL cholesterol assessment. In addition, the parti-

cipants answered questions on smoking and physical exercise. They also completed a self-administered food frequency questionnaire. To assess the effect on psychosocial factors questions on loneliness, support systems, ability to cope, depression and insomnia, as well as the use of tranquillizers and sleeping pills, were also included in the questionnaire. The respondents were unaware that the effect of intervention was being measured through their responses to the 1986–1987 Tromsø Health Survey.

Statistical methods

Analysis was done using the Statistical Package for the Social Sciences (SPSS). High-risk men were compared with other men in Tromsø in 1979–1980 to estimate the degree of elevated risk. Likewise, the high-risk wives attending the screening in 1979–1980 were compared with other married women of the same age in Tromsø. No data from 1979–1980 were available on the children.

The effect of intervention was tested in both high-risk men, their wives and children by comparing the intervention and control groups for blood lipids, blood pressure, body mass index (BMI) (g/cm^2), coronary risk score[17], smoking and dietary habits in 1986–1987. Age-adjusted means and standard deviations were calculated for risk factors given as continuous variables for the intervention and control groups separately. Differences were tested using the 2-tailed t-test and the direct method of standardization was used for age adjustment. For smoking, dietary and other life-style habits, variables were dichotomized and age-adjusted proportions calculated. Age adjustment and statistical analysis of differences between the 2 groups were done using analysis of variance with age as the covariate.

Differences in the numbers exceeding pre-set risk factor limits and the numbers practising 'good' or 'bad' dietary habits were tested using the Chi-square statistic.

RESULTS

At baseline, the high-risk men had significantly higher levels of total cholesterol, triglycerides, systolic and diastolic blood pressure and lower HDL and relative HDL as compared to men of the same age in the general population *(table 1)*. They also had fewer years of education and a larger proportion were daily cigarette smokers, drank 5 or more cups of coffee per day and used butter/hard margarine as spreading fat. A smaller proportion were physically active during leisure time and fewer ate fruits and vegetables daily. Based on the serum lipids, blood pressure and smoking habits, it was estimated that the high-risk men had a 20% increased risk of coronary heart disease as compared to other men of the same age.[13] The findings among the wives were similar *(table 2)*, although of smaller magnitude. They had significantly higher total serum cholesterol, lower relative HDL and higher BMI. They also had fewer years of education, a greater proportion were smokers, consumed 5 or more cups of coffee, used butter/hard margarine as spreading fat and used whole-fat milk and a lower proportion ate fruits and vegetables daily. It was estimated that the high-risk wives had an increased CHD risk of 19%.[16] Children were not screened in the 1979–1980 population and therefore there are no baseline values comparing children of high-risk men with children in the general population. However, children 12–19 years old in the control group were compared with children of the same age in the general population at the time of rescreening in 1986–1987 *(table 3)* and it was found that they had higher serum cholesterol and triglyceride levels and higher BMIs as well as lower HDL cholesterol and relative HDL. Based on the coronary risk score, the high risk children had a 20% higher risk of coronary heart disease compared to children in the general population. Their diets, however, differed only with respect to the proportion of teetotalists and the use of fresh fruit and salty dinners.

Assessment of intervention effect was done using data collected at the 1986–1987 screening. The differences between the high-risk men in the intervention and con-

Table 1 Mean (SD) age and age-adjusted risk factor levels and life-style characteristics (per cent) at baseline in the high-risk men and in symptom-free men of the same age in the general population 1979-1980

	High-risk group n=1,373		Other symptom-free men n=4,130		
Risk factor	X	SD	X	SD	p
Age at baseline	41.1	7.4	39.2	7.1	***
Total cholesterol (mmol/l)	7.43	1.17	5.85	0.96	***
HDL-cholesterol (mmol/l)	1.22	0.54	1.55	0.42	***
Relative HDL (%)	16.23	5.65	26.91	7.48	***
BMI (g/cm^2)	2.57	0.29	2.43	0.26	***
Triglycerides (mmol/l)	2.33	1.18	1.46	0.83	***
Systolic BP (mmHg)	131.77	14.4	130.14	13.8	***
Diastolic BP (mmHg)	85.20	9.75	82.98	9.90	***
Years of education	10.47	3.64	11.11	3.90	***
Daily smokers	59.2%		45.7%		***
Relatives with CHD	35.1%		31.2%		***
Sedate in leisure time	25.3%		18.4%		***
Active in leisure time	25.5%		34.9%		***
Teetotalists	7.5%		5.8%		*
Using					
≥5 Cups coffee/day	70.3%		61.7%		***
Butter/hard margarine	30.1%		25.5%		**
Fruits/vegetables daily	40.5%		44.1%		*

* p<0.05, ** p<0.01, *** p<0.001

trol group have been reported earlier.[14] Men in the intervention group had a significantly greater change in total cholesterol, triglycerides, BMI and coronary risk score as compared to the control group. There was also a larger decrease in the number exceeding pre-set risk factor limits and the intervention group reported significantly 'better' dietary habits, especially with respect to the use of coffee, table fat, salt and dairy fat. No statistically significant difference in risk factors or change in risk factors was found between the wives in the intervention and control groups.[14] However, the wives in the intervention group reported less use of both butter/hard margarine, meat for dinner and whole-fat milk and more use of soft margarine/oil, fresh fruit, vegetables and skimmed milk. There was also a larger increase in the proportion who were physically active during leisure.

To evaluate the effect of the intervention on the children and adolescents who were 5–17 years old when the intervention started, the findings between the 12–24 year olds in the intervention and control groups in 1986–1987 were compared. No difference was found in the traditional coronary heart disease risk factor levels *(table 4)*. However, a smaller proportion of children in the intervention group reported use of whole-fat milk, butter/hard margarine and meat as a dinner entree. A larger proportion reported use of soft margarine/oil, bread, lean fish and skimmed milk. A larger proportion of children in the intervention group reported practising 7 or more 'good' dietary habits (203 versus 156, $p<0.01$) and 88 versus 154 practised 3 or more of

Table 2 Age-adjusted mean (96% CI) levels of CHD risk factors and dietary and life-style variables among wives of coronary high-risk males and wives of the same age in the general population 1979-1980

	Wives of high-risk men n=911		Married women in general population n=4,271		
Risk factor	X	(95% CI)	X	(95% CI)	p
Total cholesterol (mmol/l)	5.98	(5.9–6.1)	5.78	(5.7–5.8)	***
HDL-cholesterol (mmol/l)	1.74	(1.71–1.77)	1.77	(1.76–1.78)	
Relative HDL (%)	30.0	(29.5–30.6)	31.6	(31.3–31.8)	***
Triglycerides (mmol/l)	1.13	(1.09–1.17)	1.10	(1.09–1.11)	
Systolic BP (mmHg)	122.5	(121.6–123.4)	121.6	(121.2–122.0)	
Diastolic BP (mmHg)	78.9	(78.3–79.5)	78.5	(78.2–78.8)	
BMI (g/cm^2)	2.31	(2.29–2.33)	2.28	(2.27–2.29)	*
Coronary risk score	4.55	(4.04–5.06)	3.81	(3.66–3.97)	**
Years of education	9.81	(9.6–10.0)	10.5	(10.4–10.6)	***
Daily smokers	47.3%		42.1%		**
Sedate in leisure time	22.5%		19.9%		
Sedentary occupation	22.6%		24.8%		
Using					
≥5 Cups coffee/day	56.8%		51.8%		**
Butter/hard margarine	28.1%		22.5%		***
Soft margarine	69.8%		75.1%		**
Whole fat milk	57.2%		50.9%		***
Non-fat milk	20.5%		26.5%		***
Fruits/vegetables daily	62.2%		67.1%		**

* p<0.05, ** p<0.01, *** p<0.001

Table 3 Age-adjusted mean (SD) of risk factor levels among children in the high-risk control group and children in the general population at rescreening in 1986-1987; age 12-19 years

	High-risk children n=367		General population n=389		
Risk factor	X	SD	X	SD	p
Total cholesterol (mmol/l)	4.50	0.89	4.25	0.79	***
HDL-cholesterol (mmol/l)	1.36	0.32	1.40	0.31	*
Relative HDL (%)	30.9	8.0	33.7	8.0	***
Triglycerides (mmol/l)	1.22	0.70	1.03	0.62	***
BMI (g/cm^2)	2.11	0.30	2.09	0.30	*
Systolic BP (mmHg)	117.84	11.5	117.08	10.4	
Diastolic BP (mmHg)	61.08	8.5	61.29	7.9	
Pulse rate/min	74.33	13.0	74.88	11.7	
Coronary risk score	4.78	5.8	3.99	3.6	**
Daily smokers	25%		23%		
Physically active leisure	46%		44%		
Relatives with MI or AP	11%		7%		**
Exceeding ≥1 risk limits	5%		1%		**
Teetotalists	40%		46%		***
Using					
Salty dinners ≥2/month	28%		20%		**
Addition of salt seldom	57%		63%		*
Fruits/vegetables daily	22%		16%		**

* p<0.05, ** p<0.01, *** p<0.001

Table 4 Mean (SD) age-adjusted risk factor levels and life-style characteristics (%) among 12-24 year old children in the intervention and control group: rescreening 1986-1987

	Intervention n=551		Control n=550		
Risk factor	X	SD	X	SD	p
Total cholesterol (mmol/l)	4.57	0.82	4.63	0.93	
HDL-cholesterol (mmol/l)	1.34	0.32	1.36	0.32	
Relative HDL (%)	30.0	7.7	30.18	8.0	
Triglycerides (mmol/l)	1.28	0.74	1.25	0.75	
BMI (g/cm^2)	2.19	0.31	2.17	0.32	
Systolic BP (mmHg)	120.5	11.1	119.3	11.7	
Diastolic BP (mmHg)	63.6	8.6	62.90	8.5	
Coronary risk score	5.5	7.4	6.1	10.8	
Daily smokers	34%		34%		
Physically active leisure	41%		39%		
Teetotalists	29%		27%		
Exceeding ≥1 risk factor limits	3%		5%		*
Using					
≥5 Cups coffee/day	13%		11%		
Butter/hard margarine					
As spreading fat	5%		11%		***
In cooking	25%		35%		***
Soft margarine/oil					
In cooking	67%		53%		***
As spreading fat	57%		53%		
Fresh fruits ≥4/week	43%		39%		
Meat dinner ≥3/week	25%		33%		**
Whole-fat milk	16%		22%		**
Low-fat milk	40%		42%		
<2 Slices bread/day	1%		3%		*
Skimmed milk	26%		18%		**
Lean fish					
<1/week	19%		29%		***
≥3/week	10%		8%		

* p<0.05, ** p<0.01, *** p<0.001

Table 5 Pre-set risk factor limits and number exceeding one or more of these limits among children of high-risk men in the intervention and control groups at rescreening in 1986-1987: age 12-24 years

Pre-set risk factor limits

	Males				Females			
Age	Cholesterol mmol/l	Systolic BP mmHg	Diastolic BP mmHg	Score	Cholesterol mmol/l	Systolic BP mmHg	Diastolic BP mmHg	Score
≤17	5.79	158	92	40	6.31	168	92	40
18	5.95	158	92	40	6.47	168	92	40
19	6.10	159	93	40	6.62	169	93	40
20	6.26	160	93	40	6.78	170	93	40
21	6.41	160	93	40	6.93	171	93	40
22	6.57	160	94	40	7.09	172	94	40
23	6.62	161	94	40	7.14	172	94	40
24	6.67	161	95	40	7.19	173	95	40

Numbers exceeding pre-set limits

Risk factor	Intervention group N=551	Control group N=550
≥1 risk factor limits	15	29 *

* p<0.05
Differences tested using the Chi-square statistic

the 9 'bad' dietary habits (p<0.001) shown in *table* 6. Twenty-nine children in the control group exceeded one or more risk factor limits (*table* 5) as compared to 15 in the intervention group (p<0.05).

There were no significant differences between the intervention and control groups in the proportion feeling lonely, lacking support, unable to cope, depressed or having insomnia. Nor were there any differences in the proportion using tranquillizers or sleeping pills, thus, giving no indication of an intervention effect on these parameters.

DISCUSSION

The purpose of this paper was to assess the effect of intervention on CHD risk in children/adolescents using the family approach. It was found that life-style intervention using the family as the unit of intervention did not change the mean risk factor levels among the children/adolescents of the family. However, intervention did affect the number of children/adolescents who exceeded pre-set risk factor limits for the same traditional CHD risk factors. Significant differences were also found between the intervention and control children with respect to dietary habits such as type of spreading fat, cooking fat, type of milk, meat, fish and fruit intake. The dietary differences reported between the children of the intervention and control groups were more or less the same as those reported by the parents, such as spreading fat, fruit, vegetables, dairy fat, meat and fish. These are core foods, which are usually purchased by the adults in the family and eaten in the setting of regular

meals (at home). Therefore, it is not surprising that the family members report similarly on their use. However, meals taken at home do not constitute the only source of food for children/adolescents. Unfortunately, there was no information about fast foods and snacks (hamburgers, ice-cream, hot dogs, etc.) which are known to be important sources of fat in the diet of children/adolescents. Information about these foods would most likely give us more information on the actual differences in the level of health consciousness and independent food choices among the young people in the intervention and control groups. It may also have been able to shed light on why there were no differences in mean risk factor levels between the intervention and control groups.

Several weaknesses can be identified in this study. Firstly, since there are no baseline measurements of risk factors on the children, it was assumed that the children in the 2 groups were similar at baseline and that any differences found at the 1986–1987 screening were due to the intervention. Because this is a randomized study with the high-risk men being allocated to intervention and control, this assumption is felt to be valid. Secondly, the food frequency questionnaire used to assess diet, had only been tested in an adult population and, thus, it is not known how valid and reliable it is among children/adolescents. Also, since the questionnaire was mainly a food frequency questionnaire, it was not very discerning about the amounts consumed. This was especially true for spreading fat and dairy fat where only the type of fat and not the frequencies of consumption were questionned.

It is difficult to explain why there is no difference in serum cholesterol levels between the intervention and control groups in spite of significant differences in reported dietary habits. One explanation could of course be, as mentioned earlier, that information is lacking about important sources of dietary fat among the teenagers and that the dietary consumption assessment tool is too crude. Another possibility is that different dietary habits do not have the same immediate impact on the serum lipid levels in teenagers as in adults. This hypothesis is supported by the almost universal finding in Western culture, that serum cholesterol increases with age, whereas that is not the case in countries with a low incidence of CHD. There is no indication that the age increase in serum cholesterol is caused by a progressively more coronary-prone life-style. Rather, the differences in dietary habits, when persisting over time, seem to result in increasingly greater differences in serum lipids.

Table 6 Nine 'good' and 'bad' dietary habits used in analysis of differences between the intervention and control group

Habit	'Good'	'Bad'
Spreading fat	None/soft margarine	Butter/hard margarine
Cooking fat	Oil/soft margarine	Butter/hard margarine
Fat on dinner	<1 per week	≥3 per week
Milk fat	Non fat/low-fat	Whole fat
Cups coffee/day	<5 cups/day	≥5 cups/day
Meat for dinner	<3 per week	≥3 per week
Use of fruit	≥4 per week	<2 per week
Add salt on foods	Seldom/never	Always
Use salty dinners	<1 per month	≥2 per week

When planning a family approach to intervention, there are several problems to be aware of. Somatization can lead to a reduction in the 'quality of life' if there is an excessive focusing on symptoms and signs from the organs; in this case the cardiovascular system. To a certain extent, it is possible to assess whether this was a problem by asking about chest pain during normal and brisk walking. In the intervention group, 1.4% of the children reported chest pain during normal walking compared to none in the control group and 2.2 and 0.4% respectively reported chest pain during brisk walking.[18] None of these differences were statistically significant. The participants were also asked how many times they had used different types of health service providers during the last year. Less than 1% of children in both the intervention and control groups had consulted out-patient clinics, physiotherapist or unorthodox health care providers whereas 1.69 and 1.55% respectively had consulted a primary health care facility during the previous 12 months.[18] Increased family tension is another potential problem when using the family approach to intervention. Family members may feel resentment towards the person who is 'the cause' of 'new' foods being introduced. There were few indications that this has been a problem in the present study, but specific surveys must be done to assess this fully. However, if separation/divorce rates in the families are used as an indicator of increased family tension, then there was no difference between the intervention and control families in this study.[18]

Two other potential problems are fanaticism and 'police function'. The definition of fanaticism is quite subjective and is often used in a setting where one tries to avoid making changes (becoming a fanatic). It can also be used as an accusation against persons who honestly try to change parts of their life-style. From the signals received, 1 wife may possibly be characterized as having become fanatic on the issue of diet. Otherwise, if anything, the participants tended to be too lax in their dealings with the advice given. However, there were at least 5 families in which the wives adopted a 'police function' towards the other family members. They would take responsibility for the health behaviour of the husband and children and, thus, start checking on them and become 'nagging', overly protective and maximizing all symptoms from the cardiovascular system.

In this study the effect of intervention among the children/adolescents was small and mainly found in reported dietary habits. Several factors may have contributed to this. Firstly, the intervention strategy focused mainly on the father by offering him new blood tests after 1 year as well as follow-up phone calls. No intervention was specially targeted at the children/adolescents in the families. Thus, it is possible that the children/adolescents have looked on the intervention as something necessary for the father and not relevant to themselves. Second, the lack of effect on smoking and physical exercise may be due to the fact that

the study attempted to intervene in 3 areas simultaneously, but emphasized diet most. And, as others have shown,[1-9] it is difficult to intervene on the life-styles of children/adolescents using the possibility of disease sometime in the future as motivation.

CONCLUSION

Using a family approach to intervention on CHD was well received and led to a change in reported dietary habits, especially for foods commonly eaten at home in a meal setting. There were also fewer children in the intervention group that exceeded pre-set risk factor limits. However, no differences were found in mean risk factor levels between the intervention and control groups. Neither was there any measurable effect of intervention on smoking and physical exercise. When planning an intervention study using the family approach, the use of specific intervention strategies especially targeted at children/adolescents is advised.

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