ISSN: 0976-3031

Available Online at http://www.recentscientific.com

CODEN: IJRSFP (USA)

International Journal of Recent Scientific Research
Vol. 9, Issue, 2(B), pp. 23815-23821, February, 2018

International Journal of Recent Scientific Research

DOI: 10.24327/IJRSR

Research Article

SOCIODEMOGRAPHIC, ANTHROPOMETRIC AND MEDICAL RISK FACTORS FOR COLORECTAL CANCER IN A MOROCCAN POPULATION CASE–CONTROL STUDY

Fatima Ezzahra IMAD[1]., Houda DRISSI[1]., Karima BENDAHHOU[3]., Nezha TAWFIQ[2]., Nadia TAHIRI JOUTI[4]., Abdellatif Benider[2] and Driss RADALLAH[1]

[1]Laboratoire de Biologie et santé, unité de recherche associée CNRST, URAC-34, Faculté des Sciences Ben M'sik Université Hassan II de Casablanca, Maroc
[2]Centre Mohamed VI Pour le traitement des cancers, CHU Ibn Rochd , Faculté de médecine et de Pharmacie, Université Hassan II, Casablanca, Maroc
[3]Registre Des Cancers de la région du grand Casablanca, Maroc
[4]Faculté de médecine et de Pharmacie, Université Hassan II, Casablanca, Maroc

DOI: http://dx.doi.org/10.24327/ijrsr.2018.0902.1542

ARTICLE INFO

Article History:
Received 26th November, 2017
Received in revised form 1st December, 2017
Accepted 15th January, 2018
Published online 28th February, 2018

Key Words:
Colorectal cancer, risk factors, aspirin, obesity, smoking; physical activity

ABSTRACT

Colorectal cancer (CRC) also known as colon cancer or rectal cancer, is the third most common cancer in the world, and is among the main causes of death related to cancer. Several risk factors for colorectal cancer have been clearly identified, but challenges remain in relation to the etiology of this disease. The aim of this work is to study risk factors related to colorectal cancer (Inactivity, diabetes, obesity, oral contraception, aspirin, tobacco and alcohol) are all risk factors or protective in patients treated at Mohammed VI center for cancers treatment in Casablanca during 2015-2016. This is a Case-Control Study that took two years, it includes colorectal cancer cases treated at the center, compared to controls that are not suffering from any cancerous disease. The statistical analysis of the results was carried out by the software R.

225 patients were treated in our center compared to 225 controls. Cases and controls were paired by age and sex. The average age of the patients was 55.49 ± 14.06 years, 119 men (52.9%) and 106 women (47.1%). The family history of colorectal cancer was found in 11,11% of the cases*vs*1,33% of the controls with a significant difference (p = 0.0001). As for the medical antecedents in our patients, Taking aspirin was found in 4, 89% of the cases *vs*12.44% of the controls; (p= 0.003; OR=0.36).The analysis of toxic habits reveals that 50.7% of the patients are smokers *vs*28.5 of the controls. This difference was highly significant (p = 0.001, OR = 2.62). Alcoholic patients accounted for 11.55% of the cases, against 1.33% of the controls. (P = 0.0001, OR = 10.81).

Moreover, the results of our study allow to conclude an association between sport activity and the reduction of the risk of colorectal cancer, the practice of medium intensity or high intensity sport activities was lower in patients with respectively 3% of the cases *vs* 12% of the controls (P = 0.01) and 6% of the cases vs 29% of the controls (P = 0.0001).

In the light of this study and examined in the literature, it is recommended that important steps be taken to combat illiteracy and promote education. As well as raising awareness among the general public about risk factors for colorectal cancer. The fight against certain risk factors on which action can be taken and effective integrated prevention that promotes healthy eating, physical activity and the fight against alcohol consumption, overweight and obesity could have an impact and reduce the incidence of colorectal cancer in the long term.

INTRODUCTION

Worldwide, Colorectal cancer (CRC) is the third cancer in terms of incidence and mortality, for men (663000 cases), and women (571000 cases) (Ferlay *et al*., 2013). It is the fourth cause of death by cancer in the world. (8% of death by cancer). In Morocco, the incidence of this cancer is low compared to developed countries, however, this incidence is constantly

Corresponding author:* **Fatima Ezzahra IMAD
Laboratoire de Biologie et santé, unité de recherche associée CNRST, URAC-34, Faculté des Sciences Ben M'sik Université Hassan II de Casablanca, Maroc

growing as showed by the evolution of death by this cancer in Casablanca region, which increased from 7.3 cases/100,000 inhabitants in 2004-2007 to 9.6 cases/1000000 inhabitants in 2008-2012 for both sexes combined. It became the first digestive cancer, outclassing stomach cancer. (RCGC, 2017).

Although, the etiology of this disease highly lethal remains uncertain, the identification of risk factors for colorectal cancers is of paramount importance to develop recommendations for prevention.

PATIENTS AND METHODS

Type of study

This is a case-control study conducted at Mohammed VI center for cancers treatment in Casablanca at the IBN ROCHD Hospital Center.

Study population

We have included in our study consecutively all patients with colorectal cancer histologically confirmed, who were treated at the center from January 2015 to January 2017. The same number of controls free from any cancer disease was included among patients admitted to the diagnostic center dermatology and ophthalmology of CHU Ibn Rochd Casablanca.

The cases were matched with controls in age and sex, using age at diagnosis for cases and age at interview for controls.

Data collection was done prospectively by a standardized questionnaire administered face-to-face and supplemented from the medical record for certain clinical variables. Each patient interviewed was asked to complete an informed consent form.

The data collected concerned:

- Sociodemographic data: age, intellectual level, area of residence, socio-economic level, occupation, health insurance.
- Personal medical history: oral contraceptive use, aspirin use, oral antidiabetic use.
- MICI's personal history: Crohn's disease, Hemorrhagic retinitis
- A history of genetic predisposition to colorectal cancer: PAF, lunch syndrome.
- Family history of cancer: degree of relationship, type of cancer, location, and treatment received
- Lifestyle: physical activity, tobacco, alcohol, ...

Statistical analyzes were carried out using software R. The Chi² test was used for group comparison. The threshold of significance retained is $p < 0.05$. We presented the Odds ratios for the different risk factors in the cases and controls.

RESULTS

We recruited a total of 225 patients presenting for colorectal cancer at Mohamed VI Center for Cancer Treatment and 225 controls free from any cancer disease during the study period.

The average age of the patients was 55.49 ± 14.06 years, with extremes ranging from 17 to 90 years, 119 men (52.9%) and 106 women (47.1%). There was a male predominance with a sex ratio of 1.12. 77.78% of cases were married at the time of diagnosis. The analysis of marital status did not find any significant difference between the two groups (Table 1).

According to the place of residence, 75.11% of the cases reside in an urban environment *vs* 83.11% of the controls and 20.8% of the cases reside in a rural environment *vs* 12% of the controls with a significant difference (p = 0. 03).

The level of education globally shows a low intellectual level in cases of colorectal cancer compared to controls. 50% of patients were an alphabetics *vs* 29.78% of controls with p = 0.001 (Table 1).

The notion of a family history of colorectal cancer was found in 11.11% of cases *vs*1.33% of controls with a significant difference (p = 0.001).

For cases, one patient had Crohn's disease, and 7 patients had sporadic polyps (5.88%), of which one patient had familial adenomatous polyposis PAF (0.84%).

Table 1 Demographic risk factorsand familiy history ofthe colorectal cancer patients and controls

	Cases(%)	Controls(%)	P valu
Average age ± standard deviation	55 ,49 ± 14,06		
Marital status			P = 0.89
Singles	25(11.11)	32(14.22)	
Married	175(77.78)	160(71.11)	
Widowers	4(1.78)	11(4.89)	
Divorced	21(9.33)	22(9.78)	
Level of studies			P = 0.0001
Analphabetics	107(47.56)	67(29.78)	
Coranic school	22(9.78)	15(6.67)	
Primary	45(20)	38(16.89)	
College	38(16,89)	55(24.44)	
Highschool	13(5.78)	50(22.22)	
Place of residence			P = 0.03
Urbain	169(75.11)	187(83.11)	
Suburban	9(4)	11(4.89)	
Rural	47(20.89)	27(12)	
Profession			P = 0.01
Physical activity	166(73.77)	131(58.22)	
Intellectuel activity	59(26.23)	94(41.78)	
Personal polyps			----
Yes	13(5.78)	0(0)	
No	212(94.2)	225(100)	
Family history of colorectal cancer			P = 0.0001
Yes	25(11.11)	3(1.33)	
No	200(88.89)	222(98.67)	
Family history of other types of cancer			P = 0.0001
Yes	39(17.33)	12(5.33)	
No	186(82.67)	213(94.67)	
Personal IBD			------
Yes	2(0.89)	0(0)	
No	223(99.11)	225(100)	
Family FAP			------
Yes	1(0.44)	0(0)	
No	224(99.56)	225(100)	

The use of oral contraceptives was found in 34.91% of the cases *vs* 37.74 of the controls (table 2).

Diabetes associated with colorectal cancer was found in 14.67%of the patients compared with 11.56% in controls. However, these results are not significant (p = 0.20). (Table2).

Taking aspirin was found in 4.89% % of the cases *vs*12.44% of controls with a significant difference (p= 0.003) (Table 2).

The recognized harmfulness of tobacco is found in 28% of the cases in our study *vs*15.11% of the controls. This difference was highly significant (p = 0.0001).

Alcohol patients accounted for 11.55% of the cases, compared with 1.33% of controls. These results indicate a significant association between alcohol and the occurrence of colorectal cancer (P = 0.0001).

The analysis of the body mass index (BMI) overall, found overweight in 36.89% % of the patients *vs*32.45% of the controls and Obesity in 28% of the patients *vs*23.11% of the controls without the difference being significant.

Table 2 BMI, toxic habits and medical history of patients and controls

	Cases (%)	Controls (%)	P Valu
Diabete			P = 0.20
Yes	26(11.56)	33(14.67)	
No	199(88.44)	192(85.33)	
Aspirin			P = 0.003
Yes	11(4.89)	28(12.44)	
No	214(95.11)	197(87.56)	
oral contraceptives			P =0.35
Yes	37(16.44)	40(17.78)	
No	188(83.56)	185(82.22)	
Smoking			P = 0.001
Non-smoker	161(71.56)	191(84,89)	
Smoker	16(7.11)	12(5,33)	
Ex-smoker	47(20.89)	22(9,78)	
Alcohol			P =0.0001
Yes	26(11.55)	3(1,33)	
No	199(88.44)	222(98,66)	
BMI			P =0.09
normal	79(35.11)	100(44.44)	
Overweight	83(36.89)	73(32.45)	
Obesity	63(28)	52(23.11)	

In our study, the practice of sports medium intensity or high intensity was lower in patients respectively 4% *vs*19.11% of controls (P = 0.001) and 3.56% of the cases *vs*8.44% % of controls (P = 0.02).

Table 3 Frequency of physical activity in patients and controls

	Cases(%)	Controls(%)	P Valu
High-intensity physical activities (work)			P = 0,01
Yes	88(39.12)	60(26.66)	
No	137(60.88)	165(73.33)	
Physical activity of moderate intensity (work)			P = 0.13
Yes	143(63.56)	155(68,87)	
No	82(36.44)	70(31,11)	
Walk at least 10 minutes in a row			P = 0.27
Yes	180(80)	186(82.67)	
No	45(20)	39(17.33)	
Sport of high Intensity			P = 0.02
Yes	8(3,56)	19(8.44)	
No	217(96.44)	206(91.56)	
Sport of moderate intensity			P = 0.001
Yes	9(4)	43(19.11)	
No	216(96)	182(80.89)	

They appear to be correlated with a lower risk of colorectal cancer. And paradoxically, the trade or predominates the high intensity physical work is correlated with a higher risk of colorectal cancer in our study since it was found in 39.12% of the cases compared with 26.66% of the controls (p = 0.01).

Univariate analysis for the different risk factors in cases and controls stratified by sex concluded that no association was found with marital status.

On the other hand, the Odds ratio for the level of study was significant. It was 0.33 (95% CI, 0.22-0.50).Any association is established between diabetes and colorectal cancer.

On the other hand, a protective association between aspirin and the risk of colorectal cancer was found in our series (OR = 0.36) Our results show the strong association between toxic habits and CCR, these results suggest that a smoking patient is twice as likely to have a CRC than a non-smoking patient (OR = 2.62). An alcoholic patient is 9 times more likely to develop colorectal cancer (Table 4).

Table 4 Odds Ratio for colorectal cancer of different risk factors

	OR	95% IC
Marital status		
Married		
Single, widowed, divorced	0.70	(0.46-1.07)
Level of studies		
Analphabetics, Coranic school, Primary		
College , Highschool	0.33	(0.22-0.50)
Placeof residence		
Urban		
Suburban, Rural	1.63	(1.03-2.59)
Diabete		
Yes		
No	0.76	(0.44-1.31)
Aspirin		
Yes		
No	0.36	(0.17-0.75)
Oral contraceptives		
Yes		
No	1.13	(0.69-1.86)
Smoking		
Non- smoker		
Smoker, Ex-smoker	2.18	(1.37-3.48)
Alcohol		
Yes		
Non	9.66	(2.88-32.43)

Table 5 summarizes the results of the analyzes of the different risk factors according to the location of colorectal tumors. However, rectal cancers (n = 114) were more frequent, followed by those on the left colon (n = 74) and then on the right colon (n = 37).

The distribution of colorectal cancer by sex shows a slight predominance of the left colon cancer in women (55.4%) compared to the right colon cancer (43.2%), rectal cancer is more common in men (57%).

Otherwise, the family history of non-colorectal cancers was present in 29.7% of cases in the right colon; 17.9% in the left colon and 13.2% in the rectum with a significant difference (P = 0.02).

In diabetic patients, the right colon cancer came first in 16.2% followed by the left colon in 13.5% of cases and the rectum last in 8.8% of cases.

In our series, smoking patients had rectal cancer in 30.7% of the cases, a right colon cancer in 27.1% of the cases and a left colon cancer in 24.3% of the cases without the difference being significant (p = 0.4).

For right colon cancer, 21.6% of the cases were alcoholic *vs*12.2% for left colon cancer *vs*7.9% for the rectum with a significant difference (P = 0.02).

Table 5 Distribution of the different risk factors according to the location of the tumor

	Rectum (n=114)	Left colon (n=74)	Right colon (n=37)	P valu
Average age ± ecart type	54 .70 ± 13.48	56 ± 14.32	59 ± 14.57	P=0.2
Sex				P=0.5
Men	65(57)	33(44.6)	21(56.8)	
Women	49(43)	41(55.4)	16(43.2)	
Family history of colorectal cancer				P=0.1
Yes	9(7.9)	11(14,9)	5(13.5)	
No	105(92,1)	63(85,1)	32(86.5)	
Family history of another type of cancer				P=0.02
Yes	15(13.2)	13(17.6)	11(29.7)	
No	99(86.8)	61(82.4)	26(70.3)	
Diabetic				P=0.1
Yes	10(8.8)	10(13.5)	6(16.2)	
No	104(91.2)	64(86.5)	31(83.8)	
Aspirin				P=0.01
Yes	1(0.9)	7(9.5)	3(8.1)	
No	113(99.1)	67(90.5)	34(81.1)	
oral contraceptives				P=0.7
Yes	22(19.3)	11(14.9)	7(18.9)	
No	92(80.7)	63(85.1)	30(81.1)	
Smoking Statut				P=0.47
Non- smoker	79(69.3)	56(75.7)	27(72.9)	
Smoker , Ex-smoker	35(30.7)	18(24.3)	10(27.1)	
Alcohol				P=0.02
Yes	9(7.9)	9(12.2)	8(21.6)	
No	105(92.1)	65(87.8)	29(78.4)	
BMI				P=0,8
normal	40(35.1)	18(24.3)	14(37,8)	
overweight	40(35.1)	34(45.9)	14(37.8)	
Obese	34(29.8)	22(29.7)	9(24.3)	

DISCUSSION

Our study identified risk factors and protective factors likely to be related to the occurrence of the CRC among the Moroccan population. The strong point of our study is the prospective character with direct interview with patients and controls. The weak point of our study is the retrospective search for risk factors and protective factors in relation to colorectal cancer. The answers of patients with cancer may be biased by culpability related to cancer and may overestimate or underestimate exposure to certain factors known by the general population.

The average age of our series is comparable to the statistics of Ayoujil (Ayoujil A.2014) with an average age of 54.2 years and that of Mrini with an average age of 55 years (Mrini, K.2009).On the other hand, our population is younger than that studied by Dreyer, where the average age is 66 years old (Dreyer, C, 2016).This difference can be explained by the young age of our population as shows the data from the cancer registry of the Greater Casablanca region or subjects under 40 represent 70% of the Moroccan population in 2010. (RCGC, 2016).

Average age at diagnosis in patients with right colon cancer, left colon and rectum is consistent with the literature or colon cancer is diagnosed at a later age compared to the rectum (Cerbelaud C. 2008; Lahmidani N, 2011). Classically, the left cancers are of precocious revelation (violent pains, emission of mucus and blood), while the right cancers are longer silent, with no sign of appeal, which explains a later treatment (Cerbelaud C, 2008; Lahmidani N, 2011).This is partly related to the diameter of the colon which is wider on the rightwith a maximum at the coecum and decreasing at the level of the left colon.

Concerning the sex ratio, the male predominance noted in our series was found in the series of Meddah (Meddah, 2009) as well as that of Kudjawu (Kudjawu Y, 2015) where the sex ratio was 1.22.The sex ratio in the colon cancer is (0.95). In rectal cancer, the sex ratio of men/ women is 1.32. In the Western literature, the sex ratio is between 0.8 and 1.4 for colon cancer and between 1.5 and 1.6 for rectal cancer, (Cohidon C, 2009).The sex ratio by location is in the range of literature for the colon. For rectal cancer, the male predominance is lower than that reported in the literature. This distinction between men and women can only be explained by hormonal variations, eating habits, smoking, alcohol, etc.

Poomphakwaen said that, there is a protective association between the level of education and the risk of having colorectal cancer with a OR of 0.59 and a P <0.05 (Poomphakwaen K, 2015). This association was found also in our study.

Several risk factors for colorectal cancer have been clearly identified; Family history of colorectal cancer increases the risk almost three times. Genetic factors associated with familial adenomatous polyposis (APC gene mutation) and Lynch syndrome (HNPCC, characterized by anomalies on genes (MSH2, MSH6 and MLH1) coding for DNA repair proteins) are among the most important risk factors for colorectal cancer. Risk is also increased in people with Chronic inflammatory bowel disease (IBD) such as Crohn's disease and ulcerative colitis (Buecher B,2012).

In our study, the percentage of patients with a family history of colorectal cancer is lower than that reported in the literature, which estimates this proportion to be 15% -20% (viguier *et al*, 2003). This low percentage can be explained by the lack of screening forms with genetic predisposition to colorectal cancer and especially Lynch syndrome.

The family history of colorectal cancer was associated with a greater risk increase in colon tumors compared to rectal cancer and particularly the right colon. Our results are therefore consistent with those of the literature, and reinforce the hypothesis of a genetic predisposition to colorectal not documented by genetic analyzes such as Lynch syndrome, which is more difficult to detect because of the absence of polyps in the colon. This hypothesis deserves to be studied especially as consanguinity in marriage authorized by the Muslim religion is common in our population. The meta-analysis on colorectal cancers also observed stronger associations on the colon than on the rectum (Johns LE,2001;Butterworth AS, 2006; Baglietto L, 2006), no difference between the right and left colon (Johns LE, 2001).

In the case of toxic habits, tobacco consumption promotes early onset of CRC and generates mutations at the DNA level of the

digestive mucous. This association found in our series joins the results of the literature where most studies confirm the increased risk of colorectal cancer as a function of tobacco consumption. (Lüchtenborg *et al*, 2007; Huxley *et al*, 2009; Tsoi *et al*, 2009) and that smokers had 8% of the increased risk of colic cancer compared to those who had never smoked (Parajuli *et al*, 2013). Our Odds ratio is consistent with a meta-analysis included 36 prospective studies (3 million subjects) or the OR was between 1.08 and 1.44 (Liang *et al*, 2009).

The association appeared to be stronger for rectal cancer compared with colon cancer (Jorgensen OD, 2002). For colon cancer, the association with tobacco is stronger with the right localization compared to the left localization found in our series and that of Sharpe and Akhter (Sharpe CR,2002;Akhter M,2007).

Alcohol is a known carcinogen which may be associated with colorectal cancer by lowering the levels of folic acid that helps prevent the transformation of normal colon cells into cancer cells. (Troche *et al*, 2016).

Our results are consistent with recent epidemiological surveys showing a positive association between colorectal cancer and alcohol consumption (Fedirko *et al*, 2011; Moskal *et al*, 2007; Steinmetz *et al*, 2007), as well as data from meta-analysis of Benoit on 27 studies (Benoit F, 2004).

In addition, a protective association between Aspirin and the risk of colorectal cancer has been found in several studies. Cao Y *et al*. demonstrated a 15% decrease (RR = 0.85, 95% CI 0.8 - 0.91) of the risk of colorectal cancer associated with regular aspirin (at least twice a week), compared to people who do not take it (Cao Y *et al*, 2016). This protective effect of aspirin is related to the selective inhibition of cyclooxygenase type 2 by preventing production of prostaglandins (Fink SP *et al*, 2014). This association was also found in our study.

Although oral contraception provides protection against CRC, related to the impact of estrogen on the synthesis and excretion of bile and, more specifically, the decrease in the concentration of bile acids in the colon. (Boufettal H, 2010). This association was not found in our study and also in a meta-analysis which concluded that the systematic use of CO was not associated with colorectal cancer. However, it has been shown that longer durations of CO (+ 5 years) were associated with a lower risk of proximal cancers, but no distal or rectal cancers (Charlton BM, 2015). This can also be explained by the different oral contraceptives used.

In addition, type 2 diabetes is associated with a moderate increase in the risk of colorectal cancer (Peeters *et al*, 2015). Hyperglycemia could promote tumor progression because of its intracellular metabolic activity and membrane transport of glucose more important in cancer cells. (Gariani *et al*, 2010). This association was not found in our population; this difference can be explained by the low sample studied in this subgroup of our population.

The link between colorectal cancer and obesity has already been mentioned in several epidemiological studies. The risk of colorectal cancer is increased by 41% in obese people suggesting a pernicious association with obesity (Kantor ED, 2015).Several explanations have been suggested for the association of a higher Body mass index (BMI) with the CRC, These include negative regulation of adiponectin; the upregulation of leptin, interleukin-6 and tumor necrosis factor alpha; increased insulin and insulin-like growth factor 1;and an altered immune response (LeviZ, 2017).

Several researchers have shown that the risk increases with BMI. Similarly, an obese person has a 33% higher risk of having a CRC than a person with a normal BMI. This significant association was established between obesity and colorectal cancer in our series.

Since 2007, at least 5 meta-analysis have been published on this subject (Moghaddam AA, 2007, Renehan AG, 2008) with conclusions similar to the report of the World Cancer Research Fund (WCRF).However, one can modulate these results according to the localization of the tumor. Indeed, in all these studies, the risk associated with obesity or overweight is higher in colon cancers than the rectal cancers.

Furthermore, two of these meta-analysis presented site-specific estimates within the colon, but did not find of the difference between the estimates for the right colon and the left colon (Harriss DJ, 2009; Larsson SC,2011).

In addition, colorectal cancer represents the cancer on which there is the most evidence of the beneficial effect of physical activity *via* the acceleration of intestinal transit (INCA, 2012).
In our study, the practice of sport was lower in patients than on controls, it appears to be correlated with a decreased risk of colorectal cancer.

This result is inconsistent with a recent meta-analysis of 52 studies having shown a 20% to 30% decrease in risk of colon cancer in physically active individuals. (Wolin KY, 2009).Studies show a protective effect of physical activity, with a decreased risk of 40-50% (Simons, 2013; Boyle, 2012).

Our results have evoked that high-intensity physical activity during labor is associated to a higher risk of colorectal cancer. This pushes us to ask us about the nature of the professional activity of these patients that was not evaluated on the questionnaire. Several hypotheses can be put forward to explain this association. It is possible that the effect of this activity can be countered by the harmful effects of certain toxic substances especially as the majority of these patients are farmers. It is possible that this population with low socio-economic and probably low intellectual level, as suggested by their manual work activity, may be subject to at high risk conditions of low food hygiene and the consumption of polluted products. In rural areas, the population is supplied with individual or collective well water polluted by nitrates and nitrites and which are never subjected to physico-chemical controls, the absence of drinking water, and preserving food in open containers may represent a risk factor for this population.

CONCLUSION

This study has highlighted the age of onset of colorectal cancer 10 years younger than the age reported in Western literature. She demonstrated the link between some known risk factors in the literature and colorectal cancer in our context: level of education, family history of cancer, physical activity, obesity, alcohol-smoking intoxication, use of aspirin. The association was less obvious for family history of colorectal cancer, diabetes, oral contraception.

For the first time, we demonstrated the relationship between intense physical work and the high risk of colorectal cancer. Our results deserve to be confirmed by wider studies. Otherwise, we can and already develop recommendations on prevention campaigns for the fight against illiteracy, to promote a healthy lifestyle with the regular practice of a diet less caloric, the regular practice of a physical activity, the fight against the consumption of the tobacco and the alcohol.

References

Akhter M., Nishino Y., Nakaya N., Kurashima K., Sato Y., Kuriyama S., and Tsuji I. 2007.Cigarette smoking and the risk of colorectal cancer among men: a prospective study in Japan, *European journal of cancer prevention*, *16*(2), 102-107.

AyoujilA., HamiH., HabibF., Soulaymani A., Mokhtari A., and Quyou A. 2014. Colorectal cancer in Morocco: a retrospective study, *European Journal of Cancer*, *50*, S13. P12.

Baglietto L., Jenkins M. A., SeveriG., GilesG., Bishop D. T., Boyle P., andHopper J. L. (2006). Measures of familial aggregation depend on definition of family history: meta-analysis for colorectal cancer. *Journal of clinical epidemiology*, *59*(2), 114-124.

Benoit F. 2004. Les conduites d'alcoolisation: du repérage précoce au réseau de prise en charge. John Libbey Eurotext, 39-40.

Boufettal H. 2014. Contraception et cancer colorectal. *Revue marocaine du cancer*, *1*(4).

Boyle T., Bull F., Fritschi L., andHeyworth J. 2012. Resistance training and the risk of colon and rectal cancers. Cancer Causes andControl, 23(7), 1091-1097.

Buecher B; Pauw A. 2012. Hereditary colorectal cancer. La Revue de médecine interne, 33, 471-474.

Butterworth A. S., Higgins J. P., Pharoah P. 2006. Relative and absolute risk of colorectal cancer for individuals with a family history: a meta-analysis. *European journal of cancer,* 42(2), 216-227.

Cao Y., Nishihara R., Wu K., Wang M., Ogino S., Willett W.,and ChanAT. 2016. Population-wide impact of long-term use of aspirin and the risk for cancer. *JAMA oncology*, 2(6), 762-769.

Cerbelaud C., Chouillet AM., Molinie F., Auffert N., Bouvier AM., Lambrail P. 2008 .Pratique de prise en charge du cancer du côlon en Loire Atlantique en l'année 2000. Revue d'épidémiologie et de santé publique; 56(5), 298.

Charlton B M., WuK., Zhang X., Giovannucci E L., Fuchs CS., Missmer SA., and Michels K. B.2015. Oral Contraceptive Use and Colorectal Cancer in the Nurses Health Study I and II. *Cancer Epidemiology and Prevention Biomarkers*, 172.

Cohidon C., andImbernon E. 2009. Santé mentale et travail, de la connaissance à la surveillance. *Bulletin épidémiologique hebdomadaire*, 25-26.

Dreyer C., Afchain P., Trouilloud I., andAndré T. 2016. Nouvelles classifications moléculaires du cancer colorectal, du cancer du pancréas et du cancer de l'estomac: vers un traitement à la carte. *Bulletin du Cancer*, *103*(7), 643-650.

Fedirko V., Tramacere I., Bagnardi V., Rota M., Scotti L., Islami F., and Boffetta P. 2011. Alcohol drinking and colorectal cancer risk: an overall and dose–response meta-analysis of published studies. *Annals of oncology*, *22*(9), 1958-1972.

Ferlay J., Steliarova-Foucher E., Lortet-tienlent J., and al. 2013. Cancer incidence and mortality patterns in Europ: estimates for 40 countries in 2012. *Eur. J. Cancer* 49, 403.

Fink S. P., Yamauchi M., Nishihara R., JungS., Kuchiba A., Wu K., and al. 2014. Aspirin and the risk of colorectal cancer in relation to the expression of 15-hydroxyprostaglandin dehydrogenase (HPGD). *Science translational medicine*, *6*(233), 233re2-233re2.

Gariani K., Tran C., and Philippe J. 2010. Diabète et cancer: une association pernicieuse. *Revue médicale suisse*, *6*(252), 1193-1194.

Harriss D. J., Atkinson G., George K., Tim Cable N., Reilly T., Haboubi N., and al .2009. Lifestyle factors and colorectal cancer risk (1): systematic review and meta-analysis of associations with body mass index. *Colorectal disease*, *11*(6), 547-563.,

Huxley R. R., Ansary-Moghaddam A., Clifton P., Czernichow S., Parr C. L., and Woodward M. 2009. The impact of dietary and lifestyle risk factors on risk of colorectal cancer: a quantitative overview of the epidemiological evidence. *International journal of cancer*, 125(1), 171-180.

INCA 2012. Fiche repère Activité physique et cancers. Collection fiches repère

Johns L E., and Houlston RS. 2001. A systematic review and meta-analysis of familial colorectal cancer risk. *The American journal of gastroenterology*, *96*(10), 2992-3003.

Jørgensen O. D., Kronborg O., and Fenger C. 2002. A randomised study of screening for colorectal cancer using faecal occult blood testing: results after 13 years and seven biennial screening rounds. *Gut*, *50*(1), 29-32.

Kudjawu Y. C., Eilstein D., Decool E., De Maria F., Beltzer N., and Chatellier G. 2015. Time to first treatment after colonoscopy in patients suffering from colon or rectum cancer in France.3T 3T Cancer epidemiology, 3T 3T 39 (6), 877-884.

LahmidaniN.2011. Etude épidémiologique, clinique, endoscopique, Radiologique, biopathologique et thérapeutique des cancers colorectaux au CHU HASSAN II Fès.

Larsson S C., and Wolk A. 2007. Obesity and colon and rectal cancer risk: a meta-analysis of prospective studies. *The American journal of clinical nutrition*, *86*(3), 556-565.

Levi Z., Kark JD, Katz LH, Twig G., Derazne E., Tzur D., Leibovici Weissman Y., Leiba A., Lipshiez I., Keinan Boker L.and Afek A. 2017. Adolescent body mass index and risk of colon and rectal cancer in a cohort of 1.79 million Israeli men and women: A population-based study. *Cancer*, *123*(20), 4022-4030.

Liang PS., Chen T Y., andGiovannucci E. 2009. Cigarette smoking and colorectal cancer incidence and mortality: Systematic review and meta-analysis. *International Journal of Cancer*, *124*(10), 2406-2415.

Lüchtenborg, M., White, KL., Wilkens L., Kolonel LN., and Le Marchand L. 2007. Smoking and colorectal cancer: different effects by type of cigarettes? Cancer Epidemiol. Biomark. Prev. Publ. Am. Assoc. Cancer Res. Cosponsored Am. Soc. Prev. Oncol. 16, 1341– 1347.

Meddah, D., Meddah, B., Tir rouil, A., Ghalek, M., and Sahraoui, T. (2009). Étude épidémiologique du cancer du côlon chez des patients de l'Ouest algérien. *J. Afr. Cancer* 1, 31-35.

Moghaddam A A., Woodward M., and Huxley R. 2007. Obesity and risk of colorectal cancer: a meta-analysis of 31 studies with 70,000 events. Cancer Epidemiology and Prevention Biomarkers, 16(12), 2533-2547.

Moskal A., NoratT., Ferrari P., and Riboli E. 2007. Alcohol intake and colorectal cancer risk: a dose-response meta-analysis of published cohort studies. *Int. J. Cancer* 120, 664–671.

Mrini K., Hrora A., El Mernissi H., Benamer A., Raiss M., Sebbah F., and Ahallat M. (2009). Epidemiological aspects of colon cancer about a Moroccan surgical unit experience. *Arab Journal of Gastroenterology*, 10(2), AB27-AB28.

Ning Y., Wang L., and Giovannucci E L. 2010. A quantitative analysis of body mass index and colorectal cancer: findings from 56 observational studies. Obesity reviews, 11(1), 19-30.

Parajuli R., Bjerkaas E., Tverdal A., Selmer R., Le Marchand L., Weiderpass E., andGram, I. T. 2013. The increased risk of colon cancer due to cigarette smoking may be greater in women than men. *Cancer Epidemiology and Prevention Biomarkers*.

Peeters, P. J., Bazelier, M. T., Leufkens, H. G., de Vries, F., and De Bruin, M. L. 2015. The risk of colorectal cancer in patients with type 2 diabetes: associations with treatment stage and obesity. Diabetes Care, 38(3), 495-502.

Poomphakwaen K., Promthet S., Suwanrungruang K., Kamsa-ard S., and Wiangnon S. (2015). Risk factors for colorectal cancer in Thailand. *Asian Pac J Cancer Prev*, *16*, 6105-09.

RCRC. 2017. Registre des cancers du grand Casablanca, données 2008-2012. Ministère de la Santé publique

Renehan A. G., Tyson M., Egger M., Heller R. F., and Zwahlen M. 2008. Body-mass index and incidence of cancer: a systematic review and meta-analysis of prospective observational studies. *The Lancet*, 371(9612), 569-578.

Sharpe C R., Siemiatycki J A., and Rachet B P. 2002. The effects of smoking on the risk of colorectal cancer. Diseases of the colon & rectum, 45(8), 1041-1050.

Simons C. C., Hughes L. A., Van Engeland M., Goldbohm R. A., Van Den Brandt P A., and Weijenberg MP. 2013. Physical activity, occupational sitting time, and colorectal cancer risk in the Netherlands cohort study. American journal of epidemiology, 177(6), 514-530.

Steinmetz J., Spyckerelle Y., GuéguenR., andDupré C. 2007. Le tabac et l'alcool augmentent le risque d'adénomes et de cancers colorectaux: Étude comparant 3292 cas avec 5456 témoins dans une population à risque moyen ayant eu un test de dépistage de sang dans les selles positif. La Presse Médicale, 36(9), 1174-1182.

Troche JR., Mayne ST., Freedman ND., Shebl FM., Guertin KA., Cross, A.J., and Abnet CC. 2016. Alcohol Consumption-Related Metabolites in Relation to Colorectal Cancer and Adenoma: Two Case-Control Studies Using Serum Biomarkers. PloS One 11, e0150962.

Tsoi KKF., Pau CYY., Wu WKK., Chan FKL., Griffiths S., and Sung JJY. 2009. Cigarette smoking and the risk of colorectal cancer: a meta-analysis of prospective cohort studies. Clin. Gastroenterol. Hepatol. Off. Clin. Pract. J. Am. Gastroenterol. Assoc. 7, 682- 688-5.

viguier J., bourlier P., karsenti D., Decalan l, and Danquechin Dorval E. 2003. Cancer du côlon. Gasto Enterol. 18.

Wolin K Y., Yan Y., Colditz G A., andLee I M. 2009. Physical activity and colon cancer prevention: a meta-analysis. British journal of cancer, 100(4), 611-616.

How to cite this article:

Fatima Ezzahra IMAD *et al*.2018, Sociodemographic, Anthropometric And Medical Risk Factors For Colorectal Cancer In A Moroccan Population Case–Control Study. *Int J Recent Sci Res.* 9(2), pp. 23815-23821.
DOI: http://dx.doi.org/10.24327/ijrsr.2018.0902.1542

****