ORIGINAL ARTICLE

Taylor&Francis healthsciences

Genetic Counselling for Cancer and Risk Perception

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Acta Oncologica Vol. 42, No. 7, pp. 726–734, 2003

The main aim was to investigate risk perception and psychological distress in individuals attending genetic counselling. A consecutive series of 86 individuals with a diagnosis and/or family history of breast, ovarian or colorectal cancer was included. Risk assessments were performed before and immediately after genetic counselling and at a one-year follow-up. Psychological distress was assessed 1 week before, and 6 weeks, 6 months and 1 year after genetic counselling. The number of individuals who correctly estimated the general risk in the population increased significantly from 35% before to 82% after counselling ($p < 0.001$). One year later, data on general risk estimates showed a significant reduction of the number of correct estimations to 51%, compared with directly after the counselling ($p < 0.005$). In total, 54% estimated their own lifetime risk correctly after the counselling, compared with 17% before ($p < 0.001$) (those with a cancer diagnosis estimated the risk of their children developing cancer). One year later, the number of correct estimations had dropped to 28%. Before the counselling, the majority of the participants overestimated both the general risk and their own/children's risk. The participants experienced moderate levels of psychological distress before the counselling and a decrease of anxiety afterwards ($p < 0.02$). However, half of the participants reported moderate or high distress. There were no differences in psychological distress between those who estimated their risk/children's risk as low, moderate or high or between those who over-, under- or correctly estimated their own/children's risk. Further investigations are needed to develop and adjust the risk information provided to the individual in order to avoid misunderstanding, especially as this information is going to be revealed to family members. Counselling support should be offered to those individuals who experience psychological distress.

Received 5 December 2002
Accepted 29 April 2003

The most common cancer diagnoses in the Swedish population are breast cancer among women and prostate cancer among men, with 6 300 and 7 350 new cases in 1999, respectively. Colorectal cancer is the second leading cancer diagnosis for both sexes. More than 5 300 individuals were diagnosed with colorectal cancer in 1999 (1). Genetic predisposition has been detected in approximately 5–10% of patients diagnosed with breast cancer (2). Two major breast cancer susceptibility genes, BRCA1 (3) and BRCA2, (4) have been identified, which has yielded the possibility to assess the likelihood of disease onset for individuals in high-risk families. Two forms of hereditary colorectal cancers have been well described, familial adenomatosis polyposis (FAP), caused by germline mutations in the APC gene (5) and hereditary non-polyposis colorectal cancer (HNPCC), caused by germline mutations in mismatch repair genes (6). Together, these two forms may account for about 5% of the overall incidence of colorectal cancer (7, 8). However, there is a large number of families who fulfil the criteria for hereditary breast and colorectal cancer, but where an inherited mutation cannot be detected in the known susceptibility genes. In the future, additional genes are likely to be identified that cause these forms of hereditary cancer.

The increasing knowledge concerning hereditary cancer syndromes and the identification of cancer susceptibility genes make it possible to provide genetic counselling to individuals with a family history of cancer. One important aspect of the counselling is to estimate the personal risk for developing the cancer in question as well as the risk for children and other relatives. Genetic counselling is a complex process including information not only about the person attending the counselling, but also about other family members and relatives who have never asked for or been interested in receiving this information. During the counselling session, a considerable amount of information is given regarding genetics, for example how genes are passed to subsequent generations, what the risks are of inheriting a mutated gene and what the risk is of developing cancer as a carrier of a mutated cancer susceptibility gene.

 ISSN 0284-186X
DOI: 10.1080/02841860310011023

The risk estimate is usually expressed in percentage and proportions. Several studies have indicated difficulties in communicating risk estimates to women with an increased risk for breast cancer. Lerman et al. (1995) demonstrated that 2/3 of the women grossly overestimated their risk for developing breast cancer and that their risk perception did not improve after counselling (9). In addition, Watson et al. (1999) showed that 45% of women overestimated their own risk for developing breast cancer before counselling and of these, 61% continued to overestimate their risk afterwards (10). However, other studies of risk estimates in women with a family history of breast cancer generally show more under- than overestimation (11, 12).

How well the risk information is understood, the way patients handle the knowledge related to family members, personal compliance and possible worry about an increased cancer risk all vary from person to person. Watson et al. (1999) demonstrated high levels of cancer-specific distress before genetic counselling, with 28% reporting that they worried about breast cancer 'frequently or constantly' and 18% considered this worry 'a severe or definite problem'. Levels of cancer-specific distress were unchanged after genetic counselling. Also, general mental health remained unchanged over time (10). Moreover, Lerman (1993) showed that approximately one-third of the women attending familial breast cancer clinics reported that their daily lives were affected by worries about breast cancer (13). Trask et al. (2001) examined cancer-specific distress in women at increased genetic risk for developing breast or ovarian cancer. About two-thirds of the sample perceived that worries about breast cancer interfered with their daily functioning. Women whose worry interfered with their functioning experienced higher levels of anxiety and confusion (14). Cull et al. (1999) found that women undergoing genetic counselling reported a higher mean trait anxiety score before the counselling (11), compared with women in a general population sample (15), but not significantly different from screening samples (16). After counselling, state anxiety scores were significantly lower than baseline scores for the women in the Cull et al. (1999) study (11).

Collins et al. (2000) investigated the changes in knowledge of familial colorectal cancer (nine items concerning bowel cancer genetics), worry about developing colorectal cancer and perceptions of personal risk in individuals after attendance at a familial colorectal cancer clinic. The number of individuals perceiving their risk as higher, or much higher, than the general population decreased by more than one-third after the genetic counselling. The total knowledge score increased significantly among both men and women after attendance at the genetic clinic. The proportion of persons reporting much worry about developing colorectal cancer decreased significantly from 49% before to 34% after counselling (17).

The aim of the present study is to investigate risk perception and psychological distress in individuals attending genetic counselling. The following specific questions were raised:

1) What estimates of the risk to develop cancer in the general population are stated by participants before, directly after and 1 year after genetic counselling?
2) What estimates of their own risk of developing cancer (those with a cancer diagnosis estimate the risk of their children) are stated by participants before, directly after and 1 year after genetic counselling?
3) What are the proportions of individuals who over-, under- or correctly estimate their risk after counselling and what are the relations between risk estimations and psychological distress?
4) To what extent do participants, 1 year after the genetic counselling, remember the screening programme recommended by the geneticist and to what extent have they talked to their relatives about the information given at the counselling?
5) To what extent do participants experience psychological distress in connection with the genetic counselling and are there changes over time in this respect?

MATERIAL AND METHODS

Participants

A consecutive series of 86 individuals attending genetic counselling at the onco-genetic outpatient clinic at the University Hospital in Uppsala were included between September 1999 and June 2001. All were referred to the clinic by general practitioners or oncologists, because they had a family history of cancer. Inclusion criteria were: at least 18 years old, with a diagnosis and/or family history of breast, ovarian or colorectal cancer, no serious mental illness and ability to complete a questionnaire. Out of the 86 eligible individuals, 77 (89%) accepted participation, 71 women and 6 men. Of those who did not participate (six women and three men), six reported other worries, one had recently gone through surgery and was in a bad physical condition and one did not want to participate because of being neighbour of one of the researchers in the project.

The mean age of the participants was 43 years (range 19–73). Forty-six individuals (60%) had no cancer diagnosis, but a family history of breast cancer (29 women and 2 men), colorectal cancer (7 women and 4 men) or ovarian cancer (4 women). Eight individuals had a known mutation in their family (3 HNPCC, 1 FAP and 4 BRCA1). In total 40% (31/77) of the participants had a cancer diagnosis, i.e. 25 had breast cancer, 4 colorectal cancer and 2 ovarian cancer. All of these were women. Two women received information that

they carried a BRCA1 mutation of their own at the time of genetic counselling; one of them had breast cancer.

Measures

Risk assessment. Participants completed a risk assessment form concerning (a) the risk to develop cancer in the general population, and (b) their own lifetime risk of developing cancer (18, 12). Those who had a cancer diagnosis estimated the lifetime risk of their children of developing cancer. The risk was expressed in terms of 12 fixed gambling odds ratios as well as percentages (e.g. 1/2 or 50%, 1/3 or 35%). There were three more questions in this form: if the individual had spoken to other members of the family about cancer risk, if the individual believed that he/she has an increased risk of developing other cancers and if screening would be helpful. The responses to these questions were given in a yes/no format. A similar form including only risk assessment was used by the geneticist. The risk assessment by the individual was compared with the risk estimated by the geneticist for each individual. The individuals were divided into three groups: those who over- or underestimated their risk and those who provided correct estimates after counselling. To be classified as correct, the participants were allowed to deviate one step above or below the geneticist's risk estimate. Any risk estimation more than one step above/below the risk estimated by the geneticist was defined as over- or underestimation, respectively. However, for the three highest risk figures (35%, 50% and 80%), the participant had to state the same risk figure as the geneticist to be classified as correct.

To investigate the agreement between the individuals' own estimated risk, and the risk figures communicated by the geneticists, the risk estimations were classified according to the Claus model: low ($<20\%$) moderate (20–34%) and high (35–80%) risk (19). The Claus model is based on the number of breast cancer cases among first- and second-degree relatives, the age of family members at disease onset and age of the woman presenting for genetic counselling.

Psychological distress was assessed by the following instruments:

1) A numerical 1–7 scale with the endpoints defined as 'no worry at all' and 'worst possible worry' was developed for this study to assess worry immediately before and after the counselling.
2) The Hospital Anxiety and Depression Scale (HADS) is designed to assess anxiety and depression and consists of two subscales: anxiety (7 items) and depression (7 items). The subscale scores range from 0 (no distress) to 21 (maximum distress) (20). The following categories have been suggested for the subscales: 'no case' (0–7), 'doubtful case' (8–10) and 'clinically significant case' (11–21) for anxiety and depression, respectively. The HAD scale has been validated for use with somatic patients (21).
3) The Impact of Event Scale (IES) was originally developed to assess levels of distress in response to a specific traumatic event (22). A modified version of this questionnaire, previously used in high-risk and general population women, was included to assess psychological responses, with specific reference to thoughts about increased risk of developing cancer (23). It consists of two subscales: 'intrusion' (7 items), scored from 0 (no distress) to 35 (maximum distress), and 'avoidance' (8 items), scored from 0 to 40. The following categories have been suggested: low (0–8), moderate (9–19) and high (20–) degrees of subjective stress for both subscales. The IES has been validated for use with women who are at increased risk for developing hereditary breast cancer (24).

Procedure

The HAD scale was sent by mail to be completed 1 week before the counselling. The risk assessment and the numerical scale for worry were completed immediately before and directly after the counselling. Immediately after the participant left the counselling room, the geneticist completed the risk assessment form. The geneticist estimated the communicated individual lifetime risk for developing the cancer screened for, as well as the individual's worry before and after the counselling. One of the members of the research group was always present during the counselling session and while participants completed their risk assessment form. This gave participants the possibility to ask about any uncertainty as regards the risk assessment form. This was done to avoid incorrect risk estimations caused by misunderstanding. Furthermore, only two geneticists with extensive experience from genetic counselling performed the counselling and all risk estimations of individuals immediately afterwards.

The HADS and the IES were completed 6 months after the counselling and 1 year afterwards. At 1 year, an interview was performed by telephone about screening recommendations given by the geneticist at the counselling and whether the participant had talked to any relatives about the results of the risk assessment (Appendix 1).

Statistical analysis

The χ^2 test was used for group comparisons. In case of statistical dependence between variables, e.g. risk estimations from the same individual on two occasions, the McNemar test was used. Comparisons of mean values between three groups were performed by ANOVA followed by post hoc Fisher LSD testing, and between two groups paired t-tests were used. Agreement of risk estimations between the individual and the geneticist was tested by the Cohen kappa statistic (k) (25). The correlation between risk

estimation by the individual and the geneticist was computed as a Spearman rank correlation coefficient. Comparisons of psychological distress over time between groups of individuals providing under-, over- or correct risk estimations were performed by repeated measures ANOVA. Post hoc testing between and within groups was performed by the Fisher LSD test.

RESULTS

Risk estimate for the general population

To determine over-, under- and correct risk estimates, the population lifetime risk of 8–10% was used for breast cancer, 5% for colorectal cancer and 1.6% for ovarian cancer, respectively.

The proportions of individuals who correctly estimated the general risk in the population to develop the cancers in question (breast, colorectal, or ovarian cancer) increased significantly from 35% (27/76) before to 82% (63/77) after counselling ($\chi^2 = 30.42$, df = 1, $p < 0.001$). Some 58% (44/76) overestimated whereas 7% (5/76) underestimated this risk before counselling. One year later, data on general risk estimates were available from 49 individuals showing a reduction of the number of correct estimations to 51% (25/49), compared with directly after the counselling (82%) ($\chi^2 = 4.76$, df = 1, $p < 0.05$). Before the genetic counselling, a total of 69/76 (91%) reported that they had discussed the cancer risk with their relatives. A total of 51% (38/75) reported that they perceived themselves to be at increased risk for developing other cancers and all but one person thought that screening would be helpful. There was no difference between those individuals who had cancer compared with those who had not in perceiving increased risk of developing other cancers. After counselling, all except one (76/77) stated that they would talk to their relatives about the information given at the counselling. Also, approximately half of the participants (48%) thought that they had an increased risk for developing other cancers. Only two individuals did not think screening would be helpful.

Personal risk estimate

The risk figures estimated by the geneticist showed the following distribution: 22 (29%) of the participants had a risk below 20%, 16 (21%) between 20 and 34% and for 37 (49%) the risk was estimated at 35–80%. Thus, almost half of the participants had a high risk. The participants' own risk estimations after counselling were: 25 (33%) estimated the risk to be below 20%, 9 (12%) between 20 and 34% and 41 (55%) estimated the risk to be 35–80%. Screening programmes for colorectal cancer are recommended at a risk above 10% and for breast cancer at a risk above 20%.

In total, 54% (39/72) estimated their own lifetime risk correctly after the counselling, compared with 17% (12/72) before ($\chi^2 = 17.8$, df = 1, $p < 0.001$). One year later, the number of correct estimations had dropped to 28% (13/46) compared with directly after the counselling ($\chi^2 = 7.64$, df = 1, $p < 0.01$). The correlation between the individual's own perceived risk, assessed before counselling, and the risk figures communicated by the geneticist was not significant (rho = 0.015, n = 72, ns). After counselling, this association was strengthened (rho = 0.68, n = 73, $p < 0.001$) but was somewhat lower one year later (rho = 0.50, n = 46, $p < 0.001$).

The proportions of individuals who under-, over- or correctly estimated their risk at each assessment are presented in Fig. 1.

To investigate the agreement of the participants' own estimated lifetime risk and the risk estimated by the geneticist, Cohen's kappa was used, showing that the agreement was good (k = 0.65) (Table 1).

Nearly half of the participants who overestimated their risk before counselling (41%, 14/34) continued to overestimate their risk 6 weeks after counselling (data not

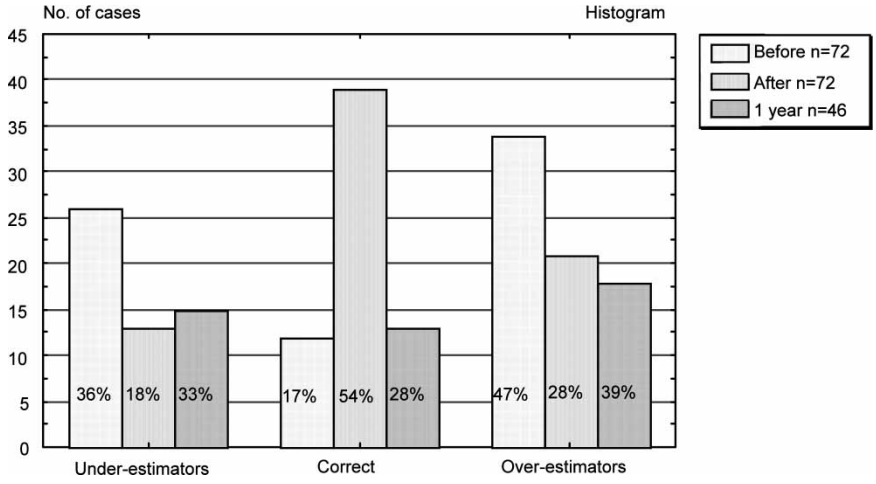

Fig. 1. The proportions of individuals who under-, over- or correctly estimated their risk at each time point.

Table 1

Risk estimations after genetic counselling, assessed by the individuals and the geneticist, classified as low, moderate and high risk (Claus 1992)

	Geneticist			
	Low	Moderate	High	
Individual				
Low	19	3	3	25 (34%)
Moderate	1	7	1	9 (12%)
High	2	6	32	40 (54%)
	22 (30%)	16 (21.5%)	36 (48.5%)	n = 74

k = 0.65.

shown). Interestingly, as many as 28/34 (82%) of the 'over-estimators' thought that their risk was very high (50–80%) and of these, 50% (14/28) continued to overestimate. In total, there were only 12 individuals (16%) for whom the geneticist estimated this high (50–80%) lifetime risk. Of those, 73% estimated their risk correctly after counselling indicating that they understood the communicated high risk. In a comparison of over-estimators between those who had a cancer diagnosis and those who had not the proportions of over-estimators were slightly higher (but not significantly so) among those who had a cancer diagnosis (52%, 14/27) compared with those who did not have cancer (43%, 20/46). After the counselling there were significantly more over-estimators among those who had cancer (13/27) compared with those who had not (7/46) ($\chi^2 = 9.28$, df = 1, $p < 0.01$).

Data from the one-year telephone follow-up were available for 55 individuals. The majority (93%, 51/55) reported that they received recommendations for screening controls for themselves and/or family members. Ten persons were already undergoing screening examinations, 16 individuals could specify both how often and what kind of examination they were undergoing, while 11 were able to report only what kind of examination was recommended but did not remember how often they were supposed to undergo it. Eight individuals reported that their children were supposed to attend controls in the future. Six individuals were recommended routine mammography controls. Altogether, 80% (41/51) had attended at least one examination whereas 9 individuals either were still too young or wanted to wait and see. All but two had the intention of participating in the future.

Breast self-examination was performed regularly by 43% (16/37) while 49% (18/37) did this occasionally but found it difficult, three never performed examinations and four women had had mastectomy performed. There was no difference in performed self-examinations between women who had breast cancer compared with those who had not. Some 13% (7/53) reported that they had changed their lifestyle after the genetic counselling, e.g. taking more regular physical exercise and attempting to reduce stress factors in their life. Nearly half of the participants (47%, 25/53) reported that they had been recommended to inform relevant relatives about the risk and all of those except two had contacted at least one relative. Twelve additional individuals reported that they had contacted at least one relative on their own initiative without recommendations from the geneticist.

Psychological distress

The participants reported a mean value of worry of 3.1 (scale 1–7) before and 2.9 after the genetic counselling. The geneticist's assessments of worry before (4.0) and after (3.3) counselling were significantly higher than those of the individuals' assessments both before ($t = 4.1$, df = 71, $p < 0.001$) and afterwards ($t = 2.2$, df = 71, $p < 0.05$).

The mean values of HAD anxiety and depression and IES intrusion and avoidance at each assessment are given in Table 2. Owing to the small number of individuals who completed the HADS and the IES 1 year after genetic counselling, repeated-measures ANOVAS were based on the remaining assessments only. There were no statistically significant changes over time in any of the variables but anxiety. HADS anxiety decreased significantly 6 weeks after (mean 5.3) compared with before the counselling (mean 6.3) ($F = 4.67$; df = 2,128; $p < 0.02$). The range of responses on the HAD scale was between 0 and 21. About 25% of the participants were classified as doubtful cases/cases of anxiety at all points of assessment and the corresponding proportion for depression was 10%. About half of the participants reported moderate to high avoidance at all points of assessment, while the number of individuals who reported moderate to high intrusion was lower. As the sample represents both men and women, which possibly could affect the anxiety ratings, we also performed an analysis with the six men excluded. However, the findings were nearly the same and the anxiety still decreased significantly 6 weeks after the counselling (mean 5.6) compared with before (mean 6.6), ($F = 4.19$; df = 2,122; $p < 0.02$).

HAD anxiety (mean 5.0) at one year was lower as compared with before counselling (mean 6.3) ($t = 2.45$; df = 47; $p < 0.02$). HAD depression at the one-year assessment (mean 2.7) did not differ from that performed before the counselling (mean 3.5). No significant changes of IES intrusion or avoidance were detected at one year compared with 6 weeks after counselling (data not shown).

A one-way ANOVA was performed to examine whether there were any relations between the individuals' own risk perception after the counselling, according to the Claus model (low, moderate and high risk) and psychological distress (HADS and IES). There were no significant differences between these groups. Also, no significant correlation was found between the risk estimated by the

Table 2

Means and SDs of HAD anxiety and depression and the number of individuals classified as cases, doubtful cases and non-cases two weeks before (T1), and 6 weeks (T2), 6 months (T3) and 1 year (T4) after genetic counselling, and mean scores of IES intrusion and avoidance and the number of individuals classified as having a low, moderate or high degree of subjective stress measured at 6 weeks (T2), 6 months (T3) and 1 year (T4) after the counselling

	T1 (n = 74)	T2 (n = 75)	T3 (n = 69)	F (df)	T4 (n = 49)
HAD					
Anxiety					
Mean	6.3	5.3*	5.7	4.67*(2 128)	5.0
SD	5.0	4.6	4.4		4.2
No case	50 (68%)	53 (71%)	53 (77%)		35 (71%)
Doubtful case	11 (15%)	14 (19%)	5 (7%)		11 (23%)
Case	13 (17%)	8 (10%)	11 (16%)		3 (6%)
Depression					
Mean	3.5	2.9	3.4	n.s.	2.7
SD	3.5	3.2	3.7		3.4
No case	65 (88%)	69 (92%)	58 (84%)		43 (88%)
Doubtful case	6 (8%)	4 (5%)	7 (10%)		5 (10%)
Case	3 (4%)	2 (3%)	4 (6%)		1 (2%)
		T2 (n = 55)	T3 (n = 54)	F (df)	T4 (n = 49)
IES[a]					
Intrusion					
Mean		9.6	8.8	n.s.	9.0
SD		8.0	7.6		8.7
Low		28 (51%)	31 (57%)		31 (62%)
Moderate		22 (40%)	16 (30%)		13 (26%)
High		5 (9%)	7 (13%)		6 (12%)
Avoidance					
Mean		9.1	9.0	n.s.	10.8
SD		7.6	7.6		9.0
Low		29 (53%)	27 (50%)		23 (40%)
Moderate		19 (34%)	21 (39%)		20 (40%)
High		7 (13%)	6 (11%)		7 (14%)

* = $p < 0.02$.
[a] The IES was not completed by the last 20 participants due to an administration failure.
The mean values at T4 are not included in the analysis of variance.

geneticists and HAD anxiety and depression. The participants who perceived their risk as high reported slightly higher mean values of both intrusion and avoidance throughout than did those who perceived their risk as low or moderate (data not shown).

Those individuals who correctly estimated their own risk after counselling reported slightly higher HAD anxiety and depression at all times of assessment than those who over- or under-estimated their own risk, but not significantly so ($p = 0.06$). However, for intrusion and avoidance, higher mean values (not statistically significant) were reported by those who underestimated their risk compared with those who did correct estimations or overestimated their risk.

DISCUSSION

The number of individuals who correctly estimated the general risk in the population of developing cancer (breast, ovarian or colorectal cancer) increased significantly from 35% (before) to 82% after the counselling. One year later, data from 49 individuals were available, showing a decrease to 51% in the number of persons with correct estimates. Watson and co-workers (1999) found a lower proportion of individuals who correctly estimated the general risk (24% before, 62% after and 52% at one year) (10). However, the pattern of an increase after the counselling and a decrease at one year was the same. A probable explanation for the higher proportions of participants with correct risk estimations in the present study is that 40% of our participants had a cancer diagnosis. They may therefore have received information about cancer at earlier appointments at the hospital. In the study by Watson et al. (1999), all of the participants had a family history of breast cancer but were not clinically affected by cancer (10). A study by Evans (1994) showed an even lower proportion of persons (33%) who correctly estimated the population risk one year after genetic counselling and only 16% did so before (12).

In the present study, the population risk before the counselling was overestimated by 58% of the participants whereas only 7% underestimated this risk. This is in

contrast with the women in the Watson et al. (1999) study who underestimated and overestimated in roughly similar proportions (40% and 63%) (10). Burke et al. (2000) found that participants overestimated both their personal risk of breast cancer and that of the average woman before genetic counselling, and that they continued to overestimate after counselling but to a lesser degree (26). The present results indicate that it is more difficult to understand, or recall, the information about one's own risk compared with that of the population, since 46% of the participants were not able to give the correct risk figures after the counselling as compared with 18% for the general risk. One year later, as many as 72% of the participants were unable to make the correct estimations of individual risk figures. There were slightly more individuals who overestimated (47%) than underestimated (36%) their own risk before the counselling. This is similar to results on the perceived risk of prostate cancer among men with a family history of this type of cancer (27). In contrast, a study performed by Cull et al. (1999) revealed that more women underestimated (39%) than overestimated (14%) their own risk for developing breast cancer before genetic counselling (11). In the present study, as many as 82% of the over-estimators thought that they had a very high risk (50–80%) of developing cancer before the counselling, and 50% of those continued to overestimate to same extent after the counselling (cancer patients estimated the risk of their children). However, such very high (50–80%) risk estimates were given by the geneticist for only 12 individuals. As many as 73% in this subgroup estimated their risk correctly after the genetic counselling and seem to have understood that they or their children have this very high risk. This is most important for motivating these individuals and their children to participate in future screening programmes.

It is hazardous to compare studies of risk estimations as the number of individuals who correctly estimate their own risk may vary with the criteria for assessing 'correct' estimation and the risk assessment forms used. In the Cull et al. (1999) study, the participants were classified as 'under-estimators' by a factor of 0.5 or less, the 'over-estimators' by a factor of 2 or higher and 'close estimators' were women who fell between these limits, 47% (11). This represents more generous criteria than in the present study. A study performed by Rothemund, Paepke and Flor (2001) revealed that 48% of the women overestimated their own risk while 39% were correct after the counselling. In that study ' correct' was defined as an estimation within 50% of the counselled risk (28).

One of the aims of genetic counselling is to provide individual screening programmes for those individuals who are found to have an increased risk for developing cancer. It is most important to provide risk information in a comprehensible way, since this may motivate participation in screening programmes. Likewise, individuals who are found to have no increased risk will benefit from the reassurance that they are not at as high a risk as they may have thought and therefore do not have to undergo additional control programmes in the future. One year after the counselling, the majority (93%) of the present participants reported that they had received recommendations for screening programmes for themselves and/or family members and of those, 80% had attended at least once during the past year. All but two had the intention of participating in future examinations. Several studies have revealed that participation in recommended screening programmes for colorectal cancer is low, even in relatives with a higher risk than the normal population of developing cancer (29–31). On the other hand, compliance is high with mammography for individuals with an increased risk for breast cancer (12). Breast self-examination was performed regular by 43% of the women; 49% found breast examination difficult and performed such examinations occasionally. Of those who were encouraged to talk to relatives (47%), almost everyone did so. However, no information is available about whether if the information given to the relatives was optimal. With such a high proportion of participants who did not understand their risk, the information (risk figures) given may be incorrect. This highlights the importance of improving the information given at genetic counselling.

Psychological distress

Worry before and after the counselling was reported by the participants to be low, while the geneticists estimated it to be higher. Similar results have been reported earlier demonstrating that nursing staff/physicians overestimate the worry experienced by their patients as compared with patients' own reports (32, 33). In the present study, we found no increase in worry before compared with after the counselling.

There were no significant changes over time in psychological distress, except for HAD anxiety, which decreased 6 weeks after compared with before the counselling. Also, HAD anxiety at 1 year was lower compared with before the counselling. This is in accordance with results from Cull et al. (1999), who found that women experienced a decrease in anxiety after compared with before the counselling (11). Watson (1999) found no statistically significant change in general mental health at follow-up compared with the pre-genetic level. However, a significant decrease of state anxiety was found after the counselling compared with before (10).

There are several possible explanations for the decrease in anxiety after the counselling, such as relief afforded by receiving risk information irrespective of whether the risk is high or not. For some individuals, the risk perceived before the counselling was found to be lower than they expected, which may have contributed to a reduction of anxiety afterwards. Although the mean value for HAD anxiety after

the counselling was rather low (5.3), about 25% of the participants were classified as doubtful cases/cases of anxiety at all points of assessment. This indicates that some individuals may need some kind of psychological support. This level of anxiety is about the same as that demonstrated in non-cured gastrointestinal cancer patients (mean 4.9) attending medical follow-up control visits (35). In a group of 517 women recalled for further examination after attending mammography screening, 27% scored as doubtful cases of anxiety (36). Thus, the level of anxiety at genetic counselling is similar to that in non-cured GI cancer patients and in women recalled for further examination after mammography screening.

Even though the mean values for both avoidance and intrusion were moderate at all points of assessment, about half of the participants reported moderate or high avoidance at all points in time (34). Some other studies have revealed lower levels of cancer-specific distress (using the IES) than in the present study (27, 37), while Watson et al. (1999) reported values similar to the present results (10).

We found no relationship between participants' own risk perception (low, moderate or high risk according to Claus (1992) (19) and the experience of psychological distress (HADS and IES). Other factors than the individual's own risk perception are likely to influence psychological distress, such as personality, coping styles and disposition for optimism and pessimism. Further investigations are needed to explore this.

In addition, there were no significant differences with regard to psychological distress after the counselling between participants who over-, under- or correctly estimated their own risk. However, individuals who correctly estimated their own risk after counselling reported slightly higher mean values of HAD anxiety and depression at all times than did those who over- or underestimated their risk. This is in contrast with Watson et al. (1999), who demonstrated that women who overestimated their risk after the counselling reported significantly higher cancer-specific distress (as measured by the IES) both before genetic counselling and at a one-year follow-up (10). The present IES data do not demonstrate this pattern. There are several differences between the two studies. In the Watson et al. (1999) study, all of the participants were women, none was clinically affected by cancer and 46% of the women had a risk higher than 20% (10). The present sample is heterogeneous with respect to possible cancer diagnoses and more of the participants (70%) had a risk higher than 20%. Thus, the experience of cancer was more common among the present participants, since 40% of them had a cancer diagnosis, which may have made them more aware of a possible high risk. Furthermore, the proportion of overestimators who continued to overestimate their risk after the genetic counselling was higher (61%) in the Watson et al. (1999) study as compared with the present study, where 41% continued to overestimate their risk afterwards (10). Further research is needed to clarify this issue.

The genetic counselling increased participants' ability to correctly estimate the population risk and their own risk for developing cancer. The majority of the participants overestimated both the general risk and their own risk before the counselling. The participants experienced moderate levels of psychological distress before the counselling and a decrease of anxiety afterwards. However, half of the participants reported moderate or high distress, which needs to be attended to. There were no differences in psychological distress between those who estimated their risk as low, moderate or high or between those who over-, under- or correctly estimated their own risk. It is important to develop and adjust the risk information provided to the individual in order to avoid misunderstanding, especially as this information is going to be revealed to family members. Those individuals who experience psychological distress should be offered counselling support.

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APPENDIX 1

- Questions asked during telephone follow-up one year after genetic counselling.
- Did you get any recommendations at genetic counselling?
- If control-programme—have you been to any examination yet?
- Are you going to participate in these examinations again?
- Own breast examination?
- Habit change during the last year?
- Encouraged by geneticist to inform any relatives?
- Have you contacted these persons?