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Can the Dietary Inflammatory Index Be an Essential Tool for Preventing Diabetes? An Overview of Clinical Studies

Diyet İnflamatuvar İndeksi Diyabetin Önlenmesinde Temel Bir Araç Olabilir mi? Klinik Çalışmalara Genel Bir Bakış

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Abstract

Low-grade, chronic inflammation is related to various non-communicable diseases, such as diabetes, obesity, cardiovascular disease, and cancers. Dietary factors are important modulators of chronic inflammation. Furthermore, diet may increase the risk of type 2 diabetes mellitus through inflammation. The dietary inflammatory index was designed to determine the inflammatory potential of diet. Considering that diet may increase the risk of type 2 diabetes mellitus through inflammation, determining the inflammation potential of individuals' diets is essential for preventing diabetes risk. Thus, it may benefit from using the dietary inflammatory index to characterize dietary inflammatory potential. In this review, a comprehensive literature search was performed for the dietary inflammatory index and studies investigating the effects of the Dietary Inflammatory Index on diabetes risk and glucose markers. Although studies evaluating diabetes and diabetes-related markers with the dietary inflammatory index have inconsistent results, the dietary inflammatory index is successful in reflecting the inflammatory potential of diet. It can be an essential tool to characterize the populations' diet and reduce the risk of chronic inflammation-related disease, including diabetes. However further clinical studies are needed to determine the relationship between the Dietary Inflammatory Index and diabetes and whether the dietary inflammatory index tool will be helpful in practice.

Keywords: Dietary inflammatory index, Inflammation, Diabetes, Type 2 diabetes, Pro-inflammatory diet

Özet

Düşük dereceli kronik inflamasyonun, diyabet, obezite, kardiyovasküler hastalıklar ve kanserler gibi çeşitli bulaşıcı olmayan hastalıklarla ilişkili olduğu bilinmektedir. Diyet faktörleri de kronik inflamasyonun önemli modülatörleridir. Bununla beraber diyet, inflamasyon yoluyla tip 2 diyabetes mellitus riskini artırabilmektedir. Diyet inflamatuvar indeksi, diyetin inflamatuvar potansiyelini belirlemek için tasarlanmış bir indekstir. Diyetin inflamasyon yoluyla tip 2 diyabetes mellitus riskini artırabileceği göz önüne alındığında, bireylerin diyetlerinin inflamasyon potansiyelinin belirlenmesi diyabet riskinin önlenmesi açısından önemlidir. Bu nedenle, diyetin inflamatuvar yükünü saptama amacıyla Diyet inflamatuvar indeksinin kullanılmasından faydalanılabilir. Bu derlemede Diyet inflamatuvar indeksi için kapsamlı bir literatür taraması yapılmış ve Diyet inflamatuvar indeksinin diyabet riski ve glukoz belirteçleri üzerindeki etkilerini araştıran çalışmalara yer verilmiştir. Diyabet ve diyabetle ilişkili belirteçleri diyet inflamatuvar indeks ile değerlendiren çalışmalarda tutarsız sonuçlar elde edilmesine rağmen, Diyet inflamatuvar indeksinin diyetin inflamatuvar potansiyelini yansıtmada başarılı olduğu ve popülasyonun diyetini karakterize etmek ve diyabetin de dahil olduğu çeşitli hastalıklarda kronik inflamasyon riskini azaltmak için önemli bir araç olabileceği gösterilmiştir. Ancak diyet inflamatuvar indeks ile diyabet arasındaki ilişkinin ve Diyet inflamatuvar indeksinin pratikte yararlı olup olmayacağının belirlenmesi için daha fazla klinik çalışmaya ihtiyaç olduğu belirtilmektedir.

Anahtar Kelimeler: Diyet inflamatuvar indeksi, İnflamasyon, Diyabet ,Tip 2 diyabet, Proinflamatuvar diyet

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Introduction

Chronic systemic low-grade inflammation, characterized by consistent presence of high levels of circulating pro-inflammatory cytokines in serum throughout life has been thought to cause the development of various non-communicable diseases (NCDs), such as diabetes, obesity, cardiovascular diseases, cancers, autoimmune disorders, and depression.[1,2] Although it is not possible to explain chronic systemic low-grade inflammation and its connection with non-communicable diseases cannot be attributed to a single mechanism, it is suggested that the increased pro-inflammatory cytokines in chronic systemic low-grade inflammation cause chronic diseases through various mechanisms, including DNA damage, alterations in gene expression and genetic instability, insulin resistance, blunted immune response, neural signaling, and vascular dysfunction.[1] In recent years, it has been approved that diet has an essential modulating role in chronic inflammation.[3] Studies have pointed out that serum levels of inflammatory markers such as IL-6, TNF-α, and CRP are affected by dietary patterns.[4,5] And many foods and nutrients affect the level of inflammation markers.[6,7] An anti-inflammatory Mediterranean diet with a higher consumption of vegetables, fruits, and fish is associated with low inflammation levels. In contrast, a pro-inflammatory Western diet with a higher consumption of saturated fat, refined cereals with a high glycemic index (GI), and processed food is related to high inflammation levels.[8,9] Further, it is suggested that diet may increase the risk of T2DM through inflammation, and various studies supported this relationship. Dietary fiber and carotenoids thought to have anti-inflammatory effects have been related to a low risk of T2DM, whereas consumption of red meat rich in saturated fatty acid thought to have pro-inflammatory effects has been associated with a high risk of T2DM.[10-13]

The dietary inflammatory index (DII®) was improved to evaluate the inflammatory potential of the diet. It was designed using data from a wide variety of human populations.[14] The dietary inflammatory index (DII) represents the first index devised for the evaluation of the inflammatory impact of dietary patterns and is applicable across diverse populations.[14] In calculating the inflammatory index scores of the diet, consideration is given to the entirety of the individual's dietary intake.[8,14] The lower DII score (negative or close to negative) represents the more anti-inflammatory diet; the higher DII score (positive or close to positive) represents the more pro-inflammatory diet.[2,8,14] Since the development of DII, the relationship between the inflammation potential of the diet, as measured by DII, and a variety of chronic non-communicable diseases (NCDs), including diabetes has been investigated.[15-18] This review aims to give brief information about DII and to evaluate whether the use of DII can be an essential tool in reducing the risk of diabetes by assessing the results of studies investigating the relationship between DII and diabetes.

Methods

In this study, a comprehensive literature search was conducted on Web of Science, PubMed, and Scopus electronic databases for studies that investigate the relationship between DII, diabetes, and glucose metabolism markers. Search terms included: (diabetes OR Type 2 diabetes OR glycemic markers OR glucose metabolism markers) AND (dietary inflammatory index OR diet inflammation index OR inflammatory potential of diet OR inflammatory diet score) Furthermore, a manual search from reference lists of all relevant studies was performed in order not to miss any relevant studies. Articles that have no available full text and are not in English were excluded. Studies investigating the association between DII, diabetes risk, and glucose markers were summarized.

Dietary Inflammatory Index (DII)

The dietary inflammatory index is literature-based. It was developed to determine the inflammatory potential of the diet. Its design is based on a scoring algorithm obtained by scoring the peer-reviewed articles published from 1950 to 2010 that investigate the effects of various dietary factors on six inflammatory markers, including CRP, TNF-α, IL-1β, IL-4, IL-6, IL-8, and IL-10. To calculate DII, a global composite food intake database created with this scoring algorithm is used.[14] The DII reflects evidence from qualitative laboratory animal and cell culture experiments, as well as many human studies with different study designs and dietary assessment methods.[19] Studies of creating DII started after the importance of diet-induced inflammation on diseases was understood and the first version of DII was published in 2009 by Cavicchia et al.[20] The second version was produced in 2014 by adding current articles to the literature information on the first index by Shivappa et al.[14] In this version, an improved scoring system has been developed using data sets of 11 countries on four continents, with a more complete literature search. The derivation and scoring algorithm steps of the DII have been previously defined in the methods paper,[14] and the steps of calculation of the DII are shown in figure 1. Briefly, first, peer-reviewed original research articles that evaluate the association of inflammatory biomarkers and various food parameters were reviewed. These articles were scored taking into account (i) the study design, (ii) the association direction of inflammatory markers and food parameters, and (iii) the association strength. Based on this article scoring system, an inflammatory effect score derived from literature was assigned to each food parameter,[2,] and a global database, containing the mean and standard deviation for each food parameter was improved.[14] In the last three steps in

calculating the DII score, this global database is used and an overall DII score is obtained for each individual.[2] This is the first index for detecting the inflammatory character of the diet and associated with a global food intake database. In the calculation, the whole diet is considered not just individual nutrients or foods.[20] In addition, individual intakes are standardized to global referent values derived from the world compound database. Therefore, the index is universal in applicability and can be used in any nutritional research with dietary data.[19] Dietary data required to calculate DII can be provided from any dietary assessment tool that determines food intake data.[14] Methods such as food frequency questionnaire (FFQ), 24-h dietary recall, and 3-7-day food record data were used in studies to date.[21-23] DII was validated in various studies and a positive relationship was found between DII and inflammation markers including TNF-α, IL-6, and CRP in these studies.[25,26] DII scores range from -8.87 to +7.98 and there is no classification for the scores. A higher DII score (positive or close to positive) corresponds to a pro-inflammatory dietary quality and a lower DII score (negative or close to negative) corresponds to an anti-inflammatory dietary quality.[14]

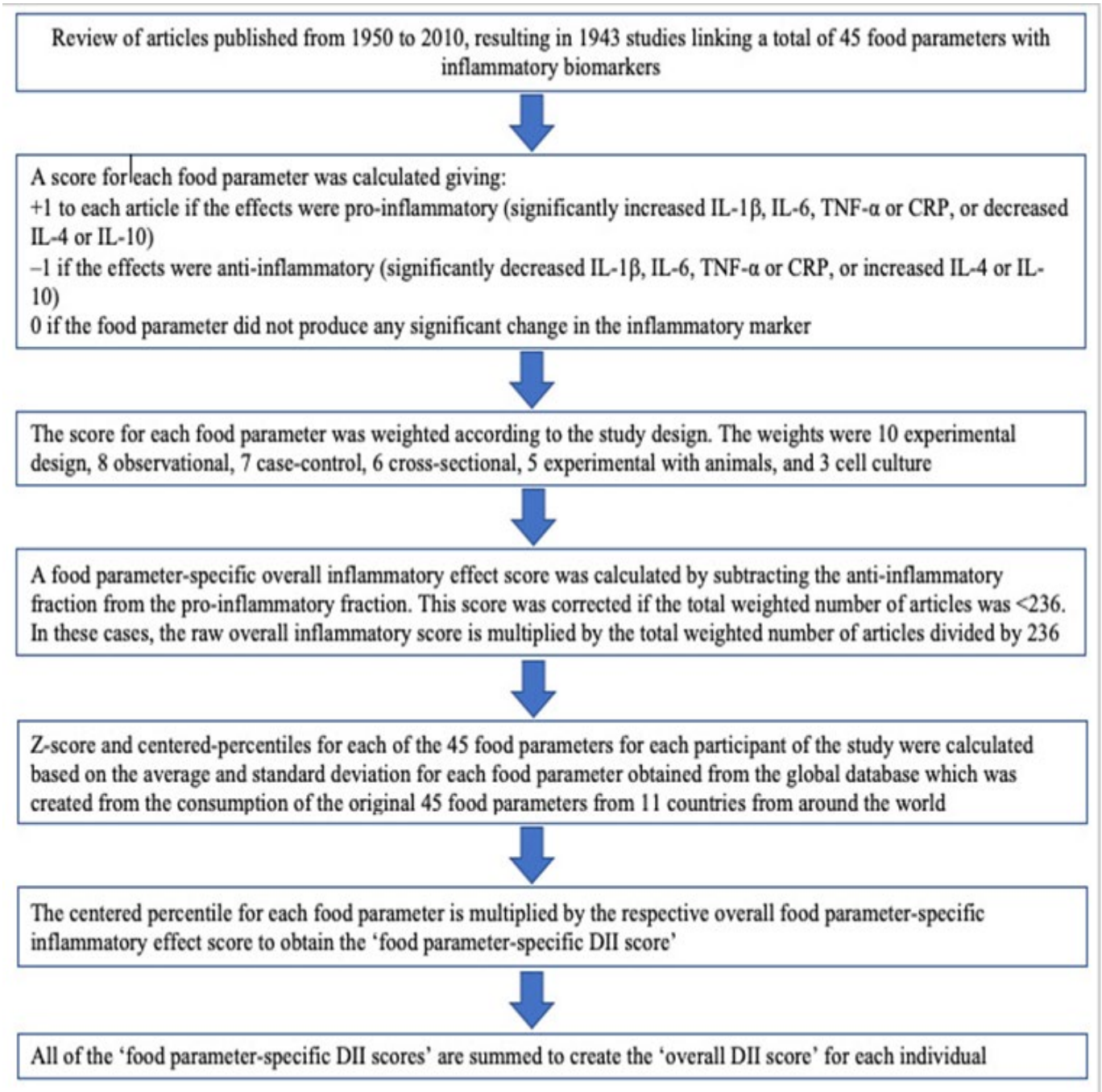

Figure 1. *Sequence of steps followed to create the dietary inflammatory index.*

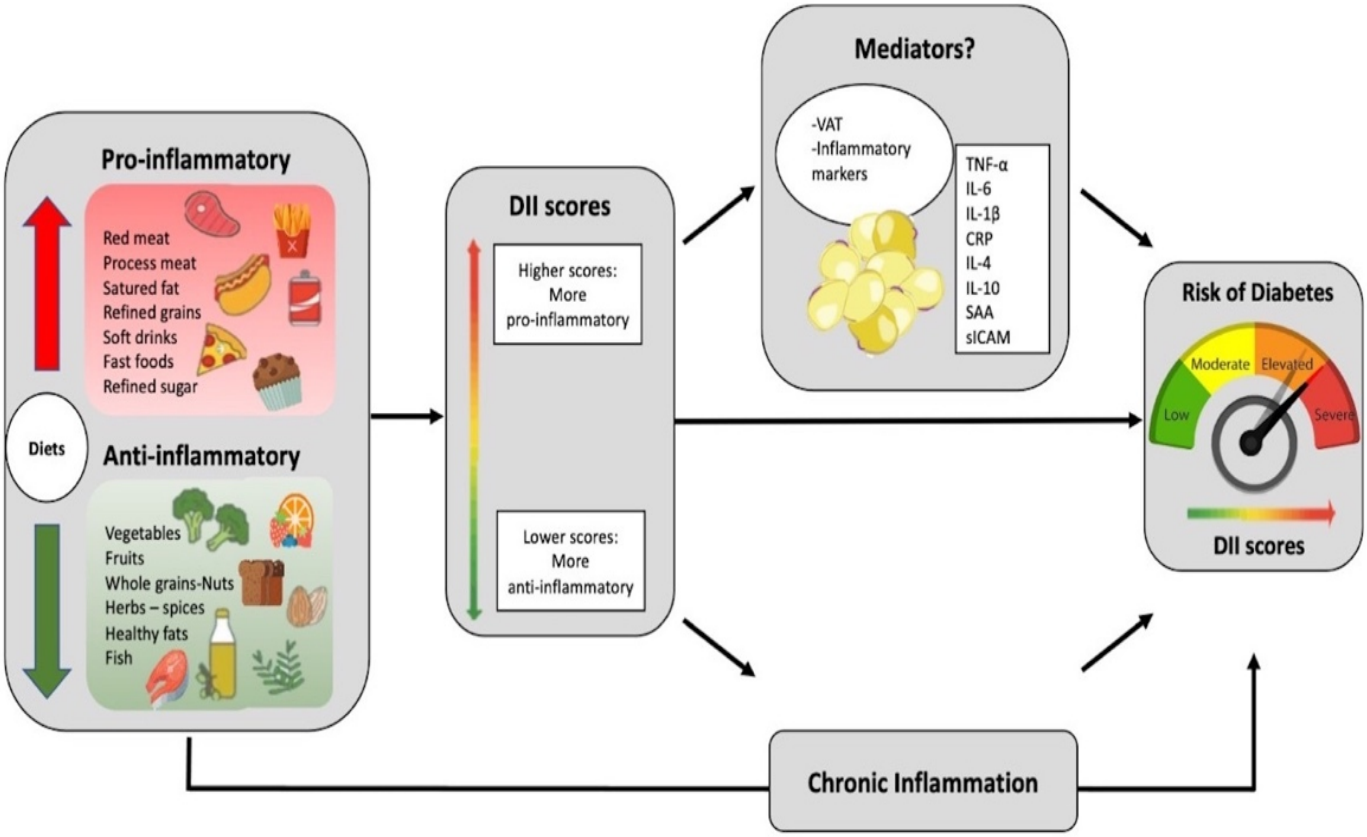

Figure 2. *Illustration of the association with DII and diabetes.*

Higher DII scores indicate a pro-inflammatory diet, whereas lower scores indicate an anti-inflammatory diet. DII may have simple direct effect on diabetes risk, or indirect effect, mediated by adiposity and/or inflammatory markers. **Abbreviations:** DII: dietary inflammatory index, VAT: visceral adipose tissue, IL-6: interleukin 6, IL-1β:interleukin 1 beta, CRP:C-reactive protein, IL-4: interleukin 4, IL-10: interleukin 10, SAA: serum amyloid A, sICAM: soluble inter-cellular adhesion molecule.

DII and Type 2 Diabetes

Recent evidence indicates that diet-induced inflammation is associated with type 2 diabetes mellitus (T2DM) risk.[10] Using the DII may be useful in understanding the role of diet-related inflammation in the T2DM pathophysiology.[9] In some studies, pointed out the role of mediators such as adiposity and/or some certain inflammatory markers in the relationship between DII and diabetes.[9,15] Figure 2 illustrates the association between DII and diabetes. To date, in studies investigating the relationship between DII and metabolic syndrome, obesity, and cardiometabolic risk factors the relationship between DII and some glucose metabolism markers was also evaluated.[16,17] However, the number of studies investigating the relationship between DII and T2DM risk is limited (Table 1).[26-29]

Table 1: *Characteristics of studies reporting the association between dietary inflammatory index (DII) and diabetes and diabetes-related markers*

First author/ Reference	Year	Country	Study design	Sex	Age range	Sample size	Dietary assessment/ Index	Results
Shivappa[31]	2018	Italy	Cross-sectional	Both	≥35	20823	FFQ/DII	Individuals in the lowest DII quintile had higher prevalence of diabetes and had higher FPG compared with highest
Denova-Guitiérrez[26]	2018	Mexico	Cross-sectional	Both	20-69	1174	FFQ/DII	Individuals in the lowest quintile of DII had significantly lower FPG and HbA1c compared with the highest
Woundenbergh[10]	2013	Netherlands	Cross-sectional	Both	Mean= 64	1024	FFQ/ADII	ADII was inversely related to HOMA-IR, FPG and post-load glucose but not associated with HbA1c
Moslei[29]	2016	Iran	Cross-sectional	Both	19-75	12523	FFQ/DII	No significant relationship was observed between glycemic markers and DII
Vahid[27]	2017	Iran	Case-control	Both	Mean= 47	214 prediabetics and 200 healthy matched controls	FFQ/DII	A correlation between DII score and higher FPG and HbA1c was observed
Alkerwi[30]	2014	Luxemburg	Cross-sectional	Both	18-69	1352	FFQ/DII	No significant relationship was observed between DII and glucose, insulin, HOMA-IR, hyperglycemia
Phillips[32]	2018	Ireland	Cross-sectional	Both	50-69	1992	FFQ/E-DII	Individuals in higher E-DII score group had higher FPG. No significant difference was reported between DII and other glucose metabolism markers
Mtintsilana[9]	2019	South Africa	Cross-sectional	Women	<65	190	FFQ/E-DII	E-DII scores were associated with FPG, insulin, HbA1c, HOMA-IR, 2h-G
King[28]	2019	USA	Cross-sectional	Both	≥20	4434	2 24-hour dietary recalls/DII	Participants in the lowest DII group had lower prevalence and severity of diabetes

FFQ: food frequency questionnaire, DII: dietary inflammatory index, FPG: fasting plasma glucose, HbA1c: glycated hemoglobin A1c, ADII: adapted dietary inflammatory index, HOMA-IR: homeostatic model assessment-insulin resistance, E-DII: energy-adjusted dietary inflammatory index, 2h-G:2-hour glucose

Table 1: *Characteristics of studies reporting the association between dietary inflammatory index (DII) and diabetes and diabetes-related markers (Continued)*

First author	Year	Country	Study design	Sex	Age range	Sample size	Dietary assessment/ Index	Results
Laouali[15]	2019	France	Prospective cohort	Women	Mean=47	70991	FFQ/ADII	Lower ADII scores were related with higher risk of T2DM. The overall association was partly mediated by BMI
Guinter[40]	2019	USA	Prospective cohort	Men	20-84	6016	3-day diet Record/DII	No significant association was observed between DII scores and incidence of T2DM. No effect modification was observed
Ren[33]	2018	China	Cross-sectional	Both	18-75	1712	24-h dietary recall/DII	No relationship was observed with DII and high FPG
Abdurahman[34]	2018	Iran	Cross-sectional	Both	19-59	300	FFQ/DII	No correlation between DII and high FPG was observed
Farhangi[8]	2018	Iran	Cross-sectional	Both	35-80	454	FFQ/DII	Individuals with higher DII scores had significantly higher HbA1c compared with the individuals with lower DII scores
Niknaz[35]	2018	Iran	Cross-sectional	Both	18-64	606	FFQ/DII	Individuals in the highest quartile of DII score had significantly higher FPG compared with the individuals in the lowest quartile of DII score.
Mirmajidi[36]	2018	Iran	Cross-sectional	Both	18-60	171	FFQ/DII	Individuals with higher DII score had significantly elevated levels of FPG, DII was positively associated with FPG in the regression model.
Kim[16]	2018	Korea	Cross-sectional	Both	19-65	9291	24-h dietary recalls/DII	The highest DII quartile was positively associated with the prevalence of hyperglycemia among men.
Mazidi[17]	2018	USA	Cross-sectional	Both	≥18	21874	FFQ/E-DII	In the highest DII quartile had higher FPG, HbA1C, HOMA-IR, insulin and 2h-PG compared with lowest.

FFQ: food frequency questionnaire, ADII: adapted dietary inflammatory index, T2DM: type 2 diabetes, BMI: body mass index, DII: dietary inflammatory index, FPG: fasting plasma glucose, HbA1c: glycated hemoglobin A1c, HOMA-IR: homeostatic model assessment-insulin resistance, 2h-G: 2-hour glucose.

Table 1: Characteristics of studies reporting the association between dietary inflammatory index (DII) and diabetes and diabetes-related markers (Continued)

First author	Year	Country	Study design	Sex	Age range	Sample size	Dietary assessment/ Index	Results
Sokol[37]	2016	Poland	Cross-sectional	Both	45-64	3862	FFQ/E-DII	Among men no increase prevalence of hyperglycemia was observed. Among women, higher DII scores were associated with a reduced hyperglycemia prevalence
Wirth[38]	2014	USA	Cross-sectional	Both	Mean= 42.4	447	FFQ/DII	No significant differences were observed between the 1st and 4th DII quartiles for FPG; however, odds of hyperglycemia was 2.03 times greater among in individuals in 4th quartile compared to 1st quartile.
Park[39]	2018	USA	Cross-sectional	Both	20-90	3733	24-h dietary recalls /DII	MHO individuals with higher DII score had higher HOMA-IR.

FFQ: food frequency questionnaire, E-DII: energy-adjusted dietary inflammatory index, DII: dietary inflammatory index, FPG: fasting plasma glucose, MHO: metabolically healthy overweight and obese adults, HOMA-IR: homeostatic model assessment-insulin resistance.

Studies investigating the association between DII and diabetes risk

The first study that researched the relationship between DII and the presence of T2DM is a cross-sectional Diabetes Mellitus Survey in Mexico City (DMS-MC) study, conducted with 1174 Mexican adults.[26] In this study, it was determined that individuals with the highest quintile of the DII scores had 3 times higher T2DM risk compared to those in the lowest quintile of DII scores. In addition to this, individuals with low DII scores had lower intakes of pro-inflammatory food including red and processed meat products and refined grains, and also had higher intakes of anti-inflammatory food including vegetables, fruits, fish, and nutrients including vitamin A, vitamin C, vitamin E, and vitamin D than those with higher DII scores.[26] A case-control study investigating the risk of DII and prediabetes conducted in Iran showed that the risk of prediabetes was 19 times higher in participants with high DII scores than those with low DII scores.[27] In addition, participants with higher DII scores had significantly higher glucose metabolism markers, including fasting plasma glucose (FPG), oral glucose tolerance test (OGTT), and glycated hemoglobin A1c (HbA1c) compared to lower DII scores. Intakes of pro-inflammatory food such as red meat and sugar were higher and anti-inflammatory foods such as onion and garlic were lower in participants with higher DII scores.[27] King et al.[28] investigated the association between the presence and severity of diabetes and DII. They found a significant relationship between both the presence and the severity of diabetes and DII. With a 1 point increase in the DII score, the risk of diabetes increased by 13%, and having HbA1c higher than >9% increased by 43%. Contrary to these studies, in a cross-sectional study based on data from the Tehran Lipid and Glucose Study (TLGS), no significant relationship was observed between DII and the incidence of T2DM. DII was found to be moderately associated with T2DM risk, but the results became non-significant after adjusting for all potential covariates.[29]

Studies investigating the association between DII and glucose metabolism markers

The number of studies evaluating the relationship between glucose metabolism markers and DII is slightly higher than the studies investigating the incidence of diabetes with DII (Table 1).[9,10,29-39] However, the results of these studies are inconsistent. In general, positive associations were found between DII and glycemic markers in these studies.[8,9,10,29,32,35-39] However, studies with no or inverse association have also existed.[29-34,37-39] In one study with a cross-sectional design conducted with South African women, the effect of DII on T2DM markers was investigated and it was found that DII was positively associated with all T2DM markers including FPG, insulin, HOMA-IR (homeostatic model assessment-insulin resistance), HbA1c, 2-hour plasma glucose (2h-PG).[9] Similar results were also reported in a study that cross-sectional analysis of 2 Dutch studies in which to measure the inflammatory potential of the diet instead of DII, the Adapted Inflammatory Index (ADII), which was developed with some modifications in the DII scoring algorithm was used. A significant positive association between ADII and FPG, HOMA-IR, and OGTT was found.[10] In contrast, no significant association between DII scores and glucose metabolism markers including FPG, fasting insulin, HOMA-IR and HOMA-B, which indicates beta-cell function, was found in a study conducted with 2975 adult individuals.[29] Only higher DII scores were weakly associated with higher 2h-PG. In the study, it was found that DII had no role in fasting insulin secretion, β-cell function, and insulin sensitivity. The slightly positive association between DII and 2h-PG suggests that the role of DII in the regulation of postprandial glucose may occur via non-hepatic mechanisms.[29] In the "Observation of Cardiovascular Risk Factors in Luxemburg" (ORISCAV-LUX) survey, none of the glucose metabolism markers (glucose, insulin, and HOMA-IR) were found to be associated with the DII score. The researchers explained this result by the fact that many of the participants have low DII scores (an anti-inflammatory diet approach). In other words, it was suggested that it might be due to the health status of the population being relatively better.[30] Besides all these studies, in one cross-sectional study conducted with 20823 participants in the south of Italy, an inverse association between DII scores and both diabetes incidence and FPG levels was observed. Participants in the highest quintile (quintile 5) had lower FPG levels and diabetes incidences than the others. The authors stated that this might be because the participants in the highest quintile are younger than the other quintile. Moreover, the older participants might be more likely to have adopted a healthier, more anti-inflammatory diet approach due to their chronic diseases.[31]

Studies investigating mediator factors in the association between DII and diabetes

Studies on DII and diabetes also investigated whether some mediator factors would play a role in a possible relationship between DII and diabetes.[9,10,15,40] Adiposity and several inflammatory markers were the factors that were generally emphasized.[9,10] Previous studies have shown that diet has effects on inflammatory markers and these effects have already been based on the design and development of DII.[6-9] Therefore, it is possible that inflammatory markers play a role as a mediator in the association between DII and T2DM, and also thought that adiposity may be a mediating factor in this association. In adiposity, increasing adipose tissue, especially visceral adipose tissue (VAT) has been associated with various chronic diseases including diabetes.[41] And also, the anatomical location of VAT may lead to the direct delivery of excess free fatty acids and pro-inflammatory cytokines to the liver through the hepatic portal system.[42,43] Increased liver fat accumulation has also been

associated with hepatic IR and increased hepatic glucose production, thus increased the risk for T2DM.[43,44] According to these theories, in a few studies some mediator factor roles have been explored.[9,10,15,40] In the Dutch study in which ADII was used, it was also investigated whether inflammation had a mediator role in the relationship between ADII and glucose metabolism markers, and was concluded that low-grade inflammation detected by six inflammatory markers including TNF-α, IL-6, IL-8, CRP, Serum AA, and sICAM might have a mediator role in the relationship between diet and insulin resistance.[10] In another study, a prospective cohort also using ADII, has been reported that a more anti-inflammatory potential diet is associated with a lower risk of type 2 diabetes and adiposity (evaluated here with BMI) is one of the main mediators that provides this association.[15] The mediating role of inflammatory markers could not be evaluated since there was no data on any biochemical parameter related to the inflammation status of the participants in the study.[15] There is another study in which adiposity acted as a mediator and was concluded that VAT mediates most of the association between Energy-adjusted DII (E-DII) and T2DM markers compared to other adiposity measures (total obesity measured by BMI). Researchers explained that the inflammatory effect of VAT is higher than other adipose tissue regions and has higher rates of lipolysis.[9] Contrary to these results, in a prospective study investigating the inflammatory potential of diet and the incidence of T2DM, neither any relationship between the pro-inflammatory diet measured by DII and the incidence of T2DM, nor a mediator effect of adiposity in this possible relationship was observed.[40]

Evaluation of Studies investigating the association between DII and diabetes

DII represents a recently emergent instrument, and the number of studies investigating the relationship between DII and T2DM is limited, with existing studies exhibiting inconsistency. Upon scrutiny of these studies, the underlying source of inconsistency could be the diverse geographical settings in which the studies have been executed. DII, essentially assesses the inflammatory potential of the daily diet of individuals and encounters potential variability due to the diverse dietary patterns prevalent in each country. The research conducted, although limited in number, covers a wide continent from America to Asia.[16,28,33,40] An important factor that is often emphasized in studies and has the potential for inconsistency in this context is the use of "DII," which is calculated by evaluating 45 parameters. In relevant studies, different numbers of parameters were used to calculate DII from one study to another; For illustration purposes, to detail an example, in one study 27 parameters were used for DII calculation,[26] while in another study calculations were carried out using 37 parameters.[29] However, a noteworthy aspect of this point of difference coincides with the emphasis of the index's developers. They have stated that DII scores should ideally be calculated using 45 parameters.[26] On the other hand, flexibility has been expressed in which the parameters obtained from diet data may also be sufficient.

In addition, standardization of individual intakes to global reference values obtained from the world compound database ensures universal applicability of the index and is valid in every country and in different populations, regardless of the number of parameters. This reflects the index's wide range of applicability, increasing the comparability of studies. This can be considered as one of the other strengths of studies in this field.[14] Likewise, one of the other strengths of the studies is the rigorous use of validated nutritional status assessment tools.[26-29] It allows a detailed evaluation of the main nutritional sources in the diet. In addition, the inclusion of a large population in many studies contributes significantly to the robustness of the studies.[15-17,29,31] Undoubtedly, it is essential to evaluate the limitations when discussing the existing studies. Although a large population was included, the fact that the studies generally had a cross-sectional design is not sufficient to strengthen the inference of causality. Therefore, it is an inevitable necessity to investigate the obtained results more thoroughly in future prospective studies. In addition, it should be noted that although the questionnaire used in studies assessing nutritional consumption has been validated, factors such as recall bias and selection bias are inevitably present.[27]

Conclusion

Studies evaluating diabetes and diabetes-related markers with DII have inconsistent results. Even if consistent results have not yet been obtained in the association between DII and diabetes risk, DII is successful in reflecting the inflammatory potential of diet.[2,19] Many studies have shown that the risk of diabetes or parameters associated with diabetes is higher in individuals consuming a pro-inflammatory diet compared to those consuming an anti-inflammatory diet. Considering diet may increase the risk of T2DM through inflammation, determination of the inflammation potential of diets is essential to prevent diabetes risk. The use of DII may help determine dietary inflammatory potential. Thus, individuals who are detected to have a pro-inflammatory diet may be recommended to change their dietary approach. Therewithal, DII can be an essential tool to characterize the diet of populations and reduce the risk of chronic inflammation-related disease, including diabetes. However, more research on this topic is needed to determine the association between DII and diabetes. It is essential to conduct studies with a prospective design to gain a more comprehensive understanding of the causal relationship between DII and diabetes. In studies, it can be tested whether the DII tool will be useful in practice and whether a diet model that is created especially by considering the DII parameters reduces inflammation and the risk of diabetes development.

Standardizing the subjects with diabetes to be included in future studies may increase the methodological clarity of the results obtained. Additionally, determining the 45 parameters used in DII calculations by more precise and comprehensive food consumption record forms could constitute a significant improvement in terms of methodological reliability.

Conflict of Interest: Authors declare that there is no conflict of interest.

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