Université de Montréal

ASSOCIATION ENTRE LE PROFIL D'ACIDES GRAS ET LA PRÉVALENCE D'OBÉSITÉ : ÉTUDES ÉCOLOGIQUE ET TRANSVERSALE

Par

Nadiah Moussavi

Département de nutrition

Faculté de médecine

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présentée par:
Nadiah Moussavi

A été évalué par un jury composé des personnes suivantes:

Dre Marielle Ledoux, Président-rapporteur
Dr Olivier Receveur, Directeur de recherche
Dr Victor Gavino, Co-Directeur de recherche
Dr François Larivière, membre de jury
Dr Peter Jones, examinateur externe
Dr Jean-Marc Lavoie, représentant du doyen

RÉSUMÉ

La littérature scientifique s'intéresse à la relation entre la prévalence d'obésité et la qualité des gras dans l'alimentation. La différence dans la structure et le degré de saturation des acides gras dans l'alimentation affectent leur vitesse d'oxydation et leur dépôt dans le corps. Malgré les mécanismes expliqués dans les études in vitro et in vivo, effectuées surtout chez les animaux ainsi qu'un certain nombre d'essais cliniques, les études épidémiologiques concernant ce sujet sont peu nombreuses et les résultats contradictoires.

D'abord, nous avons entrepris une étude de revue sur le sujet afin d'examiner ce qui a été rapporté dans la littérature depuis 1970 au niveau épidémiologique. Également, nous avons considéré les études cliniques et fondamentales afin de vérifier la plausibilité biologique rapportée par ces études. Nous avons trouvé des études expérimentales in vitro et in vivo faites chez les animaux qui ont trouvé que les acides gras à chaîne courte (SCFA) ainsi que moyenne (MCFA), l'acide gras oléique, l'acide gras eicosapentaenoique, le docosahexaenoique et les ratios riches en acides gras polyinsaturés sur ceux de saturés (PUFA/SFA), en monoinsaturés (MUFA) sur ceux de SFA, (MUFA/SFA) ainsi que oméga-6/oméga-3 s'oxydent plus rapidement que les acides gras à chaîne longue (LCFA) et particulièrement les saturés à chaîne longue (LCSFA), l'acide gras palmitique ainsi que le stéarique dans des diètes isocaloriques. Dans les études cliniques, nous avons observé une tendance de ce qui a été rapporté dans les résultats démontrés chez les animaux. Les études

épidémiologiques rapportent qu'un apport élevé en gras, en MUFA, PUFA ainsi que SFA augmente la prévalence d'obésité, mais les résultats sont contradictoires. Cette étude nous a permis d'élaborer les hypothèses pour nos deux autres devis.

Nous avons donc décidé de faire une étude écologique incluant 168 pays (données disponibles), en prenant les données de l'indice de masse corporelle (IMC $\geq$30 kg/m^2) de l'Organisation Mondiale de la Santé (OMS) (2002) pour les femmes, âgées de 15 ans et plus, et en sélectionnant les bilans alimentaires du site *Food Agriculture Organization statistics* (FAOSTAT) entre 1998 et 2002. Par la suite, nous avons testé les associations entre la prévalence d'obésité, les calories, le gras total, les PUFA, les MUFA, les SFA ainsi que les « autres gras » *per capita* pour chaque pays. La moyenne de cinq années pour ces variables alimentaires et l'énergie ont été effectuées pour chaque pays. Nous avons trouvé une association positive et significative entre les calories totales, le gras total, les SFA, les PUFA ainsi que les « autres gras » disponibles à la consommation avec l'IMC. Tandis que nous avons trouvé une association inverse et significative entre les MUFA disponibles à la consommation et la prévalence d'obésité (β=-0,68, p<0,0001). Les fichiers alimentaire Canadien 2007a et celui Américain (*United States Department of Agriculture*, 2006) ont été utilisées pour les analyses statistiques.

Pour approfondir ce sujet, nous avons décidé d'utiliser des données collectées, par Santé Canada en 1990, chez les Cris de la Baie-James ou la prévalence d'obésité est différente et le profil nutritionnel diffère des autres Canadiens. Un rappel de 24

heures validé a été effectué chez 833 adultes en santé. Cependant, le fichier alimentaire Canadien de 1997 a été utilisé pour les analyses. Nous avons testé les mêmes associations citées dans le devis précédent, gras total, les SFA, les MUFA et les PUFA. De plus, avec cette étude nous avons pu identifier des types des gras spécifiques : les MCFA, les LCFA, les LCSFA, les trans, l'acide gras myristique, palmitique, stéarique, oléique, eicosapentaenoique, docosahexaenoique, linoléique, linolénique, les ratios PUFA/SFA et MUFA/SFA et oméga-6/oméga-3, en association avec l'obésité (devis transversal), IMC divisé en quatre niveaux selon l'OMS. Nous avons trouvé des associations positives et significatives pour les pourcentages entre les apports en gras total, en gras trans, en MUFA, en LCFA et particulièrement l'acide gras palmitique et l'obésité chez les femmes seulement. Nous n'avons observé aucun résultat statistiquement significatif chez les hommes.

Mots-clés : obésité, type d'acides gras, étude écologique, étude transversale, gras trans, gras monoinsaturés, saturés, polyinsaturés

ABSTRACT

Some investigators studied recently the association between dietary fat composition and obesity prevalence. Different types of fatty acids display different metabolic behaviours such as oxidation and deposition rate differences that may contribute to weight change. The structure dietary fatty acids, chain length, degree of unsaturation, position and configuration of the double bonds seem to affect their metabolic fate. Despite the mechanisms reported in litterature for these different associations in vitro and in vivo, experimental, and clinical studies, results from epidemiological designs are few and controversial.

We undertook a review which focused on the possible association between types of fatty acids and weight change from 1970s until now. It examined the biological plausibility underlying these associations and the evidence obtained to date from clinical trials and experimental studies. We reported that animal studies have shown that dietary short- (SCFA) and medium-chain fatty acids (MCFA) possess a higher oxidation rate in the body when compared to long-chain fatty acids (LCFA). Similarly, specific fatty acid, oleic, eicosapentaenoic, docosahexaenoic, high ratio of polyunsaturated to saturated (PUFA/SFA), monounsaturated fatty acids (MUFA) to SFA (MUFA/SFA), omega-6 to omega-3 (n-6/n-3) have higher oxidation rates when compared to LCFA and especially to long chain saturated fatty acids (LCSFA), palmitic and stearic fatty acids in isocaloric diet. Although results are conflicting, human studies follow the general trend reported in animal studies. These trials

suggest that some fatty acids are prone to oxidation and some others lead to fat storage when comparing isocaloric diets. Moreover, n-3 polyunsaturated fatty acids (PUFA), eicosapentaenoic and docosahexaenoic acids are preferentially oxidizied to other PUFA but results remain inconsistent. Epidemiological studies concerning this issue reported that total dietary fat, which includes MUFA, PUFA, and SFA could increase the prevalence of obesity, but results are few and conflicting. This review allowed us to draw up our hypothesis for the other epidemiological studies.

The purpose of our ecological study was to test the existence of an association between obesity prevalence and the types of fat available all over the world, in 168 countries. Data on the prevalence of obesity (body mass index $\geq$30 kg/m^2) for women over 15 years of age were obtained from the World Health Organization Global InfoBase. Food Balance Sheets for the years 1998 to 2002 were obtained from Food Agriculture Organization Statistics database. Five-year means for energy, total fat, MUFA, PUFA, SFA and "other fat" per capita were calculated, with their standard deviations, for each country. Dietary energy supply, total fat, SFA, PUFA and "other fat", per capita, were positively and significantly, associated with the prevalence of obesity. We found that populations with a lower prevalence of obesity seem to consume greater amount of MUFA (B=-0,68 ; p<0,0001). We used the Canadian Nutrient File, 2007a and the United States Department of Agriculture (USDA), 2006, for the statistical analysis.

The aim of our second study was to examine, using secondary data, the association between type of fatty acids and prevalence of obesity in a population, Indigenous Cree people of the Baie-James where the prevalence of obesity is high and the nutritional pattern is different from the rest of Canadians. A validated 24 hour recall was done in 833 healthy adults in 1990. We used the Canadian Nutrient File of 1997 in order to extract the following variables: total fat, SFA, MUFA, PUFA as for the ecological study and more specifically MCFA, LCFA, LCSFA, trans fat, myristic, plamitic, stearic, oleic, linoleic, linolenic, eicosapentaenoic, docosahexaenoic fatty acids, PUFA/SFA ratio, MUFA/SFA ratio and n-6/n-3 ratio to test their association with BMI categorized in four levels according to World Health Organisation. The following variables were positively and significantly different in the groups of women: percentages of total fat, trans fat, MUFA, LCFA and particularly palmitic fatty acid intake. There was no statistical difference in men for the studied variables across BMI groups.

Keywords: obesity, type of fatty acids, ecological study, cross-sectional study, trans fat, monounsaturated, polyunsaturated and saturated fat

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Apo A-IV	Apoprotéine A-IV
ASPQ	Association pour la Santé Publique du Québec
CCK	Cholescystokinine
CG	Charge Glycémique
CSF II	Nationwide Food Consumption Survey II
FAOSTAT	Food Agriculture Organization Statistics
G	gramme
IG	Indice Glycémique
IMC	Indice de Masse Corporelle
Kcal	kilocalories
LCFA	Long Chain Fatty Acids ou acides gras à chaîne longue
MCFA	Medium Chain Fatty Acids ou acides gras à chaîne moyenne
MUFA	Monounsaturated Fatty Acids ou acides gras monoinsaturés
NFCS	National Food Consumption Survey
OMS	Organisation Mondiale de la Santé
PPAR	Peroxisome Proliferator-Activated Receptors ou récepteur prolifératéur activé peroxisome
PUFA	Polyunsaturated Fatty Acids ou acides gras polyinsaturés
QFA	Questionnaire de Fréquence alimentaire
SCFA	Short Chain Fatty Acids ou acides gras à chaîne courte
SFA	Saturated Fatty Acids ou acides gras saturés
USDA	United States Department of Agriculture

VLDL	Very Low Density Lipids ou lipides à très faible densité
TIA	Tétradé-acyl-thio-acetique

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INTRODUCTION

Depuis les dernières années, la prévalence de l'obésité a augmenté à travers le monde. Ce fait a pour conséquences une augmentation de morbidité telle l'hypertension artérielle (MacCahon et al., 1987), le diabète de type 2 (Colditz et al., 1990) et une mortalité (Pedone et al., 2005) prématurée (Lew et Garfinkel, 1979). Aux États-Unis, 30,9% de la population possède un indice de masse corporelle (IMC) d'au moins 30 (Fifth Report, 2004). Au Canada, 15% de la population a un IMC d'au moins 30 (Fifth Report, 2004). Selon le dernier rapport de l'association pour la santé publique du Québec (ASPQ), 57% de la population québécoise possède un excès de poids. 35% présente un embonpoint, IMC entre 25 et 30 kg/m^2 et 22% présente de l'obésité, IMC supérieur à 30 kg/m^2 (ASPQ, 2005). On remarque aussi une prévalence élevée de l'obésité chez les Premières Nations et particulièrement les Cris qui semblent avoir un très haute prévalence d'obésité, d'hypertension artérielle et aussi de diabète type 2 (Thouez et al., 1990). Les principales causes de cette épidémie restent incertaines. Le rôle de l'alimentation en association avec cette maladie est de plus en plus étudié (Jéquier et Tappy, 1999). La relation entre les lipides, plus particulièrement, et l'obésité a été considérée dans la littérature. Plusieurs recherches cliniques et épidémiologiques se sont penchées sur la question. Cependant, les résultats de ces études sont contradictoires.

Nos questions spécifiques sont :

1. Existe-il au niveau international une association entre la prévalence d'obésité et le type de gras consommés : SFA, MUFA et PUFA (étude écologique)?

2. Dans une population à forte prévalence d'obésité (Crie de la Baie-James), la présence d'obésité est-elle associée à un type de gras consommé : SFA, MUFA, PUFA, gras trans, ratio PUFA/SFA, ratio MUFA/SFA et/ou acides gras myristique, palmitique, stéarique, oléique, eicosapentaenoique, docosahexanoique, oméga-3, oméga-6 et le ratio de ces derniers gras (étude transversale)?

Dans cette thèse, nous allons voir une revue de littérature sur les causes de l'obésité, particulièrement l'alimentation et spécifiquement les lipides. Ensuite, nous allons présenter trois articles qui ont examiné la relation entre l'obésité et le type de gras. Le premier conçu comme partie clé de la revue de la littérature présente les études expérimentales et épidémiologiques permettant d'examiner la plausibilité biologique d'une association entre le type d'acides gras et la prévalence d'obésité. Le second et le troisième concernent directement nos deux questions de recherche. Finalement, une discussion générale intègre les chapitres de ce travail afin d'en faire ressortir la cohérence et les implications.

CHAPITRE 1 REVUE DE LA LITTÉRATURE

1.1 DÉFINITION DE L'OBÉSITÉ

L'excès pondéral est défini comme un IMC ≥25 kg/m² et l'obésité comme un indice ≥30 kg/m² (OMS, 2005). L'obésité est considérée comme étant le résultat d'un déséquilibre chronique entre l'apport et la dépense énergétique (Harrison, 1997; Bray et Popkin, 1998; Jéquier et Tappy, 1999). Le gain de poids se fait, le plus souvent, d'une façon progressive et augmente avec l'âge. En effet, les deux tiers des obèses deviennent obèses entre l'âge de 20 à 50 ans, à raison de 0,45 à 0,91 kg par année ce qui correspond à un excédent de 15 à 30 kcal par jour (Williamson et al., 1990). Un déséquilibre si subtil de l'équation énergétique pourrait donc être le résultat de phénomènes métaboliques réduisant l'efficacité d'oxydation des substrats plutôt que d'un comportement conscient de contrôle de la prise alimentaire.

1.2 LES CAUSES DE L'OBÉSITÉ

Certains facteurs probablement responsables de l'obésité tels la génétique (Bouchard et al., 1990), l'activité physique et le déséquilibre entre les apports et les dépenses énergétiques dans la diète ont été étudiés profondément dans la littérature (Harrison, 1997; Willett, 1998; Neumark-Sztainer et al., 1999). Toutefois, le rôle relatif de ces facteurs et les mécanismes impliqués est encore controversé.

1.2.1 Les facteurs génétiques

Des recherches rapportent que la génétique influencerait l'obésité (O'Rahilly et Farooqi, 2006) de 40% à 50% dans une même population (Bouchard et al., 1990). Certains gènes ont été identifiés tel le gène *fat mass and obesity associated* (FTO), responsables de l'obésité chez les humains (Li et Loos, 2008; Loss et Bouchard, 2008) et ce domaine de recherche est très actif (Atkinson, 2008; Loss et Bouchard, 2008). Le métabolisme de base est influencé par la génétique et régulé en partie par des peptides anorexigéniques comme l'hormone thyrotropine (*thyrotropine releasing hormone*), la corticotrophine (*corticotrophin releasing hormone*), l'oxytocine et la mélanine (*melanin concentrating hormone*) (Valassi et al., 2008). Certains signaux de satiété, tels la CCK, le *glucagon-like peptide-1* et les peptides YY (PYY) originent du tractus gastro-intestinal durant l'ingestion d'un repas, vont au cerveau, à l'hypothalamus, via les réseaux sensitifs ou le sang afin de signaler la satiété (Valassi et al., 2008). Aussi, le nerf vague peut informer le cerveau par la distension gastrique, le signal clé de satiété. Des concentrations sanguines élevées de leptine et d'insuline sont aussi impliquées dans les signaux de satiété durant l'ingestion d'un repas (Valassi et al., 2008). Des auteurs ont trouvé que la balance énergétique est corrélée avec des concentrations élevées de leptine qui sont corrélées avec la grosseur du tissu adipeux corporel (Jéquier, 2002; Valassi et al., 2008). Chez les obèses ces niveaux sont plus élevés. Par contre, des auteurs rapportent qu'il y a présence d'une résistance à la leptine chez les obèses à différents niveaux (Jéquier, 2002; Valassi et al., 2008). Certaines hormones (des neuropeptides, des peptides, des récepteurs chimiques) sont

impliquées dans l'obésité via des signaux de satiété ainsi que d'appétit au niveau du système nerveux périphérique et central.

Malgré la génétique qui joue un rôle important dans la régulation de la balance énergétique (Hernandez et Perlemuter, 1997, Jéquier, 2002), l'augmentation fulgurante de la prévalence d'obésité à l'intérieur d'une même population telle que celle observée aux États-Unis pointe clairement vers d'autres causes (Willett, 1998) d'ordre environnementale (Association pour la Santé Publique du Québec, ASPQ, 2005; Mascie-Taylor et Goto, 2007; Bellisar, 2008).

1.2.2 Les facteurs environnementaux

1.2.2.1 Activité physique

Des études ont démontré qu'une augmentation de l'activité physique a pour effet de stabiliser la masse corporelle et peut même conduire à une modeste réduction de poids (Willett, 1998). Une étude longitudinale (Klesges et al., 1992) a trouvé une association positive entre l'activité physique et la perte de poids chez les femmes seulement. Des auteurs ont aussi remarqué une diminution de poids chez les femmes sédentaires ayant un surplus de poids. Ces femmes suivaient une diète hypocalorique et la durée de l'exercice était d'au moins 150 minutes/semaine (Chambliss, 2005). D'autres auteurs, ont rapporté que cette association entre activité physique et réduction de poids était similaire chez les obèses et les non obèses (Meijer et al., 1992; Jéquier et Tappy, 1999) et donc cela ne constituait pas une raison de surplus de

poids chez les obèses. Il est à remarquer que la plupart de ces études ont été effectuées à court terme (Moore, 2000).

1.2.2.2 Facteurs alimentaires potentiellement liés à l'obésité

Les fibres alimentaires (Yao et Roberts, 2001), le calcium (Zemel, 2002), la fréquence de la consommation des repas (Louis-Sylvester et al., 2003), l'apport énergétique (Bray et Popkin, 1998), l'apport en gras (Bray et Popkin, 1998) et la densité énergétique ainsi que l'apport en sucres (Bray et al., 2004) semblent influencer l'obésité chez différentes populations.

1.2.2.3 Fibres alimentaires

Des études ont rapporté qu'un apport élevé en fibres alimentaires semble aider à la perte de poids à court et à long terme (Yao et Roberts, 2001). Des auteurs ont suggéré qu'un apport en methylcellulose (fibre insoluble) peut représenter un traitement pour l'obésité (Keenan et al., 2006). Les fibres alimentaires semblent favoriser la satiété par leur contribution à l'augmentation du volume ingéré et une diminution proportionnelle de la densité énergétique. De plus, des études ont rapporté que l'apport en fibres alimentaires est inversement proportionnel avec les niveaux d'insuline sanguins, hormone qui favorise le dépôt adipeux (Koh-Banerjee et Rimm, 2003).

1.2.2.4 Calcium

Dernièrement, une attention particulière a été accordée à l'association entre l'apport en calcium et l'obésité. Des études épidémiologiques et cliniques ont trouvé que la consommation élevée de calcium semble diminuer le risque d'obésité chez des jeunes hommes de race blanche (Brooks et al, 2006) et chez les humains, en général (Zemel, 2002). Cette dernière étude a souligné que la cure de perte de poids semble être plus rapide avec un apport élevé en calcium alimentaire. Un apport élevé en calcium semble conserver davantage la thermogenèse alimentaire et favoriser la lipolyse lors de suivi d'une diète. D'autres recherches n'ont toutefois pas démontré la présence d'une association entre perte de poids et apport en calcium alimentaire (Venti et al., 2005).

1.2.2.5 Fréquence de la consommation des repas

Des études ont considéré l'association entre l'obésité, la fréquence ainsi que la durée de consommation des repas chez certaines populations et leurs résultats sont contradictoires. Une étude effectuée en Suède chez les femmes a démontré que le nombre de repas pris dans une journée chez les obèses était plus élevé que chez les non obèses. De plus, les obèses semblaient consommer à des heures plus tardives dans la journée que les non obèses (Bertus et al., 2002). Des auteurs ont rapporté qu'une fréquence de consommation élevée des repas était un facteur protecteur contre l'obésité chez les enfants (Toschke et al., 2005). Par contre, une autre étude, effectuée

en Angleterre, n'a trouvé aucune association entre la fréquence de consommation des repas et l'obésité (Summerbell et al., 1996). D'autres études ont rapporté qu'une fréquence élevée des repas, pour le même apport énergétique dans la journée, est associée à un maintien ou même une perte de poids modeste à long terme (Louis-Sylvester et al., 2003).

1.3 APPORT ÉNERGÉTIQUE ET EN MACRONUTRIMENTS

1.3.1 Apport énergétique

Examinons les aspects physiologiques d'apports et d'utilisation énergétique. L'apport énergétique chez les individus est constitué des substrats qui sont les carbohydrates, les protéines, les graisses et l'alcool. Le taux de métabolisme de chacun de ces substrats diffère dans le corps (4 kilocalories (kcal) par gramme (g), 4 kcal/g, 9 kcal/g et 7 kcal/g respectivement) (Butte et Caballero, 2005). L'efficacité d'absorption de ces macronutriments varie peu chez les sujets sans problèmes de santé et l'obésité ne peut être causée par une absorption excessive de l'un ou de l'autre (Jéquier et Tappy, 1999). Selon les études (Raben et al., 2003) le taux d'oxydation des substrats est par contre dans l'ordre suivant : alcool>protéine>carbohydrate>gras. L'apport en alcool stimule la dépense énergétique mais supprime l'oxydation en gras et la libération de la leptine (Raben et al., 2003).

Les dépenses énergétiques sont la thermogenèse alimentaire (±10%), l'activité physique (±30%) et le métabolisme de base (±60%) (Willett et Stamfer, 1998). La thermogenèse alimentaire est l'énergie nécessaire afin que les substrats ingérés soient métabolisés dans le corps et varie peu chez les sujets (Willett et Stampfer, 1998).

Le facteur principal qui semble jouer un rôle au niveau de l'obésité est le déséquilibre entre l'apport et la dépense énergétique (Harrison, 1997; Bray et Popkin, 1998; Anderson et Butcher, 2006; Rosengren et Lissner, 2008). Les résultats des études épidémiologiques sur ce sujet sont toutefois contradictoires. Certaines de ces études ont rapporté une diminution de l'apport total en énergie chez des populations où la prévalence de l'obésité a augmenté. À titre d'exemple, les NFCS et CSFII ont noté une diminution de 7% de l'apport énergétique consommé en 1994 comparé aux années 1977 à 1978 (Harnack et al., 2000). Par contre, d'autres sondages ont souligné le contraire. En effet, le « *US Food Supply Series* » a démontré que l'énergie par capita a augmenté entre 1970 à 1995, de 15,2% (Harnack et al, 2000). Ces résultats conflictuels s'expliquent, en partie, par des méthodes différentes utilisées dans les études mais aussi par le fait que les gens obèses sous-estiment davantage (de 30 à 55%) leur apport énergétique que les gens non-obèses (de 0 à 20%) (Lissner and Heitmann, 1995). Il est à noter que la magnitude de cette sous-estimation est possiblement exagérée par le fait qu'une partie importante de la population atteinte d'excès de poids est à tout moment donné en phase active de perte de poids. Un autre point qui a été relevé par Wansink et Chandon (2006), est le fait que les plus grosses portions sont sous-estimées, en terme de calories, par la population obèse et non

obèse. Ces auteurs ont reporté ce résultat à la suite d'ajustement pour le poids corporel.

Les études cliniques ont démontré des résultats positifs pour cette association. L'étude d'Alfieri et al, (1997) a considéré l'association entre l'apport énergétique total, le gras total et l'IMC chez des sujets modérément ($27,1 \leq IMC \leq 39,9$) et sévèrement obèses ($IMC \geq 40,0$) ainsi que chez des sujets non obèses ($20,0 < IMC \leq 27,0$). Ils ont trouvé une association positive entre l'apport en gras total et l'obésité chez le groupe des sujets modérément et sévèrement obèses. Alfieri et al. (1997) ont alors souligné qu'une diète riche en gras contribue à l'obésité, indépendamment de son contenu en calories. D'autres études cliniques ont démontré qu'un apport élevé en énergie est associé avec un gain de poids (Gothelf et al., 2002).

1.3.2 Macronutriments

Selon Alfieri (1997) une consommation élevée en gras alimentaire semble favoriser l'obésité. Toutefois, des auteurs soulignent qu'il est difficile d'affirmer que les obèses consomment nécessairement davantage d'énergie, sous forme de gras, que les non obèses (Sidell, 1998). Chez une même population, plusieurs recherches transversales et prospectives ont trouvé des résultats contradictoires (Willett, 1998). Les études épidémiologiques rapportent qu'une diète riche en gras influence le développement de l'obésité d'une façon suggestive mais non définitive (Lissner et Heitmann, 1995; Khor, 2004).

Les études cliniques démontrent une relation positive d'un gain de poids avec une diète riche en gras (George et al., 1990). Certaines études écologiques ont trouvé des associations positives et d'autres négatives pour la relation entre le gain de poids et la consommation de gras (Sidell, 1998). Paradoxalement, certains auteurs rapportent que les diètes traditionnelles (faibles en gras) pour la perte de poids ont un taux faible de réussite (Bahadori et al., 2004). L'évidence dans les études expérimentales concernant la relation existante entre l'obésité et l'apport en gras dans la diète est davantage rapportée que dans les études épidémiologiques (Lissner et Heitmann, 1995).

Les études épidémiologiques sont peu et les résultats conflictuels pour ce sujet. Dans la majorité des cas les outils pour la collecte des données et les modèles statistiques sont différents dans ces études. Aussi les facteurs confondants inclus dans les études ne sont pas les mêmes. L'explication, des différents résultats rapportées par les études épidémiologiques sur le sujet, réside aussi dans les méthodes des collectes des données, dans la sélection de l'échantillon, biais entre les interviewers et aussi une sous-estimation d'énergie rapportés par les obèses (Bouchard, 1990; Lissner et Heitmann, 1995). Ces résultats peuvent aussi être dus aux méthodes analytiques utilisées. On devrait tenir compte de l'apport énergétique total en épidémiologie de la nutrition et particulièrement en relation avec l'obésité pour trois raisons (Willett et Stampfer, 1998):

1) Le niveau d'apport énergétique total peut constituer lui-même un déterminant important de l'obésité.

2) Des apports énergétiques totaux différents chez les individus font varier aussi l'apport dans les différents nutriments consommés, ce qui peut engendrer des erreurs lorsqu'on analyse les données puisque la majorité des nutriments sont corrélés avec l'apport énergétique total.
3) Lorsque l'apport énergétique total est associé significativement à une maladie mais ne constitue pas une cause directe, l'effet d'un nutriment spécifique peut être confondu par l'apport énergétique. L'apport énergétique peut constituer un facteur confondant.

Dans les études épidémiologiques en nutrition, l'apport en différents nutriments est positivement corrélé avec l'apport énergétique dans la majorité des cas (Lyon et al. 1983; Gordon et al., 1984). Il faut idéalement alors ajuster pour l'énergie totale afin d'étudier l'effet d'un nutriment spécifique en association avec la maladie (Willett et Stampfer, 1998).

L'apport énergétique total en relation avec l'obésité a fait l'objet de plusieurs études épidémiologiques. Les résultats de ces études sont contradictoires (Willett, 1990; 1998). Les auteurs attribuent aussi ces résultats aux méthodes utilisées telles le recueil d'informations, rappel 24 heures et/ou questionnaire de fréquence alimentaire (QFA) et/ou les biais inter- ou intra individuel des interviewers ou des sujets à l'étude (Harrisson, 1997; Bray et Popkin, 1998; Willett, 1998). Kumanyika et al., 2003 a comparé trois outils de mesures : un QFA, un rappel 24h et un journal alimentaire de trois jours, et ont trouvé que l'apport énergétique était plus élevé chez les sujets avec

le journal de trois jours, ensuite le QFA et le rappel de 24h a pris la dernière position. Des auteurs ont rapporté que pour une étude transversale, le rappel de 24h représente une méthode valide pour examiner l'apport nutritionnel chez un groupe (Zoellner et al., 2005). Une autre étude effectuée à Taiwan a rapporté qu'un QFA était approprié pour les nutriments consommés chez une population tandis que pour l'apport énergétique un rappel de 24h était plus adéquat. Ces auteurs (Lyu et al., 2007) ont souligné que les résultats rapportés étaient acceptables pour la reproductibilité et validité avec l'utilisation de ces deux outils.

Le modèle statistique utilisé devrait être approprié à l'outil en question. Les facteurs confondants tels l'âge (Willett, 1998) sont très importants à considérer dans les études épidémiologiques car ils peuvent exagérer ou atténuer une relation. L'utilisation des modèles statistiques tels la régression ou corrélation devraient être appropriés aux données recueilles et/ou disponibles et aux questions de recherche. Lorsqu'un nutriment est corrélé lui-même avec l'apport énergétique ou les nutriments sont corrélés entre eux et à l'apport énergétique, nous pouvons avoir une exagération ou une diminution de l'association entre maladie et nutriment. Il faut alors ajuster pour l'énergie totale et/ou les autres nutriments dans le modèle statistique. Par contre, cela n'est pas approprié avec un seul rappel de 24h dont l'analyse se fait au niveau des groupes et non des individus. Ajuster pour l'âge, une variable individuelle, est toutefois possible dans la comparaison de groupes.

Récemment, la densité énergétique a été considérée comme étant le seul facteur convainquant qui influence l'apport énergétique alimentaire ainsi que la balance énergétique (Swinburn et al., 2004). La densité énergétique est le taux d'énergie métabolisée par unité de volume ou de poids d'aliment (Livesey, 1991). Toutefois, la densité énergétique est considérée de deux façons dans la littérature. En effet, elle peut référer à l'énergie par gramme d'aliment ingéré ou encore au pourcentage de l'énergie métabolisée provenant des lipides par rapport à l'énergie totale (Livesey, 1991). La méthode de mesure de la densité énergétique n'est pas claire jusqu'à ce jour (Cox et Mela, 2000). Dans les aliments, les principaux déterminants de la densité énergétique sont l'eau, le gras et les fibres alimentaires (Yao et Roberts, 2001).

Une diète riche en gras possède une densité énergétique élevée, à cause du taux métabolique élevé d'un gramme de gras qui est de 9 kilocalories par gramme (McCance et Widdowson, 1991). Il est à remarquer qu'une densité énergétique élevée ne veut pas nécessairement dire un apport élevé en gras. Un apport élevé en hydrates de carbone peut conduire aussi à une densité énergétique élevée (Poppitt et Prentice, 1996). Des études (Macdiarmid et al., 1998) se sont intéressées à l'apport en gras et/ou en carbohydrates indépendamment de leur densité énergétique en association avec l'obésité. Ces auteurs ont classifié les aliments selon cinq principales sources. Ils ont trouvé que l'apport en gras était relié à l'obésité chez les hommes. L'apport en gras et en carbohydrates était relié à l'obésité chez les femmes. Une autre étude (DeGonzague et al., 1999) a considéré l'apport alimentaire et la prévalence de l'obésité chez deux communautés Ojibwe. Elle a trouvé une association positive entre

le pourcentage d'énergie provenant de gras (hommes 37% et femmes 40%) et l'obésité dans cette communauté Ojibwe. De plus, une autre étude effectuée chez les Inuits au Canada (Kuhnlein et al., 2004) a trouvé une corrélation positive entre l'obésité chez les hommes et le pourcentage de l'énergie dérivée des lipides. Toutesfois autres études n'ont rapporté aucune association entre l'apport en gras et l'obésité (Togo et al., 2001).

En ce qui concerne l'association entre la satiété, le type de macronutriments et la densité énergétique, Poppitt et Prentice (1996) ont souligné que les protéines et les glucides alimentaires semblent avoir un plus grand effet de satiété que les gras. Poppitt et Prentice (1996) ont rapporté qu'indépendamment du contenu en gras de la diète, une densité énergétique faible fournit une plus grande satiété comparée à une diète élevée en densité énergétique, ce qui confirme le fait qu'un signal de régulation clé de satiété est le poids ou le volume des aliments. D'autres auteurs ont aussi rapporté que le volume gastrique joue un rôle dans la sensation de satiété (Rolls et Roe, 2002). Olin et al. (1996) rapportent que les gens consomment approximativement le même volume d'aliments à chaque repas. Ello-Martin et al. (2005) ont souligné qu'une diète à faible densité énergétique (riche en fruits, légumes et soupes) augmente la satiété et contrôle la faim chez les sujets tandis qu'une diète faible en gras favorise la perte de poids. À long terme, une diète faible en densité énergétique semble donc permettre une perte de poids (Yao et Roberts, 2001).

L'indice glycémique (IG) est la mesure du glucose sanguin à la suite de deux heures d'un repas contenant 50 grammes de sucre (Kolset, 2003). La littérature rapporte qu'une diète à faible IG peut être bénéfique à long terme pour la perte de poids (Kolset, 2003). Il est à remarquer que d'autres études (Solth et al., 2004) n'ont pas trouvé de relation entre le poids et l'IG. Le rôle de l'IG et de l'appétit dans la régulation de poids n'est pas clair dans la littérature. Récemment la charge glycémique (CG) a été considérée dans la littérature (Livesey, 2005). Cette charge tient compte des fibres alimentaires et de la quantité de glucides disponibles dans une portion d'aliment (Livesey, 2005). CG permet de mesurer la réponse glycémique totale après la prise d'un aliment ou d'un repas (CG= IG/100 x grammes de glucides par portion). Une diète contenant un CG faible suggère une perte de poids, à long terme. Cela s'explique par une satiété élevée ainsi qu'une diminution d'apport énergétique (Livesey, 2005). D'autres études sont nécessaires pour confirmer la relation entre ces indices et l'obésité (Livesey, 2005).

Une étude récente (Bray et al., 2004) s'est intéressée à la qualité des sucres dans une diète. Ces auteurs ont rapporté que, puisque contrairement au glucose, le fructose n'agit pas sur la sécrétion de l'insuline et de la leptine, deux hormones clés dans la régulation des signaux afférents de l'apport alimentaire, une consommation élevée en fructose pourrait contribuer à l'augmentation d'un apport énergétique élevé ainsi qu'au gain de poids chez un sujet. Des auteurs (Segal et al., 2007) ont suggéré, récemment, d'utiliser un indice pour le fructose aussi, afin de catégoriser les aliments selon cet indice en relation avec l'obésité. Toutefois, des auteurs ont rapporté que la

réserve des sucres est limitée dans le corps (Flatt, 1987 et 1995). Une consommation élevée en protéines et en carbohydrates semble se réguler par le corps d'une façon équilibrée (Bray et Popkins, 1998). La surconsommation de ce dernier groupe aboutit dans un premier temps à un dépôt de glycogène, puis le surplus est transformé en gras mais cette dernière voie n'est pas favorisée par le corps (Jéquier et Tappy, 1999), tandis qu'un apport élevé en gras ne semble pas affecter l'oxydation des gras et le corps semble posséder un pouvoir illimité de dépôts adipeux (Bray et Popkins, 1998).

1.4 TYPE D'ACIDES GRAS ET OBÉSITÉ

Des études récentes ont rapporté que les différents types de gras alimentaire sont métabolisés à des niveaux différents dans le corps (Clandinin et al., 1995; German et Dillard, 2004). La littérature rapporte que la composition différentielle de gras dans la diète peut influencer le développement de l'obésité indépendamment de l'apport énergétique provenant des gras (Jones et Schoeller, 1988; Hill et al, 1993; DeLany et al., 2000; Khor, 2004). Cette affirmation a beaucoup d'implications dans l'obésité et dans les recommandations nutritionnelles.

Nous avons donc entrepris une revue de la littérature, considérant toutes les études qui ont été effectuées depuis 1970. Ces recherches comportent les études expérimentales effectuées chez les animaux et humains. De plus, certaines études ont été faites in vitro. Les devis épidémiologiques ont été aussi rapportés dans cet article. Ce manuscrit résume toutes les associations rapportées entre l'apport en acides gras

ou type de gras et le changement de poids. De plus, il considère les mécanismes impliqués, la plausibilité biologique, ainsi que les différentes hypothèses mises de l'avant par les auteurs pour ces associations.

1.5 ARTICLE I

Could the quality of dietary fat, and not just its quantity be related to risk of obesity? A review

Nadiah Moussavi[1], Victor Gavino[1] and Olivier Receveur[1]

[1] Department of Nutrition, Université de Montréal.

Abbreviations: BMI, body mass index; FFQ, food frequency questionnaire; MCFA, medium chain fatty acids; MUFA, monounsaturated fatty acids; PPAR, peroxisome proliferators-activated receptor; P/S, polyunsaturated on saturated ratio; PUFA, polyunsaturated fatty acids; RQ, respiratory quotient; SCFA, short chain fatty acids; SFA, saturated fatty acids; SREBP, sterol regulatory element-binding proteins

Key words: weight change, dietary fatty acids, MUFA, PUFA, SFA

Correspondence to Dr Olivier Receveur, Nutrition Department, Medicine Faculty, Université de Montréal, C.P 6128, Succ. Centre-Ville, Montréal, Qc. H3C 3J7 (Email[redacted].

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1.5.1 Abstract

This review focuses on the possible association between types of fatty acids and weight change. It examines the biological plausibility underlining these associations and the evidence obtained to date from clinical trials and epidemiological studies. Animal studies have shown that dietary short- and medium-chain fatty acids compared to long-chain fatty acids appear to promote weight loss. Similarly, monounsaturated fatty acids (MUFAs) appear to favor weight loss compared to saturated fatty acids (SFAs) in human studies. The structure of fatty acids seems to affect their degree of oxidation and deposition. Although results are conflicting, human studies follow the general trend reported in animal studies. These trials suggest that some fatty acids are prone to oxidation and some others lead to fat storage when comparing isocaloric diets. For instance, n-3 polyunsaturated fatty acids (PUFAs), eicosapentaenoic and docosahexaenoic acids are preferentially oxidizied to other PUFA but results remain inconsistent. Epidemiological studies concerning this issue reported that total dietary fat, which includes MUFA, PUFA, and SFA could increase the risk of obesity, but results are few and conflicting. The rising biological plausibility linking dietary fat quality and risk of obesity, together with the rather recent addition of fatty acids content in food composition tables, support the need for major epidemiological studies in that area.

1.5.2 Introduction

Obesity is a growing health problem, with its prevalence increasing all over the world and particularly among young age groups (1). The exact etiology of obesity remains unclear but it appears to be a complex combination of genetic, metabolic and environmental factors (2). Several genetic defects contributing to obesity have been proposed (3). However, in the short-term, increased prevalence of obesity (4, 5) is influenced largely by environmental factors such as lack of physical activity and dietary behaviors (5, 6). Nutritional factors including energy excess, high energy density, an increasing consumption of carbohydrates and fat in the diet have been reported to lead to obesity (7–10). In particular, increases in the amount of fat in the diet have been shown to be associated with the risk of obesity (11, 12) and the pathway linking obesity and dietary fat has been the subject of several studies. Some authors proposed that high-fat diets can lead to obesity because fat is an energy dense macronutrient (13, 14). Furthermore, dietary fat can contribute to obesity via passive over consumption, because this macronutrient is less satiating than either carbohydrates or proteins (13, 14).

When comparing the evolution of fat consumption between Paleolithic nutrition and today's diet, it seems that humans have evolved from an animal protein diet from wild game to an animal protein containing more fat per unit of weight (15–17). Moreover, fat from wild species of animals were rich in polyunsaturated fatty acids (PUFAs) and contained an appreciable quantity of ω-3 (4%) (18). Beef meat in

today's diet contains undetectable amounts of this type of fatty acid (18). Western diet contains more polyunsaturated fat rich in n-6, particularly arachidonic and linoleic fatty acids (17), because of the recommendations throughout the last fifty years, aiming at minimizing saturated fat intake in the diet.

Some investigators proposed recently that dietary fat composition, in addition to the amount of fat or energy intake, could affect the development of obesity (19,20). Different types of fatty acids display different metabolic behaviors such as oxidation and deposition rate differences that may contribute to weight change. The structure of dietary fatty acids, chain length, degree of unsaturation, position and configuration of the double bonds seem to affect their metabolic fate (21–23). Results focusing on this subject are controversial. For instance, some authors reported that fish oil promotes weight loss in animals (24) and in humans (25, 26) but other reports showed no difference in weight change as a result of consuming a diet rich in fish oil (27). When comparing dietary monounsaturated fatty acid (MUFA) intake to saturated fatty acid (SFA) in humans, results showed a decrease in fat deposition with a diet rich in MUFA (28). On the other hand, Christiansen *et al.* (29) showed no difference between groups on isocaloric diets when given avocado fat, mixed oil, or fatty acids derived from trans fats. Lovejoy et al. (30) demonstrated that trans fatty acids are highly oxidized compared to oleate or palmitate. Moreover, Coelho et al. (31) reported that adding 30% of peanut oil (from a MUFA source) to the diet, led to a weak and significant weight gain (2.3 kg) in overweight subjects after 8 weeks, but this weight gain was less than the amount that was theoretically predicted. Among

epidemiological studies, some cross-sectional studies showed a positive association between the consumption of PUFA, MUFA, and SFA and the risk of obesity (32–35). Nevertheless, some investigators did not find any relationship between the consumption of PUFA, MUFA, or SFA and the risk of obesity (36–38). Results from epidemiological studies are very controversial when considering the association between different types of fatty acids and weight change.

Qualitative changes in food for fatty acid composition and its association with weight change have not so far been thoroughly investigated. Thus, the evidence linking the types of fatty acids to change in body weight is the focus of this review. In this study, we will first present the biological plausibility underlying a potential association between types of fatty acids and the risk of obesity. We will then undertake a review of epidemiological studies, which have examined this association.

1.5.3 Experimental studies

1.5.3.1 In vivo and in vitro studies

In vivo and in vitro studies have shown that PUFA, but not MUFA or SFA, seem to suppress the expression of genes involved in lipogenic transcription by decreasing the expression of the sterol regulatory element–binding proteins (SREBPs) (39–41). These studies were conducted on cells from hamster ovary or rat hepatocyte cell. The study of Worgall et al. (39) included PUFA from C18:2 to C22:6 and oleate. They found that oleate and PUFA decrease the transcription of SRE-

regulated genes by 20–75%. Mater et al. (40) considered an isocaloric diet with different types of fat taken from lard, olive oil, corn, walnut, and fish oil in male rats. They demonstrated that all PUFAs down-regulate hepatic SREP-1 gene expression in vivo. The fatty acids administered by the Hannah et al. study (41) were oleate, linoleate, and arachidonic fatty acids. These authors demonstrated that PUFAs administrations down regulate the expression of SREP-1a and SREBP-1c. The mechanism explained by most of these studies, concerning the suppression of the expression of genes involved in lipogenic expression in SREBP, is that SREBP activates genes involved in adipocyte differentiation, in cholesterol synthesis and metabolism (42). Most fatty acids activate all of the peroxisome proliferator-activated receptor family (PPAR) at micromolar concentrations (43, 44). PPARs transcription factors are key mediators of gene regulation in lipid metabolism (42, 45). Moreover, PUFAs and particularly eicosapentaenoic and docosahexaenoic fatty acids seem to function as activator ligands for PPARs (45–47). PPARs genes are involved in adipocyte differentiation and lipid storage (45). They decrease adipose tissue mass and suppress development of obesity when activated (45). Although the induction of a high-fat oxidation rate by the consumption of PUFAs could influence energy utilization and fat deposition, the quantitative meaning of this assertion is presently unknown in humans (42). Results have demonstrated that PUFAs are better activators of PPAR-γ than MUFA or SFA. It seems that the highest binding affinity is completed with fatty acids containing 16–20 carbons. Also it has been reported that PUFAs are better activators of PPAR-α than MUFA or SFA (43, 44, 48). The activities of all of these transcription factors have to be determined precisely as a

function of the type of fatty acid intake. In vitro studies are helpful to explain the mechanisms of the associations observed in studies. They cannot be extrapolated in all cases to human or animal studies.

1.5.3.2 Animal studies

Animal studies have shown different rates of weight change in response to different types of fats in the diet (49, 50). Rats were shown to oxidize linoleate more than palmitate (49). In rat liver, the oxidation rate of different fatty acids was as follows: linoleate > butyrate > linolenate > acetate > stearate (50). The general trend in animal studies is that the oxidation rate of the SFA decreases with increasing length of the carbon chains: laurate (72%) > myristique (58%) > palmitate (41%) > stearate (28%) (51).

For unsaturated long-chain fatty acids, there are suggestions in the literature that n-6 and n-3 PUFA have higher oxidation rates than does long-chain SFA (49). Additionally, after absorption, n-6 PUFA from safflower oil produces a greater thermogeneic effect (52), more oxygen consumption (53) and more sympathetic nervous stimulation (54). Some authors (52, 54) have reported that when comparing safflower oil and beef tallow intake in rats, the latter group had a higher carcass fat content and a lower sympathetic nervous activity as well as a lower diet-induced thermogenesis.

PUFAs from fish oil can also decrease fat mass gain (55) and increases weight loss in rodents (56). Madsen et al. (23) stated that PUFAs from the n-3 group induce more effective control on PPAR-α and γ in activating their genes than does n-6 PUFA. Moreover, n-3 PUFAs from marine sources seem to reduce adiposity more than n-3 PUFAs from plants such as α-linolenic acid (57). On the contrary, Ailhaud et al. (17) reportedthat the inclusion of α-linolenic acid in an isocaloric diet rich in linoleic acid prevented the increase of fat mass in a group of pups. These authors highlighted that these data were consistent with their previous results reported in vitro in which they were comparing the adipogenic effect of n-6 PUFA to that of n-3 PUFA. The mechanism proposed by investigators (who reported that n-3 PUFAs from marine sources reduce obesity in rodents) is that fish oil intake has been shown to increase the activity of carnitine acyltransferase I in rats (58), in mice (46), Syrian hamsters (59), and to inhibit malonyl-CoA activity (60) a key signal of satiety (61). The activation of carnitine acyltransferase I, as well as the inhibition of the synthesis of malonyl-CoA, seem to promote fatty acid availability for oxidation (47, 59, 62). Some authors demonstrated that with higher n-3/n-6 ratio intake, there was an increase in peroxisome and mitochondria B-oxidation in rodents (62). This was due to an increase in carnitine acyltransferase I activity. To note, not all studies demonstrated a decrease in fat mass and body weight in rats fed on fish oil (27).

There is some data that have shown that fat oxidation is higher with an increase in polyunsaturated/saturated (P/S) ratio intake (63, 64). The explanation reported by most of these authors is that the morphology of villi in both rat jejunum and ileum is

also affected by the ratio of P/S in the diet (63, 64). In fact, villus surface area is increased with a diet high in PUFA (63). The study of Thomson et al. (63) was conducted in adult female rats. Isocaloric diets rich in palmitic, stearic, oleic, linoleic, and linolenic fatty acids were given. They demonstrated that jejunal uptake of medium-chain acid (C8:0–C12:0) was higher with the oleic acid than with other diet groups. Sagher et al. (64) reported that maize and olive oil increase villus height in both jejunum and ileum compared to butter oil in a group of rats fed isocaloric diets.

Few experiments studied the influence of dietary fatty acids with their deposition in Zucker fed a cafeteria diet (65). In Zucker obese rats, cafeteria feeding resulted in an alteration of enzymatic activities particularly in the conversion of linoleic fatty acid into dihomo-linolenic acid (65). Fat deposition was higher in rats given the cafeteria diet. In fact, cafeteria diet in lean and obese rats had shown a direct incorporation of dietary fatty acids into the rat lipids. On the contrary, the chow feeding in both groups activated lipogenesis and favored the deposition of shorter chain and more SFAs. Dang et al. (66) examined the effect of different diets (with 20% of energy derived from saturated, unsaturated or menhaden oil), on the phospholipid fatty acid composition and in vitro δ-5-desaturase activity of hepatic microsomes in the normal or streptozotocin-induced diabetic rat. The normal rat fed the saturated fat or menhaden oil diet had significantly decreased arachidonate levels in phospholipids, consistent with decreased δ-5-desaturase activities. On the other hand, the unsaturated fat diet decreased dihomo-γ-linolenate and increased arachidonate levels in phospholipids, without increased δ-5-desaturase activity (66).

Low levels of arachidonic acid, particularly in phospholipids, were observed in obese animals (45). Furthermore, PUFAs only, and not MUFAs or SFAs, seem to activate an isoform of SREBP, SREBP-1c, mRNA. The mechanism is still unknown (42). In rat liver, δ-5-desaturase is one of the targets of SREBP-1c. Docosahexaenoic fatty acid is the product of this desaturase when increased and is mostly incorporated into phospholipids and not in triglycerides (42).

Another mechanism that affects the utilization of SFA is the difference in stereoisomeric configuration of fat molecules (67, 68). It seems that saturated dietary fat molecules, occupying sn-1 and sn-3 positions (coconut and palm oil), may be preferentially absorbed in comparison to those that are esterified on the sn-2 positions (milk and lard) (67). In fact, the former group may form insoluble calcium soaps in the human intestine (67). Some authors highlighted that insoluble calcium soaps lead to a decrease in the incorporation of fatty acids in chylomicrons, which will in turn deliver less lipids to the tissues and the plasma lipids (69). In contrast, some other investigators have not shown any significant difference in the absorption of dietary fatty acids subsequent to modifications of their stereoisomeric configuration structure (70). Most of these studies have been undertaken on animals and their physiological significance in humans is unclear. Additional research in this area is needed in human populations.

One should notice that animals, in most of these studies, are fed a standard laboratory diet supplemented with different fatty acids for various periods of time. The period of time of a given diet is an important factor to consider in animal studies. For instance, Marette et al. (49) reported a weight loss in rats only after 13 weeks when the rats were fed corn oil in comparison to the group of rats fed on a lard diet. In these studies, the quantity and the type of fat were well controlled. Authors tend to imitate the human diet when using laboratory animals such as rats, mice, or chicken but the human diet contains a great diversity of fatty acid profiles. Also, animals do not possess the exact same metabolism as do humans (23).

1.5.4 Human studies

1.5.4.1 Metabolic studies

In Table 1, we summarized all the studies reported in literature that reported a positive, a negative, or an absence of association between dietary fat composition and weight change. We described outcomes as oxidation rate, weight change or fat deposition, energy expenditure and thermogenesis.

It is well known that short-chain fatty acids (C2:0–C4:0) and medium-chain fatty acids, MCFAs (C6:0–C12:0) are preferentially oxidized compared to long-chain fatty acids, C14:0–C24:0 (LCFAs), in humans as well as in animals (22, 71–75). Short-chain fatty acid and MCFA are transported directly to the liver via the portal

system (76, 77). Short-chain fatty acid and MCFA are preferentially oxidized because they are not incorporated into esterified lipids and their transport to the mitochondrial matrix is not carnitine-dependent (76, 77). Because of these properties, short-chain fatty acid and MCFA may lead to increased energy expenditure and contribute to a loss of adipose tissue in humans (74, 78). Dulloo et al. (78) reported that when substituting LCFA with MCFA, in humans, at 15–30 g of fat/day, the energy expenditure is stimulated by 5% (500 kJ), which is partially mediated by the activation of the sympathetic nervous system. Therefore, these authors and others (79) stated that consumption of a diet rich in MCFA could lead to a reduction in final body weight. Others reported that MCFAs are poorly deposited within human tissues (80). In the adipose tissue, MCFAs are absent and they are less incorporated into hepatocytes and into adipocyte triglycerides than LCFA in humans and in animals (81–83). Some investigators also demonstrated (84) that the net energy value of MCFA is 5 kcal/g, which is different from the usual amount of energy coming from LCFA, 9 kcal/g.

The high rate of oxidation, the poor deposition of MCFAs in adipose tissue, their lower level of energy per gram available for metabolism, may explain the final body weight reduction reported in some studies when these types of fatty acids are consumed.

For long-chain unsaturated fatty acids, the MUFA type found in the human diet are: myristoleic (C14:1), palmitoleic (C16:1), oleic (C18:1), gadoleic (C20:1) and erucic (C22:1) acids. Oleic acid is the one that has been studied the most in literature for its association with weight change. This is the major fatty acid in olive oil, which in turn is one of the major components of the Mediterranean diet (85, 86). Several studies in humans have shown that oxidation of MUFA, especially oleate, was higher than linoleate, which in turn was greater than stearate or palmitate (87–89). In addition, several clinical trials with one exception (30) reported that oleate is oxidized to a greater extent than palmitate, particularly in obese individuals (90). In their randomized study Lovejoy et al. (30) administered a diet with 28% of energy from fat. Authors compared MUFA (oleic), SFA (palmitic) and trans fatty acids (elaidic) in 25 healthy men and women.

In studies that reported a higher oxidation rate with MUFA intake (87–89), the mechanism proposed by most of the authors is that MUFA intake increases diet-induced thermogenesis which in turn stimulates the sympathetic nervous system. Abdominally, obese subjects may be more responsive to the stimulation of the sympathetic nervous system because they have an increased density and sensitivity of B-adrenoreceptors (91). Furthermore, abdominal, more than femoral adipocytes, have greater lipolytic response to catecholamines (91). However, some authors compared the substitution of a high MUFA diet with an energy-restricted diet (92, 93) but did not find any significant difference in body weight in either group. Clifton et al. (92) reported a conservation of lean body mass with a diet rich in MUFA. In this last randomized study, 62 women with type 2 diabetes

were observed for 12 weeks. Thirty-five percent of energy was given to the group of women following the MUFA rich diet and 12% of total fat was given to the group of women following the low-fat diet. Pelkman et al. (93) stated that inclusion of MUFA food sources in the diet may increase the compliance for a calorie-reduced diet during weight loss programs and promote weight-maintenance after a weight loss regimen. However, some studies have not shown a positive or negative association between BMI and MUFA intake (94, 95) and specifically olive oil (96).

For the other unsaturated LCFA family, n-6 PUFA, Cunnane et al. (97) reported that α-linolenate is the most oxidized 18-carbon chain PUFA, and arachidonic fatty acid is the least. Moreover, these authors stated that docosahexaenoate undergoes less β-oxidation than do α-linolenate or linoleate. When studying n-3 PUFA, Krebs et al. (26) have shown a weight reduction with the consumption of fish oil in humans. However, fish oil intake was combined with an energy-restricted regimen in this trial (26). In this study 35% of energy was derived from fat. A capsule of n-3 PUFA (1.3 g of eicosapentaenoic and 2.9 g of docosahexaenoic fatty acid) was given to the group that had been following the fish oil diet for 12 weeks. Couet et al. (98) also reported that with a consumption of daily fish oil (6 g/day) there was less fat deposition and an increase in the rate oxidation in non-obese subjects. In this last study, 32% of total energy was derived from fat.

Some data demonstrated that the P/S ratio in the diet affects the utilization of fatty acids in the diet. For instance, Clandinin et al. (99) found that on increasing the P/S ratio,

a high oxidation rate was observed after consumption of both low- and high-fat diets (30 and 40% of energy coming from fat). These authors pointed out that since the intake of palmitic acid occurs from the upper third of the villus, increasing the P/S ratio in the diet seems to be associated with an increased rate of uptake of palmitic acid.

Recently, desaturases activity is being studied in association with obesity. Warensjo et al. (100) reported that BMI and waist circumference were positively correlated with increases in δ-6 and δ-9 desaturase activities and with elevated serum proportions of palmitic, palmitoleic, stearic, linolenic, and arachidonic acids. On the other hand, δ-5 activity and linoleic acid concentration in serum were inversely correlated with obesity markers. Their results suggest that a modification in the fatty acid profile in serum cholesteryl esters, which reflects to a certain extent the composition of dietary fatty acids and endogenous fatty acid synthesis, is correlated with desaturase activities and associated with obesity.

When observing Table 1, one can notice that clinical trials seem to have some conflicting outcomes. The main problem associated with these trials is uncontrolled conditions such as physical activity. Note that clinical trials in humans concerning the association between PUFA and especially fish oil intake and weight change are few.

1.5.5 Epidemiological studies

In Table 2, we have summarized all epidemiological studies reported in literature. In this table we have included the purpose of the studies and their outcomes. The epidemiological studies addressing this issue are few. We identified eight cross-sectional and six cohort studies. We did not find any ecological or case/control studies. In this table, one can note that there are four cross-sectional studies, which reported a positive association between BMI and MUFA, SFA or PUFA (32–35). On the other hand, two studies have shown a negative association between BMI and the Mediterranean diet (101–102). It is interesting to note that two studies have not demonstrated any association between BMI and an increase in the intake of MUFA or olive oil (94, 95). Williams et al. (34) showed a negative association between BMI and fish oil intake. Three cohort studies reported a positive association between BMI and MUFA or Mediterranean diet and SFA (36–38). Another cohort study has shown a negative relationship between BMI and the Mediterranean diet (103). On the other hand, two cohort studies have not demonstrated any association between BMI and oil olive intake (96, 104). It is interesting to note that the purpose of these studies as observed in the table is not always to study primarily the association between weight change and type of fatty acids. From the 14 epidemiological studies, 9 studies had as their main objective to test for an association between weight change and the type of fatty acids; three studies reported a positive association between BMI and the type of fatty acid; three studies did not reveal any relationship between BMI and a high intake of olive oil, and the other three studies have shown a negative result for this association. These results are conflicting.

In epidemiological studies, authors do not take into consideration physical activity, which is a major component of the energetic equation (105). At any rate, the measurement of intensity and duration in physical activity constitute a matter of conflict (105, 106). Another problem in these types of studies is the adjustment of total energy intake (105, 106) in populations. One can notice that when using several statistical methods for investigating the association between types of fatty acids in the diet and the prevalence of obesity different results may be achieved (105,106); one might acknowledge that total energy intake and the serving sizes reported are just estimated in most studies and may therefore impair the capacity of detecting an effect if present. There is also always the possibility of residual confounding factors which could in theory affect the observed association.

1.5.6 Conclusion

Although animal studies point to a possible association between the type of fatty acid consumed and obesity, and metabolic studies in humans follow that trend, the issue appears complex. In the nine epidemiological studies that directly examined the association between dietary fatty acid composition and body weight, the results have not been conclusive. In conclusion, given the potential public health implications of this question and the number of hypotheses that could be drawn about the consumption of different fatty acids and their association with obesity, there is an urgent need for conducting more studies—particularly case–control studies and analyses of large data sets (cross-sectional or longitudinal). Such studies were not feasible in the early 90s when

the specific fatty acid content of numerous foods items was largely unavailable in food composition tables. Today these studies are certainly feasible.

Table 1a. Metabolic studies in human on the association between dietary fat composition with change in body weight/body fat composition, rate oxidation, energy expenditure or thermogenesis

Authors	**Description**	**Out comes**			
		oxidation rate	**weight change/ fat deposition**	**energy expenditure(EE)**	**thermogenesis**
Scalfi et al. (107)	6 lean and 6 obese men. 38 grams (g) LCT and 30 g MCT and 8g of LCT				↑ with the MCT diet
Dulloo et al (78)	8 men. 4 type of diet: MCT/LCT ratio (0:30, 5:25, 15:15, 30:0)			Increase with ↑ MCT/LCT ratio	
Binnert et al (108)	8 obese and 8 control. Olive oil and a mixture of LCT, MCT (30 g of oil was given for 6 hours.	MCT>LCT in both groups			
Papamandjaris et al. (109)	12 women, normal to overweight. 40% of energy from fat. MCT (butter and coconut oil). LCT (beef tallow) for 14 days	MCT ↑ endogenous oxidation of LCT (saturated)			
St-Onge et al (74)	17 obese women. 40% of fat MCT or LCT for 27 days			↑ with MCT diet	
St-Onge et al (76)	19 overweight men. 40% of fat MCT or LCT for 4 weeks	↑ with MCT diet in men with high weight		↓ with MCT diet in men with higher weight	
Matsuyama et al (110)	11 males and female (obese children) Diet rich in diacylglycerol (DAG) or		↓ total and subcutaneous		

	Triacylglycerol (TAG) For 5 months in daily use cooking oil.		fat areas with DAG intake		
Hainer et al. (72)	11 obese. 15 ml/day of a mixture: MCT for 2 weeks			MCT administration prevent the decline of EE.	
Piers et al. (28)	14 normal to overweight men. 43% of total fat MUFA (olive oil) or SFA (cream) for 1-2 weeks.	↑ with MUFA intake			↑ with MUFA intake
Piers et al (111)	8 overweight men SFA, MUFA, PUFA for 4 weeks		with MUFA intake ↓		
Clifton et al. (92)	62 women. Isocaloric diet. 35% of energy coming from MUFA or very-low fat diet with 12% of energy coming from fat during 12 weeks		No difference between groups		
Soares et al (88)	12 women, normal to obese, for 5 hours Cream (40% of energy coming from fat) and olive oil extra virgin (41% of energy coming from fat).	↑ with olive oil in obese women.			↑ with olive oil in obese women.
Pieterse et al. (112)	55 men and women. Isocaloric diet with 30% of energy coming from fat, avocado or mixed oil, during 6 weeks		No difference between groups		
Jones et al. (113)	7 non obese and 8 obese men. P/S ratio and L/O ratio (linoleic/oleic)	Decrease with a ↓ P/S ratio in obese			

MacDougall et al (114)	8 men. 40% of energy from fat. Butter, tallow or corn oil for 11 days	Myristic acid> Palmitic acid		
Christiansen et al. (29)	16 obese patients. 20% of energy coming from trans fatty acids or saturated fatty acids during 6 weeks	No change		
Kien et al. (89)	43 adults. 40% of energy derived from fat. Oleic or palmitic fatty acids for 28 days.	↑ with oleic fatty acid		↑ with oleic fatty acid
Couet et al (98)	6 non obese men. 6g/ day of fish oil (FO) for 3 weeks. 32% of energy was derived from fat.	Increase with FO diet	↓ fat deposition with FO diet	
Delany et al (30)	4 men. 40% of total fat. Laurate, palmitate, stearate, oleate, elaidate, linoleate, linolenate Test duration for 9 hours	Laurate >others LCFA ↓ with ↑of carbon length		

Table 1b. Epidemiological studies to determine whether fat composition is associated with change in body weight/body fat composition, rate oxidation or energy expenditure

Authors	Study design	Purpose of study	Outcomes
Kafatos et al. (36)	Cohort study 245 men	Relation between heart disease risk-factor status and dietary change in a Cretan sample	+ association between BMI, MUFA intake and concentration of adipose palmitic acid
Doucet et al. (32)	Cross-sectional study 128 men in Quebec	Relation between dietary fat composition and adiposity in men	+ association between BMI and total fat, SFA and MUFA intake
Leung et al. (104)	Cohort study 124 child	Relation between dietary fat intake of Chinese and of Hong Kong children and obesity and biochemical markers	Null association between obesity and type of fat
Gonzales et al. (115)	Cross-sectional study 23289 women and 1437 men	Relationship between BMI, obesity and the consumption of different types of fat and olive oil	+ association between BMI and SFA, MUFA, PUFAs intake
Williams et al. (34)	Cross-sectional study 334 women and 311 men	Association between dietary patterns and glucose intolerance and other features of metabolic syndrome	+ correlation between central obesity and fried/sausages and - correlation between central obesity and fish intake.
Brunner et al. (35)	Cross-sectional study 869 women and men	Effects of PUFAs and carbohydrates intake on obesity and metabolic variables	+ association between waist/hip ratio and PUFA intake in women
Sundstrom et al. (37)	Cohort study 475 Sweden men	Predictors factors associated with echocardiographic left ventricular hypertrophy and geometric subtypes at age 50 years and after 20 years of follow up	+ association between BMI and SFA, MUFA intake - association between BMI and PUFAs (linoleic acid) intake

Trichopoulou et al. (94)	Cross-sectional study 27862 men and women	Effect of energy intake on body mass index depending on the source of energy	Null association between BMI and MUFA
Schroder et al. (116)	Cross-sectional study 1547 men and 1615 women	Relation between BMI, obesity and the traditional Mediterranean (Med) diet	- association between BMI and Med diet
Shubair et al. (102)	Cross-sectional study 759 adults	Association of the Med diet with healthy body weight	- association between BMI and Med diet
Trichopoulou et al. (95)	Cross-sectional study 23597 men and women	Adherence to a traditional Med diet is associated or not with BMI and waist-to-hip ratio	Null association between BMI or waist to hip ratio and Med diet rich in olive oil
Bes-Rastrollo et al. (96)	Cohort study 7368 Spanish males & females	Association between dietary olive oil and the incidence of overweight or obesity	Null association between BMI and higher amounts of olive oil intake
Panagiotakes et al. (117)	Cohort study 1514 men, 1528 women	Association between Med diet and weight gain	- association between BMI and Med diet
Sanchez-Villegas et al. (38)	Cohort study 6319 subjects	Association between Med diet and weight gain	+ association between BMI and Med diet

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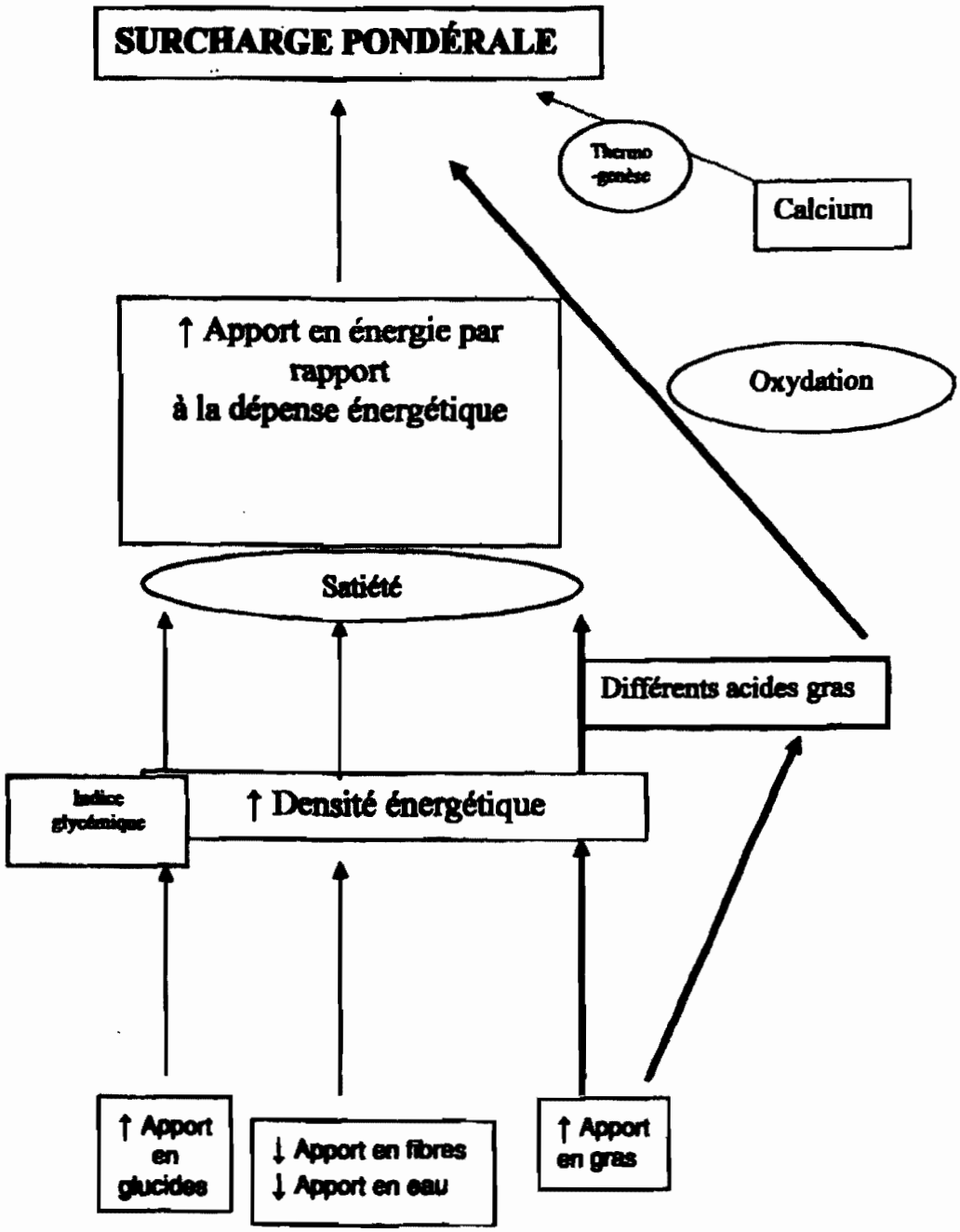
CHAPITRE 2 PROBLÉMATIQUE, OBJECTIFS ET HYPOTHÈSES

2.1 PROBLÉMATIQUE

Le cadre conceptuel (figure a) illustre les mécanismes nutritionnels principaux qui pourraient être impliqués dans le développement de l'obésité. Dans ce schéma, les facteurs alimentaires qui ont été étudiés dans la littérature en relation avec la surcharge pondérale sont présentés. Un apport en glucides élevé et un apport élevé en gras, un apport faible en eau ainsi qu'en fibres alimentaires agissent sur la densité énergétique d'une façon positive qui agit sur la satiété. Une surconsommation énergétique peut alors être observée. Cette dernière mène à la surchage pondérale. La quantité des lipides pourrait jouer un rôle dans le poids via la densité énergétique mais aussi la qualité des gras peut jouer un rôle dans le corps via l'oxydation. Certains acides gras tels les chaînes courtes ainsi que moyennes s'oxydent plus rapidement que ceux à chaînes longues. Les acides gras insaturés semblent de plus s'oxyder plus rapidement que les acides gras saturés. Finalement, le calcium pourrait affecter la surcharge pondérale via la thermogenèse. Dans cette thèse, nous avons examiné la relation entre la quantité totale de gras ainsi que les différents acides gras alimentaires et l'obésité (les flèches épaisses dans la figure a.).

Figure a. Cadre conceptuel soulignant l'importance de l'alimentation dans l'obésité



Parmi les différents facteurs impliqués dans le développement de l'obésité, tel que vu précédemment, des études avec des modèles animaux et des études métaboliques chez les humains suggèrent que le type de gras consommé pourrait avoir un effet sur le développement de l'obésité. Il semblerait qu'une alimentation riche en acides gras insaturés, en MCFA ainsi que SCFA, en acide gras oléique et avec des ratios riches en PUFA/SFA et oméga-6/oméga-3 soit associés à un risque moindre d'obésité. De plus, les LCFA et particulièrement l'acide gras palmitique (C16 :0) et stéarique (C18 :0) ont été rapportés dans la littérature comme étant associés à un risque élevé d'obésité tandis que l'opposé semble possible.

2.2 OBJECTIFS

Le but de notre étude est de caractériser à l'aide de devis écologique et transversal la présence d'association entre le profil d'acides gras et la prévalence d'obésité dans différentes populations.

D'abord, notre étude considère l'association possible entre la prévalence de l'obésité et la consommation de gras (quantité et qualité) dans différents pays (devis écologique). Ensuite, nous avons sélectionné une population où le taux d'obésité est élevé et analysé en détail le profil d'acides gras (rappel de 24 heures) dans leur alimentation, en lien avec la prévalence d'obésité (étude transversale).

Parmi les différents facteurs impliqués dans le développement de l'obésité, tel que vu précédemment, des études avec des modèles animaux et des études métaboliques chez les humains suggèrent que le type de gras consommé pourrait avoir un effet sur le développement de l'obésité. Il semblerait qu'une alimentation riche en acides gras insaturés, en MCFA ainsi que SCFA, en acide gras oléique et avec de ratio riche en PUFA/SFA et faible en oméga-6/oméga-3 soit associés à un risque moindre d'obésité. De plus, les LCFA et particulièrement l'acide gras palmitique (C16 :0) et stéarique (C18 :0) ont été rapportés dans la littérature comme étant associés à un risque élevé d'obésité tandis que l'opposé semble possible.

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2.3 QUESTIONS SPÉCIFIQUES

1. Existe-il au niveau international une association entre la prévalence d'obésité et le type de gras consommés : saturés, monoinsaturés et polyinsaturés (étude écologique)?
2. Dans une population à forte prévalence d'obésité (Crie de la Baie-James), la présence d'obésité est-elle associée à un type de gras consommé : trans, saturés, monoinsaturés, polyinsaturés, ratio PUFA/SFA, MUFA/SFA et/ou acides gras myristique, palmitique, stéarique, oléique, eicosapentaénoique, dcosahexahéñoique, oméga-6, oméga-3 et le ratio de ces derniers gras (étude transversale)?

2.4 RÉSUMÉ DES MÉTHODES UTILISÉES

2.4.1 Étude écologique

Celle-ci consiste en 168 pays ayant des données sur la prévalence d'obésité et les bilans alimentaires. La prévalence d'obésité est obtenue chez les femmes âgées de 15 ans et plus, car davantage de données existent pour les femmes que pour les hommes. La prévalence d'obésité en pourcentage dans chaque pays a été collectée du *Fifth Report, WHO* (OMS), 2003. En ce qui concerne l'apport différentiel d'acides gras qui existe dans chaque pays, les données de FAOSTAT sont analysées. Ces données sont des estimés de la nourriture accessible à la population de chaque pays à partir desquelles il est possible d'estimer la quantité d'acides gras saturés, mono- et

polyinsaturés disponibles à la consommation. Pour ce faire, la quantité moyenne de gras disponible quotidiennement de chaque aliment est divisée en quantité d'acides gras saturés, mono- et polyinsaturés disponibles à la consommation à partir de leur contenu documenté dans le fichier d'aliments Canadien et/ou Américain. Pour chaque pays, la moyenne des calories totales, des lipides totaux et de chaque type de gras SFA, MUFA et PUFA par capita sont calculées pour les cinq années de 1998 à 2002.

Des corrélations simples non paramétriques (Spearman) entre la prévalence d'obésité, les calories totales, le gras total, et le type de gras, SFA, MUFA, PUFA seront effectuées. Ces corrélations de Spearman protègent contre l'influence excessive d'un ou de quelques pays dans cette petite base de données. Les mêmes corrélations seront reproduites en contrôlant pour les calories disponibles dans chaque pays.

2.4.2 Étude transversale

La banque de données de Santé Québec des Cris de la Baie James offre la possibilité de tester les associations d'intérêt dans une population (N=833) à haute prévalence d'obésité. L'IMC de chaque individu sera la variable dépendante catégorisée selon les recommandations de l'OMS en quatre groupes : IMC dans l'intervalle du poids santé (18,5-25 kg/m^2), IMC dans l'intervalle de l'embonpoint (25-30 kg/m^2), IMC dans l'intervalle de l'obésité (30-35 kg/m^2) ainsi qu'IMC $\geq$35

kg/m^2. Après stratification pour le sexe, étant donné que la relation entre l'alimentation et la prévalence d'obésité a été rapportée comme variant entre les hommes et les femmes, des ANOVA seront testées pour les calories totales, le gras total, le type de gras (SFA, MUFA et PUFA), le ratio (PUFA/SFA), celui de (MUFA/SFA) ainsi que le type d'acides gras spécifique consommé calculés à partir des rappels de 24-h et du fichier Canadien d'aliments. Les acides gras spécifiques d'intérêt sont les myristique, palmitique, stéarique, oléique, linoléique, linolénique, eicosapentaenoique, docosahexaenoique, oméga-6, oméga-3 et le ratio de ces deux derniers acides gras ainsi que les MCFA et LCFA total seront considérés. Les analyses seront finalement ajustées pour l'âge.

Des valeurs de $P < 0.05$ seront considérées comme statistiquement significatives et celles de < 0.10 comme indicatives de tendances.

Au niveau éthique, aucune évaluation n'était nécessaire par le comité de la recherche de la faculté de médecine puisqu'il s'agit là des analyses secondaires de données publiques.

CHAPITRE 3 ARTICLE II

Is obesity related to the type of dietary fatty acids? An ecological study

Nadiah Moussavi. Dtp., Msc. (PhD candidate)
Victor Gavino. PhD (Professor of Nutrition, Department of Nutrition)
Olivier Receveur. PhD (Professor of Nutrition, Director of Graduate studies, Department of Nutrition)
Department of Nutrition, Faculty of Medicine, Université de Montréal, Canada.

Key words: Dietary fatty acids, Obesity, Ecological study

Nutrition Department, Faculty of Medicine, Université de Montréal, C.P 6128, Succ. Centre-Ville, Montreal, Qc. (Email [redacted]).

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3.1 ABSTRACT

Background: Animal studies and a few clinical trials lend credibility to the hypothesis that not all types of fatty acids carry the same potential for weight gain. Only a few epidemiological studies concerning this issue are currently available and results are conflicting. Aim: The purpose of the present ecological study was to test the existence of an association between obesity prevalence and the types of fat available in 168 countries. Methods: Data on the prevalence of obesity (BMI ≥30 kg/m2) for women over 15 years of age were obtained from the WHO Global InfoBase. Food balance sheets for the years 1998 to 2002 were obtained from the FAOSTAT database. Fiveyear means for energy, total fat, MUFA, PUFA, SFA and 'other fat' per capita were calculated, with their standard deviations, for each country. Bivariate correlations and a multiple linear regression model were used to test for the association between prevalence of obesity and types of fat available in these countries. Results: Not surprisingly, dietary energy supply, SFA, PUFA and 'other fat' were positively associated with the prevalence of obesity. We also found, however, a strong negative association between MUFA availability and obesity prevalence (ß=-0,68, p<0,0001). Conclusion: Populations with a lower prevalence of obesity seem to consume a greater amount of MUFA. Considering the partial correlations between variables, our results suggest that in countries with higher obesity prevalence, it is the shift from MUFA to PUFA that particularly appears to be associated with the risk of obesity.

Keywords: Dietary fatty acids, Obesity, Ecological study

3.2 INTRODUCTION

The prevalence of obesity has increased all over the world (1). Obesity may lead to morbidity such as hypertension (2) and type 2 diabetes (3), and premature mortality (4, 5). Some authors have stated that dietary fat can contribute to obesity via passive over-consumption, because this macronutrient is less satiating than eithercarbohydrates or proteins (6) and is the most energy-dense macronutrient (6, 7). Recently, attention has been drawn to the type of fatty acids in the diet because of their differential metabolism, which is explained mostly by their chain length, saturation degree and stereoisomeric configuration (8–11). Some investigators have proposed that dietary fat composition, independently of the amount of fat intake, can affect the development of obesity (8).

Authors have suggested that short- and medium-chain fatty acids have a higher oxidation rate (8–11) and may prevent obesity (11–13). Others have reported that dietary MUFA (14, 15), particularly oleic acid such as found in olive oil(16), and PUFA(17–19), especially those found in fish oil, may promote weight loss. Although outcomes are not always consistent (20–22), these results are reported mostly in animal studies and in a few clinical trials. Few studies in epidemiology concerning this issue are currently available and the results are conflicting (22–29). To examine the general trend in the world on the relationship between type of fat available for human consumption and obesity, an ecological study was conducted. The purpose of

the study was to test for the existence of an association between obesity prevalence and types of fat available in 168 countries.

3.3 METHODS

An ecological study of 168 countries was conducted. Data on the prevalence of obesity (BMI ≥30 kg/m2) among women aged 15 years and over were obtained from the WHO Global InfoBase (30). Food balance sheets (FBS) for the years 1998 to 2002 were obtained from the FAOSTAT database (31). Five-year averages for energy, total fat, MUFA, PUFA, SFA and 'other fat' per capita were calculated. The category 'other fat' is a category in the FBS regrouping all oils that have not been listed separately as other items. Their fatty acids content cannot therefore be estimated. We used the US Department of Agriculture Nutrient Data Laboratory database (32) and the Canadian Nutrient File (33) to derive fatty acids from the types of fat available for human consumption in each country. One hundred and sixty-eight countries were selected according to the availability of FBS in the FAOSTAT database and BMI percentage in the WHO website. According to FAOSTAT, the FBS presents a comprehensive picture of a country's food supply pattern during a specific period. For each food item the FBS shows what is potentially available for human consumption, referring to the sources of supply and utilisation. Furthermore, the FAOSTAT database also gives the per capita supply of each food item available for human consumption, obtained by dividing the respective quantity by the population actually consuming it. These 'per capita' figures refer to oneyear

availability of food supply. All data are presented as means with standard deviation. Spearman correlations between obesity prevalence and potential predictors (energy, total fat, PUFA, MUFA, SFA and 'other fat' in g/capita per d) were performed. To elucidate the relationship between the type of fat available in these countries and obesity prevalence we conducted multiple linear regression models. Statistical significance was accepted at the 5% level. All analyses were performed using the Statistical Analysis Systems statistical software package version 8 (SAS Institute, Cary, NC, USA).

3.4 RESULTS

The characteristics of the various countries are presented in the Appendix. The prevalence of obesity ranged from 0% in Ethiopia to 49,2% in Kuwait. There was a wide variation in total fat consumption, from 10,5 g/capita in Burundi to 159,1 g/capita in Belgium. Means and standard deviations for the variables studied, together with Spearman correlation coefficients for the association of dietary variables with the prevalence of obesity, are presented in Table 1. Significant positive correlations were observed between obesity prevalence and energy (0,48), total fat (0,51), MUFA (0,41), PUFA (0,43), SFA (0,45), and 'other fat' (0,41). Furthermore, the types of fat were correlated positively with each other, with energy and total fat, and all results were statistically significant. The contribution of each fat group is also presented in Table 1 as a percentage of total energy intake, since recommendations are often reported in such terms. Similarly to the absolute contributions, the percentage

contribution of each type of fat also increased in countries with higher obesity prevalence, but the correlations, although all still significant, were weakened slightly.

Table 1 Mean and standard deviation of variables studied in 168 countries and Spearman correlations (r) between obesity prevalence and energy, total fat, SFA, MUFA, PUFA and 'other fat'.

Variables	Mean±SD	Rho (P*)
% obesity	16.1± 11.7	
Calories (kcal)	2683±502	0.48 (<0.0001)
Total fat (g)	76.6±35.4	0.51 (<0.0001)
% energy from tot fat	24.7±7.9	0.43 (<0.0001)
Saturated fat (g)	25.5±13.9	0.45 (<0.0001)
% energy from SFA	8.2±3.7	0.36 (<0.0001)
Monounsaturated fat (g)	23.4±13.7	0.41 (<0.0001)
% energy from MUFA	7.4±3.3	0.33 (<0.0001)
Polyunsaturated fat (g)	16.5±8.4	0.43 (<0.0001)
% energy from PUFA	5.4±2.2	0.37 (<0.0001)
Other fat (g)	11.5±5.3	0.41 (<0.0001)
% energy from other fat	3.7±1.5	0.27 (<0.0003)

*P value <0.05 are significant

Table 2 Results of multiple linear regression analyses of dietary variables v. obesity prevalence (percentage of women in the population with BMI ≥30 kg/m2) as dependent variable in 168 countries

Variables (Per capita)	Adjusted regression coefficient	Standard Error	P*
Calories (kcal)	0.007	0.003	0.02
Saturated(g)	0.38	0.09	<0.0001
Monounsaturated(g)	-0.68	0.13	<0.0001
Polyunsaturated(g)	0.68	0.15	<0.0001
Others fat (g)	0.44	0.18	0.02

*P value <0.05 are significant.

R^2=0.32

We conducted multiple linear regression analyses to separate the relationships of each type of fat with obesity prevalence controlling for per capita energy intake. Note that the sum of all four types of fat (SFA, MUFA, PUFA and 'other') equals the total fat per capita and therefore this last variable was not included in the model. As expected, SFA (B=0,38; P<0,0001), PUFA (B=0,68; P<0,0001) and 'other fat' (B=0,44; P=0,02) were significantly positively associated with obesity. However, we found a significant negative association (B=-0,68; P=0,0001) between MUFA availability and the prevalence of obesity (Table 2).

3.5 DISCUSSION

The main result of the present paper is that, in spite of the significant positive association between obesity prevalence and total fat availability, MUFA availability is significantly negatively associated with the prevalence of obesity. It suggests that populations with lower obesity prevalence seem to consume greater amounts of MUFA, but such association cannot be taken as causal with our ecological study design. Nevertheless, this finding supports results from a few epidemiological studies reporting that the Mediterranean diet seems to be beneficial to weight loss (16, 34, 35). In these studies, the authors specifically considered the consumption of olive oil, and not all types of MUFA in the diet. In contrast, other studies have reported that olive oil or the Mediterranean diet may promote weight gain (21, 24, 25). Yet other investigators have not shown any relationship between a high consumption of MUFA and the prevalence of obesity (29, 36, 37). Some clinical trials (15, 38–41) but not all

(21) have demonstrated that MUFA have a higher oxidation rate than SFA. In fact, the mechanism underlying this negative relationship, according to these studies, is that MUFA intake increases diet thermogenesis, which in turn stimulates the sympathetic nervous system (39), and abdominally obese subjects may be more responsive to stimulation of the sympathetic nervous system because they have an increased density and sensitivity of b-adrenoreceptors (42). Similarly, some studies (43) in mice demonstrated that MUFA consumption might have an anti-obesity action. These authors reported that MUFA intake may stimulate fat utilisation through activation of the nuclear receptor, PPAR-a. Others (44) have demonstrated that rats with a high MUFA intake may gain weight.

Our multivariate model also suggests that, in countries with higher prevalence of obesity, dietary MUFA tend to give place to some SFA and more so to PUFA consumption. In fact, it has been reported that a high PUFA intake may promote weight gain (25, 27). When comparing eighty-eight children from Crete and Cyprus, two Mediterranean islands, regarding the association of adipose tissue arachidonic acid content with BMI and overweight status, Savva et al. (45) found higher mean levels of arachidonic acid, dihomoglinolenic acid and DHA in overweight and obese children. A positive association between adipose tissue arachidonic acid and BMI was noted. On the other hand, Ailhaud et al. (46) reported that the inclusion of a-linolenic acid coming from PUFA in an isoenergetic diet rich in linoleic acid prevents increase of fat mass in pups. The authors highlighted that these data were consistent with their previous in vitro results comparing the adipogenic effect of n-6 PUFA and

n-3 PUFA. Concerning SFA consumption and weight change, Doucet et al. (23) and Gonzalez et al. (25) reported a higher consumption of SFA in obese populations. Furthermore, some clinical trials (15, 40) have demonstrated a higher oxidation rate in subjects who were consuming MUFA than in a group with SFA intake, for an isoenergetic diet. Kien et al. (15) suggested that a high SFA intake (palmitic acid) may increase the obesity rate. Sanders (47) demonstrated that populations with higher MUFA consumption tend to have lower intake of SFA, but we did not find such an association at the ecological level.

The present study has some positive points. First, the data on obesity prevalence were derived for all countries from the same recent WHO data set (30). FBS were also derived from one online database, FAOSTAT. These FBS represent the pattern of a country's food supply during one year. Moreover, according to FAOSTAT, the quantity of foodstuff produced in a country added to the total quantity imported and adjusted to any change in stocks during a period of time gives the availability of supply during that period. These tables provide a useful reference for fat consumption for all countries (31).

For the statistical analysis we carried out multiple linear regression analyses to adjust for energy and estimate the respective contribution of each group of fats. This model explains 32% of the variance found in the prevalence of obesity.

However, there are some limitations. Obviously, we cannot assume a negative cause-and-effect relationship between MUFA intake and obesity prevalence because the potential bias of ecological fallacy is always possible. This relationship may be totally or partially confounded by other unmeasured variables such as physical activity, geographical situation, consumption of dietary fibre, and fruit and vegetable intake. We are conscious of the fact that the FBS gives the food supply availability for the entire population in a country but obesity percentages taken into account in the present paper only include women aged 15 years and over. Consequently this relationship might be different for men, but the prevalence of obesity among men and women in a country is probably highly correlated. Another potential limitation is utilisation of the FBS, which is an estimation of the food supply available for human consumption in a given country, and that the validity of national reports may vary from country to country. The potential consequences of these variations in our analysis cannot be estimated. Also, the 'other fat' category that we had to use must have added imprecision to our estimates. An associated bias is nevertheless unlikely since its absolute contribution is small and represents probably a variety of fats. Finally, the availability for human consumption of more specific types of fatty acids and the n-6:n-3 ratio could not be taken into consideration for statistical analysis in the present paper, because of the imprecision and missing values of some particular items in the FBS.

This is the first ecological study to consider the type of fat and the prevalence of obesity in a large data set of 168 countries, since data on obesity from the WHO

became available only recently. Our analysis suggests that additional studies on the potential role of MUFA in obesity are needed. Future use of online data sets is also encouraged.

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3.7 Annexe I. Characteristics of 168 countries

Country	Obesity (%)	Tot calories (kcal)	Tot fat (g)	SFA (g)	MUFA (g)	PUFA (g	Other fat (g)
Albania	23,8	2818	83,4	29,5	22,4	19,4	12,1
Algeria	11,9	2986	69,2	15,9	20,3	24,1	8,9
Angola	5,9	2009	41,3	12,3	12,5	10,9	5,6
Antigua	21,5	2313	81,8	24,8	22,7	16,4	17,9
Argentine	27,1	3116	109,7	36,1	33,9	34,5	6,7
Armenia	19,8	2195	41,3	14,5	11,6	8,2	7
Australia	22,5	3077	133,6	48,8	50,4	18,7	16,2
Austria	20,4	3724	162	52,6	54,7	28,2	26,8
Azerbaijan	24,9	2403	38,4	15,6	11,7	7,3	3,8
Barbados	46,7	3024	98,3	28,4	27,9	19,4	22,6
Bahamas	25,6	2675	93,9	29,5	28,2	10,4	25,8
Bangladesh	0,1	2262	26,1	6,9	8,4	7,5	3,3
Belarus	32,2	3053	100,2	35,3	30,9	20,4	13,6
Belgium	9,5	3605	159,1	60,2	50,6	26,1	22,2
Belize	17,2	2795	67,3	22,3	21	10	14
Benin	6,2	2495	46,3	12,3	13,9	14,2	5,9
Bolivia	28,8	2169	54	13,7	15,8	16,1	8,4
Bosnia-Herz	21,5	2649	56,3	17,8	16,4	11,9	10,2
Bostwana	12,9	2158	50,9	13,9	11,7	14,8	10,5
Brazil	15	2995	91	29,1	26,8	26	7,4
Brunie Dar	25,9	2842	74	25,5	22,1	12	14,4
Bulgaria	19	2811	94	27,5	27,4	30,5	8,6
Burkina Faso	1,1	2393	53	11,5	17,4	16,1	8
Burundi	1,2	1638	10,5	2,4	2,1	2,6	3,4
Cambodia	0,1	1998	29,4	10,4	10,4	4,8	3,8
Cameroon	9,2	2231	45,7	14,1	13,1	10	8,5
Canada	22,2	3530	147	41,9	59,2	29,8	15,5
Cape Verde	11	3194	98,6	25,8	29,1	29	14,7
Czech Rep	20	3145	114,6	34	44,2	22,5	14
Central Africa	1,1	1935	60	15,5	18	12,2	14,3
Chad	1,3	2121	66,6	12,6	25,5	16,6	11,9
Chile	27,2	2831	83,1	24,2	26,9	23,3	8,7
China	1,5	2962	84,1	24,1	32,7	18,6	8,5
Colombia	20,3	2567	65,3	25,4	19,9	12,8	7,2
Comoros	5,8	1750	40,1	28	4,9	2,9	4,3
Congo	2,7	2009	49,5	13,2	13,6	11,6	11,1
Congo demo	0,6	1660	26,7	8,4	8,1	4,5	5,7
Costa Rica	22,7	2839	76	28,5	22,2	16,8	8,5
Cuba	20,7	2973	50,6	17,6	14,6	14,2	4,2
Coted'Ivore	4,8	2614	57,2	20,7	18,2	9,6	8,7
Croatia	15,4	2722	79,1	26,5	29,1	22,3	1,6

Cyprus	20,7	3238	128,9	37,6	43,3	28,3	19,6
Denmark	6,4	3399	137,8	54,1	49,1	22,1	14,3
Djibouti	5	2126	62,8	25,6	18,9	8,8	9,5
Dominica	41,8	2742	75,3	29,8	18,9	10,3	16,3
Dominica Rep	27,8	2301	80,5	26,3	21,3	24,3	8,6
Ecuador	15,4	2717	93,2	38,3	31,3	16,2	7,4
Egypt	39,3	3342	58,4	14,9	14,4	17	12,5
Elsalvador	16,5	2486	57,1	18,8	15,6	15	7,7
Eritrea	0,1	1534	24,8	4,6	5,2	5	10
Estonia	8,4	3094	96,7	35,6	26,1	12,6	22,4
Ethiopia	0	1791	25,3	4,2	3,4	3	14,7
Fiji I	29,8	2876	97,4	41,4	24,5	20,8	13,2
Finland	17,5	3127	124,3	46,4	45,3	16,5	15,2
France	6,1	3616	167,7	57,3	55,8	29,9	22,5
Gabon	24,5	2617	59,1	15,1	17,9	11,2	14,9
Gambia	1,9	2266	74,4	14,8	25,3	24,6	9,7
Germany	19,2	3442	144,8	50,5	47,3	24,7	20
Georgia	13,4	2398	45	14,5	11,6	8,7	10,2
Ghana	3,5	2578	38,5	8,3	7,1	3,4	19,7
Grenada	19,8	2865	101,1	36	22,8	15,8	26,5
Greece	23,4	3671	149,8	36,9	64,5	27,5	18,9
Guatemala	25	2211	47	11,3	12,2	15,5	8
Guinea	4,2	2343	54,9	16,2	19	11,6	8,1
Guinea-Bis	2,4	2076	49,9	17,2	15,2	9,6	7,9
Guyana	15,6	2678	51,7	27	12,2	6,6	5,9
Haiti	8,2	2064	41,4	8,4	9,5	12,6	10,9
Honduras	13,1	2358	65,3	27	21,3	11,3	5,7
Hungary	16,1	3421	141,3	47,2	49	33,6	11,5
Iceland	22	3186	126,9	51,5	40,4	20,6	14,4
India	1,1	2406	49,5	16,2	14,2	10,4	8,7
Indonesia	2	2875	58,8	27,4	15,3	9,6	6,5
Iran	25	3079	61,4	16,1	14,4	20,4	11,7
Ireland	8,4	3666	135,4	55,3	47,7	21,9	10,8
Italy	12,2	3676	157,1	47	64,5	30,1	16,7
Jamica	36,4	2646	73,3	24,4	19	20,7	9,2
Japan	1,5	2784	85,3	21,3	29,2	23,4	11,4
Jordan	40,2	2648	79,8	22,2	27	22,1	8,8
Kazakhastan	13,1	2479	69,5	23	18,5	13,9	14,1
Kenya	1,8	2131	48,7	18,4	14,8	8,7	6,8
Kiribati	37,9	2820	100,8	67,8	11,6	5,1	16,3
Korea	9,5	3034	74,2	20,7	21	22,5	10,1
Kuwait	49,2	3063	109,1	38,3	31	25,5	14,1
Kyrgystan	14,2	2999	54,3	22	15,6	8,6	8,1
Laos	9,2	2264	27,9	6,4	9,2	6	6,3
Latvia	15	2926	98,3	33,6	36,3	18,6	9,9

Lebanon	23,9	3162	112,6	25,9	31,6	29,8	25,3
Lesotho	33,2	2592	36	5,8	8	9,1	13,1
Liberia	9,6	2042	55	22,5	16,7	6,5	9,3
Libyan A Jam	21,1	3314	105,5	20,4	35,1	31,5	18,5
Lithuania	13,9	3306	91,7	29,4	30,6	16,8	14,9
Macedonia	24,3	2736	87,9	23,1	21,9	26,5	16,4
Madagascar	1,5	2043	29,6	7,4	9	5,8	7,4
Malawi	1,6	2143	29,7	5,2	8,5	9,9	6,1
Malaysia	6,8	2875	84	37,7	22,2	11,4	12,7
Maldives	20,2	2522	62	24,7	8,4	5,9	23
Moldova Rep	11,2	2682	52,1	13,4	11,8	15	11,9
Mali	3,4	2223	45,8	11,5	12,7	12,3	9,3
Malta	33,8	3520	111,8	40,2	37,5	20,4	16,6
Mauritania	20,6	2759	68,8	23	20,9	16	8,9
Mauritius	16,1	2945	82,9	21,6	21,4	29,7	10,6
Mexico	31,6	3142	86,4	25,1	25,2	22,6	14,5
Mongolia	24,6	2185	82,2	31,4	28,6	7	15,2
Morocco	19	3064	60	12,7	19,1	19,2	9,6
Mozambique	2,7	2000	33,4	9,2	8,1	10,1	6
Myanmar	8	2851	46,4	11,1	15,6	12,2	7,5
Namibia	4,9	2195	48,9	14,2	13,6	12,3	8,8
Nepal	0,2	2386	35,4	10,3	11,4	6,9	6,8
Netherlands	10,7	3304	143,7	55,7	46,1	24,7	17
NewZeland	26,7	3206	113,7	41,1	38,3	20,7	14,4
Nicaragua	28,3	2265	45,8	13,6	12,4	10,9	8,9
Niger	1,9	2154	37,2	10	11,5	9,7	6
Nigeria	4,9	2726	63,4	20,3	19,1	12,2	11,8
Norway	8,6	3404	139,8	50,2	45,5	28,1	14,4
Pakistan	2,9	2439	65,7	27,4	19,1	11,9	7,3
Panama	18,3	1850	68,3	21,9	20,5	17	8,9
Paraguay	15,8	2543	84,3	26,1	27,1	26,1	5
Peru	28,9	2535	46,5	13,7	12,4	14,9	6,3
Philippines	2,8	2366	47,3	21,6	15,7	6,7	3,3
Poland	18	3365	111,9	40,3	42	15,8	13,8
Portugal	14,6	3719	137,8	46	48,7	25,9	17,2
Romania	12	3370	93,5	28,8	25,6	26,6	13,5
Russia	23,6	2954	78,9	26,8	23,4	22	7,5
Rwanda	1,2	1944	15,6	4,5	4	2,9	4,2
StKitts Nevis	22	2619	82,5	28,3	20,3	15,1	18,8
St Lucia	30,5	2918	79	35,7	23,5	9,4	10,4
St Vincent G	17,8	2463	66,7	23,9	16,7	11	15,1
Samoa	55	2814	128,1	73,7	29,5	9,5	15,4
Saotome P	3,7	2330	68,9	44,8	7,5	3,7	12,9
S. Arabia	32,8	2835	82,7	30,2	24,4	15,5	12,3
Senegal	7,8	2270	68,4	14,7	22,2	25,1	6,4

Serbia Mont	20,6	2772	120,2	41,3	41,3	26,3	11,3
Sierra Leone	10,9	1939	46,6	19,6	14,4	6,5	6,1
Slovakia	21,3	2930	110	33,8	42,2	23,2	10,8
Slovenia	23,7	3003	105,8	36	34,3	22,3	13,2
Solomon Ist	13,4	2441	42,4	25,3	7,2	2,9	7
South Africa	34,3	2883	73,8	17,7	20,4	27,5	8,7
Spain	14,5	3345	150	39	58,3	37,3	16,5
Srilannka	0,1	2372	44,6	29,7	5,4	2,6	6,9
Sudan	4,3	2285	71	23,4	24,2	16	7,4
Sweden	10	3131	125,4	41,3	46,4	19,8	17,9
Swaziland	11,8	2357	45,4	12,7	11,7	8,8	12,2
Switzerland	16,4	3394	150,1	53,6	49,5	26	21,8
Syria	20,8	3051	103,9	27,1	29,5	24,8	22,5
Suriname	15,8	2638	68,9	21,2	18,5	23,8	5,4
Tajikistan	9,2	1815	35,4	10	7,8	12,8	4,8
Tanazania	2,8	1945	29,7	10,1	7,8	6,4	5,4
Thailand	7	2397	50,6	19,3	14,8	10	6,5
Timor Leste	14,2	2665	38,8	9,3	10,9	7,9	10,7
Togo	4,3	2298	44,8	15,4	12,4	10,4	4,6
Trin & Tob	41,9	2693	73,7	26	19,9	20,8	8
Tunisia	28,8	3309	97,7	22,6	34,1	29,5	12
Turkey	32,1	3358	89	25,3	24,4	27,8	11,9
Turkmenistan	15	2657	70,4	24,7	19	18,7	8
Seychells	35,8	2442	75,2	25,2	16,4	20,8	12,8
UAE	37,9	3184	96,6	32,7	27,4	20,3	17,8
Uganda	1,3	2335	31,4	7,5	10,1	7,9	5,9
U Kingdom	21,3	3386	144,8	45,6	54,8	26,3	13,6
Ukraine	19,4	2909	74,1	24,2	21	21	7,9
Urguay	19,6	2826	93,7	28,5	30,9	19,7	14,7
USA	37,8	3751	152,2	43,5	45,9	47,2	13,9
Uzbekistan	13,5	2349	66,4	22,3	16,6	20,5	7
Venezuela	22,4	2383	67,7	18,3	19,6	22,6	7,2
Vanuatu	23,4	2559	89	48,3	19	7,5	14,2
Vietnam	0,2	2504	41,5	14,1	14,8	6,2	6,4
Yemen	4,4	2034	38,8	14	11,8	8,3	4,7
Zambia	1,6	1892	30,8	6	8,4	11	5,4
Zimbabwe	14,1	2017	52,7	12,3	15,9	19,3	5,2

*total fat, calories, MUFA, PUFA, SFA and other fat represent all the quantities of fat available to human consumption per capita.

*Obesity prevalence (BMI $\geq$30kg/m^2) variable is presented in percentage in a country.

CHAPITRE 4 ARTICLE III

Is obesity related to the type of dietary fatty acids? A cross-sectional study in the Baie-James, Cree population.

Nadiah Moussavi. Dtp., Msc. (PhD candidate)

Victor Gavino. PhD (Professor of Nutrition, Department of Nutrition)

Jill Torrie MA (Cree Board of Health and Social Services of James Bay)

Olivier Receveur. PhD, MPH, Dtp (Professor of Nutrition, Department of Nutrition)

Department of Nutrition, Faculty of Medicine, Université de Montréal, Canada.

Key words: Dietary fatty acids, Obesity, Cross-sectional study

Nutrition Department, Faculty of Medicine, Université de Montréal, C.P 6128, Succ. Centre-Ville, Montreal, Qc. (Email [redacted]).

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4.1 ABSTRACT

Obesity rates are increasing worldwide. The exact etiology remains unclear but appears to be a combination of genetic, metabolic and environmental factors. Recently, dietary fatty acids in association with the risk of obesity has been considered. It has been reported that the Cree people of James Bay in Québec are experiencing a nutritional transition with changing dietary pattern and high obesity prevalence when compared to the rest of Canadians.

To examine, using secondary data, the association between type of fatty acids and risk of obesity in Cree population. A validated 24 hour recall was done in 833 healthy adults in 1990. We used the Canadian Nutrient File of 1997 in order to extract the following variables: total fat, trans fat, saturated fat (SFA), monounsaturated fat (MUFA), polyunsaturated fat (PUFA), medium chain fatty acids (MCFA), long chain fatty acids (LCFA), long chain saturated fatty acids only (LCSFA), myristic, palmitic, stearic, linoleic, linolenic, eicosapentaenoic, docosahexaenoic fatty acids, PUFA/SFA ratio, MUFA/SFA ratio and n-6/n-3 ratio, and test their association with BMI categorised in four levels according to World Health Organisation. We proceeded by ANOVA and then by ANCOVA, adjusting for age and stratifying for gender in two models. P value was considered significant at 0.05. In ANCOVA analysis, percentages of total trans, MUFA, palmitic, LCFA and fat intake were significantly different in women. There was no statistical difference in men for the studied variables across BMI different groups.

Despite the cross-sectional design of our study, we demonstrated an association between MUFA, palmitic, LCFA, trans fat, and total fat intake (in percentage) and obesity in women. More research is needed in order to better understand the association between types of fatty acids in the diet and risk of obesity.

Keywords: dietary fatty acids, obesity, cross-sectional study

4.2 INTRODUCTION

Obesity increases morbidity and mortality from several diseases, including cardiovascular diseases (1), diabetes type 2 (2), hypertension (3) and some forms of cancer (4). The exact etiology of obesity remains unclear and appears a complex combination between genetic, metabolic and environmental factors (5). Recently, some investigators took into consideration via animal experiments (6, 7) and human studies (8, 9), the association between obesity and the intake of specific types of fatty acids intake. Investigators reported that the types of fatty acids in the diet are metabolised differently in the body and this fact is explained mostly by their chain length, their degree of saturation and their stereoisometric configuration (10, 11). Nevertheless, there appears to be no agreement among authors on these relationships and the results from the few epidemiological studies (12-17) are controversial.

Cree people living around James Bay in Quebec, have a higher prevalence of excess body weight compared to the rest of Canadians (18, 19) and particularly for Cree women compared to other women in Quebec province (20). It has been reported in the literature previously that Indigenous people are experiencing a nutritional transition with a change in their dietary pattern (19). The objective of this study was to examine the association between types of fatty acids in the diet and prevalence of obesity in a population where the prevalence of obesity is high and the nutritional pattern and food choices are different from the rest of Canadians.

4.3 METHODS

Validated 24 hour recall was used to derive estimates of total fat, specific fatty acids and energy intakes. The recalls took place in the summer of 1991. The interviews for these 24 hours recalls were distributed in the seven days of the week. These recalls were precise, with the quantities, the food brand, the recipes and the cooking directions reported. Data were entered and treated with the CANDI program (Thompson, 1990). The sample included 833 healthy adults, men and women, aged from 18 to 74 years old. Weight in kilograms and height in meters were measured by the interviewers. The food items consumed by each respondent were coded according to the Canadian Nutrient File of 1990 (22). Details on the methodological aspects of data collection and data management have been previously published (21).

In order to analyse the data, we recoded the food items using the Canadian Nutrient File of 1997, because it contained more specific fatty acids, for more food items, than the one of 1990. Our variables of interest were total calories, total fat, trans fat, saturated fat (SFA), monounsaturated fat (MUFA), polyunsaturated fat (PUFA), medium chain fatty acids (MCFA), long chain fatty acids (LCFA), long chain saturated fatty acids only (LCSFA), myristic, palmitic, stearic, linoleic, linolenic, eicosapentaenoic, docosahexaenoic fatty acids, PUFA/SFA ratio, MUFA/SFA ratio and n-6/n-3 ratio. We included the above variables in our study because of our study's hypothesis (23).

We categorized BMI at four levels, according to World Health Organisation (WHO): 18,5-24.9 kg/m^2; 25-29,9 kg/m^2; 30-34,9 kg/m^2; 35 kg/m^2 and more.

In the first analysis we proceeded by ANOVA and in the second by ANCOVA adjusting for age, stratifying for gender (SPSS, version 13). In ANCOVA, we considered percentages of energy coming from the type of fat in association with each group of BMI.

P value at <0.05 was considered statistically significant, and representing a trend if <0.10.

4.4 RESULTS

Table 1 and 2 show the results for ANOVA analysis by gender. All the studied variables are higher in men than in women since energy intake is higher. BMI tends to increase with age for both men and women.

In women with a higher BMI both energy and fat intake increase. Total MUFA, PUFA, SFA and trans fat intake, as well as LCFA, LCSFA, palmitic, stearic, oleic, linoleic, and docosahexaenoic fatty acids are significantly different across BMI categories.

The first group of men, young men, had the highest energy intake and total fat intake. This group also had higher total MUFA, PUFA, SFA and trans fat intake than the others. Other differences, in this group, are for LCFA, LCSFA, palmitic, stearic, oleic and linoleic fatty acids.

We proceeded in Table 3 by ANCOVA adjusted for age, considering percentages of each type of fat in term of calories, in order to adjust in part for differences in energy intake across BMI categories. In women, one can notice some variables tend to increase with increasing BMI: total trans fat, MUFA and fat intake were significantly different (P=0.05). Percentage of LCFA intake increases with higher BMI (P=0.03) and palmitic fatty acid intake as well (P=0.05). There is no statistical difference in men for the studied variables in different groups in this table. The overall pattern of types of fat intake and specific fatty acids intake, by gender, are illustrated in figure 1a (for women) and b (for men).

Table 1. ANOVA of studied variables (mean± standard deviation) with BMI (kg/m^2) categories for women

	Group 1 BMI 18.5-24.9 N=57	Group 2 BMI 25.0-29.9 N=129	Group 3 BMI 30.0-34.5 N=133	Group 4 BMI ≥35.0 N=121	Total N=440	P value
Age (years)	27.7±10.9 a	34.4±14.4 b	33.9±13.8 b	39.7±14.0 c	34.8±14.2	**<0.01**
Calories (kcal)	1989±882	1877±677	1872±701	2116±1090	1955±847	0.08
Total fat (g)	76.6±45.1 ab	75.9±32.3	80.7±37.7 ab	93.6±71.7 b	82.3±49.5	**0.02**
Trans total (g)	3.55±3.98 ab	3.25±3.58 a	4.07±4.22 ab	4.95±4.98 b	4.01±4.28	**0.01**
Total SFA (g)	24.3±15.4	24.3±11.4	25.0±12.8	28.8±24.0	25.7±16.7	0.13
Total MUFA (g)	32.2±20.0 ab	32.2±14.7 a	35.0±18.4 ab	40.6±29.9 b	35.4±21.7	**0.01**
Total PUFA (g)	12.8±8.06 ab	12.4±6.11 a	13.2±6.15 ab	15.9±12.4 b	13.7±8.62	**0.01**
Tot MCFA (g)	0.94±0.78	1.04±1.19	1.05±1.00	0.99±0.99	1.02±1.03	0.89
Tot LCFA (g)	62.0±48.7 ab	60.9±37.5 a	64.4±45.3 a	83.3±68.6 b	68.3±52.0	**<0.01**
Tot LCSFA (g)	23.6±14.5 ab	23.3±10.6 a	24.6±12.2 ab	29.4±23.4 b	25.4±16.1	**0.01**
C14 :0 (g)	1.72±1.22	1.73±1.18	1.75±1.21	1.78±1.31	1.75±1.23	0.98
C16 :0 (g)	15.0±9.45 ab	14.74±6.69 a	15.8±8.06 ab	19.0±15.7 b	16.2±10.7	**0.01**
C18 :0 (g)	6.86±4.21 ab	6.76±3.27 a	7.03±3.60 ab	8.49±6.81 b	7.33±4.74	**0.02**
C18 :1 (g)	30.0±18.7 ab	29.8±13.8 a	32.4±17.4 ab	37.2±27.7 b	32.7±20.3	**0.02**
C18:2 (g)	10.8±7.23 ab	10.5±25 a	11.0±5.41 ab	13.1±11.0 b	11.4±7.60	**0.03**
C18 :3 (g)	1.60±0.89	1.66±1.06	1.83±1.17	1.90±1.17	1.77±1.10	0.20
C20:5(g)	0.05±0.11	0.06±0.15	0.08±0.26	0.13±0.32	0.08±0.24	0.07
C22:6 (g)	0.18±0.45 ab	0.15±0.38 a	0.19±0.35 a	0.43±1.12 b	0.25±0.68	**<0.01**
PUFA/SFA ratio	0.58±0.27	0.58±0.32	0.60±0.30	0.66±0.48	0.61±0.36	0.32
MUFA/SFA ratio	1.40±0.56	1.42±0.51	1.48±0.54	1.56±0.68	1.47±0.58	0.21
n-6/n-3 ratio	3.06±12.21	5.51±37.46	4.09±23.99	5.31±14.97	4.71±25.75	0.92

P value <0.05. Means not sharing the same subscript are significantly different.

Table 2. ANOVA of studied variables (mean± standard deviation) with BMI (kg/m²) categories for men

	Group 1 BMI 18.5-24.9 N=86	Group 2 BMI 25.0-29.9 N=155	Group 3 BMI 30.0-34.5 N=112	Group 4 BMI ≥35.0 N=40	Total N=393	P value
Age (years)	29.3±11.8 a	34.3±13.5 c	39.2±13.1 b	43.5±14.8 b	35.5±13.8	**<0.01**
Calories (kcal)	2984±1220 a	2411±988 b	2582±1071 a b	2648±1107 a b	2609±1095	**<0.01**
Total fat (g)	134.3±67.8 a	106.0±56.3 b	115.6±59.8 a b	114.3±65.4 a b	115.8±61.6	**0.01**
Trans total (g)	7.36±7.38	5.24±6.25	5.99±5.89	6.53±6.49	6.05±6.47	0.10
Total SFA (g)	41.5±24.0 a	33.0±18.4 b	35.4±19.1 a b	34.2±21.8 a b	35.7±20.5	**0.02**
Total MUFA (g)	57.6±29.7 a	45.6±26.1 b	50.0±28.0 a b	50.6±30.3 a b	50.0±28.1	**0.02**
Total PUFA (g)	22.3±14.8 a	17.7±11.3 b	19.6±11.5 a b	19.2±10.4 a b	19.4±12.2	**0.05**
Tot MCFA (g)	1.49±1.74	1.16±1.30	1.10±1.00	1.09±1.24	1.21±1.33	0.16
Tot LCFA (g)	118±68.0 a	89.9±59.6 b	101±62.9 a b	98.2±72.6 a b	100±64.5	**0.01**
Tot LCSFA (g)	41.8±23.1 a	33.1±17.7 b	35.3±19.1 a b	35.1±21.6 a b	35.8±20.0	**0.01**
C14 :0 (g)	2.75±2.39	2.16±1.83	2.11±1.40	2.21±2.21	2.28±1.91	0.08
C16 :0 (g)	26.8±15.0 a	21.3±11.6 b	22.6±12.2 a b	22.8±13.9 a b	23.0±12.9	**0.02**
C18 :0 (g)	12.1±6.86 a	9.49±5.11 b	10.3±5.98 a b	10.1±6.13 a b	10.4±5.94	**0.01**
C18 :1 (g)	53.5±27.9 a	41.5±22.8 b	46.2±26.3 a b	46.8±28.2 a b	46.0±25.9	**0.01**
C18:2 (g)	19.1±14.0 a	14.5±9.18 b	16.6±10.8 a b	15.9±8.75 a b	16.2±10.9	**0.02**
C18 :3 (g)	2.73±1.44 a	2.19±1.51 b	2.40±1.42 a b	2.36±1.65 a b	2.38±1.49	0.07
C20:5(g)	0.11±0.32	0.19±1.61	0.13±0.28	0.16±0.29	0.15±1.03	0.93
C22:6 (g)	0.30±0.72	0.45±2.67	0.32±0.65	0.39±0.74	0.38±1.76	0.92
MUFA/SFA ratio	1.50±0.50	1.47±0.58	1.49±0.60	1.56±0.64	1.50±0.57	0.82
PUFA/SFA ratio	0.62±0.38	0.59±0.30	0.60±0.31	0.63±0.30	0.61±0.32	0.89
n-6/n-3 ratio	4.08±20.29	9.60±50.94	6.73±27.40	3.66±16.94	6.97±36.82	0.66

P value <0.05. Means not sharing the same subscript are significantly different.

Table 3. ANCOVA of total SFA, MUFA, PUFA, trans and specific fatty acids in percentage (means ± standard error) by gender (adjusted for age)

	BMI (kg/m²)				
	Group 1 18.5-24.9	**Group 2 25.0-29.9**	**Group 3 30.0-34.9**	**Group 4 ≥35**	**P value**
W	N=57	N=129	N=133	N=121	Total:440
M	N=86	N=155	N=112	N=40	Total:393
Total Fat (%)					
W	34.4±8.67 a	36.6±8.97 ab	38.3±8.55 ab	38.4±9.64 b	**0.05**
M	40.3±10.0	39.1±10.4	39.7±8.44	37.6±9.55	0.10
Total SFA (%)					
W	10.9±0.49	11.7±0.32	11.8±0.31	11.6±0.33	0.41
M	12.3±0.41	12.1±0.30	12.1±0.36	11.1±0.60	0.38
Total MUFA (%)					
W	14.7±0.63 a	15.6±0.41ab	16.4±0.41 ab	16.6±0.43 b	**0.05**
M	17.8±0.57	16.9±0.42	16.8±0.83	15.9±0.83	0.27
Total PUFA (%)					
W	5.89±0.31	6.05±0.20	6.43±0.20	6.59±0.21	0.14
M	6.85±0.28	6.62±0.20	6.63±0.24	6.28±0.41	0.73
Total Trans (%)					
W	1.67±0.24 ab	1.57±0.16 b	1.96±0.16 ab	2.16±0.17 a	**0.05**
M	2.31±0.21	2.03±0.16	2.01±0.19	2.00±0.31	0.68
Tot MCFA (%)					
W	0.41±0.06	0.51±0.04	0.51±0.04	0.43±0.04	0.30
M	0.41±0.05	0.42±0.03	0.41±0.04	0.39±0.07	0.98
Tot LCFA (%)					
W	27.5±1.98 a	28.36±1.30 a	29.16±1.28 ab	33.30±1.36 b	**0.03**
M	35.99±1.54	32.60±1.12	33.15±1.32	28.84±2.27	0.07
Tot LCSFA (%)					
W	10.65±0.43	11.23±0.28	11.67±0.28	11.90±0.30	0.08
M	12.62±0.37	12.23±0.27	11.98±0.32	11.14±0.55	0.17
C14:0 (%)					
W	0.75±0.07	0.83±0.04	0.84±0.04	0.76±0.05	0.40
M	0.78±0.06	0.78±0.04	0.76±0.05	0.75±0.08	0.98

C16:0 (%)					
W	6.78±0.29 a	7.11±0.19 ab	7.46±0.19 ab	7.65±0.20 b	**0.05**
M	8.12±0.25	7.91±0.18	7.66±0.21	7.17±0.37	0.16
C18:0 (%)					
W	3.09±0.13	2.49±0.09	3.32±0.09	3.43±0.09	0.19
M	3.66±0.12	3.51±0.09	3.50±0.10	3.19±0.17	0.17
C18:1 (%)					
W	13.67±0.61	14.41±0.40	15.22±0.40	15.21±0.42	0.10
M	16.51±0.54	15.51±0.40	15.55±0.47	14.56±0.80	0.23
C18:2 (%)					
W	4.92±0.28	5.03±0.18	5.34±0.18	5.42±0.19	0.30
M	5.72±0.26	5.47±0.19	5.54±0.22	5.27±0.38	0.80
C18:3 (%)					
W	0.74±0.05	0.80±0.04	0.88±0.04	0.83±0.04	0.13
M	0.87±0.04	0.83±0.03	0.84±0.04	0.75±0.07	0.55
C20:5 (%)					
W	0.03±0.01	0.03±0.01	0.04±0.01	0.06±0.01	0.17
M	0.04±0.02	0.05±0.02	0.05±0.02	0.05±0.03	0.98
C22:6 (%)					
W	0.14±0.04	0.08±0.03	0.11±0.03	0.18±0.03	0.06
M	0.11±0.04	0.14±0.03	0.11±0.03	0.12±0.06	0.91
PUFA/SFA ratio					
W	0.60±0.05	0.58±0.03	0.61±0.03	0.64±0.03	0.61
M	0.62±0.04	0.60±0.03	0.60±0.03	0.63±0.05	0.90
MUFA/SFA ratio W	1.44±0.08	1.43±0.05	1.49±0.05	1.53±0.05	0.52
M	1.55±0.06	1.48±0.05	1.46±0.05	1.51±0.09	0.77
n-6/n-3 ratio					
W	2.84±4.27	6.72±2.72	4.57±2.60	6.40±2.68	0.85
M	4.16±4.71	10.22±3.73	7.80±3.94	5.06±6.83	0.73

W: women, M: men

LCFA include fatty acids from C14:0 to C22:6.

Figure 1a. Total fat, type of fat and specific fatty acids intake, in percentage of energy intake, according to BMI categories for women

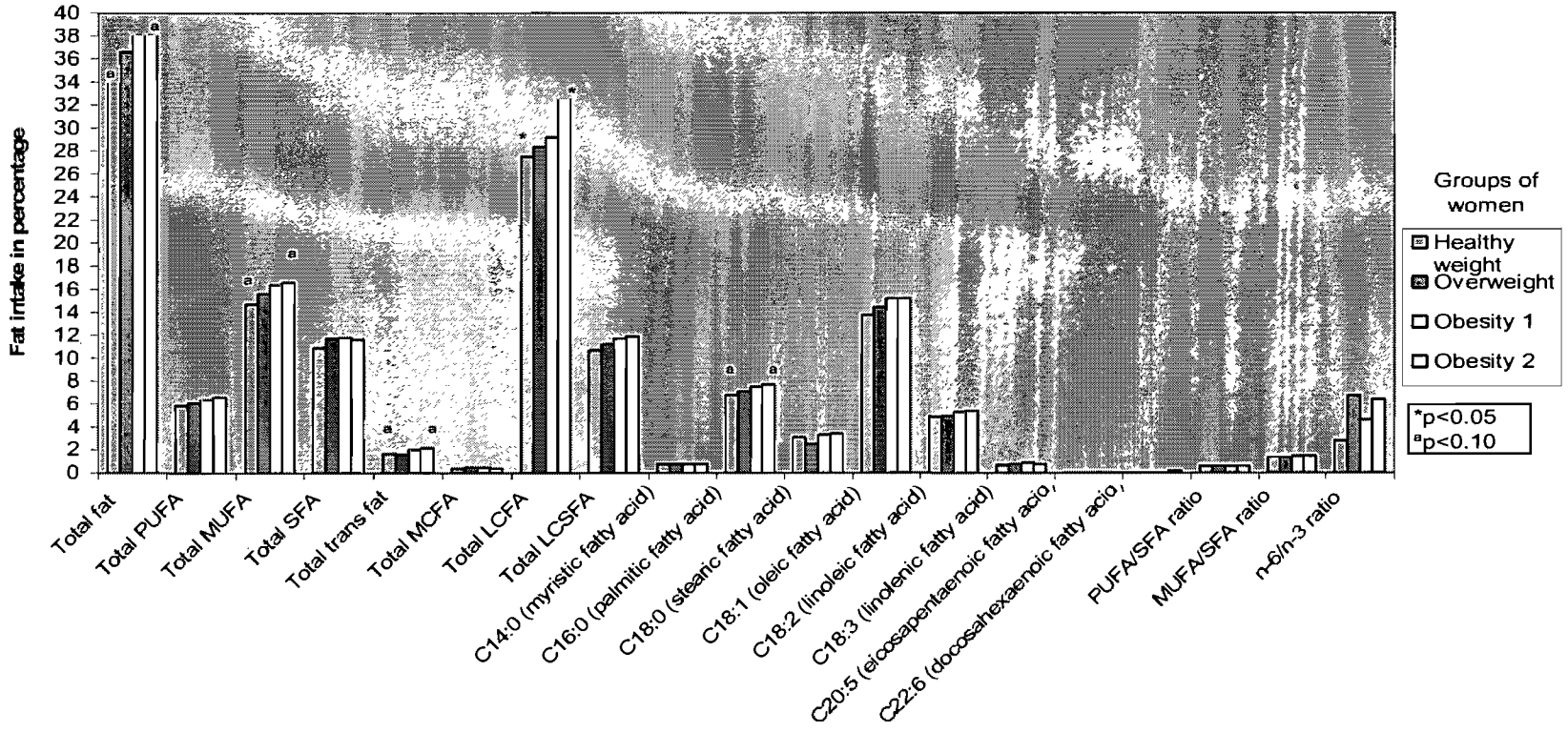
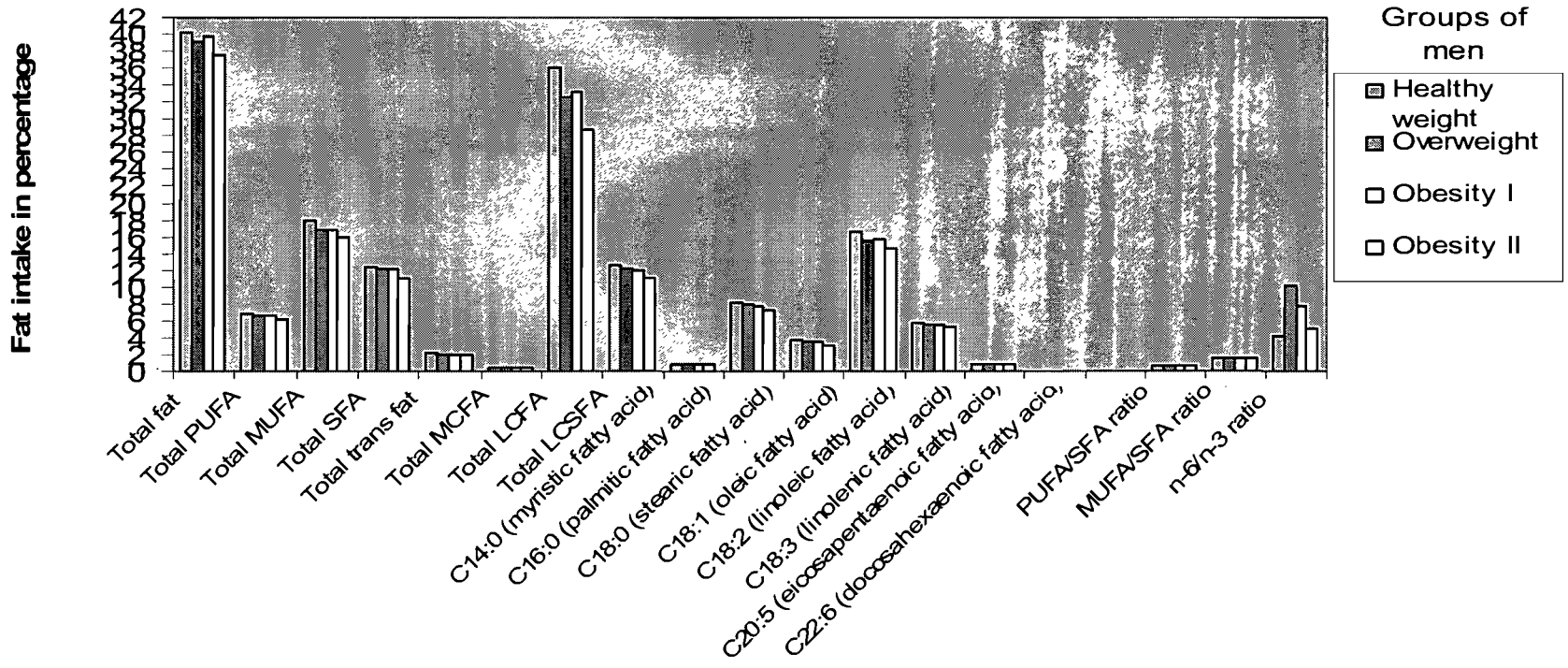


Figure 1b. Total fat, type of fat and specific fatty acids in percentage of energy intake, according to BMI categories for men



4.5 DISCUSSION

The main result shown in this paper is that, total MUFA intake appear to be positively associated with BMI in women (P=0.05). It suggests that the group with higher prevalence of obesity consume more MUFA. In this group 16.4% of total energy comes from MUFA in the diet. This finding supports results from the few epidemiological studies that reported Mediterranean diet and specifically olive oil may promote weight gain (12, 16). On the other hand, some epidemiological studies demonstrated that the Mediterranean diet or olive oil might improve weight loss (24-26). Some other investigators have not shown any relationship between a high consumption of MUFA and the prevalence of obesity (27-29). Some clinical trials (30, 31) but not all (32) have demonstrated that MUFA have a higher oxidation rate than SFA. These authors suggested that MUFA might prevent weight gain. The proposed mechanism underlying this phenomenon is that MUFA intake increase thermogenesis which in turn stimulates the sympathetic nervous system (33) and abdominally obese subjects may be more responsive to the stimulation of sympathetic nervous system, because they have an increased density and sensitivity of B-adrenoreceptors (34). Note that all of these studies done in humans (30, 31) have not demonstrated any weight loss but a postprandial increase in fat oxidation when comparing it to the other diet, mostly cream intake (SFA) on isocaloric diets. Most of these studies used 30 to 40% of fat coming from MUFA intake, principally olive oil (30, 31). Lovejoy et al., (32) used 28% of energy from MUFA or palmitic fatty acid. They did not report any weight loss between the groups nor a higher oxidation rate in

the group of subjects who were consuming the MUFA diet. Similarly, some animal studies demonstrated that in rats, MUFA consumption might have an anti-obesity action (35). These authors reported that MUFA intake may stimulate fat utilization through activation of nuclear receptor peroxisome proliferators-activated receptor alpha. Others (36, 37) demonstrated that with a high MUFA intake, rats may gain weight. Note that in the first study the authors considered 60% of total energy coming from MUFA and in the second, Buettner et al. (37) gave 42% of total energy from olive oil to the group of rats followed for 12 weeks.

First, our results can be explained by the fact that they are not adjusted for total fat intake. That might also explain our positive association reported between MUFA intake and obesity prevalence. In fact, the group of women with a higher BMI seems to consume more total fat (38.4±9.6%) than the other groups, when adjusting for age (P=0.05). This is in concordance with other studies (38, 39). Fat is an energy dense macronutrient (9 kilocalories per gram) comparatively to protein or carbohydrates (4 kilocalories per gram) (40). Furthermore, dietary fat can contribute to obesity via passive over-consumption, because this macronutrient is less satiating than either carbohydrates or proteins (40, 41).

Second, Indigenous people in Canada do not follow a Mediterranean diet, which include other aspects (42) besides MUFA and olive oil. When looking at the main sources of MUFA intake in Cree women, we noticed that animal fat, lard, tub

margarine and unspecified vegetable oils are the most reported. These sources are rich in SFA and in trans fat as well. Their profiles, in term of types of fat, are different compare to the one of olive oil, used in most clinical trials.

Also, we found a positive association between total trans fat and obesity prevalence in women (Table 3). This result is in agreement with the recent study of Kavanagh et al. (2007) using 42 male Africans monkeys which demonstrated for the first time that trans fatty acids increase intra abdominal fat deposition even without energy excess. We are conscious nevertheless that the positive association demonstrated between total fat and obesity prevalence might influence positively the relation between trans fat intake and obesity prevalence in the group of women. Also the main dietary sources of trans fat- shortening, household and unspecified vegetable fat are rich in MUFA and in SFA, so that there might be interactions between these types of fat that enhance the positive association observed between trans fat and obesity prevalence in Cree women.

Finally, we have observed a positive association between percentage of LCFA and palmitic fatty acid intake, and obesity, in women. These results are in agreement with the literature (31, 44-46). Authors explained this association by the fact that short chain fatty acids, (SCFA) and medium chain fatty acids (MCFA) are preferentially oxidized compared to LCFA in humans as well as in animals (45-48). SCFA and MCFA are transported directly to the liver via the portal system (44, 49). The latter type of fatty acids are not incorporated into esterified lipids and their transport to the mitochondrial matrix is not carnitine-dependent (44, 49). Some

investigators also demonstrated (50) that the net energy value of MCFA is 5 kcal/g which is different from the usual amount of energy coming from LCFA, 9 kcal/g. In parallel, authors reported that palmitic fatty acid may increase body weight because it is a saturated and a LCFA (31, 45).

In our paper, results for men and women are different in this population. Some articles reported that older individuals misreport their energy intake (51). The EPIC study reported that women rather than men underestimate their intake in the vast majority of cases (52). These authors reported that underreporting seem to be related to age and weight. They highlighted the complexity this creates in analysing diet between and within groups. Furthermore, there seem to be a gender difference for nutritional preferences. Macdiarmid et al. (53) reported that obese women tend to choose more high fat and sweet foods than men. However, one can also presume that women in this population were reporting the recipes and quantities used in their cooking more precisely.

A positive point of our study is that the 24 hour recall has given us access to the specific fatty acids profile of the James Bay Cree population. In the early 90s, people were less aware of trans fat and the type of fat intake in their diet, so data reporting by subjects might be more reliable than if the same data collection were done at the present time.

Our study contains some limitations. First, it is based only on one 24 hour recall which might not be representative of all the food intake of this population and this method of reporting has its limitations as has been stated elsewhere (54, 55). The fact that we did not have multiple 24-hour recalls restrained us from adjusting for intra-individual variation and therefore limited our ability to detect differences across groups. It also prevents us from using these data at the individual level and therefore from conducting multivariate analysis. For example, adjusting the identified relationships between BMI and MUFA, trans fat, total fat intake for each other as well as for other types of fat would be needed. Furthermore, our results might be partially confounded by some unmeasured variables such as physical activity. Certainly, we cannot talk about a causal effect because of the cross sectional design of our study.

4.6 CONCLUSION

Despite the cross-sectional nature of the data, we demonstrated an association between percentage of total fat, MUFA, LCFA and palmitic fatty acid intake, and obesity, in women. We also demonstrated a positive association between BMI and total trans fat intake. Most of the studies have considered trans fat intake with cardiovascular diseases, sudden death and diabetes mellitus but not with weight change in humans (56). More studies using BMI as the dependent variable and trans fatty acids as a risk factor are needed.

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CHAPITRE 5 DISCUSSION

Cette étude de devis observationnels visait à détecter la présence d'associations entre le type d'acides gras et la prévalence d'obésité. Nos deux études révélèrent une association entre le gras total et la prévalence d'obésité : gras total disponible dans l'étude écologique, et pourcentage de l'énergie dérivée des lipides, chez les femmes, dans l'étude transversale. Ces observations vont dans le sens de la littérature (Alfieri et al., 1997; Sidell et al., 1998) qui identifie plus généralement la densité énergétique comme facteur alimentaire le plus convainquant dans l'étiologie de l'obésité au niveau des populations (Swinburn et al., 2004). Nos observations corroborent donc les recommandations nord américaines de limiter l'apport en lipides à moins de 35% de l'énergie.

Au niveau des associations possibles entre le type de lipides et la prévalence d'obésité, l'étude écologique révéla que dans les pays où la prévalence d'obésité est plus élevée, le type de gras disponible inclut plus de gras saturés, plus de gras polyinsaturés, et moins de gras monounsaturés que dans les pays où la prévalence d'obésité est moindre. Par contre ces associations ne sont pas apparues dans l'étude transversale où ni les acides gras saturés, ni les polyinsaturés n'ont montré d'association, et les acides gras monoinsaturés ont montré une association inverse (leur consommation augmentant avec les niveaux d'IMC). Certes les limites respectives des devis utilisés sont nombreuses comme présentées dans la revue de la

littérature ainsi que les articles respectifs, limites propres aux devis et méthodes de collecte de données (Hennekens et al., 1987). Il est tout à fait possible que l'association observée dans le devis écologique et dans l'étude transversale ne représente pas un effet causal des MUFA mais plutôt l'action de facteurs confondants par exemple, l'apport en fibres ou bien même des différences dans le niveau d'activité physique entre les pays.

À cause de ces limites, les possibilités que l'une ou l'autre ou même ces deux associations opposées entre monoinsaturés et obésité soient artificielles ne peuvent être écartées. Toutefois, considérer la possibilité que ces conclusions opposées sont en faites existantes reste théoriquement concevable : cela reviendrait à considérer nos résultats comme un exemple de paradoxe de Simpson (Simpson, 1951). Il est en effet possible qu'une relation négative à un niveau d'analyse (étude écologique) soit compatible avec une relation inverse à un sous-niveau (groupes d'IMC dans l'étude transversale). Cela s'expliquerait par l'effet de facteurs confondants et d'une étendue restreinte et inégale des niveaux d'exposition entre les pays. Quand bien même ce serait le cas, la littérature tend à démontrer que si relation il y avait entre les apports en monoinsaturés et la prévalence d'obésité, cette relation tendrait à être protectrice. Ceci nous pousse donc à penser que l'association positive observée dans l'étude transversale n'est nullement causale et reflète plutôt l'effet de l'apport total en gras. Notre impossibilité d'ajuster l'association entre les apports en monoinsaturés et l'IMC pour l'apport en gras total est intrinsèque à l'utilisation d'un seul rappel de 24h. Cette méthode permet des comparaisons au niveau des groupes mais pas au

niveau des individus étant donnée la magnitude de la variation intraindividuelle par rapport à la variation interindividuelle (Beaton et al, 1979; Beaton et Chery, 1986). Cette limite affecte aussi les deux autres associations détectées dans l'étude transversale entre les LCFA (particulièrement l'acide palmitique), les acides gras trans, et la prévalence d'obésité. Il se pourrait donc aussi que ces associations ne reflètent que l'effet du gras total. Si le pourcentage de gras total n'avait pas été relié à l'IMC, nous aurions été mieux en position de considérer la possibilité que ces relations soient causales. Également, étant donnée que nous n'avons pas pu ajuster un type de gras par rapport à un autre dans le modèle, la présence d'interaction entre les variables pourrait exagérer les associations positives observées.

Toutefois la littérature supporte la possibilité d'une relation positive causale entre les LCFA (particulièrement l'acide palmitique) et la prévalence d'obésité. Cela pourrait s'expliquer par le fait que les LCFA sont moins rapidement oxydés que les SCFA et les MCFA et par conséquent semblent se déposer davantage dans le corps que les SCFA ou MCFA (Delany et al., 2000; Kien et al., 2005). St-Onges et al., (2003) ont même observé que la dépense énergétique moyenne était plus élevée et la masse du tissu adipeux diminuée avec une diète riche en MCFA comparée à celle à chaîne longue chez les humains. Pour l'acide palmitique en particulier, son association positive avec l'IMC peut être reliée au degré de saturation de ce type d'acides gras à chaîne longue (Leyton et al., 1987). En effet, la vitesse d'oxydation diminue avec l'augmentation du nombre de carbones dans les LCFA et les saturés (DeLany et al., 2000).

La dernière association observée dans notre étude transversale entre les apports en acides gras trans et l'IMC mérite finalement tout particulièrement d'être considérée. En effet seul Kavanagh et al., (2007) très récemment, ont rapporté chez les singes un effet positif des acides gras trans sur le poids corporel ainsi que sur leur tendance à favoriser le dépôt graisseux dans la région abdominale. La majorité des études sur le sujet a considéré l'apport en acides gras trans en association avec les maladies cardiovasculaires, sans tenir compte de l'IMC (Lichtenstein et al., 2001; Sundram et al., 2003). Certaines études ont même ajusté pour l'IMC (Roos et al., 2001; Oh et al., 2005).

Examinons maintenant les sources de gras les plus rapportées dans nos deux études. Dans le devis écologique les sources végétales les plus rapportées sont l'huile de soya, de tournesol, de palme et d'olive. Les sources animales les plus rapportées sont le gras animal et le beurre. Dans l'étude transversale la margarine, le shortening, le saindoux ainsi que le beurre constituent les principales sources. Les huiles végétales sont moins rapportées dans ce devis, que les sources précédentes de gras. Il est à remarquer que dans le premier devis nous avions les sources de gras non transformées et disponibles à la consommation tandis que dans le deuxième, nous avions les sources spécifiques de gras consommées par la population Crie. Cela rend la comparaison entre les deux études difficile puisque nous ignorons la proportion de gras transformé en margarine et /ou shortening dans chaque pays dans le premier devis. Aussi, nous savons qu'à la suite de transformation commerciale, la composition en acides gras de certaines sources change.

Dans l'étude transversale, considérer que les sources du gras rapportées sont associées à l'obésité malgré les limites déjà citées est possible. Les huiles de soya et de tournesol sont riches en PUFA tandis que l'huile de palme est riche en SFA et en MUFA et plus spécifiquement en acide gras oléique (Table a). En ce qui concerne le gras animal et le beurre, ces sources sont riches en SFA mais aussi en MUFA (acide gras oléique). En examinant ce tableau (Table a) de près, on note que les ratios du SFA par rapport au MUFA dans un même aliment ne sont pas pareils dans les diverses sources.

Table a. Tableau de composition d'huiles alimentaires riches en type de gras spécifique, dans une portion de 100 grammes selon le fichier Canadien 2007 (www.hc-sc.gc.ca/fn-an/nutrition/fiche-nutri-data/index_f.html)*

	Étude transversale		Étude écologique					
	Shortening**	Margarine molle***	Saindoux porc, lard	Beurre	Huile de soya	Huile de tournesol	Huile de palme	Huile d'olive
Énergie (kcal)	884	716	902	717	884	884	884	884
SFA (g)	26.1	8.9	40	51.4	14.4	10.3	49.3	13.5
C12:0	0.0	0.0	0.2	2.6	0.0	0.0	0.1	0.0
C16:0	15.1	3.6	23.8	21.7	10.3	5.9	43.5	10.9
C18:0	9.9	4.3	13.5	10.0	3.8	4.5	4.3	1.9
MUFA (g)	42.7	47.7	45.1	21.0	23.3	19.5	37.0	73.9
C16:1	0.0	0.1	2.7	1.0	0.2	0.0	0.3	1.2
C18:1	42.7	46.8	41.2	20.0	22.8	19.5	36.6	72.3
PUFA (g)	21.1	14.5	11.2	3.0	57.9	65.7	9.3	10
C18:2	19.5	10.5	10.2	2.7	51.0	65.7	9.1	9.2
C18:3	1.6	4.0	1.0	0.3	6.8	0	0.2	0.8
Trans total	16.9	15.4	1.3	4.6	1.8	0.7	----	0.05

*Recherche en ligne des aliments dans le Fichier canadien sur les éléments nutritifs, version 2007b.
**Végétale, domestique, non précisée
***Avec huile végétale non précisée

En examinant le profil de l'huile d'olive, souvent utilisée dans la littérature, et celui des sources observées chez la population Crie le shortening, la margarine et/ou le saindoux, nous remarquons que malgré leur apport en calories presque identique ainsi que leur richesse en MUFA, ces sources possèdent un profil différent en acides gras spécifiques. En effet, les sources rapportées chez la population Crie, contiennent davantage de SFA et de gras trans.

Les résultats des études épidémiologiques rapportent des résultas contradictoires (positifs, négatifs ou nuls) pour la relation entre la consommation des MUFA et l'obésité, tel qu'observé dans le premier article de revu dans le tableau d'études épidémiologiques. La plupart de ces études ne considèrent pas les sources des MUFA séparément et leur premier objectif n'est pas la relation de ce dernier type de gras avec l'obésité. De plus, certaines de ces études épidémiologiques considèrent la diète Méditerranéenne dans sa globalité. Nous savons que cette dernière ne contient pas que l'huile d'olive comme constituant. Également la population Crie ne suit pas une diète Méditerranéenne. Cette dernière diète possède ses caractéristiques citées ailleurs (Esposito et al., 2006). En effet, cette diète (Keys, 1956) inclut les produits céréaliers à grain entier, les légumes ainsi que les fruits frais, les légumineuses et les noix. Cette diète contient moins de viande, plus de fromage, du poisson, de la volaille et des œufs.

Il est à remarquer qu'aucune étude n'a démontré une diminution significative du poids, chez les humains, en relation avec l'apport en MUFA. La majorité d'études expérimentales publiées dans la littérature concernant le sujet ont fournit aux groupes expérimentaux au moins 30% d'un type de gras (crème, suif ou huile d'olive) de l'énergie totale dans des diètes isocaloriques. Ce qui rend l'interprétation d'une diète composée de multiples sources de gras très difficile.

Des études ont considéré la satiété en relation au type des gras consommés. L'étude de Alfenas et Mattes (2003) n'a trouvé aucune différence significative au niveau de satiété entre une diète riche en acides gras saturés (beurre) et une autre

riche en acides gras mono-insaturés (huile d'arachide et de canola). Toutefois, les acides gras à chaîne moyenne semblent avoir un effet de satiété plus grand que les acides gras à chaîne longue (Krotkiewski, 2001). Le mécanisme d'action de ce type d'acides gras semble être dans leur métabolisme. En effet, les acides gras à chaîne moyenne sont oxydés préférentiellement (dans le foie et autres tissus) et sont capables de traverser la membrane mitochondriale indépendamment de l'acylcarnitine transférase (Bremer, 1983). L'oxydation élevée dans le foie est rapportée comme étant un signal de satiété (Langhans, 1996). Aussi, les acides gras à chaîne longue semblent stimuler les fibres vagues afférents nerveux mésentériques (une voie d'information de satiété du tractus gastrointestinal) via la cholescystokinine (CCK), alors que les acides gras à chaîne courte, tel l'acide butyrique, stimulent directement ces fibres (Lal et al., 2001). Il semble que le degré de stimulation de CCK soit relié au degré de saturation des acides gras. L'ordre de cette stimulation va dans le sens suivant, du plus élevé au plus bas : l'huile de maïs (riche en C18 :2, acide linoléique), l'huile d'olive (riche en C18 :1 acide oléique), le gras du bœuf (riche en C18 :0, C16 :0; acide stéarique et palmitique) (Bearshall et al., 1989). L'étude de Alfenas et Mattes (2003) n'a trouvé aucune différence significative au niveau de satiété entre une diète riche en acides gras saturés (beurre) et une autre riche en acides gras mono-insaturés (huile d'arachide ou de canola).

Conclusion

D'autres études sur l'apport en différents types de gras et particulièrement en MUFA, acide gras palmitique, ainsi qu'en gras trans en association avec l'obésité sont nécessaires. Cependant, la relation positive rapportée par Kavanagh et al. (2007), pour l'apport en gras trans et l'obésité ne peut être étudiée davantage en utilisant des cohortes ou des essais cliniques puisque des recommandations existent quant au besoin de limiter les apports journaliers en gras trans. Toutefois, la possibilité de revisiter des études passées où les apports auraient été mesurés à l'aide de rappels de 24h multiples pourrait être explorée. En effet, de telles données permettraient de tester dans des modèles de régression multiple l'association possible entre les apports en gras trans et l'IMC tout en contrôlant pour les apports en gras total et l'apport énergétique, ce qui ne fut pas possible dans notre étude transversale car nous n'avions qu'un seul rappel de 24 h. Il serait également intéressant, dans l'optique que cette relation soit causale, de suivre l'évolution des taux de prévalence de l'obésité dans les pays où une législation limitant la consommation de gras trans existe (le Canada par exemple). Tout le reste étant égal, on pourrait s'attendre alors à une diminution de la prévalence d'obésité dans les années à venir. Dans l'immédiat, l'identification de banques de données existantes pourrait cibler les études qui furent effectuées pour mieux comprendre les risques de maladies cardiovasculaires. Dans ces études, l'IMC était considéré comme facteur de confusion (Roos et al., 2001; Oh et al., 2005) et deviendrait maintenant la variable dépendante principale. Il serait aussi souhaitable que de telles banques de données contiennent également des données sur des facteurs

de confusion potentiels qui n'ont pas été considérés dans nos analyses, tel le niveau d'activité physique.

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ANNEXES