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ANA CLÁUDIA DUARTE

EVIDÊNCIAS CIENTÍFICAS VERSUS CONHECIMENTO POPULAR A RESPEITO DO BENEFÍCIO CARDIOMETABÓLICO DO CONSUMO DIETÉTICO DE ÓLEO DE COCO

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Tese de Doutorado apresentada como requisito parcial à obtenção do título de doutora em Ciências Médicas: Endocrinologia da Universidade Federal do Rio Grande do Sul.

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“Não sois máquina! Homens é o que sois!”

Charles Chaplin

RESUMO

Dados recentes da literatura sugerem que a ingestão alimentar de óleo de coco, composto em 92% por ácidos graxos saturados (AGS), não resulta em benefícios cardiometabólicos, como melhora do perfil antropométrico, lipídico, glicêmico e de parâmetros de inflamação subclínica. Apesar disso, seu consumo aumentou nos últimos anos em todo o mundo, fenômeno que pode ser explicado, possivelmente, por um aumento da orientação, por parte de profissionais da área da saúde, de que este seria um óleo tão ou mais saudável que os demais para consumo, além da divulgação em redes sociais destas recomendações.

A fim de se entender os efeitos do óleo de coco na saúde cardiometabólica, desenvolveu-se essa tese, com uma introdução (referencial teórico) para apresentar os diferentes aspectos nutricionais e epidemiológicos relacionados ao óleo de coco, sua relação com a saúde metabólica e possíveis hipóteses sobre as razões do seu alto consumo.

No artigo 1 desenvolveu-se uma revisão sistemática com meta-análise de ensaios clínicos randomizados (ECR), realizados em adultos, e que comparavam o consumo alimentar de óleo de coco com outros óleos e gorduras. Um total de 17 ECRs foram incluídos na revisão sistemática e 7 apresentaram dados suficientes e foram incluídos na meta-análise. A análise dos dados mostrou que a ingestão de óleo de coco comparado a outros óleos e gorduras não é diferente em relação a parâmetros antropométricos, de perfil glicêmico, pressão arterial e inflamação subclínica. Em relação ao perfil lipídico, também não se demonstrou diferenças no efeito do consumo alimentar de óleo de coco em relação a outros óleos e gorduras nos níveis de colesterol LDL-C, triglicerídeos e na relação CT/HDL-C. Observou-se um aumento

estatisticamente significativo, mas clinicamente pouco relevante, dos níveis de colesterol HDL-C com o consumo alimentar de óleo de coco em relação a outros óleos e gorduras. Análises de subgrupo para os diferentes parâmetros cardiometabólicos descritos não demonstraram diferenças. Ao se analisar a qualidade metodológica dos ECRs incluídos na meta-análise, se observa que a maioria apresenta número pequeno de participantes, tempo de seguimento curto, e aplicação de co intervenções, em uma proporção dos estudos, incluindo dieta com restrição calórica e prática de atividade física, fatores que nos levam a interpretar os dados com cautela por poderem impactar no efeito cardiometabólico atribuído ao óleo de coco. Não havendo aparente superioridade do óleo de coco em comparação a outros óleos e gorduras em parâmetros cardiometabólicos, e sabendo-se dos riscos associados do consumo elevado de AGS para a saúde como piora do perfil lipídico, o incentivo do uso deste óleo como sendo de primeira escolha para consumo, deve ser desencorajado.

O objetivo do artigo 2 foi avaliar o consumo, padrões, motivos e crenças relacionados ao uso dietético do óleo de coco e seus benefícios na saúde, por meio de uma pesquisa online com duas populações do sul do Brasil: uma composta por estudantes de diferentes cursos de pós-graduação da Universidade Federal do Rio Grande do Sul e outra por pessoas que acessam a página do Hospital de Clínicas de Porto Alegre no Facebook®. Assim, realizamos um estudo antes/depois usando um questionário online com 11 perguntas. Os participantes que indicaram consumir óleo de coco receberam uma intervenção, que consistia na exposição dos dados de meta-análise recente sobre os efeitos do óleo de coco. O objetivo foi avaliar a possibilidade de mudança de conceitos e crenças a respeito do efeito metabólico e cardiovascular relacionados ao consumo do óleo de coco e aumentar a alfabetização sobre os efeitos deste óleo na saúde. Obtivemos 3160 respostas válidas. A maior parte da amostra

consumia óleo de coco (59,1%). Destes, 82,5% o consideravam saudável e 65,4% o utilizavam pelo menos uma vez por mês. Apesar de considerá-lo saudável, 81,2% dos participantes que utilizavam o óleo, não observaram nenhuma melhora na saúde com o seu uso. Após serem expostos às conclusões de uma meta-análise mostrando que o óleo de coco não apresenta benefícios superiores à saúde quando comparado a outros óleos e gorduras, 73,5% daqueles que consideraram o óleo de coco saudável não mudaram de opinião sobre os seus benefícios. Conclui-se que o consumo do óleo de coco é motivado pelas próprias crenças pessoais, possivelmente incentivadas por recomendações de profissionais da área da saúde de que este seria um óleo que contribuiria para manutenção/melhora da saúde cardiometabólica, mesmo com as evidências científicas mostrando o contrário e, curiosamente, com os participantes não observando melhoras em sua saúde com o consumo de óleo de coco. O fato dos participantes não terem mudado sua percepção sobre os benefícios deste óleo após serem expostos a informações científicas nos revela o quanto pode ser difícil mudar conceitos errados sobre alimentação após estes serem amplamente divulgados e praticados pela população. A desinformação em saúde precisa ser amplamente estudada e encarada como um problema de saúde pública. Estratégias para orientar a população das melhores opções quanto a escolha de óleo para consumo alimentar, bem como de que o óleo de coco não apresenta benefícios definidos (diferentemente de outros óleos de consumo alimentar), devem ser elaboradas e difundidas principalmente para população alvo e de risco para a saúde cardiometabólica. Encorajamos que mais estudos neste formato sejam desenvolvidos a fim de colaborar no combate à desinformação na área da saúde, especialmente na área de nutrição e de hábitos de vida saudáveis.

Palavras-chave: Óleo de coco. Mídia social. Pesquisa. Online. Informação em saúde. Internet. Ácidos graxos saturados. Perfil lipídico. Perfil antropométrico.

ABSTRACT

Recent data from the literature suggest that dietary intake of coconut oil, composed of 92% saturated fatty acids (SFA), does not result in cardiometabolic benefits, such as improvement in anthropometric, lipid, glycemic and subclinical inflammatory parameters. Despite this, its consumption has increased in recent years all over the world, a phenomenon that can be explained, possibly, by an increase in the concept, from health professionals, that this oil would be as healthy or healthier than the others for consumption, in addition to the dissemination of these recommendations on social networks.

In order to understand the effects of coconut oil on cardiometabolic health, this thesis was developed, with an introduction (theoretical framework) to present the different nutritional and epidemiological aspects related to coconut oil, its relationship with cardiometabolic health and possible hypotheses about the reasons for their high consumption.

In article 1, a systematic review was developed with a meta-analysis of randomized clinical trials (RCTs) carried out in adults, which compared the dietary consumption of coconut oil with other oils and fats. A total of 17 RCTs were included in the systematic review and 7 had sufficient data to be included in the meta-analysis. Data analysis showed that the intake of coconut oil compared to other oils and fats does not cause a better metabolic control in relation to anthropometric parameters, glycemic profile, blood pressure and subclinical inflammation. Regarding the lipid profile, no differences were shown in the effect of dietary intake of coconut oil in relation to other oils and fats on LDL cholesterol and triglycerides levels, as well as the CT/HDL ratio. A statistically significant but clinically insignificant increase in HDL cholesterol levels was observed with the dietary consumption of coconut oil in relation to other oils

and fats. Subgroup analyzes for the different cardiometabolic parameters described showed similar findings. When analyzing the methodological quality of the RCTs included in the meta-analysis, it is observed that most of them have a small number of participants, short follow-up time, and application of co-interventions, in a proportion of the studies, including a calorie-restricted diet and advice for physical activity, factors that lead us to interpret the data with caution as they may impact the cardiometabolic effect attributed to coconut oil. As there is no apparent superiority of coconut oil compared to other oils and fats in cardiometabolic parameters and knowing the associated risks of high consumption of SFA for health as a resultant worsening of the lipid profile with its intake, encouraging the consumption of this oil as the first choice should be discouraged.

The aim of article 2 was to evaluate consumption, patterns, motives and beliefs related to the dietary intake of coconut oil and its health benefits, through an online survey with two populations in Southern Brazil: one composed of students from different courses at the Federal University of Rio Grande do Sul and another by people who access the Hospital de Clínicas de Porto Alegre page on Facebook®. Thus, we conducted a before/after study using an online questionnaire with 11 questions. Participants who indicated consuming coconut oil received an intervention, which consisted of exposing recent meta-analysis data on the effects of coconut oil. The objective was to evaluate the possibility of changing concepts and beliefs about the metabolic and cardiovascular effects related to the consumption of coconut oil and increasing literacy about the effects of this oil on health. We got 3160 valid responses. Most of the sample consumed coconut oil (59.1%). Of these, 82.5% considered it healthy and 65.4% used it at least once a month. Despite considering it healthy, 81.2% of the participants who used the oil did not observe any improvement in health with its

use. After being exposed to the findings of a meta-analysis showing that coconut oil does not have superior health benefits when compared to other oils and fats, 73.5% of those who considered coconut oil healthy did not change their opinion about its benefits. These results lead us to conclude that the intake of coconut oil is motivated by own personal beliefs, possibly encouraged by recommendations from health professionals, even with the scientific evidence showing the contrary and, interestingly, with the participants not observing improvements in their health with this consumption. The fact that the participants did not change their perception of the benefits of this oil after being exposed to scientific information reveals how difficult it can be to change misconceptions about dietary habits after they are widely disseminated and practiced by the population. Health misinformation needs to be widely studied and considered as a public health problem. Strategies to guide the population on the best options regarding the choice of oil for alimentary consumption, as well as that coconut oil does not have defined benefits, unlike other oils for food consumption, should be developed and disseminated mainly to the target population and risk to cardiometabolic health. We encourage that more studies to address this issue be developed in order to collaborate in the fight against misinformation in the health area, especially in the area of nutrition and healthy lifestyle habits.

Keywords: Coconut oil; Social media; Survey; Online; Health information; Internet. Coconut oil. Saturated fatty acids. Lipid profile. Anthropometric profile.

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LISTA DE ABREVIATURAS E SIGLAS

AGS: ácidos graxos saturados

C12:0: ácido láurico

C8:0: ácido caprílico

C10:0: ácido cáprico

C14:0: ácido mirístico

C16:0: ácido palmítico

AG: ácidos graxos

AGM: ácidos graxos monoinsaturados

AGP: ácidos graxos poli-insaturados

C18:2: ácido linoleico

C18:3: ácido linolênico

AGE: ácidos graxos essenciais

AGCM: ácido graxo de cadeia média

AGCL: ácido graxo de cadeia longa

TG: triglicerídeos

VLDL: proteínas de muito baixa densidade

CT: colesterol total

LDL-C: lipoproteína de baixa densidade

ECR: ensaios clínicos randomizados

HDL-C: lipoproteína de alta densidade

CETP: cholesterol ester transfer protein

AGI: ácidos graxos insaturados

TLR: toll like receptor

NF-Kβ: fator nuclear Kappa

COX2: ciclo-oxigensase-2

PCR: proteína C reativa

IL-6: interleucina 6

INF-γ: interferão-gama

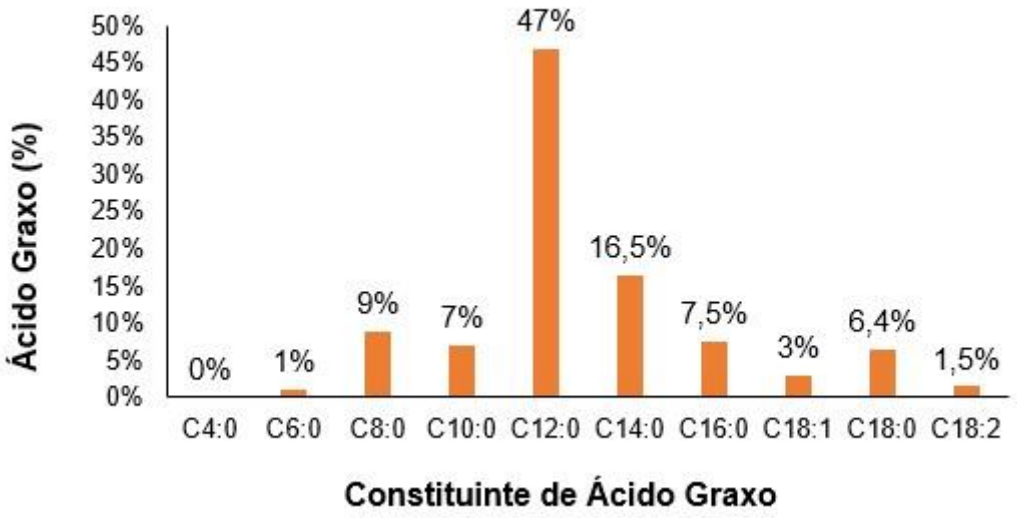
SUMÁRIO

CAPÍTULO 1 - REFERENCIAL TEÓRICO

ÓLEO DE COCO: CARACTERÍSTICAS E METABOLISMO

Aproximadamente 92% do óleo de coco é composto de ácidos graxos saturados (AGS), dentre os quais, 45-50% correspondem ao ácido láurico (C12:0), 9% ácido caprílico (C8:0), 7% ácido cáprico (C10:0), 16% ácido mirístico (C14:0) e 8% ácido palmítico (C16:0). Os ácidos graxos (AG) remanescentes são monoinsaturados (AGM) (6%) e polinsaturados (AGP) (2%). O óleo de coco apresenta baixa concentração de ácido linoleico (18:2) e não contém ácido linolênico (18:3) [ambos ácidos graxos essenciais (AGE)] (Figura 1) (1).

Figura 1. Composição de ácidos graxos do óleo de coco



C4:0 = butírico; C6:0 = ácido capróico; C8:0 = ácido caprílico; C10:0 = ácido cáprico; C12:0 = ácido láurico; C14:0 = ácido mirístico; C16:0 = ácido palmítico; C18:0 = ácido esteárico; C18:1: = ácido oleico; C18:2 = ácido linoleico

As gorduras alimentares são compostas por uma mistura de AGS, AGM e AGP, variando a composição de AG dependendo da sua fonte: se de origem animal ou vegetal. Gorduras com maior proporção de AGM e AGP geralmente são líquidas em temperatura ambiente e são denominadas como óleos. Já as gorduras com maior proporção de AGS, especialmente os de cadeia longa, geralmente são sólidas em temperatura ambiente e são denominadas como gorduras (2,3). Por conta disso, “óleo de coco”, como popularmente é conhecido, não seria o termo correto a ser empregado por ser uma gordura sólida em temperatura ambiente devido à sua alta quantidade de AGS, significativamente maior que a maior parte dos outros óleos de consumo alimentar, sendo considerado, portanto, uma gordura sólida para fins nutricionais (2).

O ácido láurico, AG mais abundante no óleo de coco, pode ser classificado tanto como um ácido graxo de cadeia média (AGCM), como ácido graxo de cadeia longa (AGCL), sendo, portanto, um AG com propriedades intermediárias entre ambos (4). Os AGCM, formados principalmente pelos ácidos caprílico (C8:0) e cáprico (C10:0), são absorvidos no intestino delgado ligados à albumina e atingem o fígado via sistema portal, não elevando a trigliceridemia (5). Já, 70 a 75% do ácido láurico é absorvido com quilomícrons (semelhante à absorção dos AGCL) (6), sendo sua presença nos quilomícrons dose-dependente (7). No geral, AGCM (C8:0 e C10:0) apresentam baixo peso molecular (512 na média), diferente dos AGCL que têm um peso molecular mais alto (o peso molecular do óleo de coco é 638). AGs com menores pesos moleculares facilitam a ação da lipase pancreática, sendo hidrolisados de maneira mais eficiente no intestino curto comparando-se com AG de cadeias mais longas (8).

Os AGCL são esterificados nos enterócitos, onde formam os triglicerídeos (TG), que são transportados até o sistema linfático, via corrente sanguínea pelos quilomícrons. A lipase lipoproteica hidrolisa os TG dos quilomícrons, liberando AG

para os tecidos periféricos, que podem ser utilizados como fonte energética ou reesterificados em novos TG para armazenamento (3). Os AGCL necessitam do transportador carnitina palmitol transferase na membrana mitocondrial externa, para serem internalizados na organela e oxidados em Acetil-Coa para servirem como fonte de energia (9,10). Os AGCM são absorvidos diretamente na corrente sanguínea, não sendo incorporados significativamente em quilomícrons e lipoproteínas de muito baixa densidade (VLDL). São considerados uma fonte rápida de energia pois, ao passarem pelos enterócitos, atingem a circulação portal, sendo transportados ao fígado ligados à albumina, onde são oxidados. Especulou-se que, devido ao seu metabolismo rápido, os AGCM estimulariam a termogênese, diminuindo a sua deposição no tecido adiposo, o que aumentaria a saciedade sem aumentar os níveis séricos de colesterol total (CT) (3). No entanto, não há estudos experimentais que comprovem estas teorias. Mesmo que houvesse, esses achados não poderiam ser extrapolados para o óleo de coco, devido a sua grande concentração de ácido láurico que não tem sua absorção, transporte e metabolismo realizados dessa forma, como exposto anteriormente.

ÓLEO DE COCO: MERCADO E CONSUMO

O coco é um alimento consumido mundialmente. O cultivo do coqueiro se dá especialmente com o objetivo fim de exploração comercial da copra dos frutos para produção de óleo de coco e coco seco desidratado (11). O continente asiático detém a maior parte da área plantada de coqueiros no mundo. Algumas populações asiáticas, como o Sri Lanka, Minangkabau e Filipinas, têm o coco como parte da alimentação diária. As Filipinas, Indonésia e Índia são os países que mais produzem

óleo de coco no mundo, extraindo o óleo de coco em duas versões: refinado, clareado e desodorizado e extraído à frio, sem processo de refinamento (óleo de coco virgem) (12).

Nos últimos anos, a produção mundial de óleo de coco, principalmente o óleo de coco virgem, não refinado aumentou expressivamente (6). Estima-se que em 2016 houve um consumo de 640 mil toneladas de óleo de coco nas Filipinas, 449 mil toneladas na Índia e 468 mil toneladas nos Estados Unidos (13). O Reino Unido é um dos países da Europa que mais importa óleo de coco do Sri Lanka (cerca de 7% em 2015) (14). Não há registros sobre o consumo do óleo de coco no Brasil, no entanto, até meados de 2010, o consumo do óleo de coco para preparo de alimentos ou uso como suplemento dietético era pouco usual.

Até meados de 2010, a utilização de óleo de coco na dieta era menos comum nas populações ocidentalizadas. Rico em AGS, gordura bem descrita na literatura como relacionada ao aumento dos níveis plasmáticos de lipoproteína de baixa densidade (LDL-C) e aumento do risco cardiovascular, o consumo de óleo de coco não era recomendado (3).

Estudos em humanos testando os efeitos do óleo de coco na saúde cardiometabólica datam da década de 1990 (15,16), mas somente após a publicação e, possivelmente, divulgação dos resultados de diferentes estudos (17,18) em encontros científicos e nas redes sociais é que ocorreu maior interesse por sua prescrição e utilização como uma opção de óleo saudável. Estes estudos de curto prazo mostraram uma leve melhora no perfil lipídico e antropométrico com a ingestão de óleo de coco em populações adultas saudáveis (17,18). No entanto, seus resultados são baseados em marcadores substitutos de saúde cardiometabólica, e nenhum estudo até o momento avaliou o benefício potencial do consumo de óleo de

coco na prevenção do diabetes e de desfechos cardiovasculares. Também, é possível, que a partir desses estudos, desenvolveu-se um interesse da indústria de alimentos em comercializar o óleo de coco como alimento saudável, ocorrendo uma disseminação dos benefícios desse óleo em sites, blogs e perfis de mídias sociais de profissionais de saúde e prática clínica desses, difundindo o óleo de coco como uma gordura saudável para ser utilizada para cozinhar, ser adicionado em saladas e outras preparações alimentícias (19) (Figura 2)

Figura 2. Modelo do processo que permeia entre a geração e interpretação de uma evidência científica até a divulgação dos seus dados

Legenda: O modelo demonstra as possíveis etapas que resultaram em um aumento do consumo alimentar do óleo de coco e que pode ser, possivelmente, aplicado em outros exemplos da construção da desinformação em saúde. Essas etapas ocorrem após a divulgação de publicações com limitações metodológicas e eventual presença de vieses. Pode resultar na interpretação distorcida de dados científicos, aplicação clínica inadequada e geração em mídia social da desinformação relacionada aos cuidados de saúde.

ÓLEO DE COCO: EVIDÊNCIAS CIENTÍFICAS EM RELAÇÃO AO SEU EFEITO EM PARÂMETROS CARDIOMETABÓLICOS

Algumas populações asiáticas, como do Sri Lanka, Minangkabau, Filipinas e das ilhas Pukapuka e Tokelau na Polinésia, antes da introdução de uma dieta mais ocidentalizada, apresentavam níveis de CT mais baixos que populações mais ocidentalizadas, além de menor morbidade por doença cardiovascular (20-23). Dentre as possíveis hipóteses aventadas para se identificar a causa, foi sugerido que o consumo de gordura de coco poderia estar, em parte, relacionado a estes achados. No entanto, é importante ressaltar que, embora estas populações apresentassem um padrão alimentar onde a maior fonte de AGS e energia era a gordura do coco, diferentemente de populações mais ocidentalizadas com maior consumo de fontes de gordura animal, elas apresentavam como maior fonte proteica o consumo de peixes e, como maior fonte de carboidratos, as frutas nativas da região, sendo um padrão alimentar saudável, habitualmente rico em fibras, pobre em alimentos ultra processados e sacarose (23).

Diferentes entidades científicas, não recomendam o uso do óleo de coco como AG de preferência para o consumo humano (2,5,24). A ingestão de óleo de coco está associada a uma piora no perfil lipídico, aumentando a concentração plasmática de LDL-C, um fator de risco bem definido para doenças cardiovasculares, sem melhora no peso corporal, controle glicêmico e parâmetros inflamatórios em comparação com outros óleos não tropicais (25-27). Esses dados levaram diferentes sociedades científicas especializadas e dedicadas à saúde metabólica a se posicionaram contra o uso da gordura de coco, também para perda de peso, por não haver na literatura,

até então, comprovação científica de que o óleo de coco possua algum mecanismo que promova a perda de peso (28).

Existe uma série de limitações metodológicas nos estudos de intervenção que avaliaram o benefício do óleo de coco em parâmetros metabólicos (Figura 2). Nas três meta-análises em que se avaliou o consumo de óleo de coco versus outros óleos em relação ao perfil lipídico, antropométrico, marcadores inflamatórios e níveis de glicemia, houve a inclusão de estudos do tipo cruzado (24-26). Dentre as limitações relacionadas à inclusão desses estudos estão: a ausência de definição do tempo de wash-out, da ordem de randomização dos grupos de intervenção e falta de clareza da existência de parâmetros de avaliação clínica, nutricional e laboratorial antes e logo após todas as intervenções.

Ainda, os ECR incluídos nas meta-análises apresentam, de uma maneira geral, tempo de seguimento curto (uma a 12 semanas) e grupos comparativos que agrupam diferentes braços, com óleos com propriedades nutricionais distintas. Por exemplo, um estudo incluído na revisão de Neelakantan e cols agrupou 3 diferentes braços de intervenção como grupo controle (óleos de chia, cártamo e soja) para comparação com o óleo de coco. Este grupo controle ignorou que os óleos demonstram respostas distintas sobre parâmetros metabólicos, eventualmente, em direções opostas, o que limitaria a capacidade de meta-analisar os dados (25). Por outro lado, um ECR indiano (198 indivíduos com doença cardiovascular, dois anos de duração), analisou parâmetros metabólicos comparando-se a ingestão de 15% do valor calórico diário com óleo de coco versus óleo de girassol no preparo dos alimentos. Não houve diferenças nos níveis de CT, LDL-C, lipoproteína de alta densidade (HDL-C), TG e VLDL, peso, circunferência da cintura, percentual de gordura e níveis de hemoglobina glicada (HbA1c) comparando-se o consumo dos dois óleos (30). Estes dados sugerem

que estudos de maior duração sejam mais adequados para demonstrar não haver benefício do consumo de óleo de coco sobre parâmetros metabólicos a longo prazo.

Além disso, o estabelecimento de conduta baseada em desfechos substitutos apresenta limitações de aplicabilidade para tomada de decisão, demonstrando ser, muitas vezes, uma medida inadequada. Como exemplo podemos citar o estudo que avaliou a eficácia do inibidor da *Cholesterol Ester Transfer Protein* (CETP), um fármaco que aumenta significativamente os níveis de HDL-C, mas que em seu estudo de segurança cardiovascular, associou-se há um maior risco de desfechos cardiovasculares, tendo que ser interrompido (31).

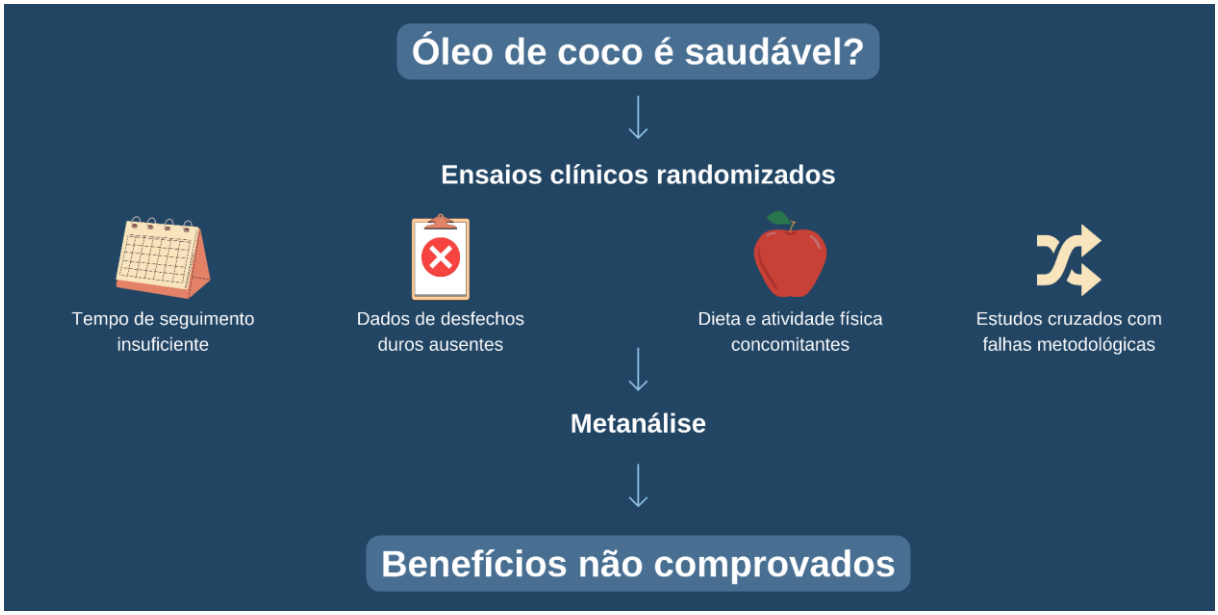
Estudo observacional mostrou que a substituição de 5% da ingestão de energia de AGS pela mesma ingestão de AGP ou AGM está associada a um risco 27 e 13% menor de mortalidade, respectivamente (32). Esses dados corroboram as recomendações da atual diretriz alimentar para americanos que preconiza a redução de AGS para menos de 10% das calorias e sua substituição por ácidos graxos insaturados (AGI) (2). Além disso, dados recentes de coortes prospectivas de longo prazo e meta-análises mostraram que as mesmas recomendações estão associadas à prevenção do ganho de peso, redução da resistência à insulina e risco de diabetes (33-36).

Tendo em vista a composição de ácidos graxos do óleo de coco, seria possível este alimento apresentar propriedades antioxidantes e seu consumo melhorar parâmetros inflamatórios? Até o presente momento, estudos que avaliaram os efeitos antioxidantes do óleo de coco são, em sua maioria, preliminares e experimentais, não podendo seus dados serem replicados em humanos (5). O óleo de coco virgem, extraído pelo processo molhado diretamente do leite de coco em ambiente controlado de temperatura, parece ter melhores propriedades nutricionais, uma vez que retém

maior quantidade de componentes insaponificáveis, como vitamina E e polifenóis, tendo, portanto, maior capacidade antioxidante (35). Todavia, o óleo de coco geralmente é obtido por processo seco, a partir do óleo de copra. Este óleo é extraído da “carne” do coco, a qual é ralada, moída e cozida em água para extrair o óleo, e é exposto a temperaturas muito elevadas ou à luz durante vários dias, até que a umidade em excesso seja removida. Essa exposição à luz solar ou altas temperaturas poderia inativar componentes reconhecidamente antioxidantes tais como tocoferóis, tocotrienóis e polifenóis (37).

Dentre todos os AGS, o ácido láurico é o que apresenta maior potencial inflamatório (38). Estudos mostram que o ácido láurico é capaz de ativar vias inflamatórias por meio da ativação de *toll like receptor 4* (TLR4), o que leva a secreção de citocinas inflamatórias e ativação de células T (36,37). Já um estudo in-vitro, com macrófagos, observou que o ácido láurico induz a ativação do fator nucelar kappa (NF-Kβ), levando a um aumento da expressão de ciclo-oxigenase-2 (COX2), pela ativação de TLR 2 e 4 (40). Meta-análise recente de ECR comparando os efeitos do consumo de óleo de coco, rico em ácido láurico, com outros óleos, não observou diferença nas concentrações plasmáticas de proteína C-reativa (PCR), um importante marcador de inflamação subclínica (25). Em um ECR, em indivíduos normocolesterolêmicos da Malásia, comparou-se os efeitos do consumo de óleo de coco com o de óleo de palma ou oliva (follow-up: 5 semanas) nas concentrações plasmáticas de homocisteína e marcadores inflamatórios como fator de necrose tumoral α, interleucina-1β, interleucina-6 (IL-6), INF-γ e–IL e não se encontrou diferença entre estes parâmetros (41). Esses dados não corroboram a divulgação de benefícios relacionados do óleo de coco sobre diferentes parâmetros metabólicos relacionados ao desenvolvimento de obesidade, diabetes e doença cardiovascular.

Figura 3. Limitações de estudos prévios sobre consumo de óleo de coco e potenciais vieses destes resultados



Legenda: Limitações metodológicas relacionadas aos resultados de estudos prévios sobre o efeito do consumo de óleo de coco em parâmetros cardiometabólicos e potenciais vieses relacionados a estes achados, resultando em ausência de evidências relacionadas ao seu consumo

DESINFORMAÇÃO EM SAÚDE

A desinformação em saúde é um problema mundial e sua prevalência aumenta com a produção de conteúdo de saúde nos meios de comunicação de massa e redes sociais, elevando a quantidade de recomendações que não são baseadas em evidências (42,43).

As mídias sociais são essenciais para o fluxo de informações, construção do conhecimento e difusão de opiniões. No entanto, com a inserção dessas novas mídias no cotidiano, a sociedade se acostumou a acessar informações de saúde em larga

escala, porém nem sempre de fontes confiáveis. Estudos mostram que as informações publicadas em sites relacionados à saúde podem apresentar imprecisões e baixa qualidade de evidência da informação disponibilizada (44-48), pois qualquer pessoa pode publicar sobre informações em saúde (49).

Dessa forma, muitas informações são divulgadas, mas pouco conhecimento está sendo construído (50). Embora as mídias sociais tenham um papel fundamental na saúde, contribuindo para a transmissão das evidências científicas para a sociedade, apresentando fatos e descobertas de forma acessível, há também grande espaço para a disseminação de informações não comprovadas ou com seus resultados apresentados de maneira distorcida. Desta forma, rumores, notícias falsas, grupos e/ou opiniões de figuras públicas baseadas em suas próprias crenças, acabam atingindo milhares de pessoas, criando teorias e verdades infundadas, o que pode impactar de maneira negativa a saúde dos indivíduos.

Em 2002, Eysenbach definiu o termo “infodemiologia” como o processo que permite a identificação de áreas onde há uma lacuna entre a tradução das melhores evidências científicas, propostas por especialistas no assunto, e como a maioria das pessoas utiliza essas informações (51). Para combater a infodemiologia é necessário traduzir a ciência da forma mais clara possível para a população em seus mais diversos graus de conhecimento e educação, utilizando uma linguagem acessível e didática por parte das autoridades, a fim de mudar crenças que podem levar a ações negativas para saúde (42,43,52). Mesmo aplicando-se grandes esforços, o impacto na mudança de conceitos enganosos pode não ser eficaz, pois os indivíduos tendem a interpretar as informações recebidas para confirmar suas crenças e não o contrário, o que é chamado de viés de confirmação (53).

Muitas pessoas apresentam dificuldade para interpretar se uma informação em saúde é de fonte confiável e, de fato, baseada em sólidas evidências científicas, o que as expõem a erros, e, até mesmo, reduzem sua participação em programas de rastreamento de doenças, além de uma menor adesão aos tratamentos propostos (54). Pessoas com menor escolaridade e/ou menor alfabetização em saúde são os grupos de maior risco neste contexto (55). Um estudo demonstrou que cerca de nove em cada dez adultos americanos não possuem as habilidades necessárias para o manejo e prevenção de doenças (56).

Alfabetização em saúde é originalmente definida como as habilidades cognitivas e sociais que determinam a motivação e a capacidade do indivíduo de obter acesso para compreender e usar as informações de maneira que promovam e mantenham uma boa saúde (58). Estudos mostram que pessoas com baixo grau de alfabetização em saúde pesquisam menos sobre saúde, escolhem diferentes fontes de informação e têm menor grau de entendimento sobre rótulos de medicamentos e informações passadas sobre saúde (58-60). Ainda, demonstrou-se que pessoas com baixo grau de alfabetização em saúde não confiam nas informações de saúde divulgadas por fontes governamentais e avaliam se a informação de saúde é confiável ou não com base na posição da página na internet nos sites de busca e pela qualidade das imagens do site (61). Em contraponto, existem evidências que mostram que as informações divulgadas pelos governos e instituições são geralmente confiáveis (62,63). Uma revisão sistemática da literatura mostra que, no geral, indivíduos com níveis educacionais mais baixos apresentam uma pior habilidade (real e auto-avaliada) para avaliar a qualidade das informações de saúde online e menor confiança nas informações divulgadas online do que seus pares com nível educacional mais elevado (49).

O desenvolvimento de estratégias para potencializar a disseminação de conceitos adequados de saúde, a fim de proteger a população contra a desinformação, é uma nova área de investigação. Algumas intervenções anti-propagação de notícias falsas relacionadas à saúde já foram testadas. Como exemplo, é possível utilizar correções algorítmicas e sociais, expondo a rede social às informações reais produzidas por um algoritmo ou por um usuário da rede social, a fim de proteger a sociedade contra notícias falsas (64). Também é possível classificar a credibilidade da fonte das informações fornecidas pelo usuário da rede social (65). Uma solução eficaz certamente será uma ampla combinação de diversas abordagens desenvolvidas por especialistas em saúde, ciências sociais e da computação trabalhando juntos em pesquisas interdisciplinares para encontrar maneiras de lidar com a desinformação sobre saúde nas mídias sociais (65-67). Também é necessária uma regulamentação do compromisso dos profissionais de saúde com a divulgação social da informação amparada nas melhores evidências científicas disponíveis, com uma análise crítica dos resultados dos estudos (43).

JUSTIFICATIVA

Nos últimos 10 anos, o consumo dietético de óleo de coco foi bastante estimulado por profissionais da saúde em meios científicos e de comunicação diversos (blogs, sites, rádio e TV, e em perfis pessoais nas mais diversas redes sociais). Em um primeiro momento, seu consumo foi estimulado baseado em ECRs de curta duração que mostram benefícios discretos do uso do óleo de coco em parâmetros antropométricos e perfil lipídico (18,19). Além disso, devido aos AGCM apresentarem um metabolismo mais rápido dos que os demais comprimentos de cadeia de AG,

pensou-se que tais benefícios poderiam ser extrapolados para o óleo de coco, estimulando o seu consumo (3).

Com a elaboração de novos ECRs sobre o tema e a publicação de diferentes meta-análises (25,26), diferentes órgãos científicos e governamentais se posicionaram contra o consumo de óleo de coco, pois seu impacto no perfil antropométrico seria nulo ou muito discreto, face ao seu potencial de elevar o LDL-C, sendo um marcador importante no desenvolvimento de DCV (6,2,24). No entanto, uma vez tendo sido amplamente divulgado como um alimento saudável em diferentes meios de comunicação e sendo incorporado nas crenças alimentares dos indivíduos como uma gordura para fins dietéticos, como desconstruir este conceito errôneo em saúde?

A integração e comparação entre o que as melhores evidências científicas nos mostram sobre o consumo do óleo de coco e as percepções da população sobre o uso dietético deste alimento nos proporcionará identificar a lacuna entre a nutrição baseada em evidências e a opinião pública, possibilitando o desenvolvimento de estratégias de saúde pública para aumentar a alfabetização de nossa população em nutrição, buscando-se com isto, uma melhora da saúde metabólica da população e a prevenção de doenças crônicas, como a obesidade, o diabetes e as doenças cardiovasculares.

OBJETIVOS

Objetivo Geral

Avaliar os efeitos do óleo de coco em parâmetros metabólicos, e identificar quais as motivações para o seu consumo como alimento em uma população do sul do Brasil.

Objetivos Específicos

1. Avaliar, por meio de uma revisão sistemática com meta-análise de ensaios clínicos randomizados, os efeitos do consumo de óleo de coco, em comparação ao consumo de outros óleos, gorduras ou placebo no perfil lipídico, antropométrico, glicêmico e inflamatório de adultos.
2. Avaliar, por meio de uma pesquisa online, o consumo de óleo de coco e a motivação para este consumo em uma população do Sul do Brasil.
3. Avaliar, por meio de uma pesquisa online, se uma população do Sul do Brasil, altera suas crenças sobre o consumo de óleo de coco após serem expostos aos resultados de uma pesquisa científica sobre o tema.

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CAPÍTULO II - The Effects of Coconut Oil on the Cardiometabolic Profile: a Systematic Review and Meta-Analysis of Randomized Clinical Trials

Original Article

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Abstract:

Background: Despite having a 92% concentration of saturated fatty acid composition, leading to an apparently unfavorable lipid profile, body weight and glycemic effect, coconut oil is consumed worldwide. Thus, we conducted an updated systematic review and meta-analysis of randomized clinical trials (RCTs) to analyze the effect of coconut oil intake on different cardiometabolic outcomes.

Methods: We searched Medline, Embase, and LILACS for RCTs conducted prior to April 2022. We included RCTs that compared effects of coconut oil intake with other substances on anthropometric and metabolic profiles in adults published in all languages, and excluded non-randomized trials and short follow-up studies. Risk of bias was assessed with the RoB 2 tool and certainty of evidence with GRADE. Where possible, we performed meta-analyses using a random-effects model.

Results: We included seven studies in the meta-analysis (n = 515; 50% females, follow up from 4 weeks to 2 years). The amount of coconut oil consumed varied and is expressed differently among studies: 12 to 30 ml of coconut oil/day (n=5), as part of the amount of SFAs or total daily consumed fat (n=1), a variation of 6 to 54.4 g/day (n=5), or as part of the total caloric energy intake (15 to 21%) (n=6). Coconut oil intake did not significantly decrease body weight (MD -0.24 kg, 95% CI -0.83kg to 0.34 kg), waist circumference (MD -0.64 cm, 95% CI -1.69 cm to 0.41 cm), and % body fat (-0.10%, 95% CI -0.56 to 0.36), low-density lipoprotein cholesterol (LDL-C) (MD -1.67 mg/dL, 95% CI -6.93 to 3.59 mg/dL), and triglyceride (TG) levels (MD -0.24 mg/dL, 95% CI -5.52 to 5.04 mg/dL). However, coconut oil intake was associated with a small increase in high-density lipoprotein cholesterol (HDL-C) (MD 3.28 mg/dL, 95% CI 0.66 to 5.90 mg/dL). Overall risk of bias was high, and certainty of evidence was very-low. Study limitations include the heterogeneity of intervention methods, in addition to small

samples and short follow-ups, which undermine the effects of dietary intervention in metabolic parameters.

Conclusions: Coconut oil intake revealed no clinically relevant improvement in lipid profile and body composition compared to other oils/fats. Strategies to advise the public on the consumption of other oils, not coconut oil, due to proven cardiometabolic benefits should be implemented.

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Keywords: coconut oil, saturated fatty acids, lipid profile, anthropometric profile

Background

Cardiovascular disease (CVD), particularly coronary heart disease and stroke, is a major public health problem, being responsible for one-third of deaths worldwide (1-4). Despite the great effort of different scientific organizations to fight against the burden of major risk factors for CVD, it is estimated that 11 million deaths and 255 million disability-adjusted life-years are attributable to dietary risk factors (5-8).

The impact of different types of dietary fats on health has been studied, and its contribution to the development of diseases, causing major burden, such as diabetes, cardiovascular diseases and cancer has been debated (6, 9). A recent report from the American Heart Association based on different prospective cohort studies, randomized clinical trials (RCTs), and meta-analyses estimated that replacing 5% of energy intake of saturated fatty acids (SFAs) with the same intake of polyunsaturated fatty acids (PUFAs) or monounsaturated fatty acids (MUFAs) was associated with a 25% and 15% lower risk of coronary heart disease, respectively (6). In light of this evidence, the most recent Dietary Guidelines for Americans recommend a reduction in SFAs to less than 10% of calories and their replacement with unsaturated fats (10). Additionally, recent data from long-term prospective cohorts and meta-analyses have shown that these recommendations are associated with weight gain prevention and reduction of insulin resistance and risk for diabetes (11-15).

Despite that, coconut oil, which is more than 90% SFA, has been widely recommended on social media for the management of obesity, diabetes, and lipid disorders, broadening its consumption all over the world (16-18). In increasing demand, the estimated consumption of coconut oil in the United States reached 400,000 tons in 2010 (19). Nonetheless, before the recent rise in coconut oil consumption in western countries, it was only mainly present in some Asian populations' diets (20-22).

A recent systematic review showed that lauric, myristic, and palmitic fatty acids - the major components of coconut oil - are responsible for the highest increase in low-density lipoprotein cholesterol (LDL-C) levels, which is a major risk factor for CVD (19). Unlike other types of oils which were consistently proven to prevent weight gain, diabetes, CVD, and mortality (23-25), studies that analyzed how coconut oil intake affects weight, lipid and glycemic levels are mostly based on small, short-term observational studies and clinical trials (16, 26, 27). In addition, there are meta-analyses including RCTs that have even demonstrated that coconut oil intake increased LDL-C in comparison to non-tropical vegetable and animal oils and did not observe differences in TG levels (28, 29).

Due to the popularity of coconut oil as a “healthy” food, its broad dietary consumption has risen all over the world. This has led to increasing difficulties to translate medical and nutritional science into adequate recommendations for physicians and health workers as well as laymen. Given this context, we conducted an updated systematic review and meta-analysis of RCTs investigating the effects of coconut oil intake on body weight and composition, lipid profile, glycemic status, blood pressure, and subclinical inflammation in adults.

Methods

This systematic review and meta-analysis was prospectively registered on PROSPERO (CRD42018081461) and was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement (30).

Search methods for the identification of studies

We searched the MEDLINE, EMBASE, and LILACS databases to identify studies analyzing the effects of coconut oil intake on weight, lipid and glycemic profiles, blood pressure, and subclinical inflammation in adults from inception to April, 2022, and searched www.clinicaltrials.gov for potentially available unpublished results (Supplementary Appendix I). The references of relevant systematic reviews were screened manually to identify further relevant citations. When an article did not present the results of interest, we contacted the authors by email requesting the data.

Study selection and data extraction

Study inclusion and data extraction were conducted independently (A.C.D., C.R.A., and C.A.). Reviewers resolved discrepancies by discussion and, when necessary, with adjudication by a third party (F.G.). Inter-rater agreement was assessed using the Kappa statistic and percentage of agreement. Kappa statistic was calculated with SPSS software (version 18.03; Chicago, USA). Data extracted were reviewed and double checked by two independent authors (B.F.S. and E.N.M.), who were blinded to the objectives of the meta-analysis.

A standard protocol for data extraction was used, including the following variables: number of participants, study design, duration of the study, interventions, demographic data, age and sex, chronic disease status, as well as exposures of interest before and after the interventions. Data was extracted to assess the effects of coconut oil on anthropometric profile (body weight, body mass index, waist circumference and body composition), lipid profile (LDL-C, HDL-C, total cholesterol [TC], TC/HDL ratio and triglycerides), glycemic profile (glucose, insulin, the homeostasis model assessment [HOMA] β and HOMA-S, HOMA-IR and glycated hemoglobin [HbA1C]), inflammatory profile (ultra-sensitive c-reactive protein [US-CRP], fibrinogen, total homocysteine

[tcHcy], interleukins [IL] 1β, IL-6, IL-8 and interferon-gamma [IFN- γ]) and blood pressure (systolic blood pressure and diastolic blood pressure).

Inclusion and exclusion criteria

Since our aim was to evaluate the isolated effect of coconut oil with no influence of dietary pattern, we considered eligible only RCTs (both parallel group or crossover randomized trials) which analyzed the effects of coconut oil intake in comparison to other fats, oils, or placebos on weight, lipid and glycemic profile, blood pressure, and subclinical inflammation of adults (≥ 18 years) published in all languages. We excluded non-randomized trials or studies with follow-ups shorter than seven days. Studies including patients with illnesses which affect metabolism, studies on animals or in vitro, and studies testing coconut products different from oils for intake were also excluded.

Assessment of bias and quality of evidence

Two pairs of authors independently assessed the risk of bias of each included trial using the revised Cochrane risk-of-bias tool for randomized trials (RoB 2) (31). RoB 2 plots were generated using the Risk-of-bias VISualization (robvis) tool (32). The overall certainty of evidence was assessed using the Grading of Recommendations Assessment, Development, and Evaluations (GRADE) (33).

Statistical synthesis

Data were synthesized both qualitatively and quantitatively. To uniformly summarize the exposure data extracted, we standardized the units of concentration by applying standard conversion factors (34, 35). Mean differences were calculated for continuous outcomes. For data collection, we prioritized intention-to-treat outcomes. Articles that expressed results as standard error had the results transformed into standard deviation. When a study did not express its results in a change from baseline manner, changes from baseline were calculated by subtracting final values from baseline values

in each group and change from baseline standard deviations were imputed using a correlation coefficient calculated from the most similar study reported in considerable detail, in accordance with Cochrane Collaboration recommendations (36). Where possible (that is, when parallel RCTs provided the baseline and final values of each outcome and when the crossover RCTs provided the order of different interventions and the measures of the variables of interest before and after each intervention), data were pooled using a meta-analytic approach. A random-effects model, with DerSimonian and Laird's variance estimator, was used, and mean differences with 95% confidence intervals were calculated. A p value ≤ 0.05 was considered statistically significant. We used I^2 statistics to assess the consistency of effects among studies (37). We did not assess publication bias with a statistical test or funnel plot because such assessment is not recommended for sample sizes of less than 10 studies (38). We used the statistical software R version 4.0.5 with the meta-version 4.18-1 package for meta-analysis.

We planned to perform subgroup analyses regarding the following factors: amount of coconut oil used, type of control group, sex, age, body mass index, geographical region where the study was conducted, studies in overweight/obesity subjects or in those with dyslipidemia, time of follow-up, and study sample size. When subgroup analysis of any forementioned factors was not possible due to the low number of studies – thus precluding our ability to quantitatively investigate the sources of heterogeneity –, this analysis was not performed and, therefore, is not mentioned in the results section of this text.

Results

After screening 1,160 potentially relevant studies, 17 fulfilled the selection criteria, of which seven studies were included in the meta-analysis. Inter-rater agreement assessed by the Kappa coefficient was 0.36 (% agreement: 91.1%) and –0.09 (% agreement: 84%) for the record screening and fulltext assessment stages, respectively. Details of the study selection are presented in Figure 1 and Table S1.

The studies comprise 721 patients (age 18–68 years, 52% females) and follow-ups varied from one week to two years. The studies were performed in Europe (n = 2), Asia (n = 3), New Zealand (n = 1), the United States of America (n = 7), and Brazil (n = 4). In four studies, coconut oil was compared predominantly to MUFAs (olive (6) and canola (1)) (39-42), in 11 studies predominantly to PUFAs (soybean (12), chia (1), safflower (7), sunflower (1), and corn (1)) (16, 17, 26, 42-49), and in six studies predominantly to SFAs (lard (1), butter (5), and palm oil (6)) (39, 40, 44, 50, 51), followed by comparisons with soybean oil + psyllium, transgenic soybean, hydrogenated soybean, and a placebo in one study each (18, 26, 45, 50). The amount of coconut oil consumed varied and is expressed differently among studies (Table 1): 12 to 30 ml of coconut oil/day (n=5), as part of the amount of SFAs or total daily consumed fat (n=1), a variation of 6 to 54.4 g/day (n=5), or as part of the total caloric energy intake (15 to 21%) (n=6). Seven studies included healthy individuals (18, 26, 39, 45, 48, 50, 51); two included subjects with hypercholesterolemia (41, 44); four, with abdominal obesity, overweight, or obesity (16, 42, 43, 49); one, in postmenopausal women (46); and one, individuals with CVD (17). The key characteristics of all included studies are in Supplementary Tables S2-S6 and summarized in Table 1.

We contacted authors from 12 trials, of whom three shared data with us (see acknowledgments).

Trials reporting the effects of coconut oil on LDL-C to HDL-C ratio, TG, TC/HDL-C ratio, glycemic control (fasting glucose and HbA1c levels) and blood glucose regulation (insulin sensitivity and β-cell function), blood pressure, and subclinical inflammation profile are described in the Supplementary Appendix II. No differences in these parameters were found between coconut oil intake and the different control oils/fats. A summary of findings of the systematic review and meta-analysis is presented in Table 2.

Coconut oil consumption and health outcomes

Anthropometric profile

Body weight

Nine studies analyzed the effects of coconut oil on body weight (16, 18, 26, 39, 42, 43, 46, 51). These studies included 533 participants (56.5% females, 18 to 68 years).

Six studies [(16, 17, 39, 42, 43, 51) were included in the meta-analysis. Overall, weight loss was similar for those receiving coconut oil in comparison to those receiving other oils or fat (Figure 2a).

The changes in body weight with coconut oil were also not significantly different in comparison to PUFA-rich oils, SFA-rich oils/fats, and MUFA-rich oils (Figure 2a).

We also performed subgroup analyses considering the type of control group, gender, geographical region, studies in subjects with overweight/obesity, time of follow-up (< 1 year vs. ≥ 1 year), and the presence of co-interventions. These analyses did not explain the heterogeneity between groups and showed no changes in the direction of the results (Supplementary Figures S1–S8).

Additionally, two crossover studies found no differences between the consumption of coconut oil and other oils and fats on body weight (26, 46).

Waist circumference

Seven studies analyzed the effects of coconut oil on waist circumference (16, 39, 40, 42, 43, 46, 49). These studies included 347 participants (80.1% females, 23 to 66 years).

Five studies were included in the meta-analysis (16, 39, 42, 43, 49). Overall, the effect of coconut oil on waist circumference was not different in comparison to other interventions (Figure 2b).

In order to understand the heterogeneity found, we performed a subgroup analysis. A small yet significant reduction in waist circumference is perceived while comparing the consumption of coconut oil with PUFA-rich oils,) but not with MUFA-rich oils (Supplementary Figure S9).

We also performed subgroup analyses considering the type of control group, gender, geographical region, studies in subjects with overweight/obesity, and the presence of co-interventions. These analyses did not explain the heterogeneity between groups and showed no changes in the direction of the results (Supplementary Figures S9–S15).

In one crossover study, the consumption of coconut oil decreased waist circumference in comparison to safflower oil (40).

Body composition

Six studies analyzed the effect of coconut oil on body fat distribution (16, 17, 39, 40, 46, 49). These studies included 460 participants (35.4% females, 29 to 68 years).

Five studies (16, 17, 39, 42, 49) were included in the meta-analysis. Overall, the effect of coconut oil intake on total body fat did not differ in comparison to other oils or fats (Supplementary Figure S16). Additionally, in comparison to PUFA- and MUFA-rich oils, the effect on total body fat was not different (Supplementary Figure S17).

Only one crossover study analyzed the effect of coconut oil on fat mass, including only postmenopausal women (n = 12, 100% females, 57.8 ± 3.7 years) (46). The comparator was safflower, and there was no difference in body fat distribution between groups.

Two studies analyzed the effect of coconut oil on lean mass (n = 41, 29% females, 35–61 years) (46, 49). The comparators were safflower and soybean oils, and, once again, coconut oil did not cause changes in lean mass in comparison with other oils (Supplementary Table S2).

Lipid profile

LDL-C

Seventeen studies analyzed the effects of coconut oil on LDL-C (16-18, 26, 39-51). These studies included 515 participants (50% females, 18 to 68 years).

Seven studies (16, 17, 39, 42, 43, 49, 51) were included in the meta-analysis. Overall, the intake of coconut oil did not change LDL-C in comparison to other oils/fats (Figure 3).

Coconut oil intake did not increase LDL-C as compared to PUFA-rich oils, SFA-rich oils/fats, and MUFA-rich oils (Figure 3a).

We performed subgroup analyses considering the type of control group, gender, geographical region, studies in subjects with overweight/obesity, time of follow-up (< 1 year vs. ≥ 1 year), and the presence of co-interventions. These analyses did not explain

the heterogeneity between groups and showed no changes in the direction of the results (Supplementary Figures S18–S25).

When analyzing the results of crossover studies, we observed that the intake of coconut oil increases LDL-C levels in comparison to butter, lard, and other oils (18, 26, 40, 41, 44-48, 50).

HDL-C

Seventeen studies analyzed the effects of coconut oil on HDL-C (16-18, 26, 39-51). These studies included 515 participants (50% females, 18 to 68 years).

Seven studies (16, 17, 39, 42, 43, 49, 51) were included in the meta-analysis. Overall, the intake of coconut oil increased HDL-C by 3.28 mg/dL (Figure 3).

We also performed subgroup analyses considering the type of control group, gender, geographical region, time of follow-up (< year vs. ≥ 1 year), and studies in overweight/obesity subjects (Figures S26-S32). These analyses did not explain the heterogeneity between groups. However, when analyzed in different comparisons, the relative type of oil in the control group and studies only conducted in women, the significant increase in levels of HDL-C no longer existed. An additional subgroup analysis demonstrated that a significant increase in levels of HDL-C with coconut oil intake in comparison to other oils/fats was only identified in studies that included lifestyle interventions (Figure S33).

While analyzing the crossover studies (17, 25, 39, 40, 43-47, 49), we observed that the intake of coconut oil increases HDL-C in comparison to butter, lard, and other oils (data not shown).

Triglycerides

Seventeen studies analyzed the effects of coconut oil on TG levels (16-18, 26, 39-51). These studies included 515 participants (50% females, 18 to 68 years).

Seven studies (16, 17, 39, 42, 43, 49, 51) were included in the meta-analysis. Overall, the intake of coconut oil did not change TG levels (Figure 3)

The effect of coconut oil on TG was also not significant in comparison to MUFA-rich oils, PUFA-rich oils, and SFA-rich oils/fats (Figure 3c).

We performed subgroup analyses considering the type of control group, gender, geographical region, studies in overweight/obesity subjects, time of follow-up (< 1 year vs. ≥ 1 year), and the presence of co-interventions. These analyses did not explain the heterogeneity between groups and showed no changes in the direction of the results (Supplementary Figures S34–S41).

Crossover studies (17, 25, 39, 40, 43-47, 49) showed that the intake of coconut oil increases TG levels in comparison to butter, lard, and other oils.

Risk of bias and certainty of evidence

Detailed results of the assessment of risk of bias are summarized in Supplementary Figures S42-S46. RCTs were overall rated either as having a high risk of bias or presenting some concerns in all analyzed outcomes. Risk of bias arose mainly from poor reporting of the randomization process and from deviations from intended interventions, in addition to carryover effects in crossover trials.

The certainty of evidence was rated as very low due to risk of bias and inconsistency in all analyzed outcomes, as follows (Supplementary Table S7).

Discussion

This systematic review and meta-analysis of RCTs shows that, compared with the dietary consumption of other types of oils and fats, the intake of coconut oil is not superior in reducing body weight or abdominal circumference nor in changing body composition, LDL-C levels, TG, and TC/HDL-C ratio. Subgroup analyses comparing coconut oil with different types of oils based on their fatty acid composition have also confirmed our findings. However, increased levels of HDL-C were observed with the intake of coconut oil in comparison with that of other oils and fats.

Regarding the outcomes that were not included in metanalyses, only two (17, 43) of the seven studies included in the systematic review that assessed glycemic control had the appropriate minimum follow-up time to analyze changes in HbA1c measures, given that the optimal timeframe to analyze alterations of HbA1c after dietary interventions is 12 weeks. Individual data from these studies do not suggest an impact of coconut oil intake on fasting glycemia, HbA1c, and estimates of β-cell function and insulin sensitivity, in line with findings from other previously published meta-analyses (29, 52).

Unlike other meta-analyses (28, 29, 52), we included studies that evaluated the effect of coconut oil on arterial blood pressure and we observed higher levels of systolic and diastolic blood pressure when coconut oil was compared with a placebo. When comparing coconut oil with olive oil and butter, only diastolic blood pressure levels increased (18, 39). Despite scarce data addressing the effect of coconut oil on blood pressure, a cross-sectional study conducted in Southern India using a seven-day food survey found that the intake of coconut oil is associated with a higher risk of hypertension (53). Although the mechanisms related to this finding remain unclear, foods rich in SFAs, such as coconut oil, can induce the development of central

adiposity and insulin resistance, both phenomena related to the development of hypertension, which might explain these findings (11-14, 54).

Regarding markers of subclinical inflammation, as in other published meta-analyses (29, 52), we did not find a reduction in US-CRP with the intake of coconut oil in comparison to soybean oil, olive oil, and butter. Studies that evaluated the antioxidant potential effect of coconut oil are mostly performed in vitro, and their data should not be extrapolated to clinical practice (55). Among the SFAs, lauric acid, which is roughly 50% of coconut oil composition, has the greatest inflammatory potential, resulting in an unfavorable rationale for conducting experimental studies evaluating the effect of the dietary consumption of coconut oil for this aim (56).

In line with previously published meta-analyses (28, 29, 52), we observed an increase in HDL-C levels with coconut oil in comparison with other oils and fats, which was also confirmed while comparing coconut oil intake with oils rich in MUFAs and PUFAs. These findings may be a result of its composition being predominantly made up of SFAs, resulting in a superior increase in HDL-C levels compared to oils/fats rich in MUFAs and PUFAs (29, 57). However, neither Mendelian randomization analyses looking at genetic variants related to higher HDL levels (58), nor a meta-analysis of 108 RCTs evaluating the effects of different interventions that increase HDL-C levels (59) demonstrated that this increment protects against cardiovascular disease. In fact, dietary fat both increases transport rate and decreases the fractional catabolic rate of HDL cholesterol esther and apo A-I, intensifying the reverse cholesterol transport, only as an adaptation to the high load of a high fat diet (60, 61). However, the consumption of SFA-rich oils, such as coconut oil, may not increase the apolipoprotein E-rich sub-fractions, which are mediators of cholesterol's reverse transport, a main mechanism by which HDL-C exerts its cardio-protective effects (62). Thus, it does not seem

reasonable to advise the intake of coconut oil based on a possible protection against CVD derived from its effect on HDL-C.

An increase in plasmatic levels of HDL-C was observed with coconut oil intake compared with other oils while analyzing different studies (Figure 3b), which could be explained by the fact that, in most of those studies, participants were exposed to co-interventions, including diet (16, 42, 43, 49, 51) and physical activity (16, 43) – which may have a significant impact on HDL-C levels (58, 59). In fact, in one of these studies, participants significantly lost more weight, and, in two of them, there was a greater reduction in waist circumference with coconut oil compared to soybean oil. These results may have been driven by the real impact of coconut oil on HDL-C levels and may explain the heterogeneity that was found (16, 43).

In this review, changes in body weight were similar between coconut oil and other oils. In only one study, the group receiving coconut oil lost more body weight (16). This result might be explained by the introduction of systematic error due to an imbalance of co-interventions, which might have been introduced as a result of lack of blinding of the staff who applied the lifestyle interventions. Similarly, among the five studies which analyzed the impact of coconut oil in comparison to other oils/fats on central obesity, the two studies which demonstrated that the coconut oil group had a more significant reduction in waist circumference also applied lifestyle co-interventions in a similar manner, possibly resulting in the same forementioned systematic error (16, 43). Subgroup analyses for studies regarding co-interventions have shown no differences in changes of body weight and waist circumference between coconut oil and diet interventions with other oils (Supplementary Figure S33 and S15).

Previous meta-analyses (29, 52) found higher LDL-C levels with the consumption of coconut oil in comparison with the intake of other oils and fats. These two reviews

included crossover trials, and, in one of them (29), oils used in different arms causing very distinct responses in LDL-C levels were grouped as a single intervention against coconut oil (16). We believe that this may explain the differences in findings between our meta-analysis and previously published ones. In line with our findings, Teng et al. (2020), in their analysis comparing coconut oil to other oils, did not find differences in levels of LDL-C, either (28). Similar to what was found previously (29, 52), we also did not identify differences in changes of TG levels, TC/HDL-C ratio, LDL-C/HDL-C ratio, and body composition between the consumption of coconut oil and other oils/fats.

LDL-C concentration is one of the main targets for cardiovascular protection. However, some subtypes of LDL-C, especially the slow dense LDL-Cs, have been associated with a higher risk of atherogenesis (63). Lipoprotein (a) (Lp[a]), a genetic variant of LDL, has also gained attention because of its considerable dyslipidemic potential (64). There is still no clear evidence that reducing Lp(a) levels results in protection for cardiovascular outcomes (65), nor do we know how nonpharmacological treatments affect Lp(a) (66). It seems that a healthy lifestyle can promote favorable changes in subclasses of lipoproteins (67), and that the characteristics of fatty acids could influence these changes (68).

None of the studies included in this systematic review assessed the subclasses of lipoproteins or Lp(a). However, a crossover trial including 31 women evaluated the effect of three different margarines, one of them containing 80% coconut oil, on plasma postprandial levels of some hemostatic variables and on fasting Lp(a). Data from only 11 subjects were evaluated, and there was a statistically significant reduction in Lp(a) in the margarine with coconut oil per se, and the total dietary composition (especially carbohydrates and total fat) was different between groups, which can influence the

results (66). New RCTs with higher methodological rigor are needed to confirm the potential of coconut oil in reducing Lp(a).

It is important to highlight that published meta-analyses about the topic included crossover studies with methodological limitations. In this meta-analysis, we only included crossover RCTs when it was possible to determine the order of the interventions and where the baselines and final averages of each arm were available. We then obtained the initial and final values of each outcome in each arm of the study before the participant was allocated to the other arm. This reduces the chance of the residual effects (carry-over) of the former intervention on the next one (36). We contacted the authors of crossover studies and received these data from the authors of one study, which we included in our analysis (51). In addition to that, we included two new RCTs (42, 49) that had not been included in the most recent meta-analysis (52).

This systematic review has some limitations. Generally, studies presented a small sample size with a short follow-up, which limits the analysis of the effects of a dietary intervention on cardiometabolic parameters. Therefore, the results must be interpreted with caution. Moreover, there was a limited number of studies analyzing the effect of the consumption of coconut oil on parameters other than lipid profile and body weight, such as body composition and glycemic and inflammatory profiles. The included studies also differ considerably from each other regarding population size and gender composition, time of follow-up, daily quantity of coconut oil consumed, type of coconut oil (virgin, extra virgin), product/vehicle for consumption (e.g.: as a capsule, as a supplement, heated as oil to cook with, or in preparations such as for muffins or crackers). Although this makes it difficult to compare different interventions, we were able to perform subgroup analyses comparing coconut oil with oils/fats with different

fatty acid content in their compositions: SFA-, MUFA-, and PUFA-rich oils/fats. We also performed subgroup analyses according to the presence of other dietary interventions and/or physical activity, which may influence the effects attributed to coconut oil on the cardiometabolic parameters which were analyzed.

Up until now, the scientific community has lacked studies with a long-term follow-up and with a significant number of participants that evaluate the effect of coconut oil consumption on cardiovascular outcomes.

Conducting new RCTs examining cardiovascular safety comparing coconut oil with PUFA- and MUFA-rich oils evaluating traditional markers does not seem to be justifiable even though coconut oil is part of the diet in South Asian countries (20-22). Moreover, in Western countries, stimulating the consumption of SFA-rich oils to the detriment of PUFA- and MUFA-rich oils may lead to an excessive intake of SFAs in populations that already have a diet rich in them (69).

Conclusions

The dietary consumption of coconut oil instead of the consumption of PUFA- and MUFA-rich oils with well-established cardio-protective effects should not be encouraged in societies that are not used to consuming it. Moreover, educational strategies should be implemented to make populations, especially those used to consuming coconut oil, aware of the potential risks related with this intake. These populations should also be informed and encouraged to replace it with cardio-metabolically healthy options linked with a reduction in rates of CVD.

List of abbreviations

CVD (cardiovascular disease), GRADE (Grading of Recommendations, Assessment, Development and Evaluations), HDL-C (high-density lipoprotein cholesterol), HOMA (the homeostasis model assessment), HbA1c (glycated hemoglobin), IL (interleukins), IFN-γ (interferon-gamma), LDL-C (low-density lipoprotein cholesterol), Lp(a) (lipoprotein [a]), MUFA (monounsaturated fatty acid), PRISMA (Preferred Report Items for Systematic Reviews and Meta-Analysis), PUFA (polyunsaturated fatty acid), RCTs (randomized clinical trials), RoB (the risk of bias), Robvis (risk-of-bias VISualization), SFA (saturated fatty acid), TC (total cholesterol), total homocysteine (tcHcy), TG (triglycerides), ultra-sensitive c-reactive protein (US-CRP).

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests

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Authors' contributions

Concept and design: ACD and FG. Acquisition, analysis, or interpretation of data: ACD, ENM, BFS, CRA, CA, VC and FG. Drafting of the manuscript: ACD. Critical revision of the manuscript for important intellectual content: all authors. Statistical analysis: ACD. Supervision: VC and FG. All authors read and approved the final manuscript.

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Table 1. Characteristics of the included studies

Source	RCT design	Population	Intervention (daily amount of coconut oil)	Control	N In	N C	Female, N (%)	Baseline lipid profile, mean (SD)				Follow up (wk)
								TC	HDL-C	LDL-C	TG	
Assunção (2009)	Parallel group	Women with abdominal obesity	30 ml	PUFA (soybean)	20	20	40 (100)	191 (32.7)	48.5 (8.7)	110.6 (28.7)	160 (81.9)	12
Cândido (2021)	Parallel group	Women with IMC between 26 and 35kg/m², % body fat >30%	25 ml	MUFA (olive oil) PUFA (soybean)	24	61	85 (100)	168.6 (9.7)	47.2 (2.7)	98.7 (9.7)	96.5 (8.9)	9
Chinwong (2017)	Crossover	Healthy individuals	15 ml	Placebo (carboxymethycellulose solution)	34	34	16 (47)	190.8 (32.3)	60.6 (9.0)	116.5 (30.1)	68.5 (23.1)	8
Cox (1995)	Crossover	Healthy individuals	39 g	SFA (butter) PUFA (safflower)	28	28	15 (53.6)	245.5 (27.5)	58 (15.5)	160.5 (29.4)	161.2 (79.7)	6
Ganji (1996)	Crossover	Healthy individuals	20% of daily calories	PUFA (soybean and soybean + psyllium fiber)	10	10	5 (50)	187.9 (30.2)	56.5 (12)	107.5 (31.3)	132.9 (40.7)	4
Harris (2017)	Crossover	Postmenopausal women	30 ml	PUFA (safflower)	14	14	14 (100)	223.1 (35.1)	64.1 (17.4)	128.7 (26.1)	105.2 (66.2)	4
Heber (1992)	Crossover	Healthy men	17.5% of daily calories	SFA (palm) Hydrogenated soybean	13	13	0 (0)	176 (4)	37 (9)	120 (7)	95 (10)	3
Khaw (2018)	Parallel group	Healthy individuals	50 g	SFA (butter) MUFA (olive)	30	66	63 (67)	229.31 (37.5)	73.47 (19.33)	138.05 (36.34)	NR	4

Lu (1997)	Crossover	Healthy women	20% of daily calories	PUFA (soybean) A16 oil	15	15	15 (100)	162.4 (17.01)	52.97 (10.82)	90.1 (15.08)	93 (38.97)	3
McKenney (1995)	Crossover	Individuals with hypercholesterolemia	Sufficient to increase in 10% the amount of daily calories from SFA	PUFA (canola)	11 (all)		5 (45.5)	222.3 (25.3)	49.8 (18.3)	149 (20.3)	117.1 (49.2)	6
Maki (2018)	Crossover	Healthy individuals	54.4 g (muffins or rolls)	PUFA (corn)	13	12	13 (52)	188 (178, 215)*	46 (38.5, 55.5)*	123 (105, 142)*	92.5 (76.5, 136)*	4
Oliveira-de-Lira (2018)	Parallel group	Obese women	6 g (capsules)	PUFA (safflower, chia, and soybean)	18	57	75 (100)	215.8 (24.2)	48.3 (8.1)	149.6 (23.7)	132.7 (41.7)	8
Reiser (1985)	Crossover	Healthy women	21% of daily calories	SFA (lard) PUFA (safflower)	19 (all)		0 (0)	NA	NA	NA	NA	5
Schwab (1994)	Crossover	Healthy women	16-26g of coconut oil/day	SFA (palm)	7	8	15 (100)	187.9 (25.5)	59.9 (10.4)	110.6 (17.8)	82.4 (34.5)	4
Vijayakumar (2015)	Parallel group	Individuals with CVD	15% of daily calories	PUFA (sunflower)	99	99	13 (6.5)	148.3 (28.3)	40.8 (9.5)	88.2 (22.2)	113.1 (51.5)	2 years
Vogel (2020)	Parallel group	Overweight men	12 ml	PUFA (soybean)	15	14	0 (0)	184.8 (44.1)	39.5 (10.1)	117 (36.1)	140.9 (67.1)	4
Voon (2011)	Crossover	Normal and overweight healthy adults	20% of daily calories	SFA (palm) MUFA (olive)	15	30	36 (80)	182.1 (25.5)	47.6 (10.8)	118.3 (22.4)	85 (39)	5

* Median (IQR). Baseline lipid values are expressed in mg/dL. Abbreviations: RCT, randomized clinical trial; N In, number of participants in the intervention arm; N C, number of participants in the control arm; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides; wk, week; PUFA, polyunsaturated fatty acid; SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; A16, transgenic soybean oil; NA, data not avaliable; CVD, cardiovascular disease.

Table 2. Summary of findings of the systematic review and meta-analysis

Outcome group		Overall result, MD (95% CI)	Risk of Bias (RoB 2)	Certainty of evidence (GRADE)
Anthropometric profile	Body weight	NS	High	Very low
	Waist circumference	NS		Very low
	Total body fat	NS		Very low
Lipid profile	LDL-C	NS	High	Very low
	HDL-C	+3.28 (0.66; 5.9)		Very low
	Triglycerides	NS		Very low
	TC/HDL-C	NS		Very low
Glycemic profile	Fasting blood glucose	NS	High	Very low
Inflammatory profile	US-CRP	NS	High	Very low

Abbreviations: MD, mean difference; CI, confidence interval; GRADE, Grading of Recommendations Assessment, Development and Evaluations; NS, non-statistically significant; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; TC, total cholesterol; US-CRP, ultra-sensitive c-reactive protein.

Figure 1. Flow chart mapping out the studies examined and included into the meta-analysis

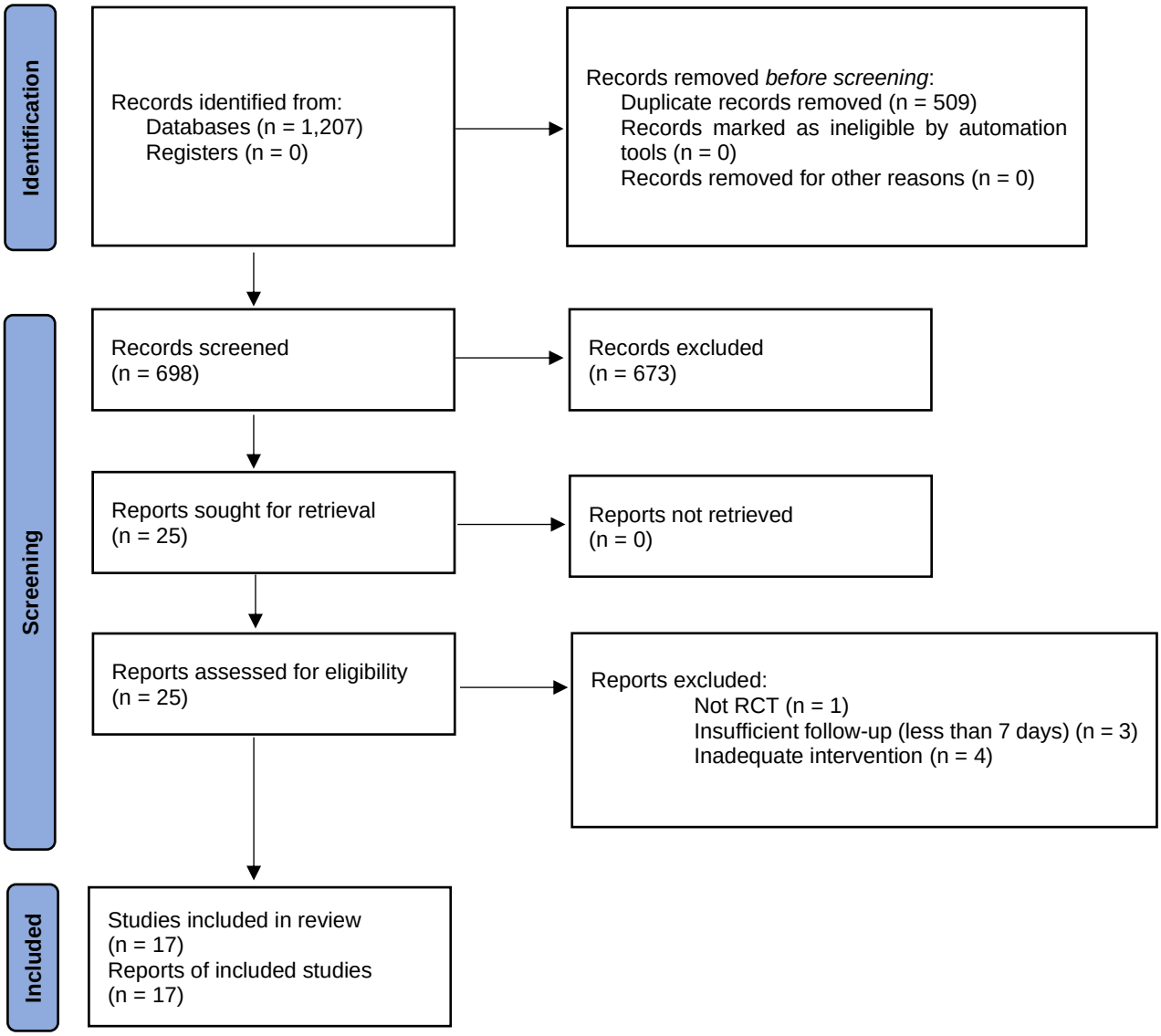
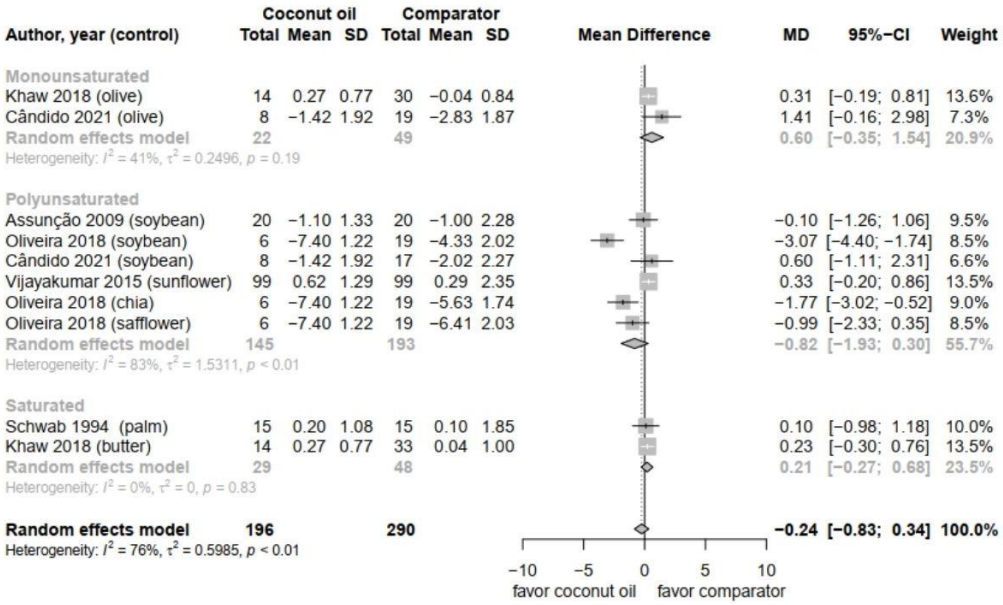


Figure 2. Forest plots of randomized controlled clinical trials investigating the effects of coconut oil intake on (a) body weight (kg); (b) waist circumference (cm). Individual trial-specific estimates and their 95% CIs are indicated by the black dots and the horizontal line, respectively. The center of the diamonds indicates the pooled estimates and the width of the diamonds indicate the corresponding 95% CI

(a)



(b)

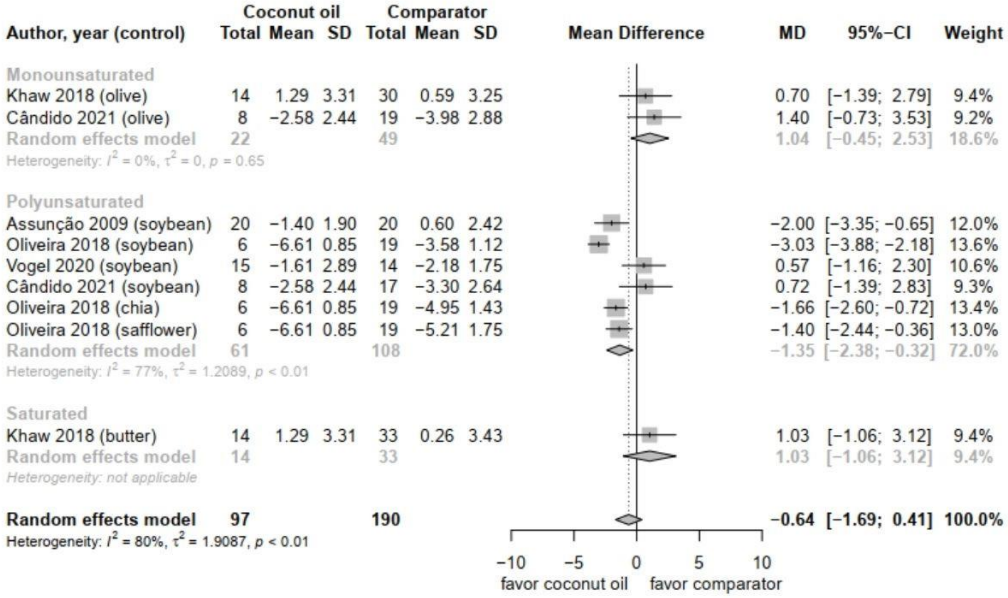
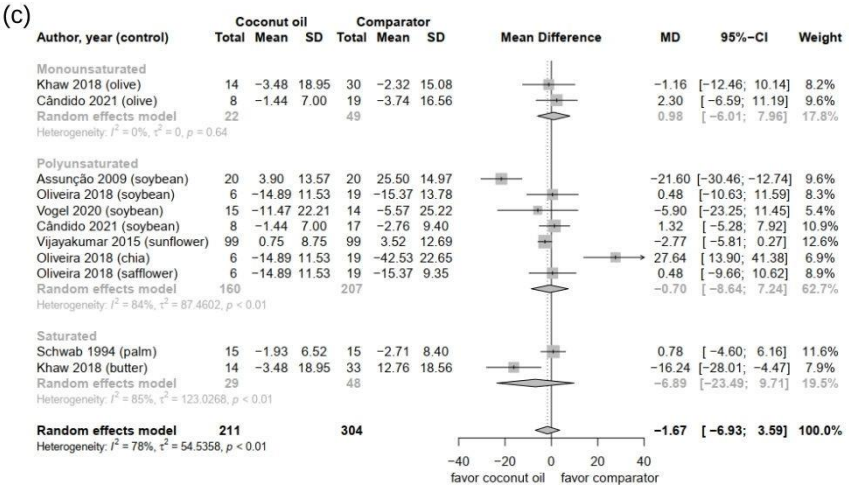
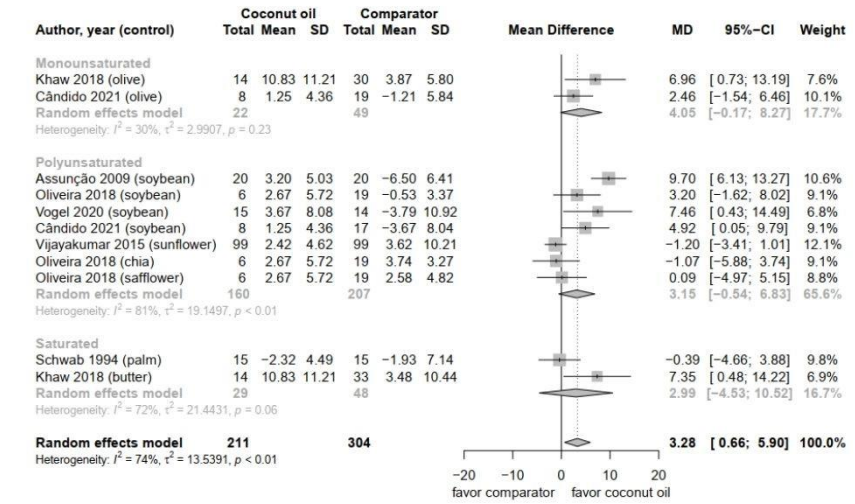


Figure 3. Forest plots of randomized controlled clinical trials investigating the effects of coconut oil intake on (c) LDL-C (mg/dL); (d) HDL-C (mg/dL); (e) TG. Individual trial-specific estimates and their 95% CIs are indicated by the black dots and the horizontal line, respectively. The center of the diamonds indicates the pooled estimates and the width of the diamonds indicate the corresponding 95% CI.

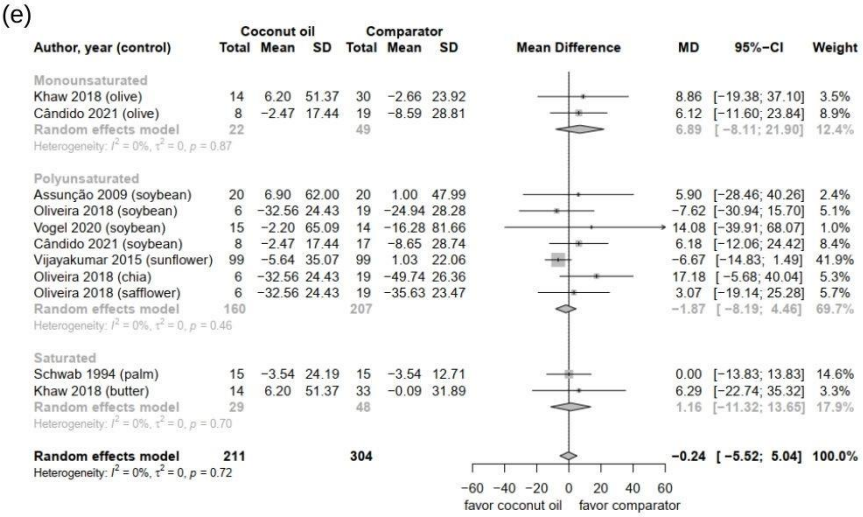
(c)



(d)



(e)



Material suplementar do Capítulo II – "The Effects of Coconut Oil on the Cardiometabolic Profile: a Systematic Review and Meta-Analysis of Randomized Clinical Trials" encontra-se no Anexo I, entre as páginas 108-176.

CAPÍTULO III - Misinformation in nutrition through the case of coconut oil: an online before-and-after study

Original Article

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Abstract:

Background and aims: Despite recent scientific evidence indicating absence of cardiometabolic benefit resulting from coconut oil intake, its consumption has increased in recent years, which can be attributed to a promotion of its use on social networks. We evaluated the patterns, reasons and beliefs related to coconut oil consumption and its perceived benefits in an online survey of a population in southern Brazil.

Methods and results: We conducted a before-and-after study using an 11-item online questionnaire that evaluated coconut oil consumption. In the same survey, participants who consumed coconut oil received an intervention to increase literacy about the health effects of coconut oil intake. We obtained 3160 valid responses. Among participants who consumed coconut oil (59.1%), 82.5% considered it healthy and 65.4% used it at least once a month. 81.2% coconut oil consumers did not observe any health improvements. After being exposed to the conclusions of a meta-analysis showing that coconut oil does not show superior health benefits when compared to other oils and fats, 73.5% of those who considered coconut oil healthy did not change their opinion. Among individuals who did not consume coconut oil, 47.6% considered it expensive and 11.6% deemed it unhealthy. **Conclusions:** Coconut oil consumption is motivated by the responders' own beliefs in its supposed health benefits, despite what scientific research demonstrates. This highlights the difficulty in deconstructing inappropriate concepts of healthy diets that are disseminated in society.

Keywords: coconut oil; social media; survey; online; health information; internet.

Introduction

1.1. Health misinformation

The real impact of information overload on the population's food preferences, lifestyle and health choices is still unknown [1]. Health misinformation, defined as false information created with no intent to cause harm [2], is a worldwide problem; its prevalence has grown with the production of health content in mass media and social networks, increasing nonevidence-based recommendations [3, 4].

Over the past 10 years, coconut oil has been promoted as a good choice for meals, being marketed as a healthy oil for cardiovascular health and for weight gain prevention. The Philippines, Indonesia and India are the main coconut oil producers in the world, Brazil being the 5th largest producer [5].

1.2. Dietary effects of coconut oil and misinformation

Coconut oil is composed of saturated fatty acids in 92% of its lipid composition [6]. Its consumption has increased substantially in the last decade as a result of the publication of small and short-term studies suggesting that its intake promotes weight loss and improves the lipid profile and glycemic control [7-9].

Before the transition to a more westernized diet, countries, such as Sri Lanka, Minangkabau, Philippines and the Pukapuka and Tokelau Islands in Polynesia, presented low rates of cardiovascular diseases despite their significant consumption of coconut oil [10-13]. Based on these findings, supposed cardiovascular benefits were attributed to coconut oil. However, it is noteworthy that these populations had a dietary pattern in which their major source of saturated fatty acids was coconut fat rather than animal fats, their major protein source was from fish and their main source of carbohydrates was from native fruits of the region, defining a dietary pattern also rich

in fibers and poor in animal fats, processed foods and sucrose [13]. Therefore, these findings are hardly applicable to populations with usual westernized dietary patterns.

When coconut oil is evaluated in light of the best available evidence, its consumption cannot be recommended. The main guidelines in Brazil [14, 15] and in the United States [16, 17], as well as recent results of different meta-analyses [18, 19] do not suggest coconut oil as a preferred fat for human consumption. Intake of coconut oil increases low-density lipoprotein (LDL) cholesterol, a very well-defined risk factor for cardiovascular disease, and results in no improvement in glycemic control and body weight compared to other oils [18, 19].

Social media is essential for the flow of information, knowledge building and the dissemination of opinions. However, with its insertion into daily life, society became used to accessing health information on a large scale, but not always from reliable sources. Studies showed that information published on health-related websites may present inaccuracies and low quality of evidence of information [20-24], as it is possible to publish about health information with or without technical knowledge in the area [25]. Thus, an impressive amount of information is disclosed, but little knowledge is being built [26].

A major challenge for the implementation of evidence-based nutrition is the discrepancy between the public opinion about health recommendations found online and evidence-based guidelines. In addition, there is a need for the development and testing of simple and affordable interventions to increase literacy on nutrition, which could be used to guide the way we disseminate science and evidence-based nutritional recommendations [27]. People find it difficult to interpret whether health information is from a reliable source and based on solid scientific evidence, causing exposure to actions that may not only be hazardous, but also leading to reductions in adherence to

screening programs and proposed treatments [28]. People with less education and/or less health literacy are the groups at greatest risk in this context [29]. A systematic literature review found that individuals with lower educational levels have less ability and confidence to assess the quality of online health information than their peers with a higher educational level [25].

1.3. Aims & hypothesis

Therefore, we conducted this before-and-after study to evaluate the patterns of and reasons for coconut oil consumption, as well as beliefs related to its benefits in an online survey of a population in Southern Brazil, given the scarcity of data on dietary consumption of coconut oil in Brazil and worldwide. Additionally, we assessed the possibility of increasing literacy on the health effects of coconut oil intake through an intervention in the same population.

Methods

This study follows the Checklist for Reporting Results of Internet E-Surveys (CHERRIES) statement (supplementary methods) [30].

Study design and population

We conducted a before-and-after study in Brazil between May and June 2020, with individuals aged 18 years or older, through an 11-item online questionnaire applied in Portuguese that contained questions regarding coconut oil consumption using Google Forms. A questionnaire summary is presented in Table 1 and detailed questionnaire development description is presented in the supplementary methods along with a translated version in English of the original questionnaire (not checked for accuracy or consistency). We analyzed two different samples: (1) university students from graduate

programs (MBA, Master's and PhD) at Universidade Federal do Rio Grande do Sul, in Porto Alegre, Brazil (University sample); and (2) individuals who accessed the Facebook page of Hospital de Clínicas de Porto Alegre, the university's main teaching hospital (Facebook sample).

Intervention

In the questionnaire (supplementary methods), participants who consumed coconut oil were exposed to the statement "A current study has reviewed scientific articles and concluded that nutritional consumption of coconut oil does not improve bad cholesterol, reduce weight or blood sugar (glucose) levels" summarizing the results of a recent meta-analysis demonstrating that coconut oil does not reduce LDL cholesterol, and does not reduce weight or blood glucose levels [18]. Subsequently, responders were asked if they still considered coconut oil intake to be healthy. To proceed to the next question, participants must necessarily answer the intervention item.

Questionnaire distribution and sampling

The final questionnaire was distributed to all graduate students registered at the University through an email list. It was also shared on the official Facebook page of Hospital de Clínicas de Porto Alegre, the University's main teaching hospital. Those interested in participating had access to the study webpage by clicking on the study invitation link. As a result, convenience sampling was used for both University sample and Facebook sample. Conflicting demographics and schooling information provided by the volunteers was checked with the responders by email.

Table 1. Questionnaire summary

Question
Have you ever used coconut oil?
Why do you use coconut oil? [a,b]
What amount of coconut oil do you intake? [a]
Which benefits did you observe by using coconut oil? [a,b]
A current study has reviewed scientific articles and concluded that nutritional consumption of coconut oil does not improve bad cholesterol, reduce weight or blood sugar (glucose) levels. Do you still consider coconut oil good for your health? [a]
Why don't you use coconut oil? [b,c]
Sex
Where do you live?
How old are you?
What is your educational level?
Do you want to receive the results of this survey?

[a.] Only answered by participants who answered "yes" or "I don't want to answer" to the question "Have you ever used coconut oil?". [b.] Question allowed multiple items to be marked. [c.] Only answered by participants who answered "no" to the question "Have you ever used coconut oil?".

Ethics Statement

The study was approved by the Research Ethics Committee of Hospital de Clínicas de Porto Alegre and Universidade Federal do Rio Grande do Sul. (GPPG-HCPA protocol 20180393 nos and CAAE nos 92144718.6.0000.5327) Participants were informed, at the study home page, that by submitting the questionnaire, they would be accepting to voluntarily participate in the survey. Additionally, the home page informed the time to complete the survey, the study objective, and the name and contact information of the principal investigator, following recommendations of the Ethics Committee. We asked each participant to provide a valid email address, to which only the main investigators of the study had access, and offered to send the results of the survey to participants who requested them.

Prevention of Multiple Entries

Entries from invalid or duplicate emails were excluded from the analysis, as well as those who reported uses of coconut oil other than as food or supplement. In case of duplicate entries, the first response was included in the analysis.

Statistical Analysis

Data was described as absolute numbers and percentages. Categorical data was compared by the chi-square test. To test for the effect of possible determinants on the patterns of consumption of coconut oil on the study samples, multiple logistic regression was performed, adjusting for gender, age and education level in different multivariate models. A 2-tailed P <0.05 was considered statistically significant. SPSS software version 19.0 (IBM, Armonk, NY, USA) was used for statistical analysis.

RESULTS

Out of 11753 registered graduate students, 3588 answered the questionnaire (30.5%). We excluded 684 entries due to invalid answers, totaling 2904 entries. Among the 279 participants coming from the hospital's Facebook page who answered the questionnaire, 23 entries were excluded due to invalid answers, totaling 256 entries. Participant flow diagram with reasons for exclusion ins presented in Figure 1. While 58.2% of the students consume coconut oil, 69.9% of the responders from the Facebook group consume it. Most of the consumers were females in both groups and distribution of responders did not change according to age (Table 2).

Table 2. Demographic characteristics of the University and Facebook samples

	University sample (n=2904)		Facebook sample (n=256)	
	Consumes coconut oil	**Does not consume coconut oil**	**Consumes coconut oil**	**Does not consume coconut oil**
Participants, n	1689	1215	179	77
Age, n				
18–19 years	--------	--------	2	3
20–29 years	764	240	38	10
30–39 years	656	218	61	19
40–49 years	179	80	40	13
50–59 years	64	26	24	51
≥60 years	26	6	14	4
Female participants, n %[a]	1239 (73.6)	527 (51.9)	166 (92.7)	67 (94.4)
Education, n				
Less than high school	--------	--------	7	2
High school	--------	--------	31	8
Incomplete undergraduate degree	--------	--------	44	16
Undergraduate degree	415	332	46	14
Graduate degree	1274	883	51	12
Brazil, %[a]	(1659) 98.2	1206 (99.3)	175 (97.8)	77 (100)

Percentage of each column. The percentage of responders who did not answer varied according to each question for the University and Facebook groups: age (22.2%) and (9.0%), respectively, gender (7.1% and 2.3%), and education (0% and 9.8%).

Most participants who consumed coconut oil in the University (82.8%) and Facebook (81%) samples considered it healthy and more than half of them used it at least once a month (Figure 2). However, most of them did not perceive any health or aesthetic benefits (Figure 2). After being exposed to the conclusions of a recent meta-analysis showing that coconut oil was not superior to other oils and fats regarding cardiometabolic health, the participants who had deemed it healthy were questioned if they still had the same opinion. In the University group, 72.7% did not change their opinion, while in the Facebook group 82.3% followed the same direction. Among individuals not consuming coconut oil, 47.3% of the University and 50.6% of the Facebook participants considered it expensive. Only 10.4% and 29.9% of them, respectively, believed that the oil was not healthy. In the University group, 14.5% of the participants reported difficulties finding coconut oil where they lived, while 5.2% of those in the Facebook group reported the same problem. In the University and Facebook groups, 10.5% and 20.8% did not like the taste of the product, respectively, and 33% and 11.7% did not use coconut oil for other reasons.

By multiple logistic regression analysis, we analyzed which factors were related to the consumption of coconut oil in different models adjusted for sex, age and educational level (Table 3). Women were more likely to consume coconut oil than men in the University sample (OR 2.7; 95% CI = 2.2 to 3.3; P = <0.001). Additionally, while those with a higher educational level had a more significant intake of this oil (OR 1.4; 95% CI = 1.1 to 1.7; P = 0.004), those aged ≥ 40 years had a lower daily consumption of coconut oil in comparison to other age groups. No differences were observed in the Facebook sample for the factors analyzed.

Table 3. Multiple logistic regression analysis for possible determinants of coconut oil consumption (yes/no) and its frequency

	University Sample (n=2904)			**Facebook Sample (n=256)**		
	OR	95% CI	*P*	OR	95% CI	*P*
Consumes coconut oil (yes/no)						
Female	2.7	2.2 – 3.3	<0.001	0.5	0.1 -2.3	0.40
18-19 years	--------	--------	--------	a	A	a
Age 20-29 years	a	A	a	0.3	0.0 – 3.6	0.38
Age 30-39 years	0.9	0.7 – 1.1	0.21	1.3	0.3 – 5.1	0.70
Age 40-49 years	0.7	0.5 – 0.9	0.03	0.9	0.3 – 3.2	0.89
Age 50-59 years	0.7	0.4 –1.2	0.24	0.9	0.2 – 3.5	0.94
Age ≥ 60 years	1.3	0.5 – 3.4	0.52	1.4	0.3 – 6.2	0.65
Education: less than high school	--------	--------	--------	a	a	a
Education: high school	--------	--------	--------	1.2	0.2 – 7.1	0.84
Education: incomplete undergraduate degree	--------	--------	--------	0.9	0.2 – 5.1	0.93

Education: Undergraduate degree	a	A	a	1.3	0.2 – 7.3	0.77
Education: graduate degree	1.4	1.1 – 1.7	0.004	0.8	0.1 – 4.6	0.80
Frequency of coconut oil intake						
Female	1.3	0.9 – 1.7	0.14	2.9	0.8 – 10.2	0.09
Age 18-19 years	--------	--------	--------	a	a	a
Age 20-29 years	a	A	a	0.8	0.0 – 19.2	0.92
Age 30-39 years	0.9	0.6 – 1.3	0.67	4.5	0.9 – 22.7	0.07
Age 40-49 years	0.4	0.2 – 0.6	<0.001	1.5	0.4 – 5.9	0.53
Age 50-59 years	0.4	0.2 – 0.7	0.002	1.0	0.2 – 3.8	0.99
≥ 60 years	0.3	0.1 – 0.8	0.01	0.9	0.2 – 3.9	0.91
Education: less than high school	--------	--------	--------	a	a	a
Education: high school	--------	--------	--------	0.6	0.1 – 3.8	0.58
Education: incomplete undergraduate degree	--------	--------	--------	0.9	0.1 – 6.2	0.96
Education: undergraduate degree	a	A	a	1.3	0.2 – 8.2	0.78

Education: graduate degree	1.1	0.8 – 1.6	0.57	0.4	0.1 – 2.9	0.39

OR: odds ratio; CI: confidence interval. a. Reference age or educational level for comparison in different multiple regression models.

Discussion

Principal Results

When applying an online survey to two different samples, one comprising adults at predominantly graduate programs of all majors in a university in Southern Brazil, and the other containing participants who accessed the Facebook page of the University's teaching hospital, we found that most of the responders routinely consumed coconut oil at least once a month (Figure 2). This is the first study analyzing the dietary consumption of coconut oil in a specific population in Brazil. We did not find the pattern of this consumption in other parts of the world. The data we find in the literature currently refer to the general domestic consumption of coconut oil, and its use is not specified (meal preparation, amount, frequency, aesthetic use, etc). The countries/regions with the highest consumption of coconut oil are: Philippines, European Union, United States of America and India (675, 645, 497 and 470 thousand tons) [5]. Most of these responders considered that coconut oil intake was capable of improving their health status, even though the majority did not perceive real health benefits with its consumption. When exposed to recent evidence provided by a meta-analysis showing no cardiometabolic benefits of coconut oil intake [18], most responders did not change their beliefs and confirmed that they would keep consuming the product. As a hypothesis-driven development, we believe that populations more exposed to social media are prone to the consumption of coconut oil. In order to test

this hypothesis, we analyzed if there was any factor related to the consumption of this oil by multiple logistic regression analysis. We found that adults 40-49 years and males were less likely to consume coconut oil and that, among consumers, adults 40 years or older were less likely to have a daily consumption. These findings do not necessarily confirm our hypothesis, hence more studies are necessary to conclude if populations more exposed to social media are more susceptible to change their nutritional behavior. Among individuals who did not consume coconut oil in the University and Facebook samples, only 10.5% and 29.9% did not consider coconut oil healthy, respectively, while 47.3% and 50.6% considered the product too expensive for consumption. It seems reasonable to assume that this population would be potential coconut oil consumers if they were able to afford it. In May 2020, the average price of coconut oil was estimated to be US$ 832/ton, while the price of soybean oil was estimated at US$595/ton [5]. These prices probably determine the final prices to the consumer and may explain these findings.

Limitations

This study has some limitations. Firstly, the study sample is composed of individuals with computer literacy from a convenience sample, resulting in a sociodemographic profile that does not totally represent the Brazilian population. As in Brazil coconut oil is usually more expensive than other oils, this population probably the one more affected by misinformation related to coconut oil intake and, consequently, an important target population to be studied. Using samples from different sociodemographic and educational backgrounds, possibly representing different profiles within the Brazilian society, we believe we have minimized this issue .The replication of these findings in different populations would allow us to understand if our

results are consistent with others. Nevertheless, we were able to identify that a significant fraction of responders, regardless of their sociodemographic and educational profile, believed that coconut oil was beneficial for their health, suggesting that their beliefs about food were so ingrained that exposure to scientific information did not result in a change of concept [18].

Secondly, it is expected that people who identify with a subject are more likely to respond to a survey, the so-called "volunteer effect" [27]. Although we were not able to control for that in our online survey, more than one-third of the University's graduate students completed the survey. Similar findings from a second sample with a different sociodemographic background reinforced that our findings may be representative of at least part of this population. Looking from another perspective, even if we assume a hypothetical scenario of maximum "volunteer effect" in the University sample (in which all of the nonparticipant graduate students did not consume coconut oil), the number of consumers would still be an alarming 14.6% of this population, considering that the main reason for coconut oil intake is its supposed health benefit. In addition, the before-and-after design of this study presents limitations such as the absence of randomization between groups and the lack of a guarantee that participants changed their opinions as a result of the performed intervention. As such, we recommend that our results should be interpreted with caution, serving as hypothesis generators for designing new studies dedicated to understanding how misinformation is changing the way we eat. The rapid exposure to scientific information through an online questionnaire suggests that this kind of intervention has a limited effect as a strategy to change concepts regarding misinformation in nutrition. Although we did not present sufficient data within the intervention question to quantify the magnitude of the consequences of coconut oil intake, which could possibly strengthen the intervention,

providing more complex information would likely reduce adherence to the intervention and, therefore, reduce the total number of participants who would benefit from it.

Coconut Oil and Misinformation

Two short-term studies showed slight improvements in lipid and anthropometric profiles with coconut oil intake in healthy adult populations [7, 31]. Based on these studies and a possible interest of third parties to promote coconut oil consumption, the dissemination of this concept was routinely propagated in websites, blogs, and health professionals' social media pages. This resulted in the addition of coconut oil consumption as a recommendation in clinical practice, indicating it as the fat of choice to be used for cooking and adding to salads and other food preparations, although neither its superiority nor its noninferiority compared to other oils were proven regarding its safety and the reduction of cardiovascular endpoints, prevention of weight gain, and the development of diabetes [32, 33]. Although social media may have played an important role in building misconceptions about coconut oil in the public opinion, an individual's beliefs about health and food are not only formed by ideas propagated on social media. Flavor, price, and convenience of access to food also influence their choices, as well as the general opinion about a certain food in the group in which the individual is inserted [1].

A significant part of our study population did not change its opinion even when exposed to information that coconut oil neither lowers LDL cholesterol levels, nor it lowers weight or blood glucose levels, continuing to consider coconut oil as a healthy and preferential oil. In 2016, a commissioned survey performed by the New York Times found that 72% of the population considered coconut oil to be good for health and 37% of nutritionists also considered it a better oil for health [34]. A study of adults aged over

59 years, who were exposed to printed information provided by a health professional on the effects of the interaction between alcohol and medication, showed that the exposure to information made them review their concepts about the topic, exerting a positive change in their level of knowledge on the subject [35]. These findings led us to question the current scope of the results obtained through scientific research in the general population, since the information and positions of respected health societies, which should have great impact on the population's health decisions, seem to have less impact than biased or unsupported scientific information spread throughout social networks.

In 2002, Eysenbach coined the term "infodemiology," helping to identify areas where there is a gap in translation of the best scientific evidence (proposed by experts on the subject) and what most people practice or believe in [36]. To combat "infodemia," it is necessary to translate science as clearly as possible for the population in its most varied degrees of knowledge and education, using an accessible and didactic language by the authorities in order to change beliefs that may lead to negative actions for health [3, 4, 37]. Even when great effort is applied into doing this, the impact in changing misleading concepts may not be effective, since individuals tend to interpret the received information in order to confirm their beliefs and not to challenge them, which is called confirmation bias [38].

The development of strategies to potentiate the dissemination of appropriate health concepts to protect the population against misinformation is a new area of investigation. Some anti-spread interventions for false references related to health have already been tested. Algorithmic and social corrections are examples that expose the social network to real information produced by an algorithm or a social network user to protect the society against unreliable content [39]. The credibility of the source

of the information provided by the social network user can also be categorized [40]. An effective solution will certainly be a broad combination of diverse approaches developed by health, social, and computer science specialists working together in interdisciplinary research to find ways to deal with health misinformation on social media [40-42].

The commitment of health professionals to the social dissemination of information supported by the best scientific evidence available, with a critical analysis of study results, should also be regulated [4]. It seems reasonable to think that we need tools to generate appropriate health concepts through statements of official scientific societies before the dissemination of a misleading concept on social media. This would be more efficient in preventing a questionable health attitude than counteracting misinformation already disseminated by social media [41].

Another aspect that needs to be assessed is the level of general and health literacy of the populations, considering the individual's abilities to obtain, communicate, and understand health information in order to make the best decisions [43]. This is an important concept, as studies indicate that around 9 out of 10 adults do not have the necessary skills for the management and prevention of diseases [43]. Populations with lower levels of health literacy have worse health outcomes [44, 45]. There are few studies that assessed the degree of health literacy in Latin American populations. Existing studies show that socioeconomic inequality, social/geographic isolation, cultural, political and language barriers are factors that affect the level of health literacy in these countries [46]. In Brazil, Maragno et al, evaluated 302 adult users of a university health clinic in the state of Santa Catarina, using a health literacy test for Brazilians, based on the functional health literacy test in adults TOFHLA, and observed that 54.6% of the sample had adequate health literacy while 26.2% has an inadequate

one [47]. Another cross-sectional study, carried out in the city of Rio de Janeiro, evaluated 150 outpatients with type 2 diabetes mellitus, using a short version TOFHLA and found that 73.3% of the participants had adequate literacy in health and 15.3%, inadequate health literacy [48]. A cross-sectional study with 248 adults in Piracicaba, state of São Paulo, showed an association between a low level of oral health literacy with a more frequent use of the dental service due to pain or just for emergency treatment, presence of dental plaque and "poor" evaluation of the dental care service [49]. It is necessary to ensure that the best scientific evidence is translated into the simplest language, and that it is easily understood by the entire population and not just by segments of society with a higher level of education and access to the best news sources [44, 45]. Since the University sample was composed of participants with a higher-than-usual level of education, our results are of great concern since they suggest that the misconception about the effects of coconut oil on health prevails in this population.

Conclusion

In this study, we used a questionnaire to analyze the pattern of consumption of coconut oil, a nutrient with a negative impact on cardiometabolic health that is widely disseminated on social networks as a healthy food. Despite the high expectancy that this should be a healthy product expressed by most of the studied population in southern Brazil, the participants did not notice health benefits with its consumption. We were not able to change their concepts with an intervention aimed at increasing literacy in this topic. These findings suggest how difficult it is to change unhealthy concepts related to our diet after the population has acquired false or wrong concepts on the topic.

For this reason, misconceptions related to diet and nutrition need to be extensively studied as a public health problem and strategies, such as algorithmic corrections in social media using reliable sources should be implemented. These are effective [39], readily available and have already been implemented to combat misinformation related to COVID-19. Furthermore, the development of interventions that are able to improve literacy regarding lifestyle healthy habits in multiple levels of education [50-52] and a fast call from action from healthcare professional societies [52] may be effective measures to reduce the impact of misinformation as promoter of the consumption of unhealthy foods linked to cardiometabolic diseases.

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Figure Legends

Figure 1. Participant flow diagram.

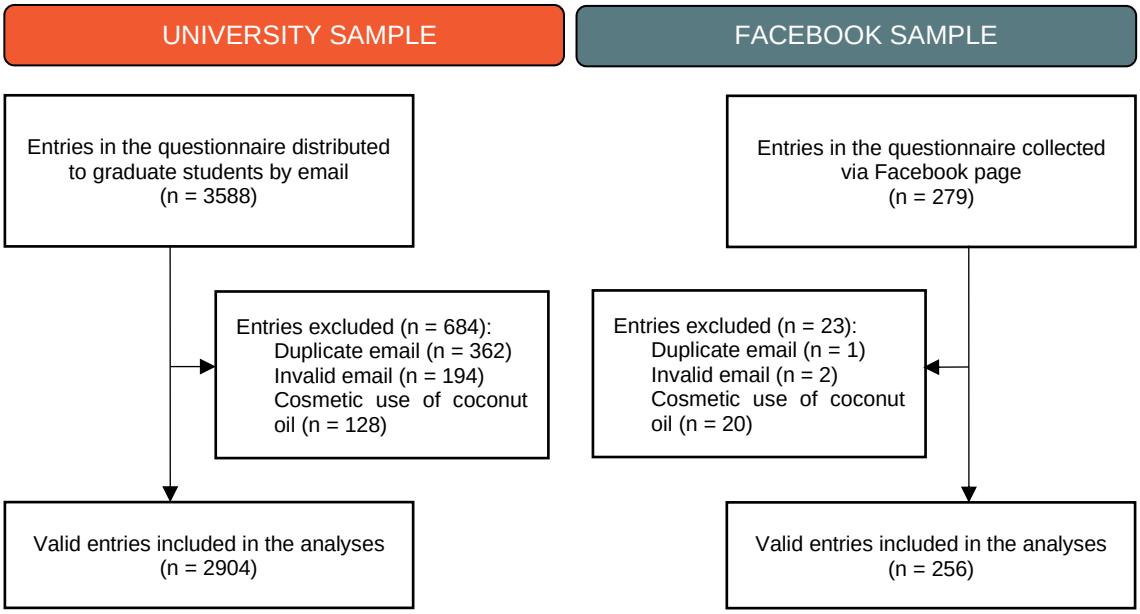
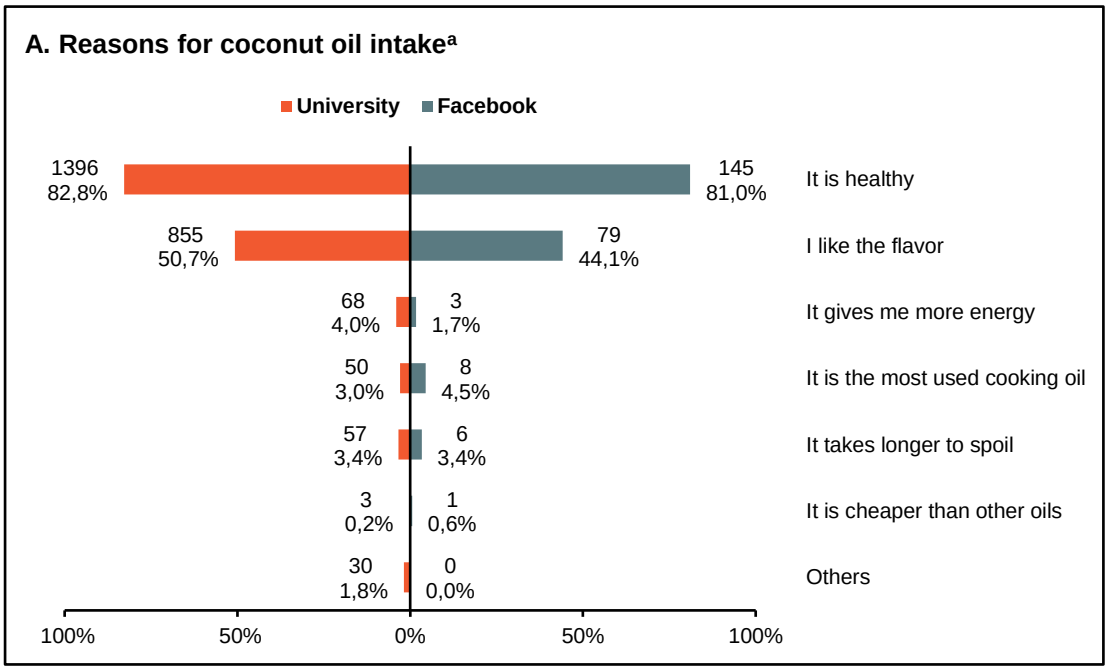
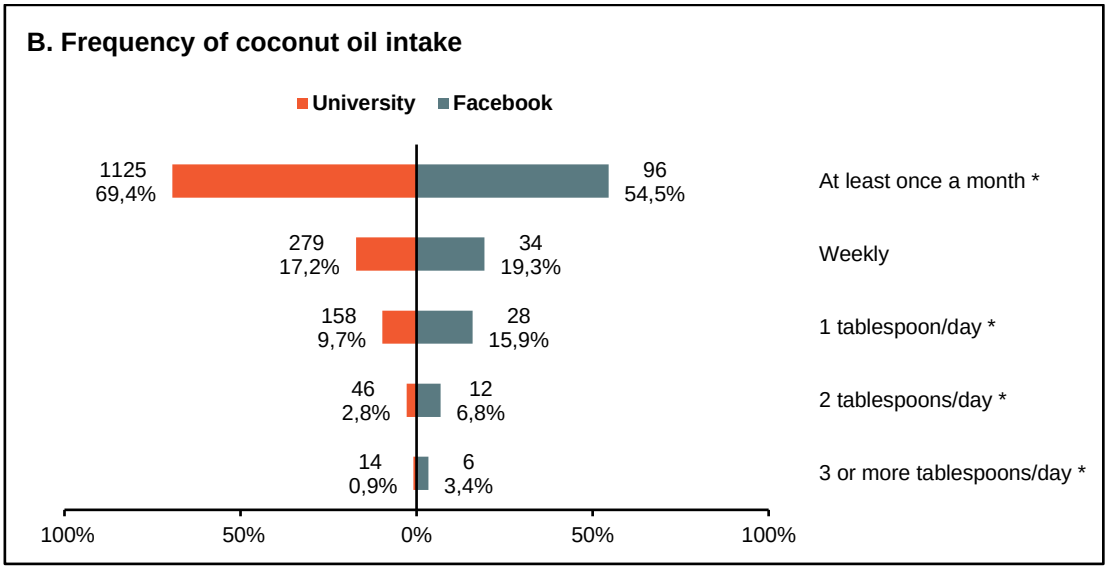
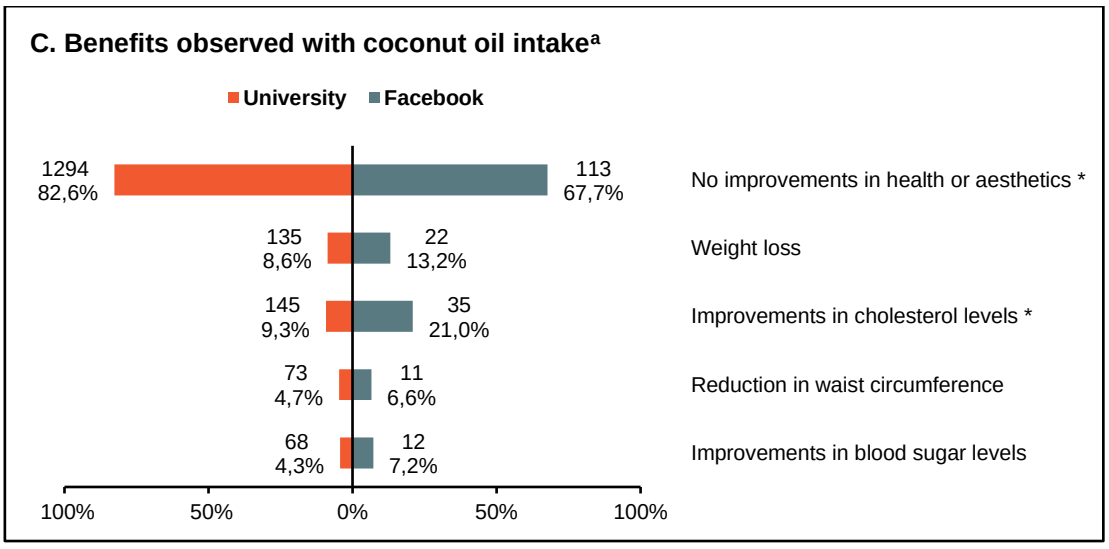


Figure 2. Patterns, reasons, and expectancy regarding the health benefits of coconut oil for those who consumed it.

[a]This question allowed the participant to mark multiple items. *p<0.001.







Material suplementar do Capítulo III – "Misinformation in nutrition through the case of coconut oil: an online before-and-after study" encontra-se no Anexo II, entre as páginas 176-186.

CAPÍTULO IV - Considerações Finais e Perspectivas Futuras

O óleo de coco é composto predominantemente por ácidos graxos saturados. É bem documentado na literatura que esta classe de gorduras deve fazer parte da alimentação habitual em menor quantidade do que os ácidos graxos mono e poli-insaturados, tendo em vista a associação desfavorável de seu consumo com a saúde cardiometabólica. Mesmo assim, o óleo de coco foi apontado em estudos científicos, por profissionais da saúde em sua prática clínica, e em mídias sociais, televisivas e impressas como sendo um óleo mais saudável.

Nós conduzimos uma meta-análise de ensaios clínicos randomizados apenas com estudos paralelos, ou com estudos cruzados com dados cedidos pelos autores e que nos permitissem excluir um possível efeito *carry-over* entre as intervenções. Observamos que o consumo alimentar de óleo de coco, em comparação com outros óleos e/ou gorduras, não exerce efeito em parâmetros antropométricos, de perfil glicêmico, nem nos níveis de pressão arterial e inflamação subclínica. Em relação ao perfil lipídico, observamos apenas um aumento estatisticamente significativo dos níveis de HDL-C, sem alteração nos níveis de LDL-C, TG, na relação LDL/HDL e CT:HDL.

Durante a elaboração deste trabalho, nos questionamos quais seriam as motivações dos consumidores para utilização do óleo de coco. Para responder esta questão, nós elaboramos uma pesquisa online antes/depois, aplicada em uma população do sul do Brasil. A maior parte das pessoas que consomem óleo de coco o utilizam por considerá-lo um óleo bom para a saúde, mesmo não observando melhoras na sua saúde ou estética com o seu consumo. Ao serem expostos aos resultados de uma meta-análise, mostrando que o óleo de coco não exerce influência

positiva sobre parâmetros metabólicos, a maioria dos participantes, continuaram considerando o óleo de coco bom para a saúde.

Observamos que há uma lacuna entre a informação científica publicada e aquela que chega e é praticada pela população. Profissionais da saúde que são agentes importantes de disseminação da informação científica nem sempre apresentam a formação necessária para analisar com cautela estudos científicos, podendo repassar informações equivocadas para a população, baseando as suas condutas em resultados de evidências científicas de qualidade metodológica limitada. Por outro lado, sabe-se que a população, de maneira geral, apresenta baixo grau de alfabetização em saúde, tendo dificuldade de desenvolver um olhar crítico para as informações divulgadas pelos profissionais de saúde e mídias em geral. Além disso, com o avanço da tecnologia, ocorreu um aumento do fluxo de informações e acesso da população a todo tipo de notícia, contribuindo para tomadas de decisões de saúde equivocadas, baseadas em achismos, crenças pessoais e populares e/ou evidência científicas de baixa qualidade.

Com base no exposto, o óleo de coco não deveria ser incentivado como óleo de primeira escolha para o consumo. Estratégias educativas deveriam ser estabelecidas a fim de conscientizar sobre o potencial risco do consumo desse óleo do ponto de vista cardiometabólico. Os equívocos relacionados à alimentação e nutrição precisam ser amplamente estudados como um problema de saúde pública e estratégias devem ser implementadas para reduzir o impacto da desinformação como promotora do consumo de alimentos não saudáveis vinculados às doenças cardiometabólicas, emagrecimento e saúde no geral.

Como perspectiva futura, esperamos que este trabalho possa estimular e servir de base para aplicação em outras questões controversas de saúde e nutrição. Há uma

lacuna entre a informação científica publicada e aquela que chega e é praticada pela população. O desenvolvimento de estudos info epidemiológicos que objetivem entender todas as fases do processo de transmissão da informação científica em saúde para a população permitirá a elaboração de protocolos de intervenção e avaliação de seu impacto na melhora da saúde da população. Com estes resultados será possível construir estratégias de saúde pública a fim de prevenir um malefício causado pela desinformação.

Anexo I – Material suplementar do Capítulo II – **“‘Effects of coconut oil on the cardiometabolic profile: systematic review and meta-analysis of randomized clinical trials”.**

Summary:

Appendix II – Figure 4. Forest plots of randomized controlled clinical trials investigation the effects of coconut oil intake vs MUFA and PUFA rich oils on fasting blood glucose (mg/dL)

Blood pressure

Systolic Blood Pressure

Diastolic Blood Pressure

Inflammatory profile

Appendix II– Figure 5. Forest plots of randomized controlled clinical trials investigation the effects of coconut oil intake on US-CRP (mg/dL)

Supplementary figures

Figure S1 - Effect on body weight of coconut oil versus PUFA and MUFA rich oils

Figure S2 - Effect on body weight of coconut oil versus olive oil

Figure S3 - Effect on body weight of coconut oil versus soybean oil

Figure S4 - Effect on body weight of coconut oil versus other oils when analyzing studies carried out in women

Figure S5 - Effect on body weight of coconut oil versus other oils when analyzing studies conducted in Brazil

Figure S6 - Effect on body weight of coconut oil versus other oils or fat in patients with overweight/obesity

Figure S7 - Effect on body weight of coconut oil versus other oils or fat without a long term study

Figure S8 - Effect on body weight of coconut oil versus other oils or fat with co-intervention

Figure S9 - Effect on waist circumference of coconut oil versus PUFA and MUFA rich oils

Figure S10 - Effect on waist circumference of coconut oil versus olive oil

Figure S11 - Effect on waist circumference of coconut oil versus soybean oil

Figure S12 - Effect on waist circumference of coconut oil versus other oils when analyzing studies carried out in women

Figure S13 - Effect on waist circumference of coconut oil versus other oils when analyzing studies conducted in Brazil

Figure S14 - Effect on waist circumference of coconut oil versus other oils or fat in patients with overweight/obesity

Figure S15 - Effect on waist circumference of coconut oil versus other oils or fat with co-intervention

Figure S16 - Effect on body composition of coconut oil versus other oils/fat

Figure S17 - Effect on body composition of coconut oil versus PUFA and MUFA rich oils

Figure S18 - Effect on LDL-C levels of coconut oil versus PUFA and MUFA rich oils

Figure S19 - Effect on LDL-C levels of coconut oil versus olive oil

Figure S20 - Effect on LDL-C levels of coconut oil versus soybean oil

APPENDIX I - SEARCH STRATEGY AND SEARCH TERMS

Full search strategy and search terms in Pubmed:

((((("coconut oil" [Supplementary Concept]) OR "coconut oil") OR coconut)) AND ((randomized controlled trial[pt] OR controlled clinical trial[pt] OR randomized controlled trials[mh] OR random allocation[mh] OR double-blind method[mh] OR single-blind method[mh] OR clinical trial[pt] OR clinical trials[mh] OR (“clinical trial”[tw]) OR ((singl*[tw] OR doubl*[tw] OR trebl*[tw] OR tripl*[tw]) AND (mask*[tw] OR blind*[tw])) OR (“latin square”[tw]) OR placebos[mh] OR placebo*[tw] OR random*[tw] OR research design[mh:noexp] OR follow-up studies[mh] OR prospective studies[mh] OR cross-over studies[mh] OR control*[tw] OR prospective*[tw] OR volunteer*[tw] NOT (animal[mh] NOT human[mh]))

Full search strategy and search terms in Embase:

('adult'/exp OR 'adult' OR 'adults' OR 'grown-ups' OR 'grownup' OR 'grownups') AND ('coconut oil'/exp OR 'coconut butter' OR 'coconut fat' OR 'coconut oil' OR 'coconut oil emulsion' OR 'copra oil' OR 'oil, coconut') AND ('randomized controlled trial'/exp OR 'controlled trial, randomized' OR 'pragmatic clinical trial' OR 'pragmatic clinical trials' OR 'randomised controlled study' OR 'randomised controlled trial' OR 'randomized controlled study' OR 'randomized controlled trial' OR 'trial, randomized controlled')

Full search strategy and search terms in LILACS:

(tw:(óleo de coco)) AND (tw:(ensaio clínico))

SUPPLEMENTARY TABLES

Table S1. Detailed reasons for the exclusion of studies in the full text assessment of eligibility stage

Record	Reason for exclusion
Francisco A O Júnior, et al., Coconut Oil Supplementation Does Not Affect Blood Pressure Variability and Oxidative Stress: A Placebo-Controlled Clinical Study in Stage-1 Hypertensive Patients. Nutrients, 2021; 28;13(3):798. doi: 10.3390/nu13030798.	Combination of interventions in groups
Mendis, S., et al. The effect of daily consumption of coconut fat and soya-bean fat on plasma lipids and lipoproteins of young normolipidemic men. Br J Nutr, 1990;63(3):547-52. doi: 10.1079/bjn19900141	Non-randomized clinical trial
Muller, H, et al. The serum LDL/HDL cholesterol ratio is influenced more favorably by exchanging saturated with unsaturated fat than by reducing saturated fat in the diet of women. J Nutr, 2003;133(1):78-83. doi: 10.1093/jn/133.1.78.	Mixing more than one oil in the same food (eg margarine, coconut oil, soy oil), which does not allow us to know the real effects of coconut oil on the outcomes studied.

Ng, T K. et al. Nonhypercholesterolaemic effects of a palm-oil diet in Malaysian volunteers. Am J Clin Nutr, 1991; 53(4 Suppl):1015S-1020S. doi: 10.1093/ajcn/53.4.1015S.	Inadequate intervention
Panth, N., et al. Medium-chain fatty acids lower postprandial lipemia: A randomized crossover trial. Clin Nutr, 2020; 39(1):90-96. doi: 10.1016/j.clnu.2019.02.008.	Insufficient follow-up (<7 days)
Sciarrilo, C M., et al. Postprandial Lipemic Responses to Various Sources of Saturated and Monounsaturated Fat in Adults. Nutrients, 2019; May; 11(5): 1089. doi: 10.3390/nu11051089.	Insufficient follow-up (<7 days)
Trepanowski J F., et al. A 21-day Daniel fast with or without krill oil supplementation improves anthropometric parameters and the cardiometabolic profile in men and women. Nutr Metab (Lond), 2012; 13;9(1):82. doi: 10.1186/1743-7075-9-82.	Data from the placebo and intervention groups were pooled, not being able to analyze the real effects of coconut oil on the outcomes of interest.
Valente FX., et al. Effects of coconut oil consumption on energy metabolism, cardiometabolic risk markers, and appetitive responses in women with excess body fat. Eur J Nutr. 2018; 57(4):1627-1637. doi: 10.1007/s00394-017-1448-5.	Insufficient follow-up (<7 days)

Table S2. Summary of randomized clinical trials investigation the effect of coconut oil intake on anthropometric profile

Author and Year	Study design (Country)	Follow-up	Sample	Intervention	Comparator	Last measurements of anthropometric profile
Assunção (2009)	Randomized clinical trial (Brazil)	12 weeks	n = 40 women with abdominal obesity Age = 29.8 ± 6.6 years BMI = 31.1 ± 3.4 kg/m²	30 ml of coconut oil should be added to the three main meals of the day, in the common preparation of meals	30 ml of soybean oil should be added to the three main meals of the day, in the common preparation of meals	Body weight (kg): soybean oil (75 ± 9.1) > coconut oil (72.1 ± 9.1)* BMI (kg/m²): soybean oil (30.7 ± 3.3) > coconut oil (30.5 ± 3.6)* Waist circumference (cm): coconut oil = soybean oil (97 ± 7)
Cândido (2021)	Randomized clinical trial (Brazil)	9 weeks	n = 52 women with BMI between 26 e 35 kg/m², %G > 30% Age = 26.81 ± 0.74	Vitamin breakfast prepared with 25 ml of coconut oil, skimmed milk powder and some fruit flavoring, chocolate or cappuccino	Vitamin breakfast prepared with 25 ml of soybean oil, skimmed milk powder and some fruit flavoring, chocolate or cappuccino	Body weight (kg): soybean oil (77.24 ± 2.08) > coconut oil (75.99 ± 2.92) > olive oil (75.81 ± 1.65) Waist circumference (cm): coconut oil (94.17 ± 2.24) >

					Vitamin breakfast prepared with 25 ml of olive oil, skimmed milk powder and some fruit flavoring, chocolate or cappuccino	olive oil (93.58 ± 1.91) > soybean oil (92.93 ± 1.87) Total fat (%): soybean oil (46.54 ± 0.90) > coconut oil (45.67 ± 1.29) > olive oil (45.27 ± 1.07)
Chinwong (2017)	Randomized crossover trial, open-label (Thailand)	8 weeks	n = 32 healthy individuals Age = 21 ± 0.7 years BMI = 20.8 ± 3.4 kg/m²	30 ml/day of coconut oil extra virgin	30 ml/day of 2% carboxymethylcellulose solution (CMC) solution	Body weight (kg): coconut oil (59.20 ± 12.57) > CMC solution (58.73 ± 12.02) BMI (kg/m²): coconut oil (20.88 ± 3.55) > CMC solution (20.71 ± 3.33)
Harris (2017)	Randomized crossover trial (EUA)	4 weeks	n = 12 postmenopausal women Age = 57.8 ± 3.7 years BMI = 26.4 ± 4.4 kg/m²	Ingestion of 30 ml of coconut oil per day in ready-made preparations (smoothies-like beverages or in the preparation of salad dressings).	Ingestion of 30ml of safflower oil per day in ready-made preparations (smoothies-like beverages or in the preparation of salad dressings).	Body Weight (kg): coconut oil = safflower oil (68.9 ± 11.5) Waist circumference (cm): safflower oil (87.1 ± 11.9) > coconut oil (85.5 ± 11) Total fat (%): coconut oil = safflower oil (37.5 ± 6)

						Fat mass (kg): coconut oil = safflower oil (25.7 ± 8) Lean mass (kg): coconut oil = safflower oil (41.5 ± 4.5)
Khaw (2018)	Randomized clinical trial (UK)	4 weeks	n = 94 healthy individuals Age = 59.9 ± 6.1 years BMI = 25.1 ± 4.2 kg/m²	Coconut oil: 50g of coconut oil incorporated in the usual daily diet in substitution of other fats or ingested as a supplement.	Butter: 50 g of butter incorporated in the usual daily diet in substitution of other fats or ingested as a supplement. Olive oil: 50 g of olive oil incorporated in the usual daily diet in substitution of other fats or ingested as a supplement.	Body weight (kg): coconut oil (74 ± 15.6) > butter (70.9 ± 11.8) > olive oil (70.4 ± 14.0) BMI (kg/m²): coconut oil (25.6 ± 4.6) > olive oil (24.9 ± 4.5) > butter (24.8 ± 3.6) Waist circumference (cm): coconut oil (86.6 ± 13.6) > olive oil (86.3 ± 12.1) > butter (84.0 ± 8.6) Body fat (%): olive oil (30.9 ± 9.5) > coconut oil (29.6 ± 10.3) > butter (29.6 ± 8.7)
Lu (1997)	Randomized crossover trial	3 weeks	n = 15 healthy women Age = 20.0 ± 2.0 years BMI = 22.6 ± 2.4 kg/m²	Coconut oil: 10% of daily VCT from coconut oil	A16 oil: 10% of daily VCT from oil A16 (transgenic soybean oil,	Body Weight (kg): coconut oil = A16 oil = soybean oil (63.30 ± 7.00) (N/S)

	(EUA)				composed of a lower ratio of 18: 3 without trans fats) Soybean oil: 10% of daily VCT from soybean oil	BMI (kg/m²): coconut oil = A16 oil = soybean oil (22.80 ± 2.50)
Oliveira-de-Lira (2018)	Randomized Clinical Trial (Brazil)	8 weeks	n = 75 obese women Age = 34.07 ± 5.4 years	Coconut oil: 6 ml/day supplemented in capsules 30 min before main meals.	Safflower oil: 6 ml/day supplemented in capsules 30 min before main meals. Chia oil: 6 ml/day supplemented in capsules 30 min before main meals Soybean oil: 6 ml/day supplemented in	Body Weight (kg): soybean oil (82.98 ± 8.09) > safflower oil (82.72 ± 7.67) > chia oil (80.6 ± 6.79) > coconut oil (79.57 ± 8.12)* BMI (kg/m²): soybean oil (32.66 ± 2.86) > safflower oil (32.33 ± 2.44) > chia oil (31.26 ± 1.96) > coconut oil (30.76 ± 2.33)* Waist circumference (cm): soybean oil (94.79 ± 2.66) > chia oil (94.68 ± 4.93) >

					capsules 30 min before main meals.	safflower (94.32 ± 6.25) > coconut oil (91.89 ± 6.05)* Body fat (%): chia oil (40.84 ± 3.33) > soybean oil (39.73 ± 3.37) > sunflower oil (39.62 ± 4.53) > coconut oil (37.57 > 4.03)* Lean mass (kg): coconut oil (62.32 ± 4.49) > safflower oil (60.38 ± 4.53) > soybean oil (60.27 ± 3.37) > chia oil (59.16 ± 3.33)*
Schwab (1994)	Randomized clinical trial (Finland)	4 weeks	n = 15 healthy women Age = 23.9 ± 4.6 years BMI = 21.4 ± 1.9 kg/m²	Refined coconut oil (16 to 26 g/day of coconut oil = 4% of the daily VCT). This diet also contained oils from other sources: rapeseed oil (5 to 8g / day), olive oil (3 to 4.5g / day)	Refined palm oil, bleached and deodorized (22 to 33 g/day of palm oil = 4% of daily VCT). This diet also contained soybean oil (2 to 5 g/day) as a source of fat.	Body Weight (kg): coconut oil = palm oil (58.9 ± 7.35)

				and sunflower oil (2 to 3.5 g/day).		
Vijayakumar (2015)	Randomized clinical trial (India)	2 years	n = 198 individuals with CVD Age = 59.0 ± 8.7 years BMI = 24.7 ± 4.7 kg/m²	15% of the daily VCT of a trademark coconut oil to be used as cooking oil.	15% of the daily VCT of a trademark sunflower oil to be used as cooking oil.	Body weight (kg): sunflower oil (64.8 ± 9.0) > coconut oil (64.23 ± 8.78) BMI (kg/m²): coconut oil (24.72 ± 3.07) > sunflower oil (24.54 ± 3.07) Body Fat (%): coconut oil (17.48 ± 2.91) > sunflower oil (17.39 ± 3.62) Waist hip ratio: coconut oil (0.97 ± 0.05) > sunflower oil (0.96 ± 0.05)
Vogel (2020)	Randomized clinical trial (Brazil)	45 days	n = 29 men with obesity I Age = between 20–59 years	Addition of 1 tablespoon (12ml) of coconut oil to dinner	Addition of 1 tablespoon (12ml) of soybean oil to dinner	BMI (kg/m²): coconut oil (32.28 ± 1.83) > soybean oil (31.17 ± 1.65) Waist circumference (cm): coconut oil (107.13 ± 4.38) > soybean oil (106.17 ± 4.60)

						Body fat (%): coconut oil (25.94 ± 3.64) > soybean oil (24.06 ± 5.01) Lean mass (kg): soybean oil (74.06 ± 3.64) > coconut oil (72.58 ± 3.46) Waist hip ratio: soybean oil (0.96 ± 0.05) > coconut oil (0.94 ± 0.05)

* Significantly different (P<0.05). BMI: body mass index; VCT: total caloric value; CVD: cardiovascular disease.

Table S3. Summary of randomized clinical trials investigation the effect of coconut oil intake on glycemic profile

Assunção (2009)	Randomized clinical trial (Brazil)	12 weeks	n = 40 women with abdominal obesity Age = 29.8 ± 6.6 years BMI = 31.1 ± 3.4 kg/m²	30 ml of coconut oil should be added to the three main meals of the day, in the common preparation of meals	30 ml of soybean oil should be added to the three main meals of the day, in the common preparation of meals	Glucose: coconut oil (82.8 ± 5.4) > soybean oil (78.5 ± 9.9) Insulin (mIu/DL): coconut oil (9.8 ± 4.1) > soybean oil (7.6 ± 2.1)

						HOMA-β: coconut oil (39.4 ± 18) > soybean oil (31.8 ± 9.8) HOMA-S: coconut oil (2 ± 0.9) > soybean oil (1.48 ± 0.45)*
Cândido (2021)	Randomized clinical trial (Brazil)	9 weeks	n = 52 women with BMI between 26 e 35 kg/m², %G > 30% Age = 26.81 ± 0.74	Vitamin breakfast prepared with 25 ml of coconut oil, skimmed milk powder and some fruit flavoring, chocolate or cappuccino	Vitamin breakfast prepared with 25 ml of soybean oil, skimmed milk powder and some fruit flavoring, chocolate or cappuccino Vitamin breakfast prepared with 25 ml of olive oil, skimmed milk powder and some fruit flavoring, chocolate or cappuccino	Glucose: coconut oil (85.69 ± 2.11) > olive oil (84.28 ± 1.19) > soybean oil (82.65 ± 0.01)* Insulin (mIu/DL): soybean oil (9.19 ± 1.12) > coconut oil (8.03 ± 0.95) > olive oil (7.99 ± 0.76)

Heber (1992)	Randomized crossover trial (USA)	3 weeks	n = 9 healthy men	35% of the calories of the day were derived from LIP and of these, 50% were from coconut oil, which was incorporated into muffins or biscuits. Each muffin or cookie provided 13.7 g of LIP of the oil test.	35% of the calories of the day were derived from LIP and of these, 50% were from palm oil or hydrogenated soybean oil which was incorporated into muffins or biscuits. Each muffin or cookie provided 13.7 g of LIP of the oil test.	Glucose: hydrogenated soybean oil (81.0 ± 6.0) > coconut oil (78.0 ± 2.0) > palm oil (69.0 ± 7.0) Insulin (mIu/Dl): coconut oil (14.0 ± 2.0) > hydrogenated soybean oil (12.0 ± 4.0) > palm oil (11.0 ± 1.0)
Khaw (2018)	Randomized clinical trial (UK)	4 weeks	n = 94 healthy individuals Age = 59.9 ± 6.1 years BMI = 25.1 ± 4.2 kg/m²	Coconut oil: 50 g of coconut oil incorporated in the usual daily diet in substitution of other fats or ingested as a supplement.	Butter: 50 g of butter incorporated in the usual daily diet in substitution of other fats or ingested as a supplement. Olive oil: 50 g of olive oil incorporated in the usual daily diet in substitution of	Glucose: butter (97.2 ± 10.8) > olive oil (95.4 ± 10.8) > coconut oil (95.4 ± 9)

					other fats or ingested as a supplement.	
Oliveira-de-Lira (2018)	Randomized Clinical Trial (Brazil)	8 weeks	n = 75 obese women Age = 34.07 ± 5.4 years	Coconut oil: 6 ml/day supplemented in capsules 30 min before main meals.	Safflower oil: 6 ml/day supplemented in capsules 30 min before main meals. Chia oil: 6 ml/day supplemented in capsules 30 min before main meals. Soybean oil: 6 ml/day supplemented in capsules 30 min before main meals.	A1c (%): chia oil (4.95 ± 0.24) > safflower oil (4.91 ± 0.30) > soybean oil (4.89 ± 0.29) > coconut oil (4.58 ± 0.21)*
Vijayakumar (2015)	Randomized clinical trial (India)	2 years	n = 198 individuals with CVD Age = 59.0 ± 8.7 years BMI = 24.7 ± 4.7 kg/m²	15% of the daily VCT of a trademark coconut oil to be used as cooking oil.	15% of the daily VCT of a trademark sunflower oil to be used as cooking oil.	A1c (%): sunflower oil (6.77 ± 1.28) > coconut oil (6.54 ± 1.32)
Vogel (2020)	Randomized clinical trial (Brazil)	45 days	n = 29 men with obesity I	Addition of 1 tablespoon (12ml) of coconut oil to dinner	Addition of 1 tablespoon (12ml) of soybean oil to dinner	Glucose: soybean oil (85.43 ± 5.93) > coconut oil (78.73 ± 10.97)

			Age = between 20–59 years			Insulin (mIu/Dl): soybean oil (9.85 ± 9.93) > coconut oil (5.13 ± 3.79) HOMA-IR: soybean oil (2.16 ± 2.17) > coconut oil (0.92 ± 0.63)

* Significantly different (P<0.05). BMI: body mass index; LIP: lipids; CVD: cardiovascular disease; VCT: total caloric value

Table S4. Summary of randomized clinical trials investigation the effect of coconut oil intake on arterial blood pressure

Author and Year	**Study design (Country)**	**Follow-up**	**Sample**	**Intervention**	**Comparator**	**Last measurements of blood pressure ( ) or changes [ ] (mm Hg)**
Chinwong (2017)	Randomized crossover trial, open-label (Thailand)	8 weeks	n = 32 healthy individuals Age = 21 ± 0.7 years BMI = 20.8 ± 3.4 kg/m²	30 ml/day of extra virgin coconut oil	30 ml/day of 2% carboxymethylcellulose solution (CMC) solution	SBP: CMC solution (117.63 ± 13.49) > coconut oil (114.84 ± 11.29)

						DBP: coconut oil (70.41 ± 6.42) > CMC solution (69.50 ± 13.28)
Khaw (2018)	Randomized clinical trial (UK)	4 weeks	n = 94 healthy individuals Age = 59.9 ± 6.1 years BMI = 25.1 ± 4.2 kg/m²	Coconut oil: 50 g of coconut oil incorporated in the usual daily diet in substitution of other fats or ingested as a supplement.	Butter: 50 g of butter incorporated in the usual daily diet in substitution of other fats or ingested as a supplement. Olive oil: 50 g of olive oil incorporated in the usual daily diet in substitution of other fats or ingested as a supplement.	SBP: coconut oil [0.18 ± 11.46] > butter [-3.79 ± 11.11] > olive oil [- 3.67 ± 8.23] DBP: coconut oil [-2.02 ± 5.71] > butter [-1.33 ± 6.24] > olive oil [-0.45 ± 8.48]

* Significantly different (P<0.05). BMI: body mass index; SBP: systolic blood pressure; DPB: diastolic blood pressure

Table S5. Summary of randomized clinical trials investigation the effect of coconut oil intake on the inflammatory profile

Author and Year	Study design (Country)	Follow-up	Sample	Intervention	Comparator	Last measurements of inflammatory profile
Assunção (2009)	Randomized clinical trial (Brazil)	12 weeks	n = 40 women with abdominal obesity Age = 29.8 ± 6.6 years BMI = 31.1 ± 3.4 kg/m²	30 ml of coconut oil should be added to the three main meals of the day, in the common preparation of meals	30 ml of soybean oil should be added to the three main meals of the day, in the common preparation of meals	US-CRP (mg/dL): soybean oil (4.2 ± 3.2) > coconut oil (3.7 ± 1.7) Fibrinogen (mg/dL): coconut oil (243.8 ± 41.9) > soybean oil (243.6 ± 43.9)
Khaw (2018)	Randomized clinical trial (UK)	4 weeks	n = 94 healthy individuals Age = 59.9 ± 6.1 years BMI = 25.1 ± 4.2 kg/m²	Coconut oil: 50 g of coconut oil incorporated in the usual daily diet in substitution of other fats or ingested as a supplement.	Butter: 50 g of butter incorporated in the usual daily diet in substitution of other fats or ingested as a supplement. Olive oil: 50 g of olive oil incorporated in the	US-CRP (mg/dL): (0.19 ± 0.2) > butter (0.16 ± 0.11) > coconut oil (0.14 ± 0.13)

					usual daily diet in substitution of other fats or ingested as a supplement.	
Vijayakumar (2015)	Randomized crossover trial (India)	2 years	n = 198 individuals with CVD Age = 59.0 ± 8.7 years BMI = 24.7 ± 4.7 kg/m²	15% of the daily VCT of a trademark coconut oil to be used as cooking oil.	15% of the daily VCT of a trademark sunflower oil to be used as cooking oil.	US-CRP (IU/L): sunflower oil (1.43 ± 1.72) > coconut oil (1.23 ± 1.59)
Voon (2011)	Randomized crossover trial (Malaysia)	5 weeks	n = 45 normal and overweight healthy adults Age: 30.1 ± 8.3 years BMI = 23.1 ± 3.7 kg/m²	Meals with 30% energy from fat, two-thirds of which was from coconut oil (20% total energy)	Meals with 30% energy from fat, two-thirds of which was from palm oil or extra virgin olive oil (20% total energy)	tcHcy (µmol/L): coconut oil (9.13 ± 3.17) > palm oil (8.88 ± 3.05) > olive oil (8.76 ± 2.96) US-CRP (mg/dL): olive oil (2.19 ± 2.36) > palm oil (2.15 ± 2.89) > coconut oil (1.96 ± 2.01) IL-1β (pg/mL): coconut oil (25.93 ± 71.05) > olive oil (23.63 ± 57.95) > palm oil (23.09 ± 57.93)

						IL-6 (pg/mL): coconut oil (9.91 ± 44.07) > olive oil (8.71 ± 31.15) > palm oil (8.52 ± 32.19) IFN- γ (pg/mL): palm oil (17.04 ± 37.78) > olive oil (16.2 ± 36.68) > coconut oil (11.53 ± 30.78) IL-8 (pg/mL): olive oil (71.02 ± 130.1) > palm oil (67.15 ± 108.46) > coconut oil (47.35 ± 85.3)

* Significantly different (P>0.005). BMI: body mass index; LIP: lipids; CVD: cardiovascular disease; VCT: total caloric value

Table 6. Summary of randomized clinical trials investigation the effect of coconut oil on changes in the lipid profile

Author and Year	**Study design (Country)**	**Follow-up**	**Sample**	**Intervention**	**Comparator**	**Last measurements of lipids ( ) or changes in lipids [ ] (mg/dL, except where specified)**
Assunção (2009)	Randomized clinical trial (Brazil)	12 weeks	n = 40 women with abdominal obesity Age = 29.8 ± 6.6 years BMI = 31.1 ± 3.4 kg/m²	30 ml of coconut oil should be added to the three main meals of the day, in the common preparation of meals	30 ml of soybean oil should be added to the three main meals of the day, in the common preparation of meals	TC: soybean oil (209.3 ± 28.5) > coconut oil (198.1 ± 39.0) * LDL-C: soybean oil (134.1 ± 28.7) > coconut oil (116.5 ± 36.8) * HDL-C: coconut oil (48.7 ± 2.4) > soybean oil (45.0 ± 5.6) TG: coconut oil (179.7 ± 93.7) > soybean oil (148.2 ± 64.8) LDL-C:HDL-C ratio: soybean oil (3.1 ± 0.8) > coconut oil (2.41 ± 0.8) *
Cândido (2021)	Randomized clinical trial (Brazil)	9 weeks	n = 52 women with BMI between 26 e 35 kg/m², %G > 30%	Vitamin breakfast prepared with 25 ml of coconut oil, skimmed milk	Vitamin breakfast prepared with 25 ml of soybean oil,	TC: coconut oil (173.50 ± 5.55) > olive oil (165.16 ± 6.22) > soybean oil (151.59 ± 5.81)

			Age = 26.81 ± 0.74	powder and some fruit flavoring, chocolate or cappuccino	skimmed milk powder and some fruit flavoring, chocolate or cappuccino Vitamin breakfast prepared with 25 ml of olive oil, skimmed milk powder and some fruit flavoring, chocolate or cappuccino	LDL-C: coconut oil (106.69 ± 4.79) > olive oil (95.89 ± 4.64) > soybean oil (85.82 ± 4.64) HDL-C: olive oil (48.26 ± 2.27) > coconut oil (46.37 ± 2.54) > soybean oil (42.27 ± 3.28) TG: olive oil (99.18 ± 8.56) >coconut oil (87 ± 7.20) > soybean oil (80.41 ± 8.35) VLDL: olive oil (19.83 ± 1.71) > coconut oil (17.40 ± 1.44) > soybean oil (16.08 ± 1.67)
Chinwong (2017)	Randomized crossover trial, open-label (Thailand)	8 weeks	n = 32 healthy individuals Age = 21 ± 0.7 years BMI = 20.8 ± 3.4 kg/m²	30 ml/day of coconut oil extra virgin	30 ml/day of 2% carboxymethylcellulose (CMC) solution	TC: coconut oil (187.7 ± 34.5) > CMC solution (183.7 ± 33.7) LDL-C: coconut oil (110.5 ± 30.5) > CMC solution (110.2 ± 32.0) HDL-C: coconut oil (64.2 ± 9.9) > CMC solution (59.0 ± 10.2)*

						TG: CMC solution (72.3 ± 28.5) > coconut oil (64.7 ± 23.5)
Cox (1995)	Randomized crossover trial (New Zealand)	6 weeks	n = 28 individuals TC: 5.5– 7.9 mmol/L TG: <3 mmol/L Age: 29 – 67 years BMI = 25.1 ± 4.2 kg/m²	Three diets, each one followed for a 6-week period. Total fat supplied 36% of energy and carbohydrate 47% of energy. Coconut diet: SFA from coconut oil supplied 20% of energy.	Butter diet: SFA from butter supplies ~20% of total energy. Safflower diet: 10% of energy from safflower oil; SFA and PUFA each 10% of total energy.	TC: butter (263.0 ± 33.0) > coconut oil (249.0 ± 29.0) > safflower oil (233.0 ± 29.0) * LDL-C: butter (175.0 ± 30.0) > coconut oil (163.0 ± 29.0) > safflower oil (151.0 ± 28.0) * HDL-C: coconut oil (57.0 ± 15.0) > butter (56.0 ± 14.0) > safflower oil (54.0 ± 13.0) TG: butter (177.0 ± 115.0) > coconut oil (159.0 ± 89.0) > safflower oil (151.0 ± 89.0)
Ganji (1996)	Randomized crossover trial (EUA)	7 days	n = 10 healthy individuals Age = 31.0 ± 5.0 years BMI = 22.3 ± 1.7 kg/m²	Coconut oil was incorporated in the preparation of a loaf, with 42 g of coconut oil, making up 20% of the VCT. Participants should	Soybean oil was incorporated in the preparation of a loaf with 42 g of soybean oil, making up 20% of the VCT. Participants	Coconut and soybean oil: TC: coconut oil (204.9 ± 32.5) > soybean oil (191.0 ± 24.0) * LDL-C: coconut oil (126.8 ± 30.2) > soybean oil (111.8 ± 23.2)

				consume 1/3 of this bread in each of the three main meals. Coconut oil plus psyllium fiber was incorporated in the preparation of a loaf, with 42 g of coconut oil, making up 20% of the VCT. Participants should consume 1/3 of this bread in each of the three main meals + 20 g of psyllium fiber per day divided into three equal doses. Soybean oil was incorporated in the preparation of a loaf with 42 g of soybean oil, making up 20% of the	should consume 1/3 of this bread in each of the three main meals.	HDL-C: coconut oil (53.3 ± 0.3) > soybean oil (52.2 ± 8.5) * TG: coconut oil (158.5 ± 53.1) > soybean oil (131.1 ± 39.0) * VLDL: coconut oil (25.1 ± 13.1) > soybean oil (25.1 ± 10.0) LDL-C/HDL-C ratio: coconut oil (2.40 ± 0.90) > soybean oil (2.20 ± 0.70) Coconut and soybean oil + psyllium fiber: TC: coconut oil (192.6 ± 28.2) > soybean oil (177.1 ± 32.1) * LDL-C: coconut oil (112.5 ± 28.2) > soybean oil (100.5 ± 28.2) * HDL-C: coconut oil (53.2 ± 9.7) > soybean oil (53.7 ± 8.9) TG: coconut oil (141.7 ± 47.9) > soybean oil (134.6 ± 54.0)

				VCT. Participants should consume 1/3 of this bread in each of the three main meals. Soybean oil plus psyllium fiber was incorporated in the preparation of a loaf with 42 g of soybean oil, making up 20% of the VCT. Participants should consume 1/3 of this bread in each of the three main meals + 20 g of psyllium fiber per day divided into three equal doses.		VLDL: coconut oil (26.2 ± 12.0) > soybean oil (23.9 ± 8.1) LDL-C/HDL-C ratio: coconut oil (2.2 ± 0.6) > soybean oil (1.8 ± 0.5)*
Harris (2017)	Randomized crossover trial (EUA)	4 weeks	n = 12 postmenopausal women Age = 57.8 ± 3.7 years BMI = 26.4 ± 4.4 kg/m²	Ingestion of 30 ml of coconut oil per day in ready-made preparations (smoothies-like beverages	Ingestion of 30ml of safflower oil per day in ready-made preparations	TC: coconut oil (237.8 ± 24.1) > safflower oil (219.3 ± 22.8)* LDL-C: coconut oil (137.5 ± 27.2) > safflower oil (126.8 ± 25.7)*

				or in the preparation of salad dressings).	(smoothies-like beverages or in the preparation of salad dressings).	HDL-C: coconut oil (70.5 ± 18.8) > safflower oil (62.9 ± 14.5)* TG: safflower oil (118.3 ± 112.7) > coconut oil (107.5 ± 80.6) CT:HDL-C ratio: safflower oil (3.8 ± 1.2) > coconut oil (3.7 ± 1.3)
Heber (1992)	Randomized crossover trial (USA)	3 weeks	n = 9 healthy men	35% of the calories of the day were derived from LIP and of these, 50% were from coconut oil, which was incorporated into muffins or biscuits. Each muffin or cookie provided 13.7g of LIP for the oil test.	35% of the calories of the day were derived from LIP and of these, 50% were from palm oil or hydrogenated soybean oil which was incorporated into muffins or biscuits. Each muffin or cookie provided 13.7g of LIP for the oil test.	TC: coconut oil (195.0 ± 21.0) > palm oil (173.0 ± 21.0) > hydrogenated soybean oil (168.0 ± 15.0)* LDL-C: coconut oil (129.0 ± 24.0) > palm oil (115.0 ± 21.0) > hydrogenated soybean oil (111.0 ± 18.0)* HDL-C: coconut oil (42.1 ± 12.0) > palm oil (41.0 ± 15.0) > hydrogenated soybean oil (39.0 ± 9.0)

						TG: coconut oil (110.0 ± 69.0) > hydrogenated soybean oil (104.0 ± 60.0) > palm oil (79.0 ± 18.0)
Khaw (2018)	Randomized clinical trial (UK)	4 weeks	n = 94 healthy individuals Age = 59.9 ± 6.1 years BMI = 25.1 ± 4.2 kg/m²	Coconut oil: 50 g of coconut oil incorporated in the usual daily diet in substitution of other fats or ingested as a supplement.	Butter: 50 g of butter incorporated in the usual daily diet in substitution of other fats or ingested as a supplement. Olive oil: 50 g of olive oil incorporated in the usual daily diet in substitution of other fats or ingested as a supplement.	TC: coconut oil (239.7 ± 34.8) > butter = olive oil (232.0 ± 38.7)* LDL-C: butter (146.9 ± 35) > olive oil (139.2 ± 39.0)> coconut oil (131.5 ± 35.0)* HDL-C: coconut oil (88.9 ± 27.0) > olive oil = butter (77.3 ± 23.2)* TG: coconut oil (97.4 ± 70.8) > olive oil (97.4 ± 53.1) > butter (88.6 ± 44.3) CT/HDL-C ratio: olive oil (3.3 ± 1.2) > butter (3.3 ± 0.9) > coconut oil (2.9 ± 0.9)*
Lu (1997)	Randomized crossover trial (EUA)	3 weeks	n = 15 healthy women Age = 20.0 ± 2.0 years BMI = 22.6 ± 2.4 kg/m²	Coconut oil: 10% of daily VCT from coconut oil	A16 oil: 10% of daily VCT from oil A16 (transgenic soybean oil, composed of a	TC: A16 oil [-19.7 ± 20.9] > soybean oil [-13.5 ± 20.1] > coconut oil [-9.2 ± 14.7]

					lower ratio of 18: 3 without trans fats) Soybean oil: 10% of daily VCT from soybean oil	LDL-C: A16 oil [-10.0 ± 19.7] > soybean oil [-4.2 ± 20.1] > coconut oil [-2.3 ± 15.8] HDL-C: A16 oil [-8.1 ± 7.0] > soybean oil [-7.0 ± 5.4] > coconut oil [-3.5 ± 8.1]* TG: coconut oil [-13.3 ± 26.6] > A16 oil [-9.7 ± 29.2] > soybean oil [-8.8 ± 28.3] LDL-C/HDL-C ratio: A 16 oil = soybean oil [0.1 ± 0.4] > coconut oil [0.1 ± 0.3]
McKenney (1995)	Randomized crossover trial (EUA)	6 weeks	n = 11 individuals with TC altered Age = 58.0 ± 8 years	Coconut oil was added as the main ingredient in oat biscuits with raisins.	Canola oil has been added as the main ingredient in oat biscuits with raisins.	TC: coconut oil (233.3 ± 19.0) > canola oil (213.1 ± 23.4)* LDL-C: coconut oil (155.4 ± 19.5) > canola oil (138.1 ± 17.0)* HDL-C: coconut oil (53.9 ± 15.9) > canola oil (51.7 ± 15.5)*

						TG: canola oil (121.3 ± 54.2) > coconut oil (120.0 ± 47.7)* CT/HDL-C ratio: coconut oil (4.30 ± 1.10) > canola oil (4.10 ± 0.80)
Maki (2018)	Randomized crossover trial (EUA)	4 weeks	n = 25 individuals Age = 45.2 ± 2.3 years BMI = 27.7 ± 0.8	Consumption of four products made with coconut oil per day, which could be three types of muffins and three types of rolls. Each product was made with one tablespoon of coconut oil (13.6 g), consisting on consumption of 54.4 g of oil per day.	Consumption of four products made with corn oil per day, which could be three types of muffins and three types of rolls. Each product was made with one tablespoon of corn oil (13.6 g), consisting on consumption of 54.4 g of oil per day.	TC: coconut oil [7.1, IC95%: -1.1; 13.1] > corn oil [-0.5, IC95%: -5.7; 9.7]* LDL-C: coconut oil [4.6, IC95%: -2,5; 17.5] > corn oil [-2.7, IC95%: -8.9; 11.5] HDL: coconut oil [6.5, IC95%: 2.7; 17.8] > corn oil [5.4, IC95%: 1.4; 10.3]* TC:HDL-C: corn oil [-4.3 IC95%: -11.7; 1.8] > coconut oil [-3.3, IC 95%: -15; 2.8]* TG: coconut oil [6, IC 95%: -3.0; 13.2] > corn oil [-2.1, IC 95%: -9.7; 20.6]

Oliveira-de-Lira (2018)	Randomized Clinical Trial (Brazil)	8 weeks	n = 75 obese women Age = 34.07 ± 5.4 years	Coconut oil: 6 ml/day supplemented in capsules 30 min before main meals.	Safflower oil: 6 ml/day supplemented in capsules 30 min before main meals. Chia oil: 6 ml/day supplemented in capsules 30 min before main meals Soybean oil: 6 ml/day supplemented in capsules 30 min before main meals.	TC: coconut oil (198.0 ± 17.6) > soybean oil (195.7 ± 26.2) > chia oil (187.1 ± 17.0) > safflower oil (182.9 ± 19.1)* LDL-C: safflower oil (130.6 ± 24.3) > coconut oil (128.3 ± 17.7) > soybean oil (127.5 ± 23.2) > chia oil (123.6 ± 18.2)* HDL-C: coconut oil (55.6 ± 6.4) > soybean oil (49.9 ± 7.1) > chia oil (49.0 ± 5.9) > safflower oil (47.1 ± 10.0)* TG: soybean oil (107.5 ± 39.2) > coconut oil (98.3 ± 29.1) > safflower oil (93.9 ± 36.5) > chia oil (88.0 ± 24.4)* VLDL: soybean oil (20.0 ± 8.0) > chia oil (18.0 ± 5.1) > coconut oil (17.8 ± 3.2) > safflower oil (15.7 ± 4.5)

Reiser (1985)	Randomized crossover trial (USA)	5 weeks	n = 19 normolipidemic male medical students (12 completed all three diets)	35% of total energy from fat, being 60% fat from coconut oil, lard, or safflower oil	Habitual diet at baseline and during washout periods	TC: coconut oil (168.0 ± 3.0) > lard (155.0 ± 3.0) > safflower oil (141.0 ± 3.1)* LDL-C: coconut oil (110.0 ± 4.1) > lard (98.0 ± 4.5) > safflower oil (90.0 ± 4.7)* HDL-C: coconut oil (46.0 ± 1.1) > lard = safflower oil (40.0 ± 1.2) * TG: lard (88.0 ± 3.5) > coconut oil (78 ± 3.6) > safflower oil (72.0 ± 3.7)*
Schwab (1994)	Randomized crossover clinical trial (Finland)	4 weeks	n = 15 healthy women Age = 23.9 ± 4.6 years BMI = 21.4 ± 1.9 kg/m²	Refined coconut oil (16 to 26 g/day of coconut oil = 4% of the daily VCT). This diet still contained oils from other sources: rapeseed oil (5 to 8g/day), olive oil (3 to 4.5g/day) and sunflower oil (2 to 3.5g/day).	Refined palm oil bleached and deodorized (22 to 33 g/day of palm oil = 4% of daily VCT). This diet still contained soybean oil (2 to 5 g/day) as a source of fat.	TC: palm oil (189.9 ± 28.5) > coconut oil (187.5 ± 24.1) LDL-C: palm oil (113.3 ± 19.5) > coconut oil (110.2 ± 18.0) HDL-C: palm oil (58.8 ± 12.0) > coconut oil (57.6 ± 10.5) TG: coconut oil (77.1 ± 30.9) > palm oil (77.1 ± 27.4)

						VLDL: coconut oil (19.7 ± 7.5) > palm oil (17.8 ± 7.5) *
Vijayakumar (2015)	Randomized clinical trial (India)	2 years	n = 198 individuals with CVD Age = 59.0 ± 8.7 years BMI = 24.7 ± 4.7 kg/m²	15% of the daily VCT of a trademark coconut oil to be used as cooking oil.	15% of the daily VCT of a trademark sunflower oil to be used as cooking oil.	TC: sunflower oil (151.6 ± 44.5) > coconut oil (149.3 ± 28.6) LDL-C: coconut oil (91.0 ± 21.9) > sunflower oil (89.6 ± 29.0) HDL-C: sunflower oil (44.4 ± 16.3) > coconut oil (43.2 ± 10.8) TG: sunflower oil (112.2 ± 45.1) > coconut oil (109.3 ± 47.1) VLDL: sunflower oil (22.5 ± 9.7) > coconut oil (21.8 ± 9.4)
Vogel (2020)	Randomized clinical trial (Brazil)	45 days	n = 29 mens with obesity I Age = between 20–59 years	Addition of 1 tablespoon (12 ml) of coconut oil to dinner	Addition of 1 tablespoon (12 ml) of soybean oil to dinner	TC: soybean oil (177.07 ± 39.44) > coconut oil (171.47 ± 49.44) LDL-C: soybean oil (116.29 ± 26.55) > coconut oil (101 ± 37.17) HDL-C: coconut oil (43.07 ± 14.86) >soybean oil (35.93 ± 7.77) TG: coconut oil (138.87 ± 78.28) > soybean oil (119.50 ± 74.13)

						VLDL: coconut oil (27.53 ± 15.74) > soybean oil (24.85 ± 16.82) TC: HDL-C: soybean oil (5.07 ± 1.35) > coconut oil (4.30 ± 1.58)
Voon (2011)	Randomized crossover trial (Malaysia)	5 weeks	n = 45 normal and overweight healthy adults Age: 30.1 ± 8.3 years BMI = 23.1 ± 3.7 kg/m²	Meals with 30% energy from fat, two-thirds of which was from coconut oil (20% total energy)	Meals with 30% energy from fat, two-thirds of which was from palm oil or extra virgin olive oil (20% total energy)	TC: coconut oil (191.4 ± 26.7) > palm oil (186.0 ± 28.6) > olive oil (179.8 ± 27.5)* LDL-C: coconut oil (127.6 ± 29) > palm oil (123.7 ± 27.5) > olive oil (118.3 ± 24.7)* HDL-C: coconut oil (53.0 ± 11.6) > palm oil (50.6 ± 10.0) > olive oil (49.5 ± 8.9)* TG: coconut oil (79.7 ± 34.5)> palm oil (75.3 ± 27.5) > olive oil (74.4 ± 32.8) TC:HDL-C ratio: palm oil (3.69 ± 0.90) > coconut oil (3.65 ± 0.95) > olive oil (3.63 ± 0.93)

* Significantly different (P<0.05). BMI: body mass index; TC: total cholesterol; LDL-C: low density lipoprotein; HDL-C: high density lipoprotein; TG: triglycerides, SFA: saturated fatty acid; VCT: total caloric value; LIP: lipids; MUFA: monounsaturated fatty acid; PUFA: polyunsaturated fatty acid; CVD: cardiovascular disease

Table S7. Grading of Recommendations Assessment, Development and Evaluations (GRADE) – Coconut oil compared to other oils, fat or placebo health outcomes

Certainty assessment							Summary of findings				
							Study event rates (%)			Anticipated absolute effects	
Participants (studies)	Risk of bias	Inconsistency	Indirectness	Imprecision	Other	Overall certainty of evidence	With other oils, fat or placebo	With Coconut oil	Relative effect (95% CI)	Risk with other oils, fat or placebo	Risk difference with Coconut oil
LDL-C											
515 (7 RCTs)	very serious[a]	serious[b]	not serious	serious[c]	none	⨁◯◯◯ Very low	304	211	-	The mean lDL-c was **0** mg/dL	MD **1.67 mg/dL lower (6.93 lower to 3.59 higher)**
HDL-C											

515 (7 RCTs)	very serious[d]	serious[e]	not serious	not serious	none	⊕◯◯◯ Very low	304	211	-	The mean hDL-c was **0** mg/dL	MD **3.28 mg/dL higher (0.66 higher to 5.9 higher)**

Triglycerides

515 (7 RCTs)	very serious[f]	not serious	not serious	serious[g]	none	⊕◯◯◯ Very low	304	211	-	The mean triglycerides were **0** mg/dL	MD **0.24 mg/dL lower (5.52 lower to 5.04 higher)**

Body weight

486 (6 RCTs)	very serious[h]	serious[i]	not serious	not serious	none	⊕◯◯◯ Very low	290	196	-	The mean body weight was **0** kg	MD **0.24 kg lower (0.83 lower to 0.34 higher)**

Waist circumference

287 (4 RCTs)	very serious[j]	serious[k]	not serious	not serious	none	⊕◯◯◯ Very low	190	97	-	The mean waist circumfer	MD **0.64 cm lower (1.69 lower**

										ence was **0** cm	**to 0.41 higher)**

Total body fat

445 (5 RCTs)	very serious[l]	serious[m]	not serious	not serious	none	⊕◯◯◯ Very low	269	176	-	The mean total body fat was **0** %	MD **0.10 % lower (0.56 lower to 0.36 higher)**

Fasting blood glucose

212 (4 RCTs)	very serious[n]	not serious	not serious	serious[o]	none	⊕◯◯◯ Very low	133	79	-	The mean total fasting blood glucose was **0** mg/dL	MD **0.82 mg/dl lower (1.18 lower to 2.82 higher)**

US-CRP

131 (2 RCTs)	very serious[p]	not serious	not serious	not serious	none	⊕◯◯◯ Very low	83	48	-	The mean total USC-RP was **0** mg/dL	MD **0.04 mg/dl lower (0.91 lower to 0.82 higher)**

CI: confidence interval; **MD:** mean difference

Explanations

a. RCTs are at risk of bias due to: blinding of participants and/or outcome (in Khaw et al. and Vogel et al.) and selective reporting (in Schwab et al.). RCTs present an unclear risk of bias in: randomization (in Oliveira-de-Lira et al., Schwab et al., Vijayakumar et al.), allocation (in Assunção et al., Oliveira-de-Lira et al., Schwab et al., Vijayakumar et al., Vogel et al.), participant blinding and/or outcome (in Candido et al., Khaw et al., Schwab et al., Vijayakumar et al., Vogel et al.) and selective reporting (in Assunção et al., Oliveira-de-Lira et al., Vijayakumar et al.).
b. Large amounts of statistical heterogeneity (I^2:78%); point estimates and confidence intervals vary considerably.
c. Imprecision due to wide confidence interval: in the worst scenario, it may increase 3.59 mg/dL; in the best scenario, it may decrease 6.93 mg/dL.
d. RCTs are at risk of bias due to: blinding of participants and/or outcome (in Khaw et al. and Vogel et al.) and selective reporting (in Schwab et al.). RCTs present an unclear risk of bias in: randomization (in Oliveira-de-Lira et al., Schwab et al., Vijayakumar et al.), allocation (in Assunção et al., Oliveira-de-Lira et al., Schwab et al., Vijayakumar et al., Vogel et al.), participant blinding and/or outcome (in Candido et al., Khaw et al., Schwab et al., Vijayakumar et al., Vogel et al.) and selective reporting (in Assunção et al., Oliveira-de-Lira et al., Vijayakumar et al.).
e. Large amounts of statistical heterogeneity (I^2:74%); point estimates and confidence intervals vary considerably.
f. RCTs are at risk of bias due to: blinding of participants and/or outcome (in Khaw et al. and Vogel et al.) and selective reporting (in Schwab et al.). RCTs present an unclear risk of bias in: randomization (in Oliveira-de-Lira et al., Schwab et al., Vijayakumar et al.), allocation (in Assunção et al., Oliveira-de-Lira et al., Schwab et al., Vijayakumar et al., Vogel et al.), participant blinding and/or outcome (in Candido et al., Khaw et al., Schwab et al., Vijayakumar et al., Vogel et al.) and selective reporting (in Assunção et al., Oliveira-de-Lira et al., Vijayakumar et al.).
g. Imprecision due to wide confidence interval: in the worst scenario, it may increase 5.04 mg/dL; in the best scenario, it may decrease 5.52 mg/dL.
h. RCTs are at risk of bias due to: blinding of participants and/or outcome (in Khaw et al. and Vogel et al.) and selective reporting (in Schwab et al.). RCTs present an unclear risk of bias in: randomization (in Oliveira-de-Lira et al., Schwab et al., Vijayakumar et al.), allocation (in Assunção et al., Oliveira-de-Lira et al., Schwab et al., Vijayakumar et al., Vogel et al.), participant blinding and/or outcome (in Candido et al., Khaw et al., Schwab et al., Vijayakumar et al., Vogel et al.) and selective reporting (in Assunção et al., Oliveira-de-Lira et al., Vijayakumar et al.).
i. Large amounts of statistical heterogeneity (I^2:76%); point estimates and confidence intervals vary considerably.
j. RCTs are at risk of bias due to: blinding of participants and/or outcome (in Khaw et al. and Vogel et al.) and selective reporting (in Schwab et al.). RCTs present an unclear risk of bias in: randomization (in Oliveira-de-Lira et al., Schwab et al., Vijayakumar et al.), allocation (in Assunção et al., Oliveira-de-Lira et al., Schwab et al., Vijayakumar et al., Vogel et al.), participant blinding and/or outcome (in Candido et al., Khaw et al., Schwab et al., Vijayakumar et al., Vogel et al.) and selective reporting (in Assunção et al., Oliveira-de-Lira et al., Vijayakumar et al.).
k. Large amounts of statistical heterogeneity (I^2:80%); point estimates and confidence intervals vary considerably.
l. RCTs are at risk of bias due to: blinding of participants and/or outcome (in Khaw et al. and Vogel et al.). RCTs present an unclear risk of bias in: randomization (in Oliveira-de-Lira et al., Vijayakumar et al.), allocation (in Oliveira-de-Lira et al., Vijayakumar et al., Vogel et al.), participant blinding and/or outcome (in Candido et al., Khaw et al., Vijayakumar et al., Vogel et al.) and selective reporting (in Oliveira-de-Lira et al. and Vijayakumar et al.).
m. Large amounts of statistical heterogeneity (I^2:75%); point estimates and confidence intervals vary considerably.
n. RCTs are at risk of bias due to: blinding of participants and/or outcome (in Khaw et al. and Vogel et al.). RCTs present an unclear risk of bias in: allocation (in Assunção et al., and Vogel et al.), participant blinding and/or outcome (in Candido et al., Khaw et al. and Vogel et al.) and selective reporting (in Assunção et al.).
o. Imprecision due to wide confidence interval: in the worst scenario, it may increase 2.62 mg/dL; in the best scenario, it may decrease 1.18 mg/dL.
p. RCTs are at risk of bias due to: blinding of participants and/or outcome (in Khaw et al.). RCTs present an unclear risk of bias in: allocation (in Assunção et al.), participant blinding and/or outcome (in Khaw et al.) and selective reporting (in Assunção et al.).

Appendix II
Additional results

LDL-C to HDL-C ratio and TC: HDL-C ratio

Three studies analyzed the effects of coconut oil on LDL-C to HDL-C ratio (n=65, 92% female, 18 to 36 years) [1-3]. The consumption of coconut oil reduced the LDL-C/HDL-C ratio in comparison to soybean and transgenic soybean oils [1,2]. Seven studies [4-10] analyzed the effects of coconut oil on TC: HDL-C ratio. These studies included 291 participants (70% females, 34 to 68 years).

Three studies [4,5,10] were included in the meta-analysis regarding TC:HDL-C ratio (-0.12; CI 95% -0.43 to 0.20; figure 1). We performed a subgroup analysis excluding a study that used a SAFs rich oil/fat as a comparator and the results did not change (vs butter**;** -0.06; CI 95% -0.46 to 0.34; figure 2) [5].

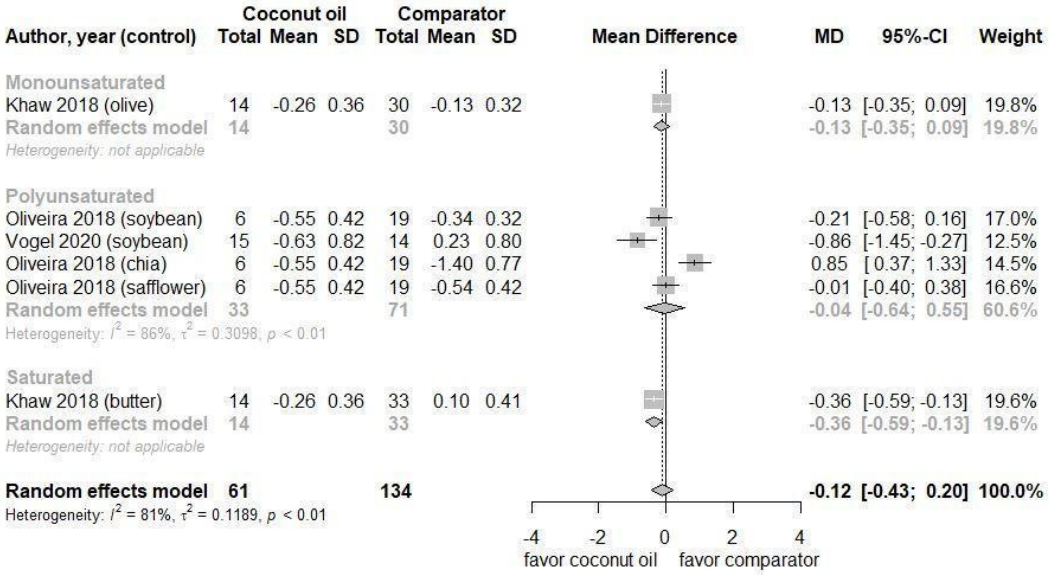


Figure 1. Forest plots of randomized controlled clinical trials investigating the effects of coconut oil intake on TC:HDL-C ratios

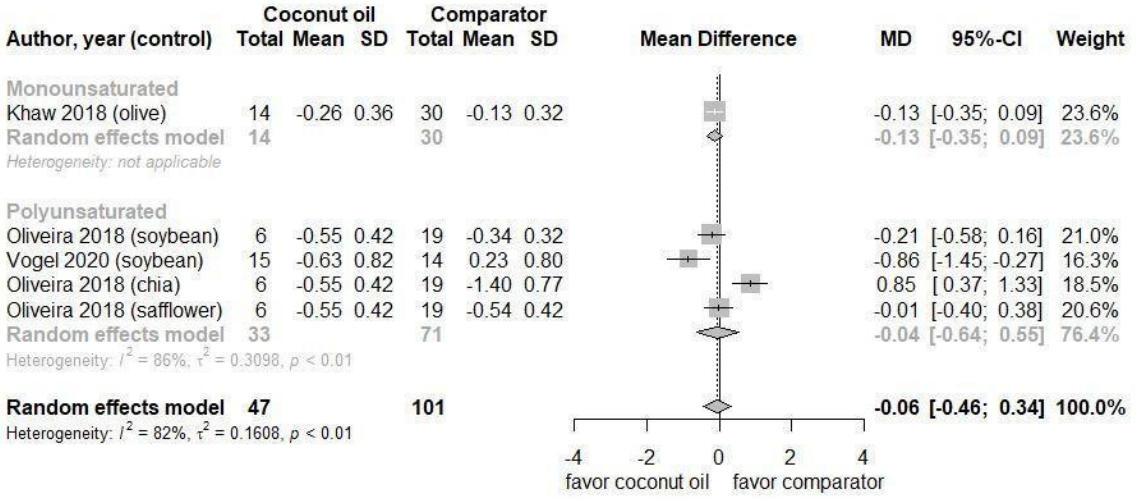


Figure 2. Forest plots of randomized controlled clinical trials investigating the effects of coconut oil intake vs MUFA and PUFA rich oils on TC:HDL-C ratios

Glycemic profile

Fasting blood glucose

Seven studies analyzed the effects of coconut oil on fasting glucose levels [2, 5, 6, 10-13]. These studies included 297 participants (69.3% females, 23 to 66 years). Four studies [2,5,10,13] were included in the meta-analysis. Overall, the effect of coconut oil intake on fasting glucose levels in comparison to other oils/fats did not differ (0.82 mg/dL; 95% CI -1.18 to 2.82 mg/dL; figure 3). We performed a subgroup analysis excluding a study that used a SAFs rich oil/fat as a comparator and the results did not change (vs butter 1.14 mg/dL; 95% CI -1.01 to 3.29 mg/dL; figure 4) [1]. The intake of coconut oil did not fasting plasma glucose did not differ in comparison to PUFAs (0.37 mg/dL; 95% CI -3.37 to 4.12 mg/dL) and MUFAs (1.91 mg/dL; 95% CI -1.48 to 5.30 mg/dL).

A crossover study (n=9) [11] demonstrated that consumption of coconut oil increases blood glucose levels more than palm oil, but less than hydrogenated soybean oil.

A1c

Two studies analyzed this outcome (n=273, 32% female, 29 to 68 years) [4,14]. An 8-week study found significantly lower values of A1c when comparing coconut oil to PUFAs [4]. A 2-year follow-up study compared the consumption of coconut oil with PUFAs and found no difference between groups [14]. Results are shown in table S2.

Effects of coconut oil on insulin levels, β-cell function and indices of insulin sensitivity

A study observed that coconut oil increased β-cell function and insulin sensitivity in comparison to the consumption of soybean oil (n=40, 100% female, 29.8 ± 6.6 years, follow-up: 12 weeks) [2]. Results are shown in table S2.

One study analyzed the insulin resistance index (HOMA-IR), comparing coconut oil with soy oil, but found no difference between groups (n=29, 100% man, 35.27 ± 11.12 coconut oil group and 39.28 ± 9.06 soybean oil group, follow-up: 45 days) [10].

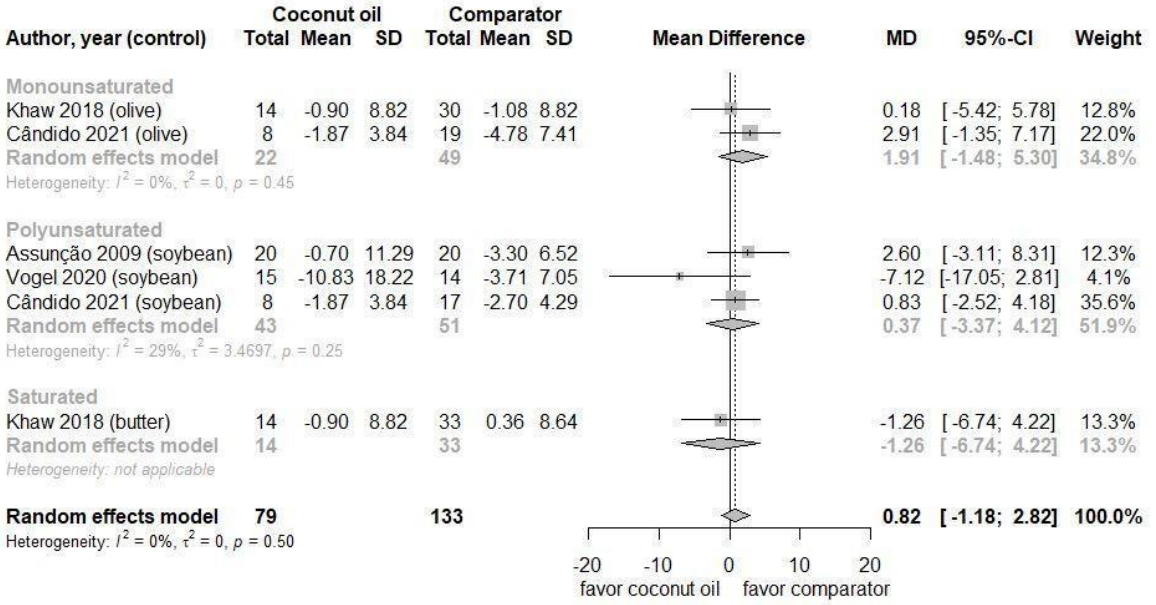


Figure 3. Forest plots of randomized controlled clinical trials investigating the effects of coconut oil intake on fasting blood glucose (mg/dL)

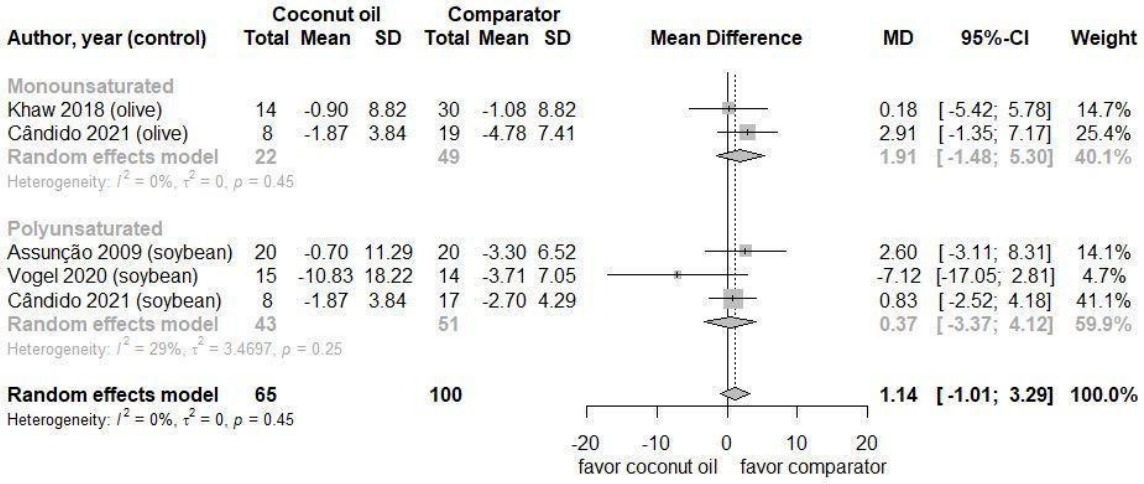


Figure 4. Forest plots of randomized controlled clinical trials investigating the effects of coconut oil intake vs PUFA and MUFA rich oils on fasting blood glucose (mg/dL).

Blood pressure

Blood Pressure Systolic

Two studies [5, 15] analyzed this outcome (n=126, 63% female, 20 to 66 years, follow-up of 4 to 8 weeks). When comparing the effect of coconut oil intake with placebo, higher levels of systolic blood pressure are observed [15]. However, when the effect of the intake of coconut oil is compared with olive oil or butter, lower levels of systolic blood pressure are observed [5]. We were not able to meta-analyze these data, since one study was a crossover trial and there was not enough data. Results are shown in table S3.

Diastolic Blood Pressure

Two studies [5,15] analyzed this outcome (n=126, 63% female, 20 to 66 years, follow-up of 4 to 8 weeks). When the effect of the intake of coconut oil is compared with placebo, olive oil and butter, higher levels of diastolic blood pressure are observed [5,15]. We were not able to meta-analyze these data, since one study was a crossover trial and there was not enough data. Results are shown in table S3.

Inflammatory profile

Four studies (follow-up 4 weeks to 2 years, n=377, 40% females, 22 to 68 years) analyzed the effects of coconut oil on US-CRP [2,5,6,14].

Two studies [2,5] were included in the meta-analysis. Overall, the effect of coconut oil intake on US-CRP in comparison to other oils/fats did not differ (-0.04 mg/dL; 95% CI -0.91 to 0.82 mg/dL; figure 5). A crossover study observed lower levels of US-CRP with the intake of coconut oil when compared to olive and palm oils [6].

One RCT study (follow-up 12 weeks, n=40, 100% female, 23.9 ± 4.6 years) analyzed the effects of coconut oil in fibrinogen. Coconut oil increased fibrinogen when compared to consumption of soybean oil [2].

A crossover study (follow-up 5 weeks, n=45, 80% female, 30.1 ± 8.3 years) observed that coconut oil consumption increased tcHcy, IL-1β, IL-6, IL-8 and *IFN- γ* when compared to the use of palm and extra virgin olive oil [6]. Results are shown in table S4.

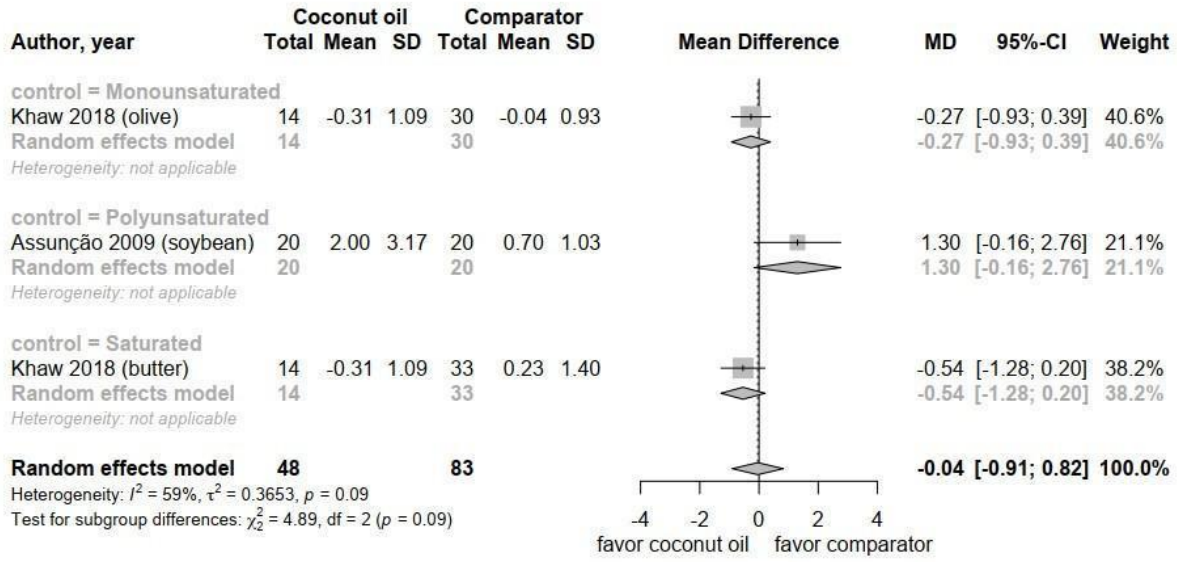
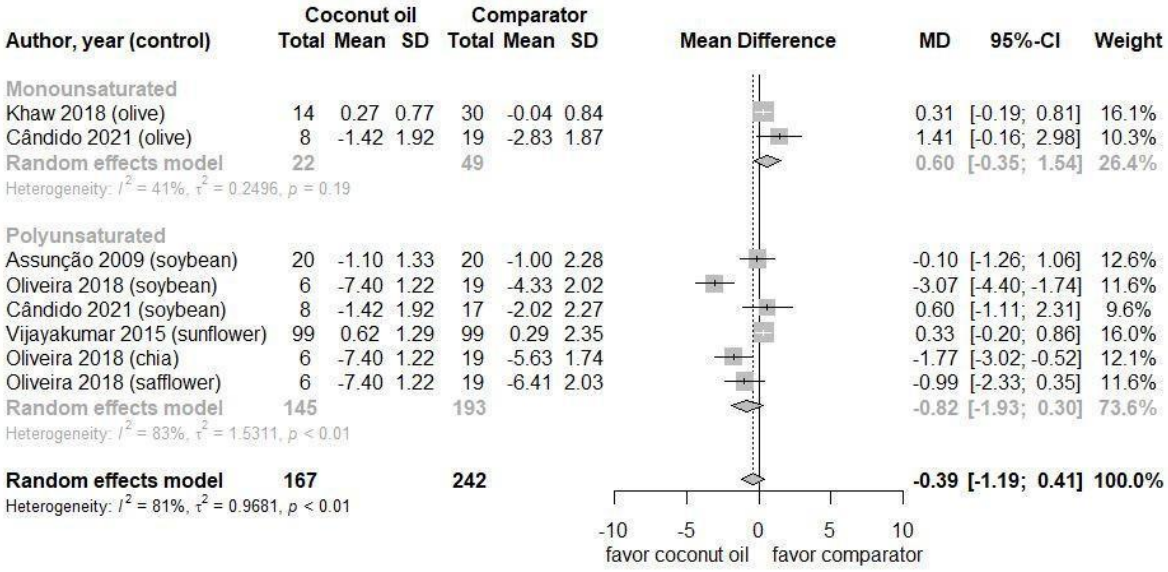


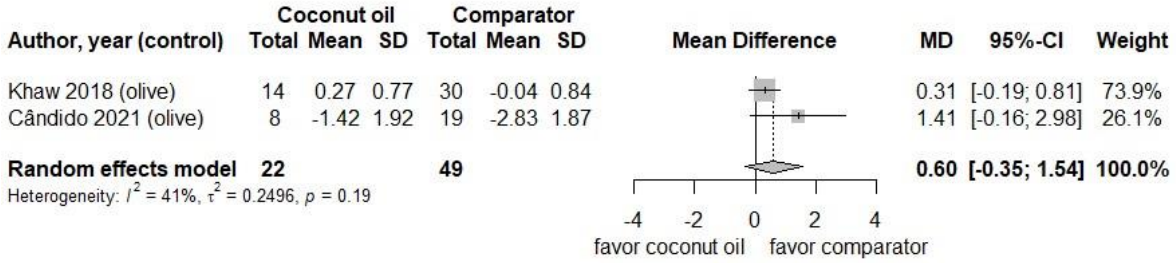
Figure 5. Forest plots of randomized controlled clinical trials investigating the effects of coconut oil intake on US-CRP (mg/dL)

Supplemental figures

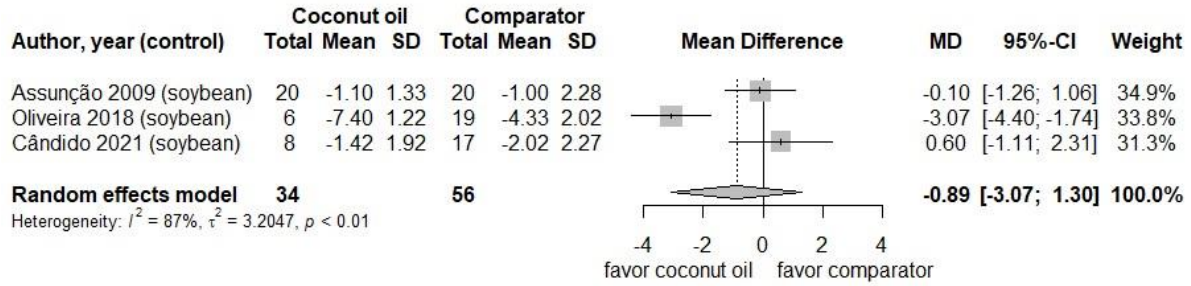
Supplemental Figure 1. Forest plot of randomized controlled clinical trials investigating the effects in body weight (kg) of coconut oil intake versus PUFA and MUFA rich oils



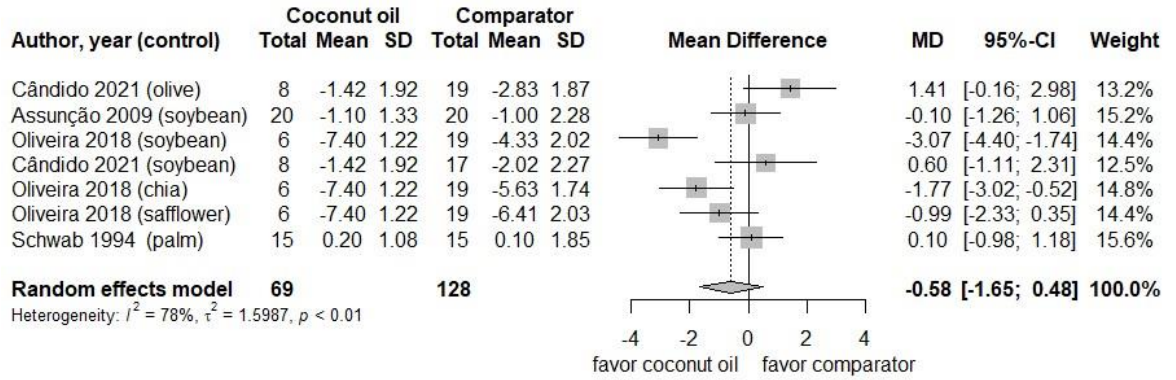
Supplemental Figure 2. Forest plot of randomized controlled clinical trials investigating the effect in body weight (kg) of coconut oil versus olive oil



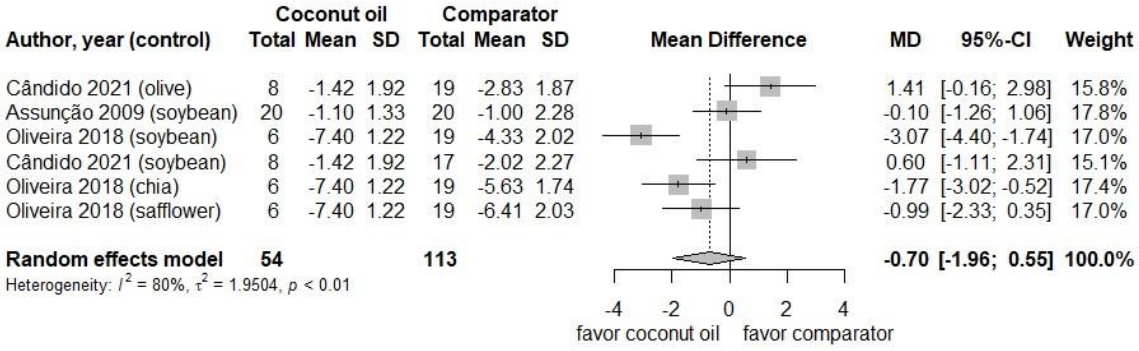
Supplemental Figure 3. Forest plot of randomized controlled clinical trials investigating the effect in body weight (kg) of coconut oil versus soybean oil



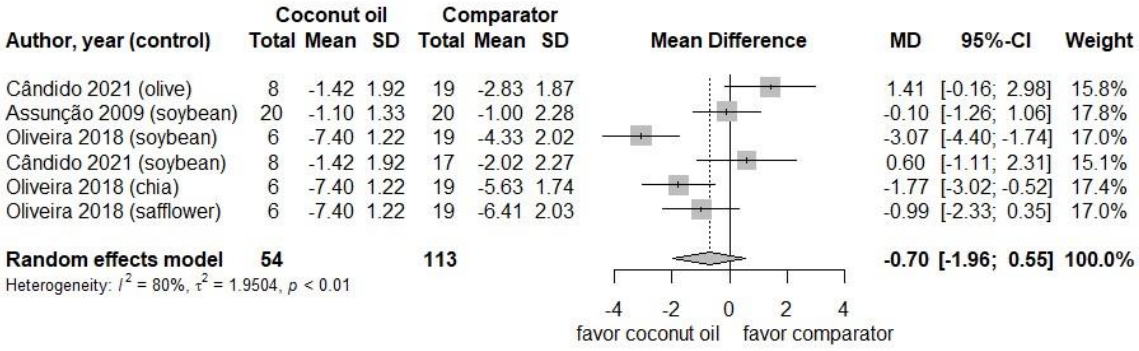
Supplemental Figure 4. Forest plot of randomized controlled clinical trials investigating the effect in body weight (kg) of coconut oil versus other oils in studies carried out in women



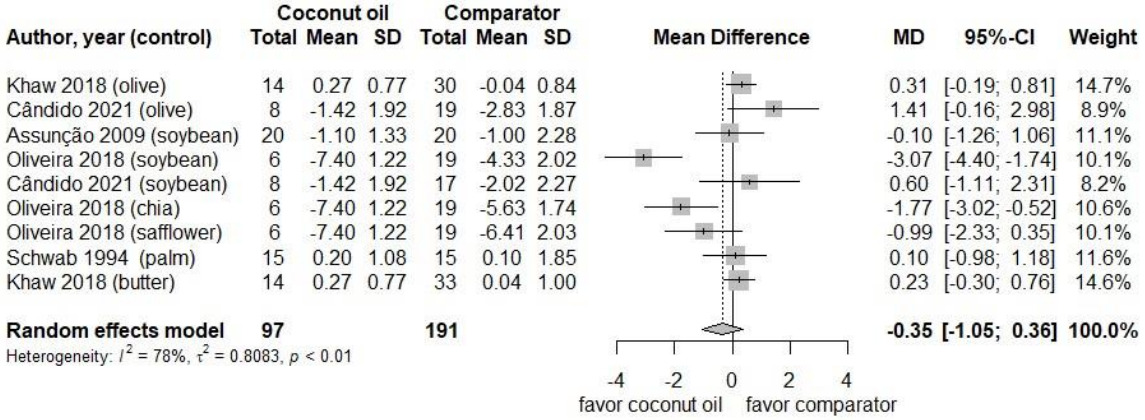
Supplemental Figure 5. Forest plot of the randomized controlled clinical trials investigating the effect of coconut oil versus other oils in body weight (kg) of studies conducted in Brazil



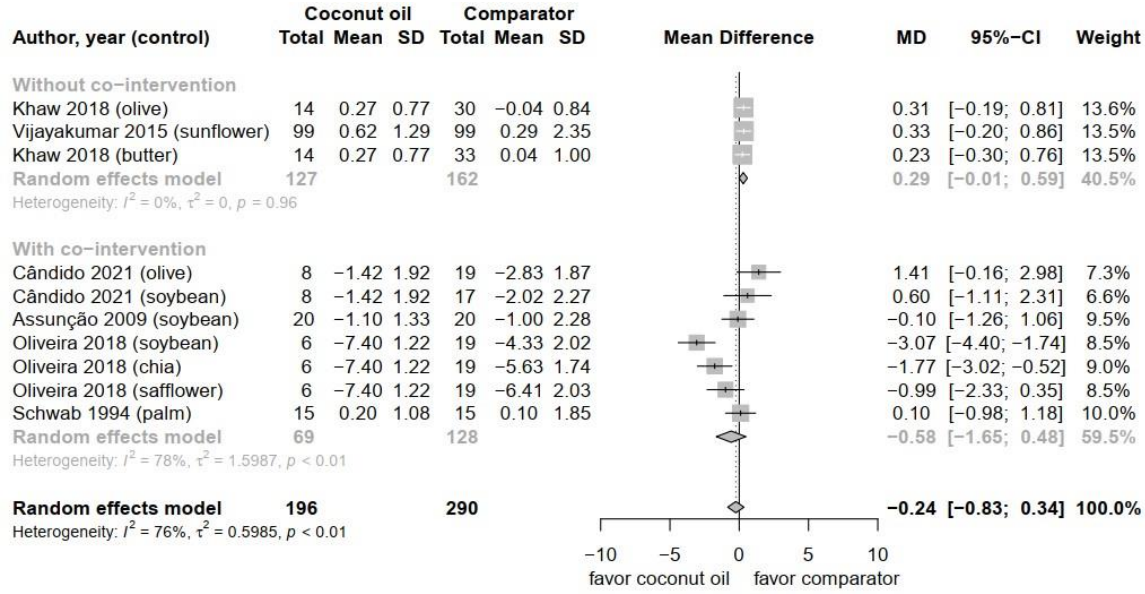
Supplemental Figure 6. Forest plot of randomized controlled clinical trials investigating the effect of coconut oil versus other oils/fats in body weight (kg) of studies carried out in patients with overweight/obesity



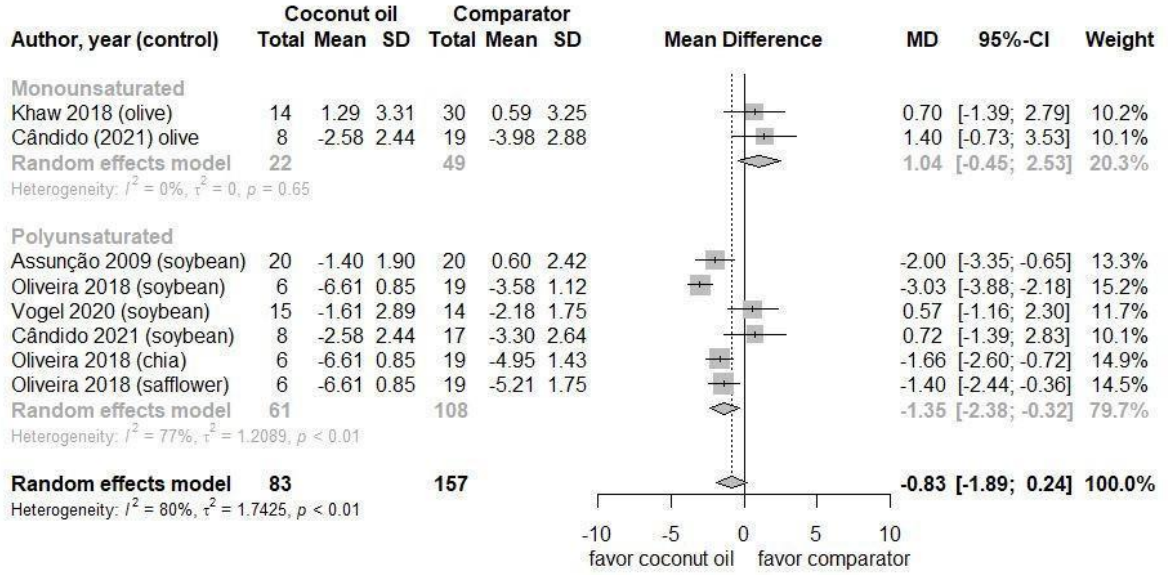
Supplemental Figure 7. Forest plot of randomized controlled clinical trials investigating the effect on body weight (kg) of coconut oil versus other oils/fats without the long term study of Vijayakumar et al.



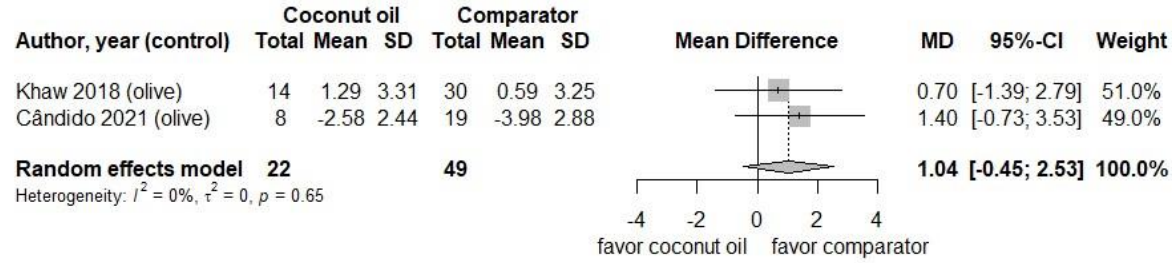
Supplemental Figure 8. Forest plot of randomized controlled clinical trials investigating the effect in body weight (kg) of coconut oil versus other oils/fats in studies including co-intervention



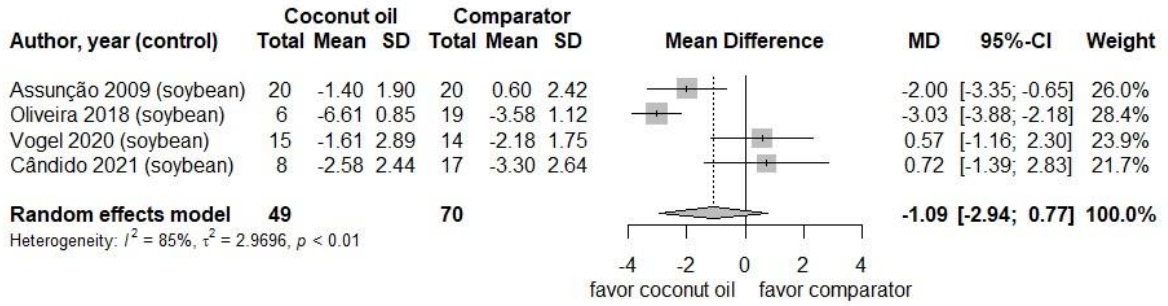
Supplemental Figure 9. Forest plot of randomized controlled clinical trials investigating the effects in waist circumference (cm) of coconut oil intake versus PUFA and MUFA rich oils



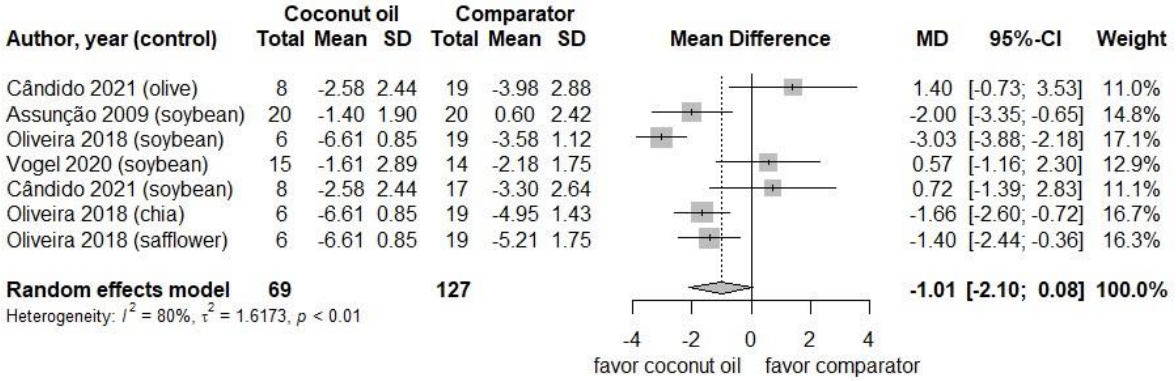
Supplemental Figure 10. Forest plot of randomized controlled clinical trials investigating the effect in waist circumference (cm) of coconut oil versus olive oil



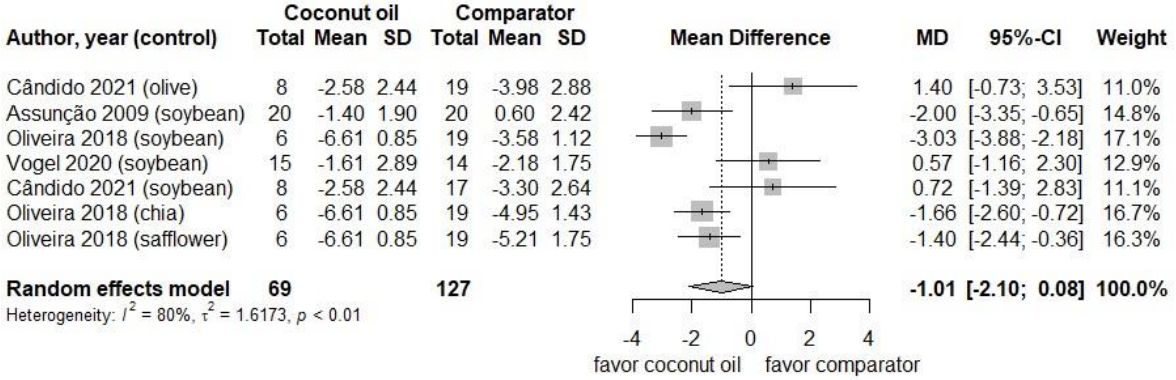
Supplemental Figure 11. Forest plot of randomized controlled clinical trials investigating the effect in waist circumference (cm) of coconut oil versus soybean oil



Supplemental Figure 12. Forest plot of randomized controlled clinical trials investigating the effect in waist circumference (cm) of coconut oil versus other oils when analyzing studies carried out in women



Supplemental Figure 13. Forest plot of randomized controlled clinical trials investigating the effect in waist circumference (cm) of coconut oil versus other oils when analyzing studies conducted in Brazil



Supplemental Figure 14. Forest plot of randomized controlled clinical trials investigating the effect in waist circumference (cm) of coconut oil versus other oils or/fat in patients with overweight/obesity

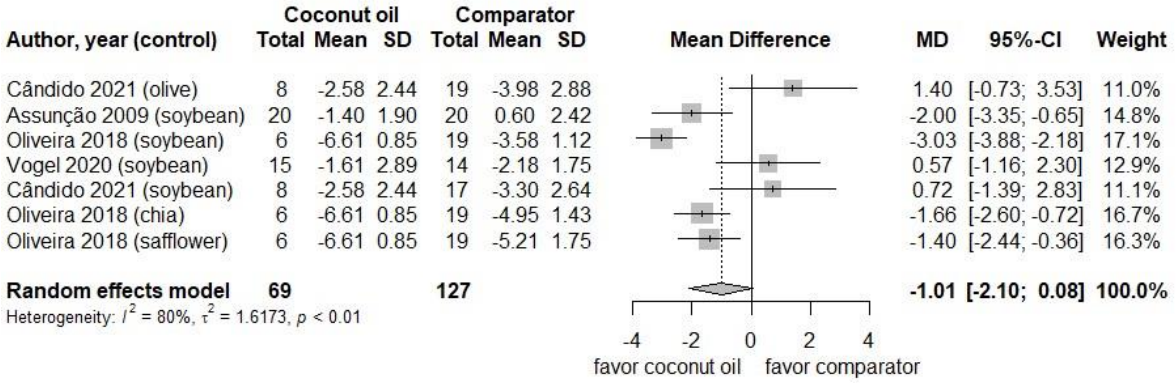
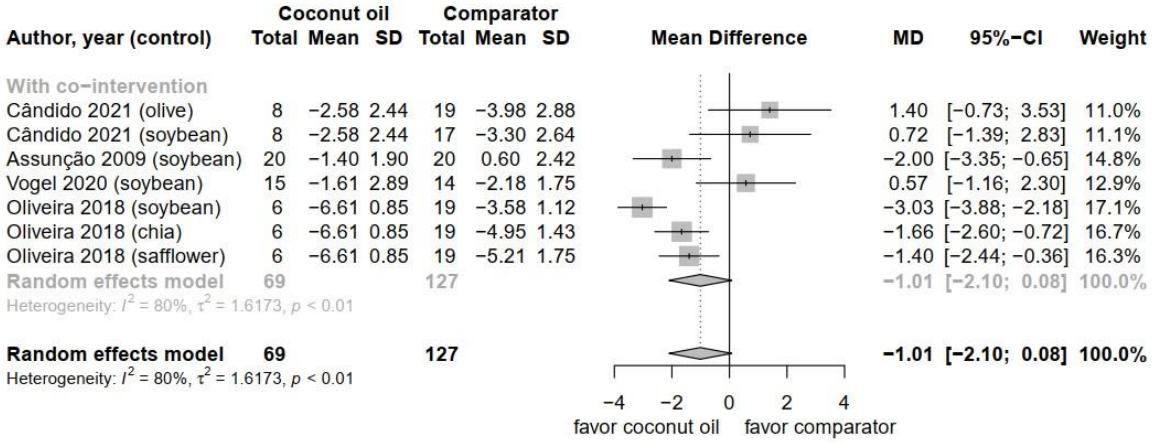
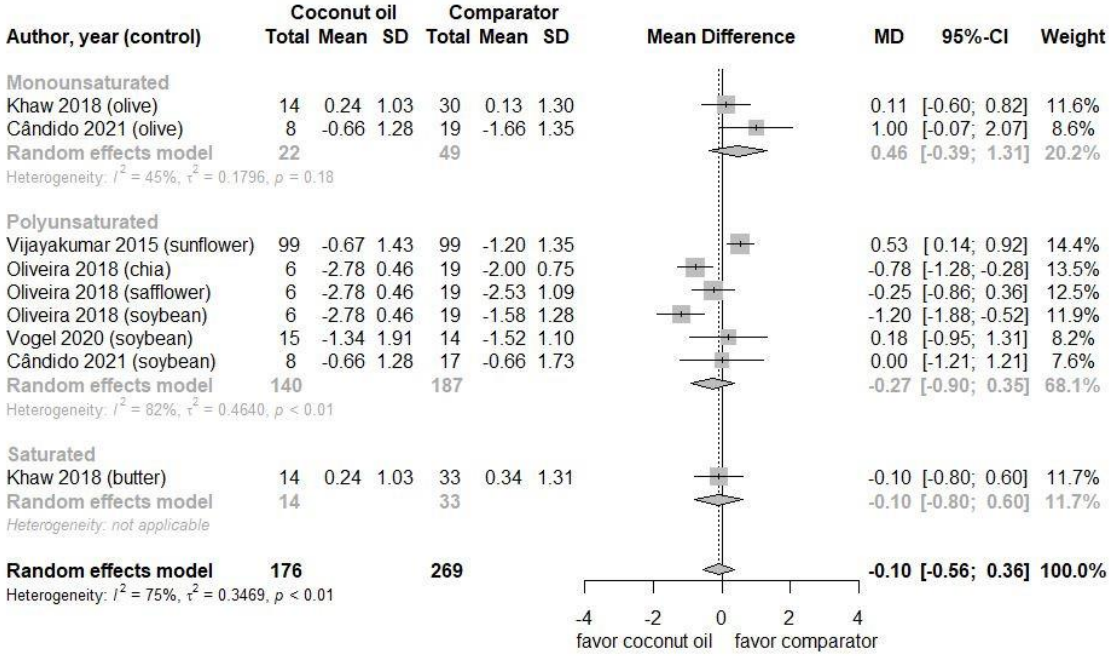


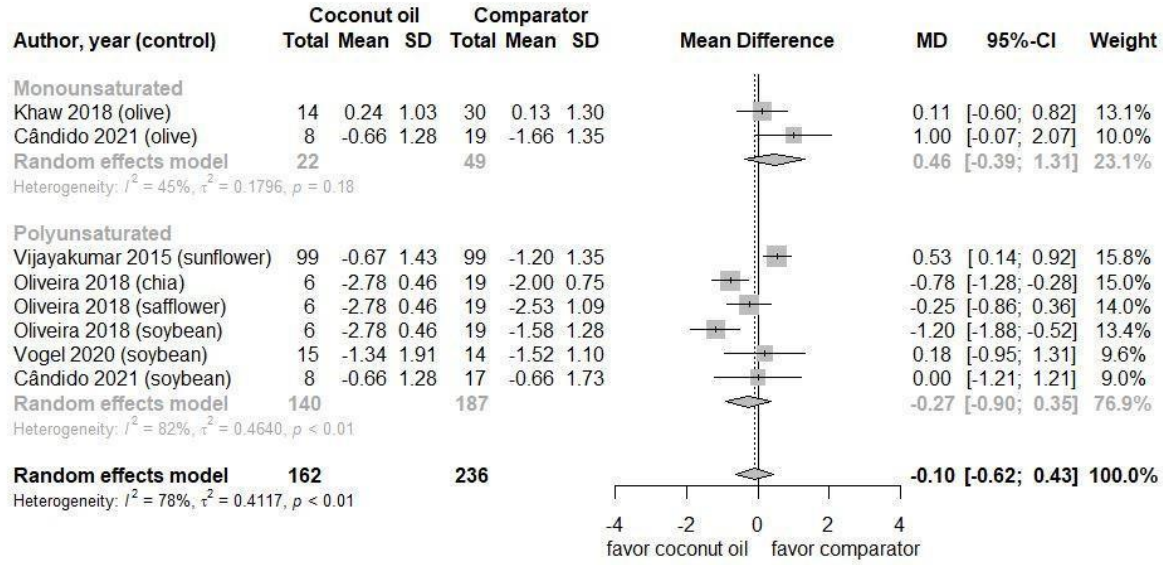
Figure S15 - Forest plot of randomized controlled clinical trials investigating the effect in waist circumference (cm) of coconut oil versus other oils/fats in studies including co-intervention



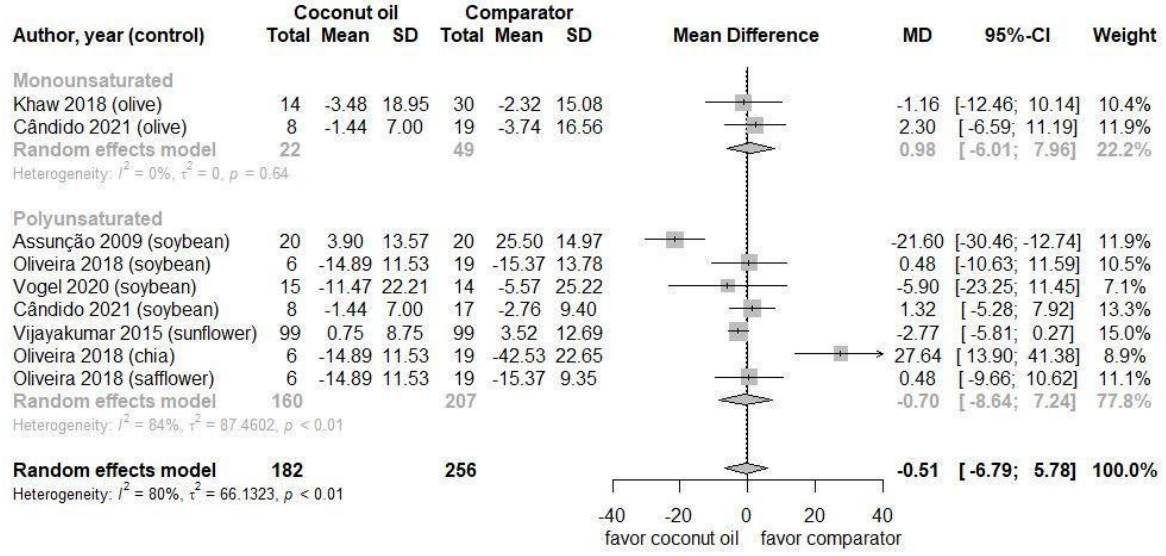
Supplemental Figure 16. Forest plot of the randomized controlled clinical trials investigating the effects in % body fat of coconut oil intake in comparison to other oils/fat



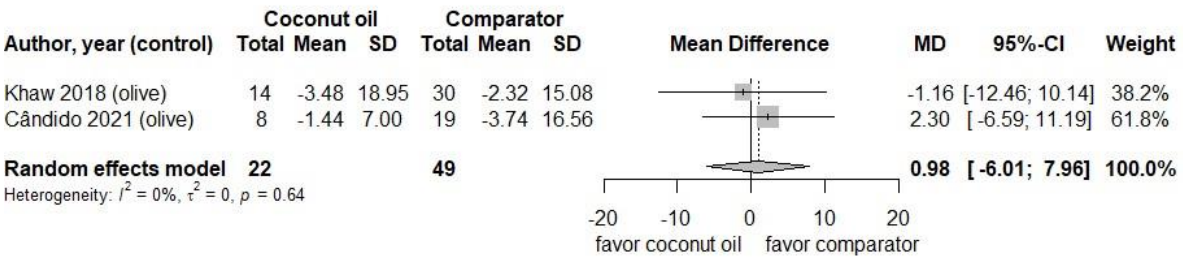
Supplemental Figure S17. Forest plot of randomized controlled clinical trials investigating the effect in % body fat of coconut oil intake vs PUFA and MUFA rich oils



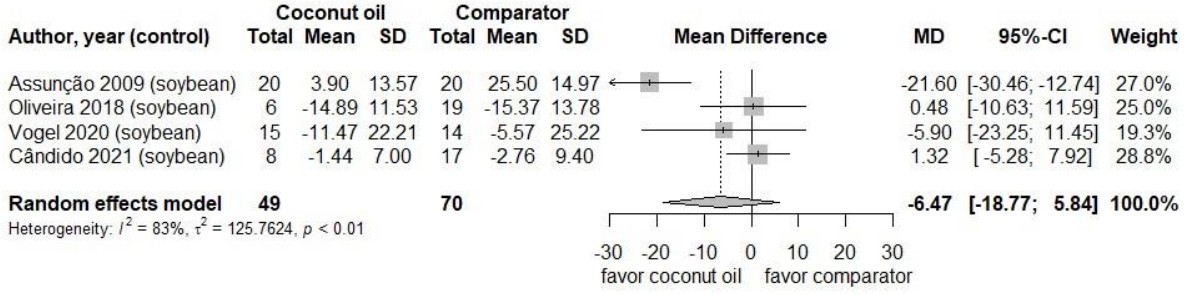
Supplemental Figure 18. Forest plot of randomized controlled clinical trials investigating the effects in LDL-C (mg/dL) of coconut oil intake vs PUFA and MUFA rich oils



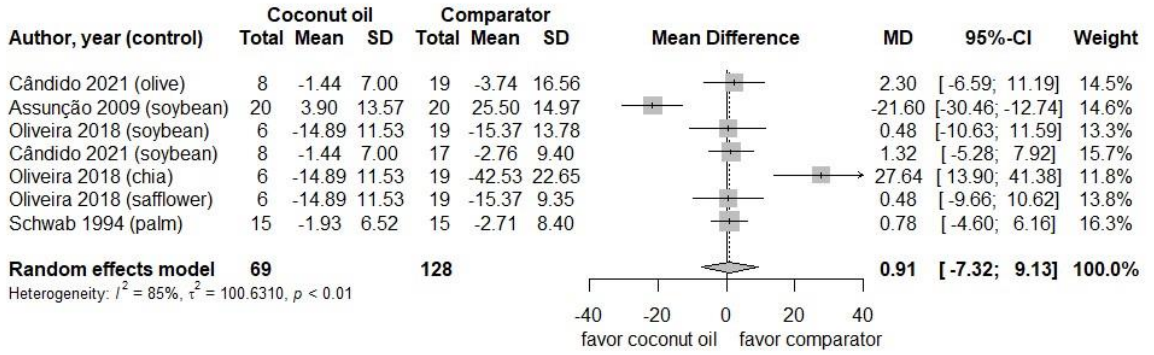
Supplemental Figure 19. Forest plot of randomized controlled clinical trials investigating the effect in LDL-C (mg/dL) of coconut oil versus olive oil



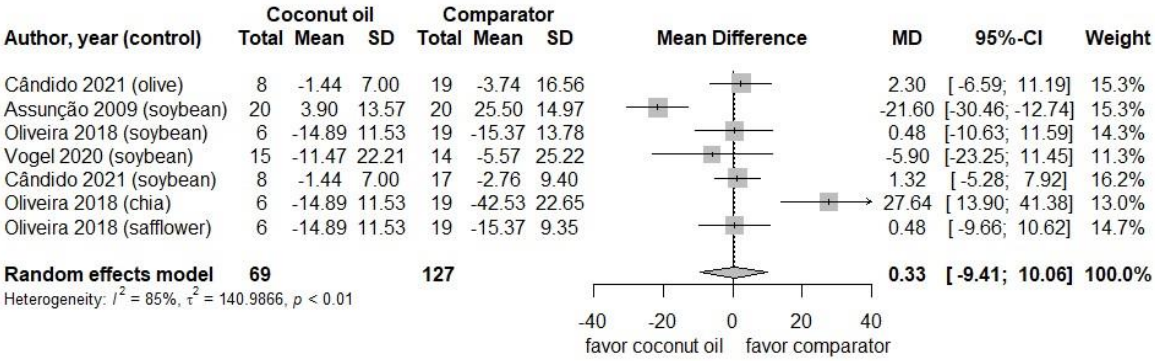
Supplemental Figure 20. Forest plot of randomized controlled clinical trials investigating the effect in LDL-C (mg/dL) of coconut oil versus soybean oil



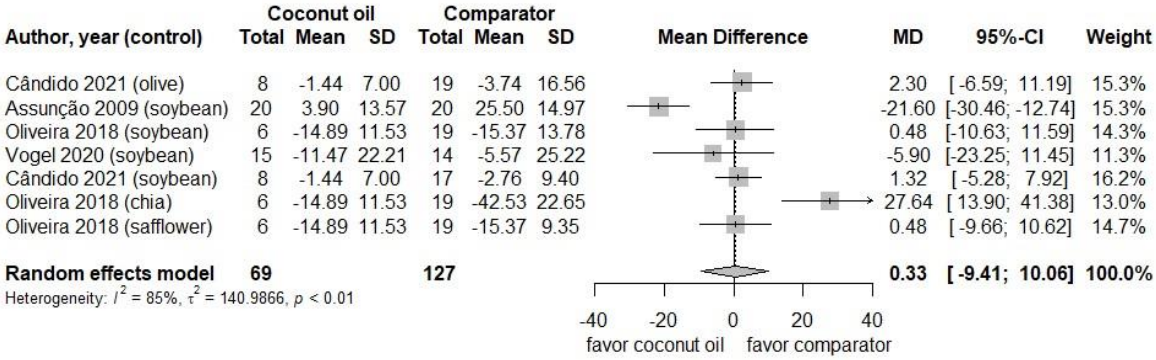
Supplemental Figure 21. Forest plot of randomized controlled clinical trials investigating the effect in LDL-C (mg/dL) of coconut oil versus other oils when analyzing studies carried out in women



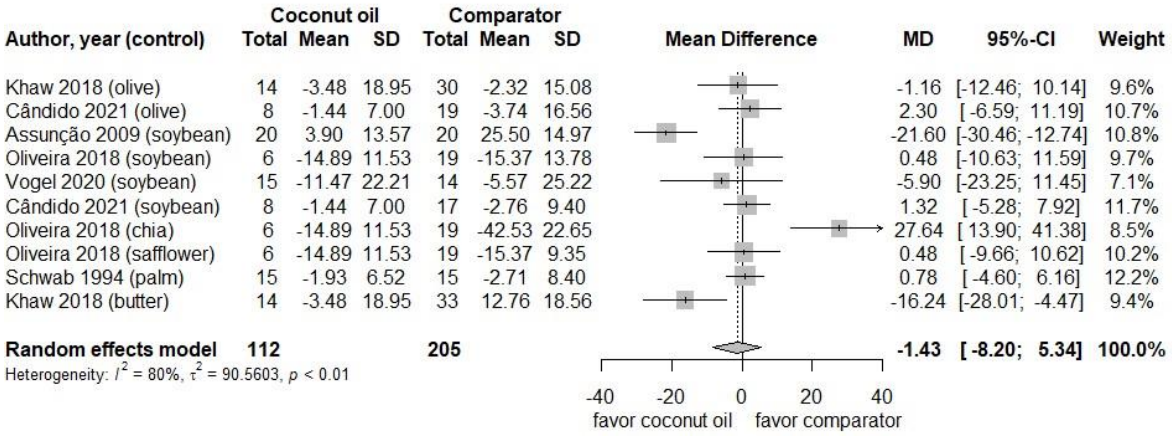
Supplemental Figure 22. Forest plot of randomized controlled clinical trials investigating the effect in LDL-C (mg/dL) of coconut oil versus other oils when analyzing studies conducted in Brazil in LDL-C



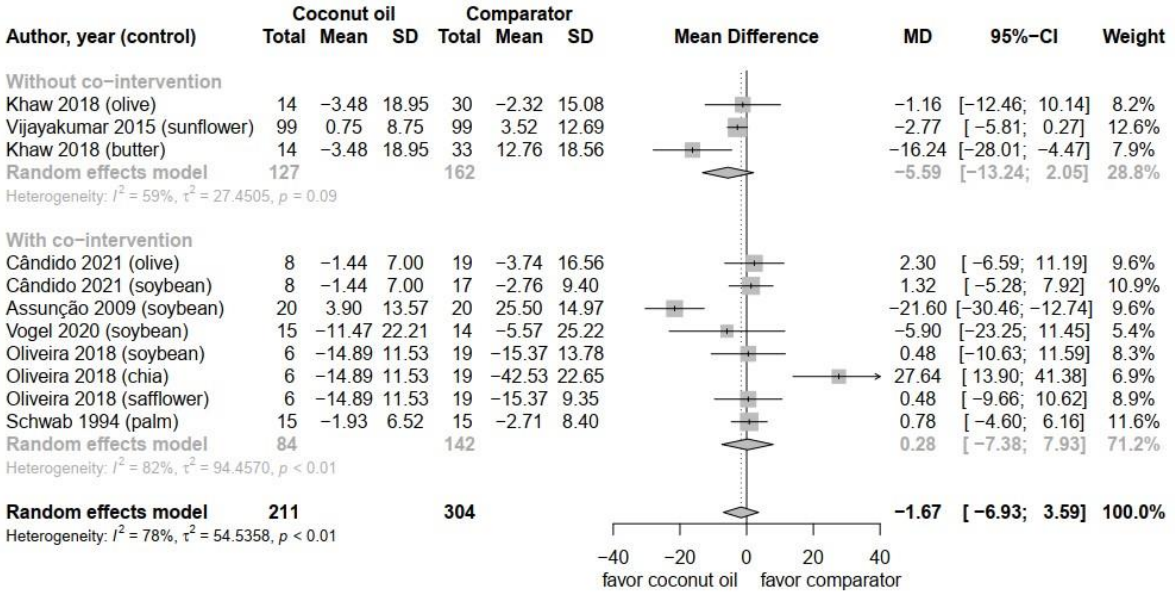
Supplemental Figure 23. Forest plot of randomized controlled clinical trials investigating the effect in LDL-C (mg/dL). of coconut oil versus other oils or/fat in patients with overweight/obesity



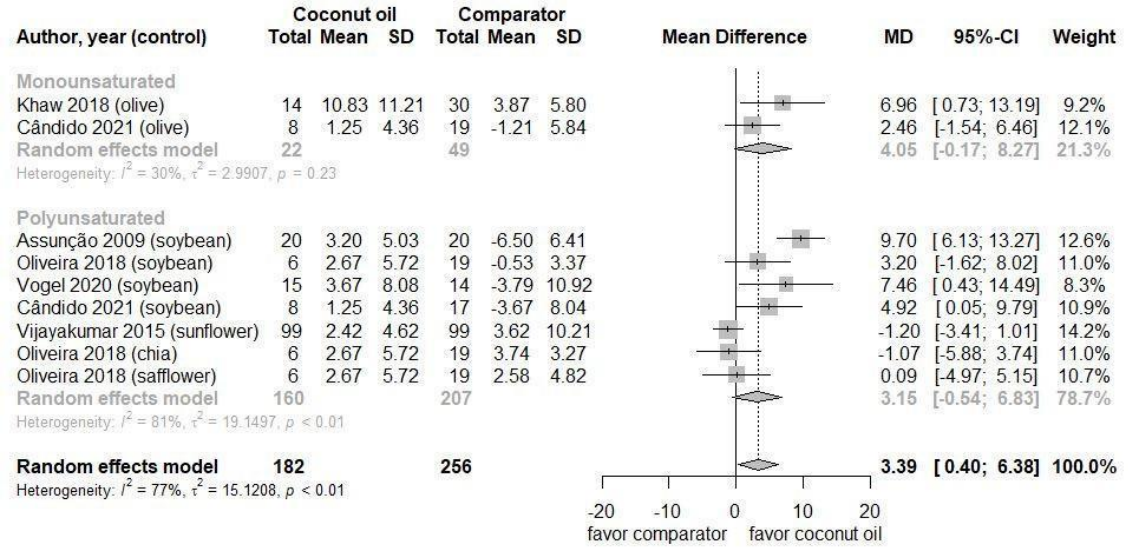
Supplemental Figure 24. Forest plot of randomized controlled clinical trials investigating the effect in LDL-C (mg/dL) of coconut oil versus other oils or fat without a long-term study (Vijayakumar et al)



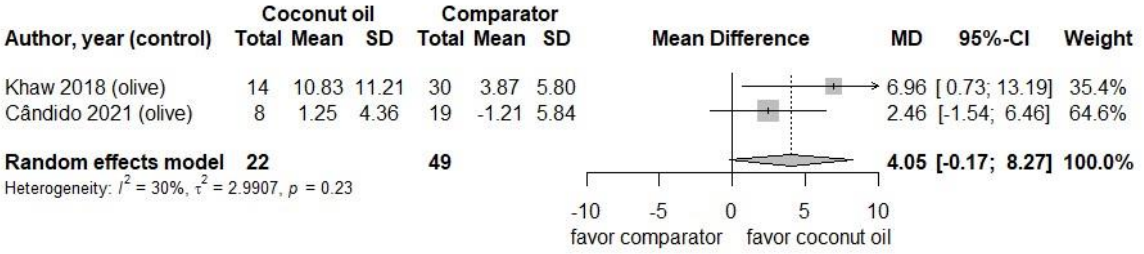
Supplemental Figure 25. Forest plot of randomized controlled clinical trials investigating the effect in LDL-C (mg/dL) of coconut oil versus other oils or fat with co-intervention



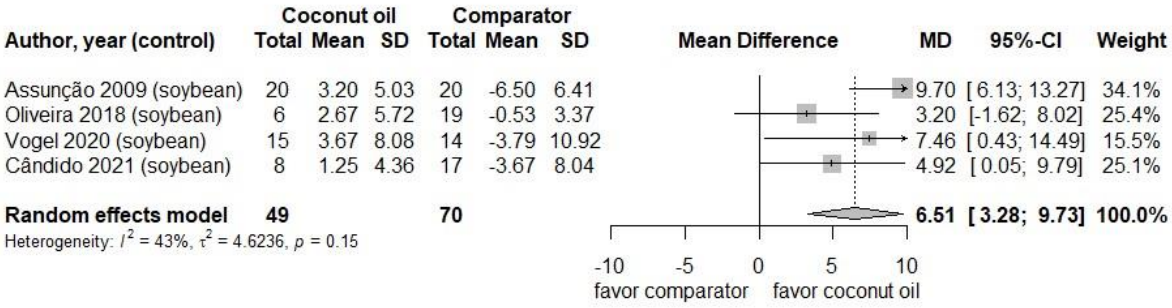
Supplemental Figure 26. Forest plot of randomized controlled clinical trials investigating the effects in HDL-C (mg/dL) of coconut oil intake vs PUFA and MUFA rich oils



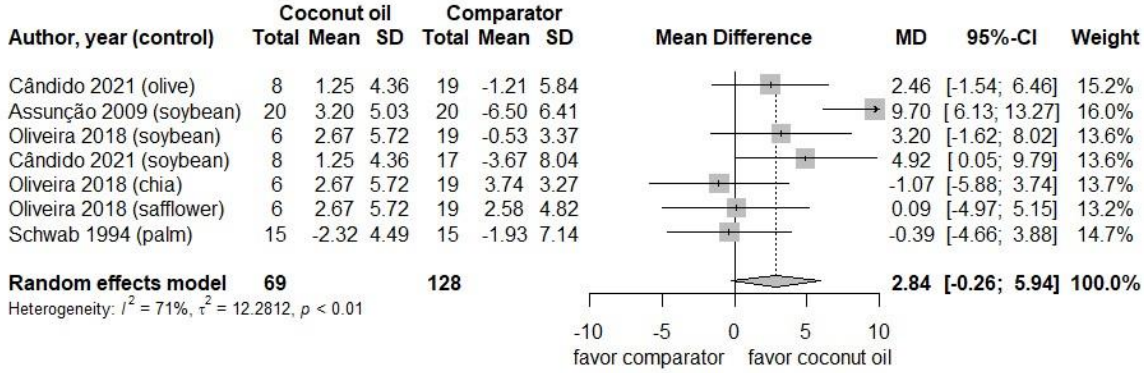
Supplemental Figure 27. Forest plot of randomized controlled clinical trials investigating the effect in HDL-C (mg/dL) of coconut oil versus olive oil



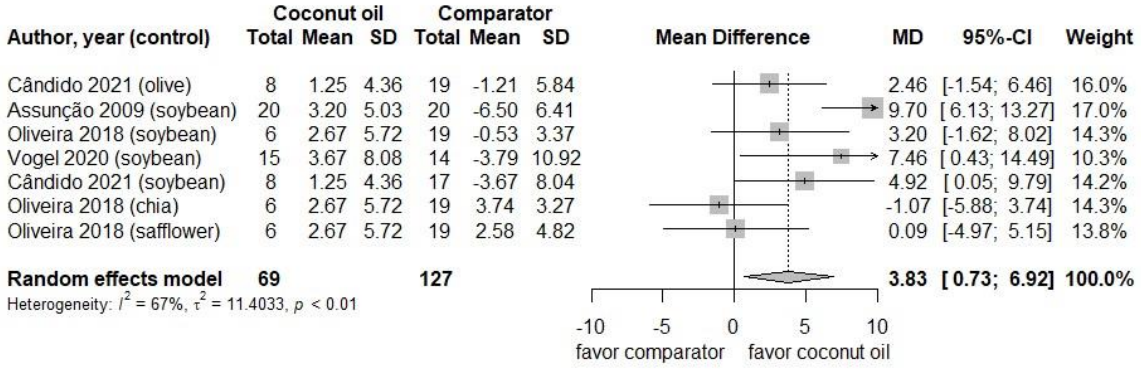
Supplemental Figure 28. Forest plot of randomized controlled clinical trials investigating the effect in HDL-C (mg/dL) of coconut oil versus soybean oil



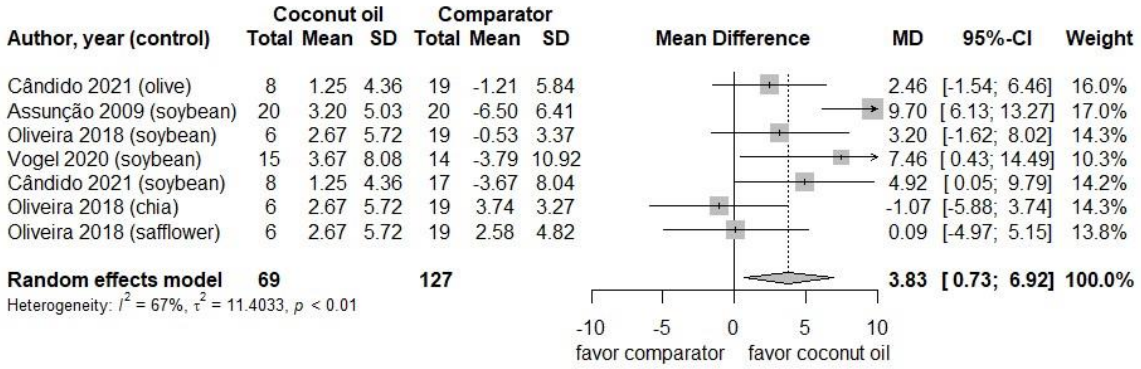
Supplemental Figure 29. Forest plot of randomized controlled clinical trials investigating the effect in HDL-C (mg/dL) of coconut oil versus other oils when analyzing studies carried out in women



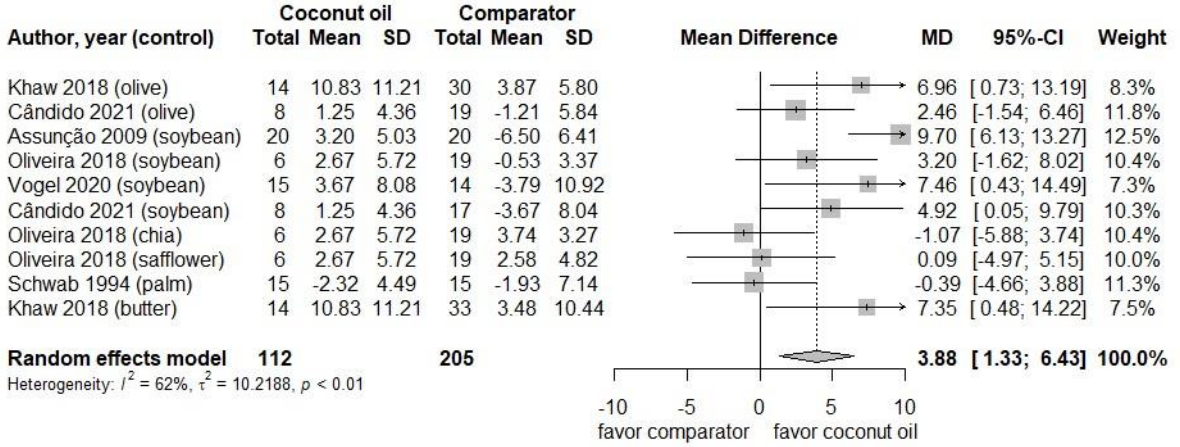
Supplemental Figure 30. Forest plot of randomized controlled clinical trials investigating the effect in HDL-C (mg/dL) of coconut oil versus other oils when analyzing studies conducted in Brazil



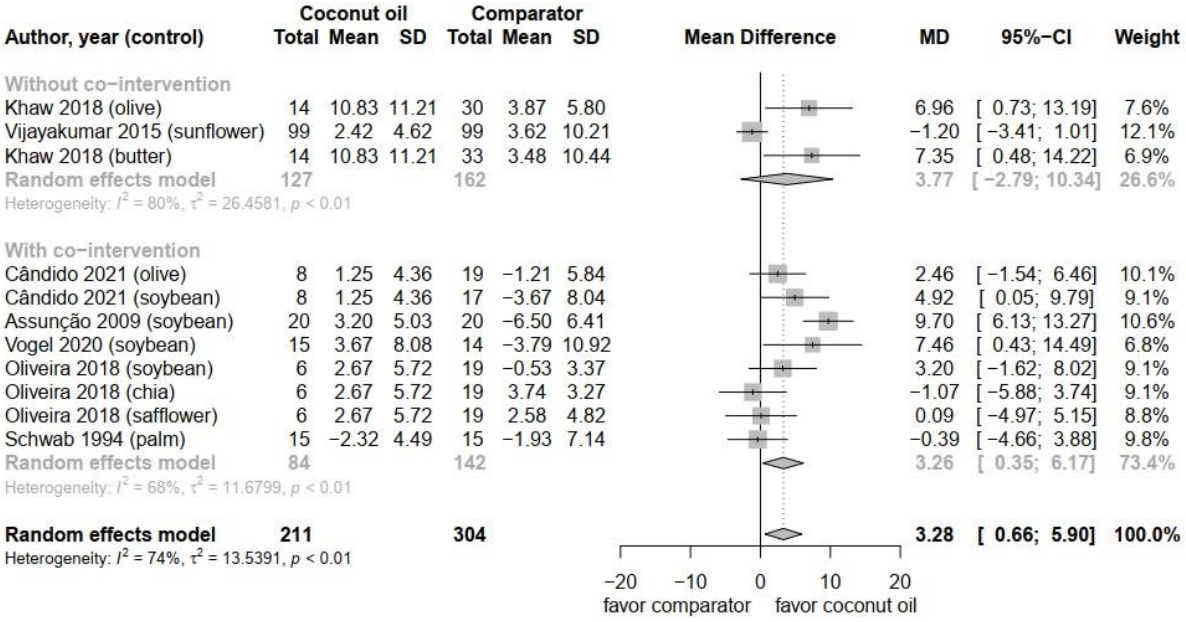
Supplemental Figure 31. Forest plot of randomized controlled clinical trials investigating the effect in HDL-C (mg/dL) of coconut oil versus other oils or fat in patients with overweight/obesity



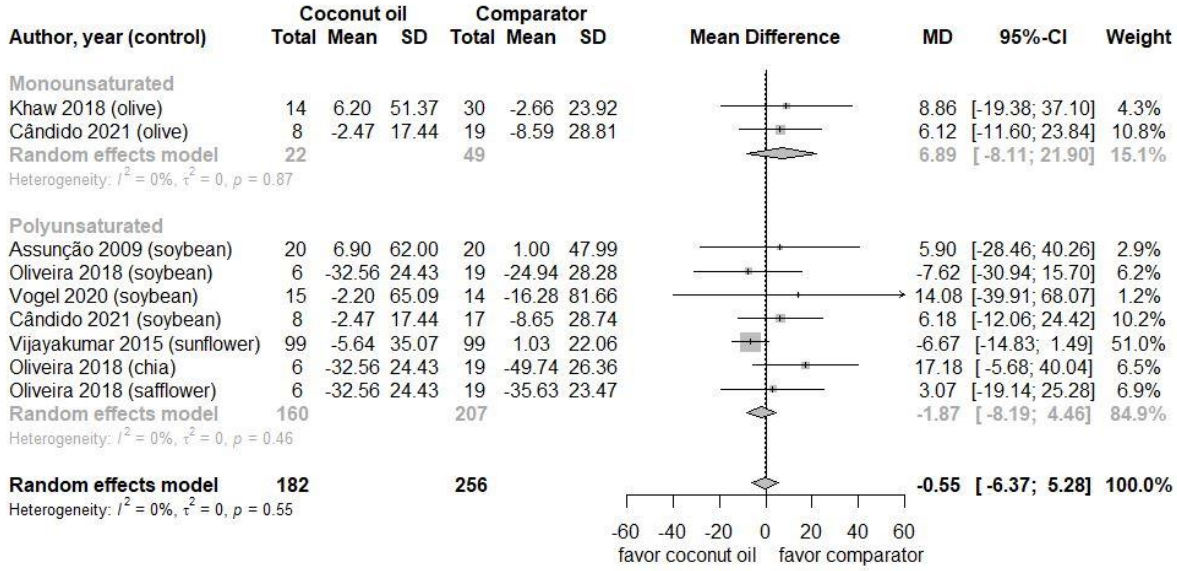
Supplemental Figure 32. Forest plot of randomized controlled clinical trials investigating the effect in HDL-C (mg/dL) of coconut oil versus other oils or fat without a long-term study (Vijayakumar et al)



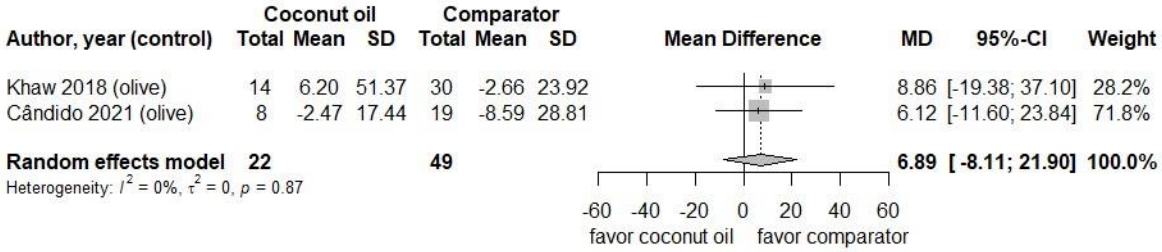
Supplemental Figure 33. Forest plot of randomized controlled clinical trials investigating the effect in HDL-C (mg/dL) of coconut oil versus other oils or fat with co-intervention



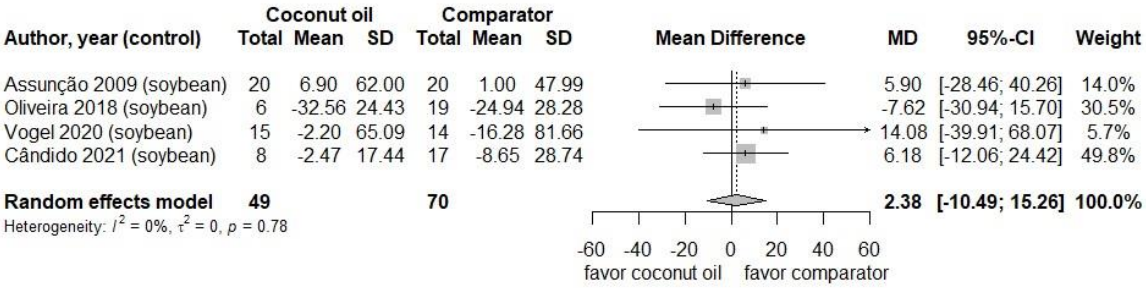
Supplemental Figure 34. Forest plot of randomized controlled clinical trials investigating the effects in TG (mg/dL) of coconut oil intake vs PUFA and MUFA rich oils



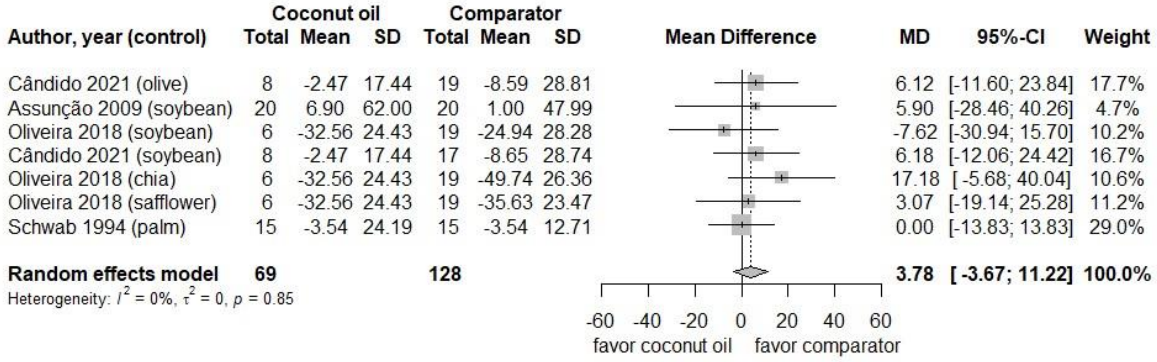
Supplemental Figure 35. Forest plot of randomized controlled clinical trials investigating the effect in TG (mg/dL) of coconut oil versus olive oil



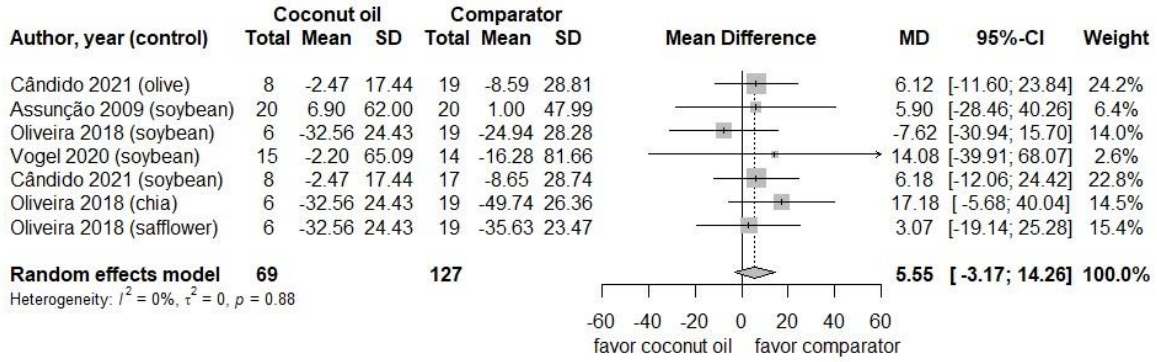
Supplemental Figure 36. Forest plot of randomized controlled clinical trials investigating the effect in TG (mg/dL) of coconut oil versus soybean oil



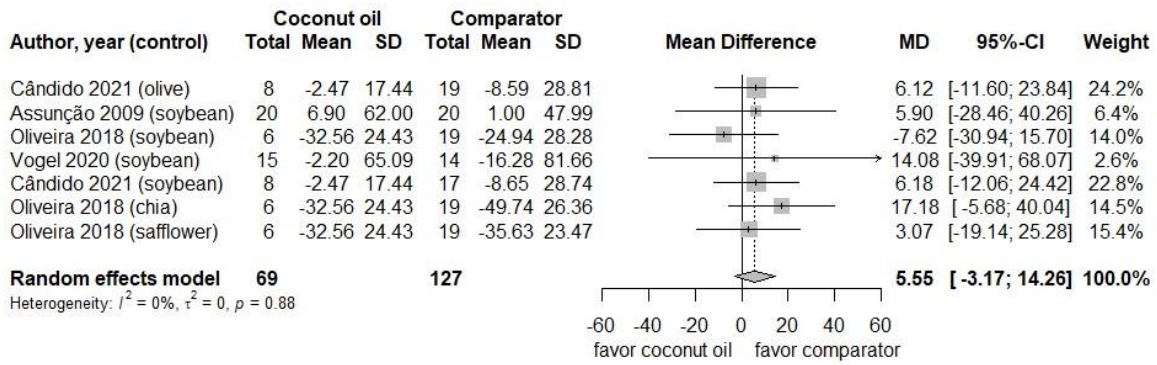
Supplemental Figure 37. Forest plot of randomized controlled clinical trials investigating the effect in TG (mg/dL) of coconut oil versus other oils when analyzing studies carried out in women



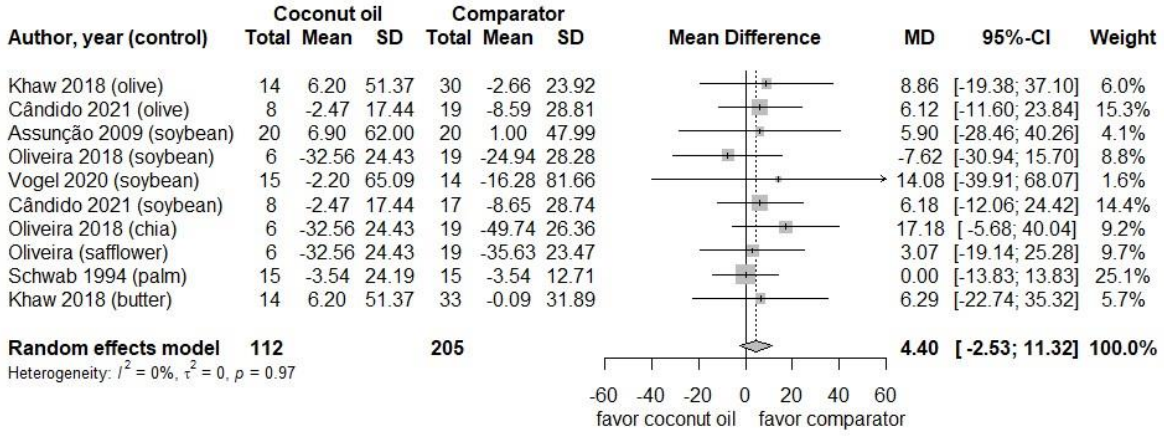
Supplemental Figure 38. Forest plot of randomized controlled clinical trials investigating the effect in TG (mg/dL) of coconut oil versus other oils when analyzing studies conducted in Brazil



Supplemental Figure 39. Forest plot of randomized controlled clinical trials investigating the effect in TG (mg/dL) of coconut oil versus other oils or fat in patients with overweight/obesity



Supplemental Figure 40. Forest plot of randomized controlled clinical trials investigating the effect in TG (mg/dL) of coconut oil versus other oils or fat without a long-term study (Vijayakumar et al)



Supplemental Figure 41. Forest plot of randomized controlled clinical trials investigating the effect in TG (mg/dL) of coconut oil versus other oils or fat with co-intervention

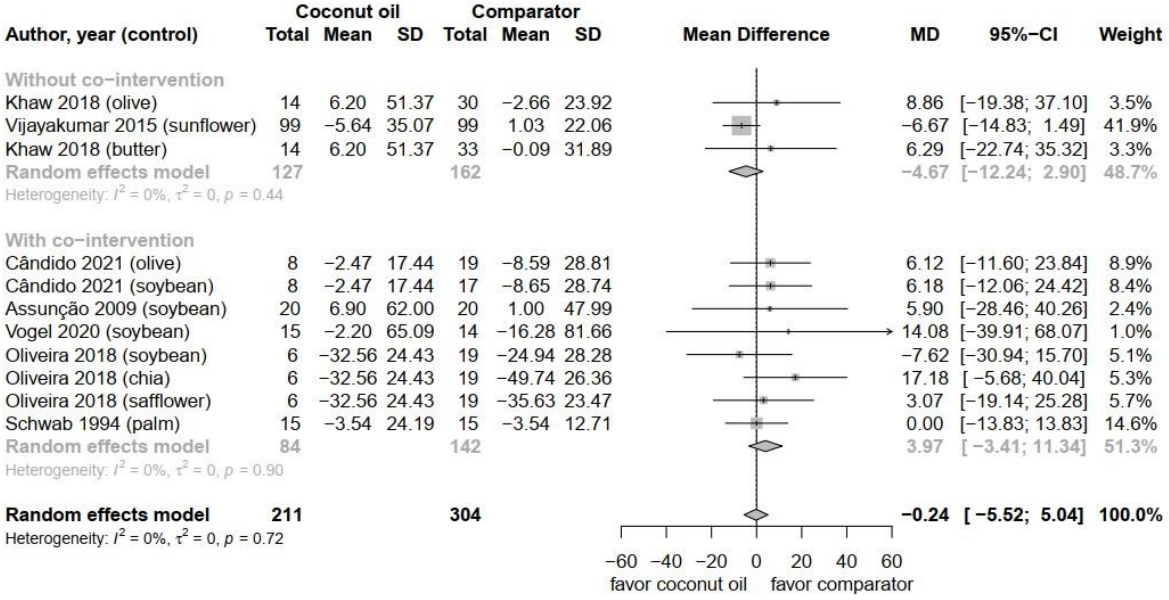


Figure S42: RoB 2.0 risk of bias in RCTs assessing the effects of coconut oil in the lipid profile

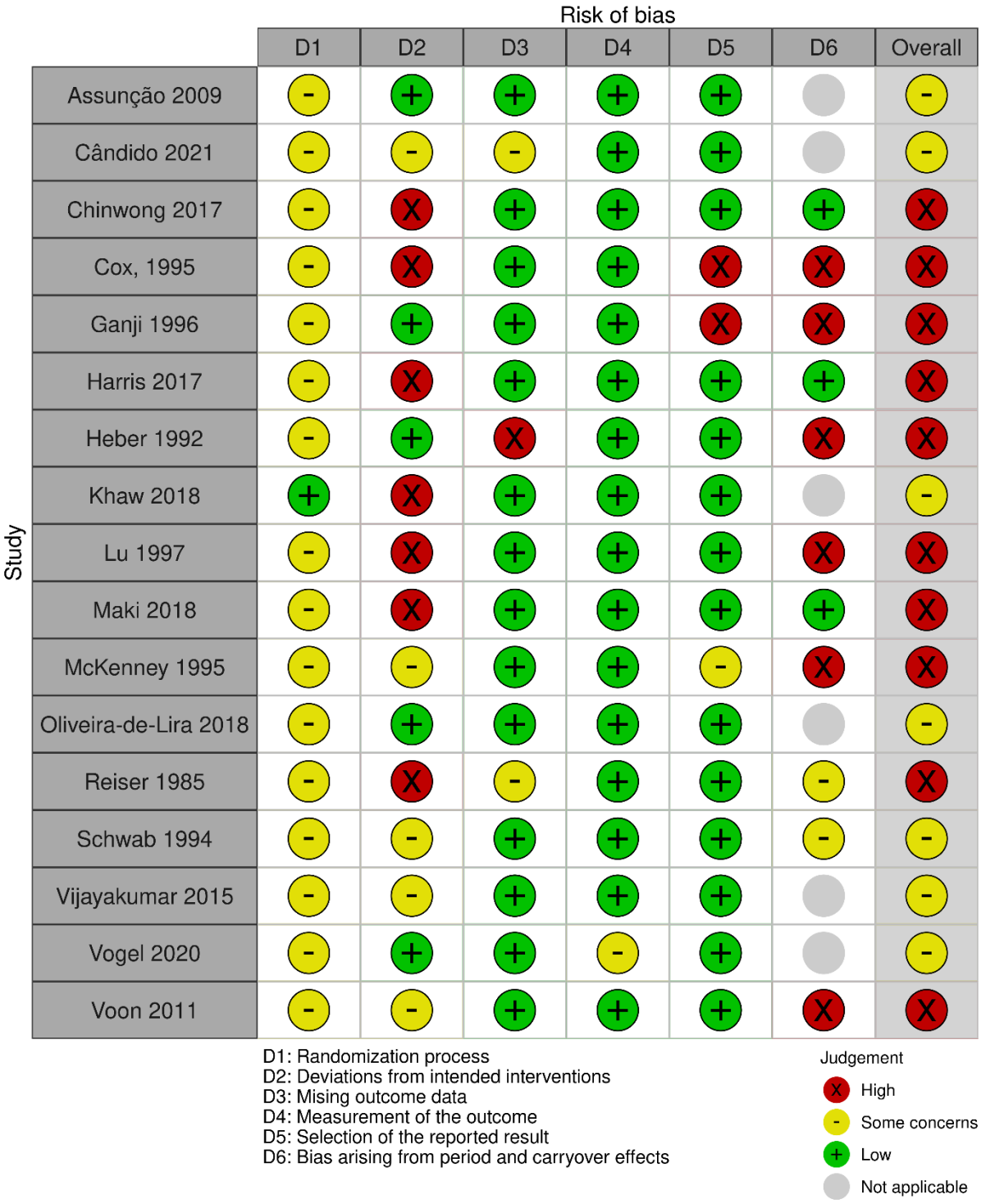


Figure S43: RoB 2.0 risk of bias in RCTs assessing the effects of coconut oil in the anthropometric profile

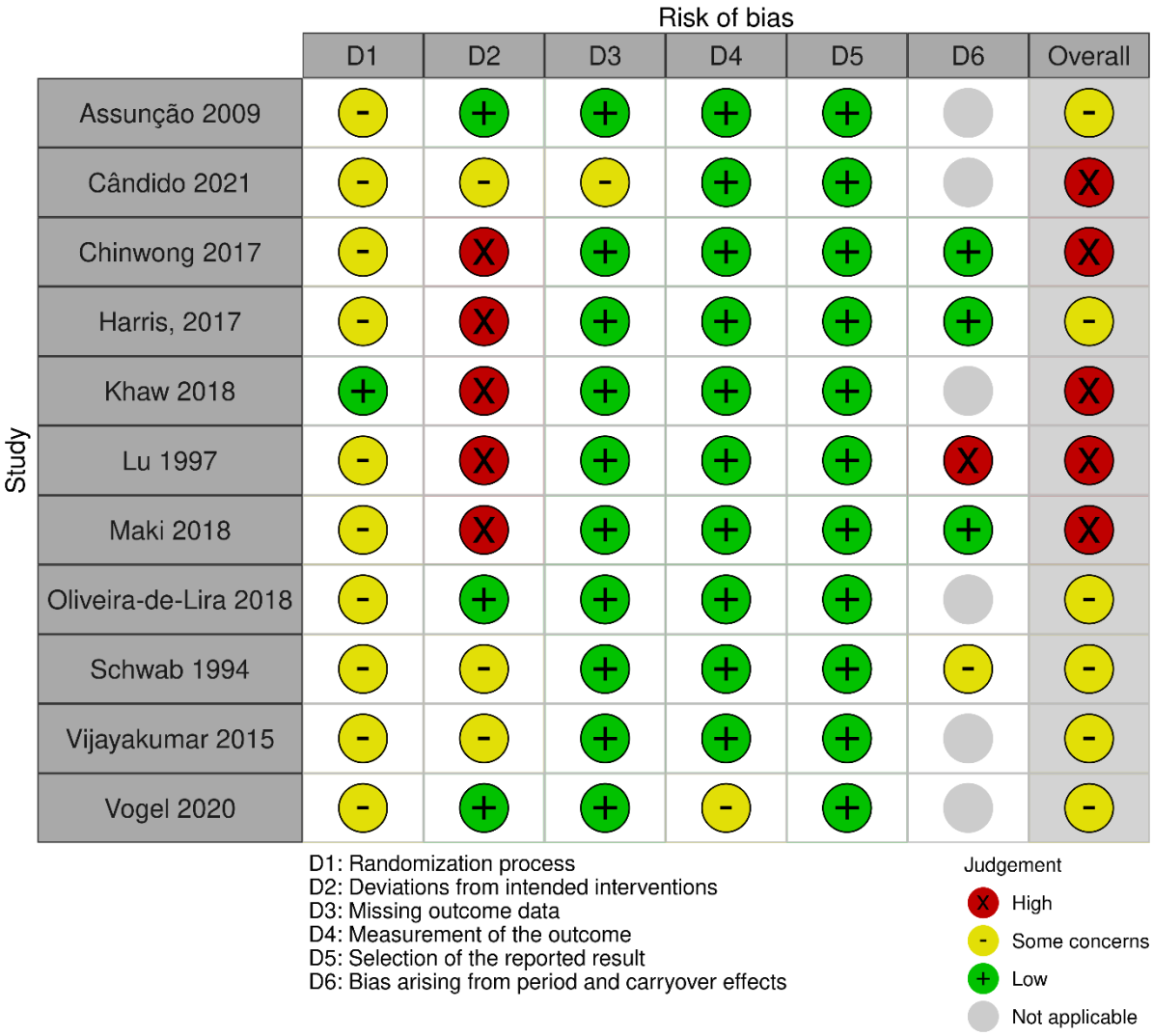


Figure S44: RoB 2.0 risk of bias in RCTs assessing the effects of coconut oil in the glycemic profile

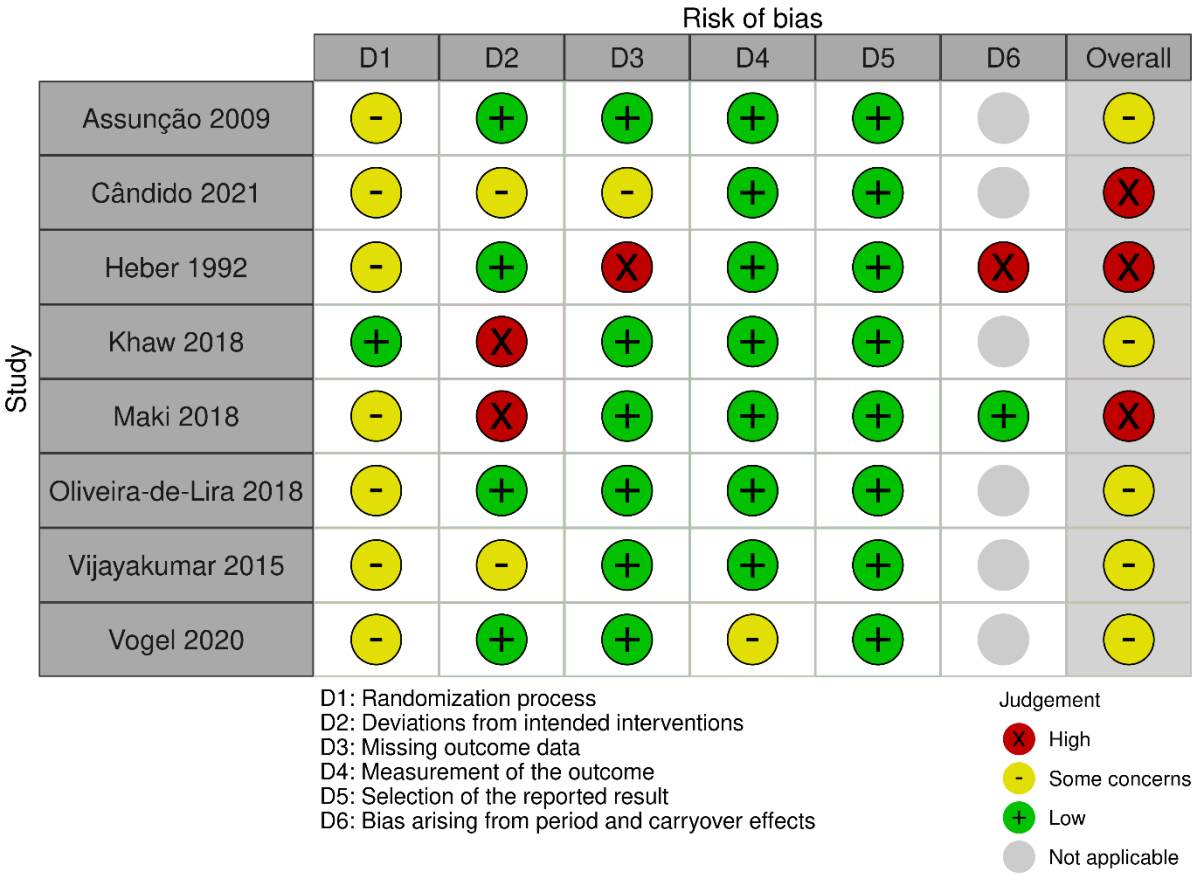


Figure S45: RoB 2.0 risk of bias in RCTs assessing the effects of coconut oil in blood pressure

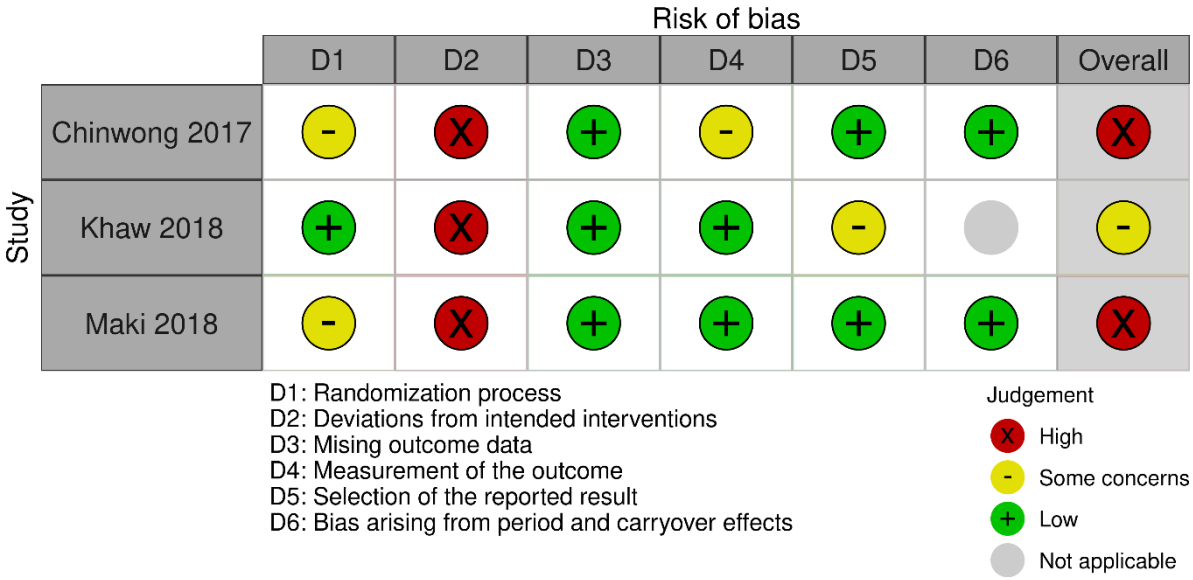
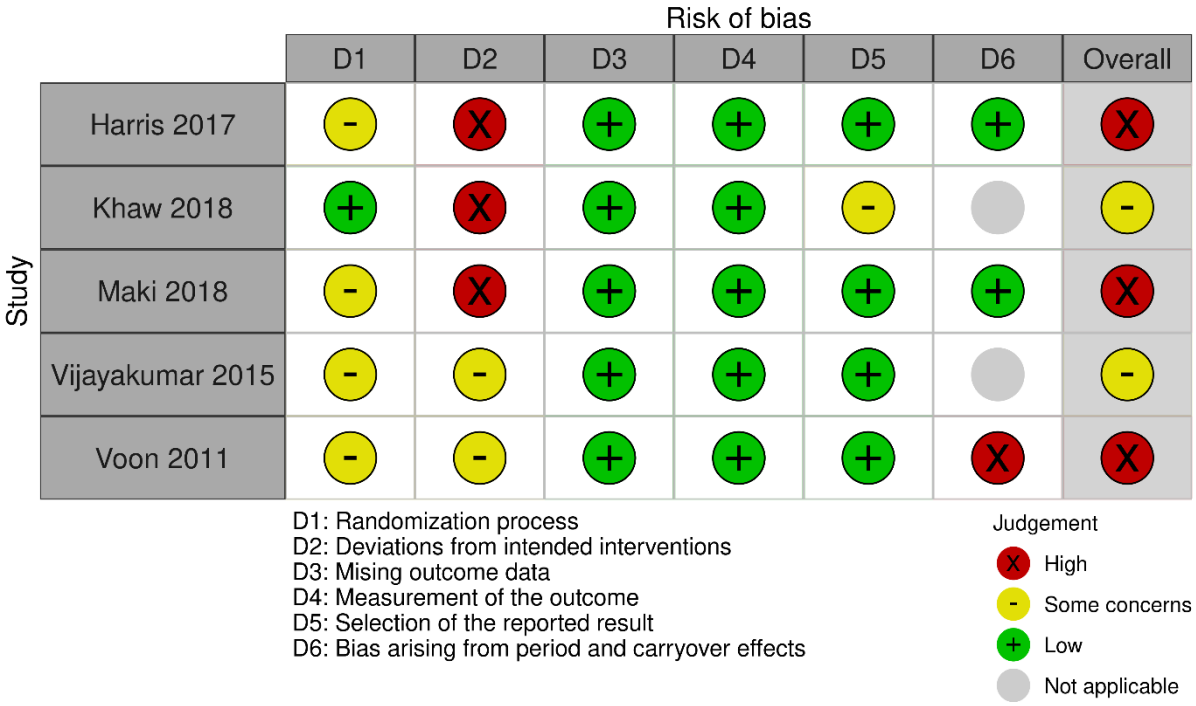


Figure S46: RoB 2.0 risk of bias in RCTs assessing the effects of coconut oil in the inflammatory profile



PRISMA 2020 CHECKLIST

Section and Topic	Item #	Checklist item	Location where item is reported
TITLE			
Title	1	Identify the report as a systematic review.	Pg. 1
ABSTRACT			
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	Pg. 2, 3
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	Pg. 4, 5
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	Pg. 5
METHODS			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	Pg. 7, 8
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	Pg. 6
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	Pg. 6
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	Pg. 6
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	Pg. 6
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	Pg. 6, 7
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	Pg. 6, 7
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	Pg. 7
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	Pg. 8
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	Pg. 7
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data	Pg. 8

Section and Topic	Item #	Checklist item	Location where item is reported
		conversions.	
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	Pg. 8
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	Pg. 8
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	Pg. 9
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	Pg. 9
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	Pg. 7
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	Pg. 7, 8
RESULTS			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	Pg. 9 and fig. 1
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	Figure 1 and Table S1
Study characteristics	17	Cite each included study and present its characteristics.	Pg. 9, 10 and table 1
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	Pg. 15 and supplementary material
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	Figures 2 and 3
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	Pg. 10-15
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	Figures 2 and 3
	20c	Present results of all investigations of possible causes of heterogeneity among study results.	Pg. 9
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	Pg. 9-15
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	Pg. 15 and Figures S42-S46
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	Pg. 15 and Table S7
DISCUSSION			

Section and Topic	Item #	Checklist item	Location where item is reported
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	Pg. 15, 16
	23b	Discuss any limitations of the evidence included in the review.	Pg. 15-21
	23c	Discuss any limitations of the review processes used.	Pg. 20, 21
	23d	Discuss implications of the results for practice, policy, and future research.	Pg. 21
OTHER INFORMATION			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	Pg. 3
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	Pg. 3
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	NA
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	Pg. 23
Competing interests	26	Declare any competing interests of review authors.	Pg. 23
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	NA

REFERENCES

[1] Lu Z, Hendrich S, Shen N, White PJ, Cook LR. Low linolenate and commercial soybean oils diminish serum HDL cholesterol in young free-living adult females. J Am Coll Nutr.1997;16(6):562-9.
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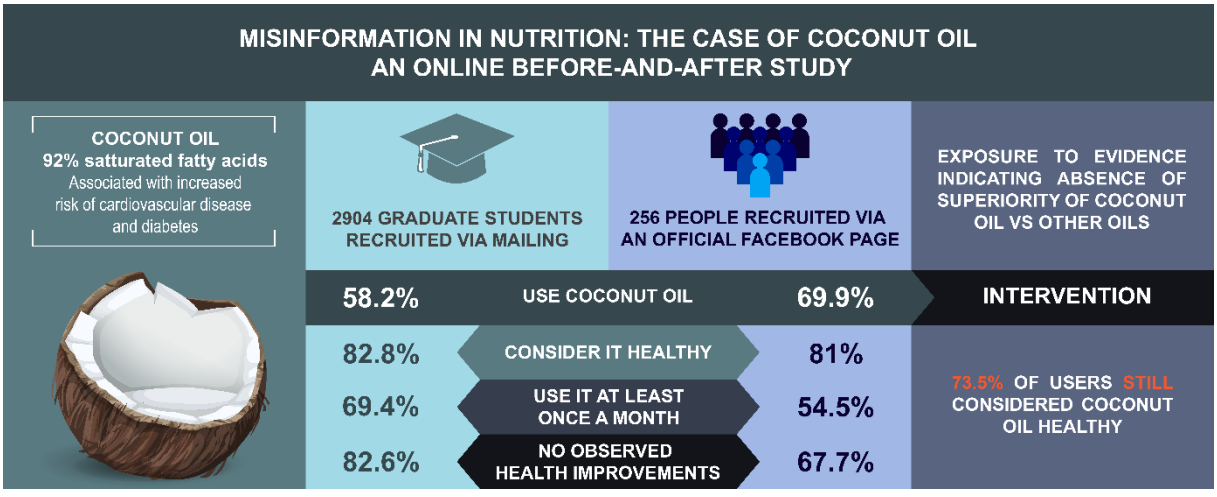
Anexo II – Material suplementar do Capítulo III – **"“Misinformation in nutrition through the case of coconut oil: an online before-and-after study”.**

Figure 1. Grafic abstract



SUPPLEMENTARY METHODS:

CHECKLIST FOR REPORTING RESULTS OF INTERNET E-SURVEYS (CHERRIES)

Item Category	Checklist Item	Description
Design	Describe survey design	The study targeted two populations of self-selected individuals aged 18 years or older: 1) University sample: students from graduate programs at Universidade Federal do Rio Grande do Sul, Porto Alegre, Brazil. 2) Facebook sample: individuals who accessed the Facebook page of Hospital de Clínicas de Porto Alegre (the main teaching hospital from the same university).
IRB (Institutional Review Board) approval and informed consent process	IRB approval	The study was approved by the Research Ethics Committee of Hospital de Clínicas de Porto Alegre and Universidade Federal do Rio Grande do Sul (approval number: GPPG-HCPA protocol 20180393 nos and CAAE nos 92144718.6.0000.5327).
	Informed consent	Participants were informed, at the study home page, that by submitting the questionnaire, they would be accepting to voluntarily participate in the survey. In the study home page, participants were informed that their

		participation would be anonymous, taking approximately 2 to 3 minutes to complete the survey. The home page also provided information about the study objective and the name and contact information of the principal investigator.
	Data protection	The only personal information requested was a valid email address, which would be only used to prevent multiple entries and to provide study results to the participants who requested it. The datasets were stored in a password protected Google Drive account, to which only the main investigators had access.
Development and pre-testing	Development and testing	The questionnaire was constructed using Google Forms, initially consisting of 13 objective questions to evaluate coconut oil consumption and public opinion on its effects on metabolic and cardiovascular health. We submitted the questionnaire to major experts in the field of metabolism and mental health as well as clinical dieticians developing research projects in the field. After changes of the constructs composing the questionnaire, a second round of submission was conducted to graduate students in order to provide feedback changes for the final training version of the questionnaire, resulting in a new version with 12 questions. The initial questionnaire was then pilot tested in 51

		individuals in Brazil (Portuguese version), India and the United States (English version) using Facebook advertisement as a way of dissemination. Based on the results of the pilot test, modifications were performed to improve question wording, response items and comprehension. The final questionnaire consisted of 10 sequential objective questions.
Recruitment process and description of the sample having access to the questionnaire	Open survey versus closed survey	This is an open survey. Distribution to University sample was made via email with an exclusive link using the mailing list of the university, while the distribution to Facebook sample was made in a post in the Hospital de Clínicas de Porto Alegre official Facebook page, containing a separate link to a Google Forms questionnaire.
	Contact mode	The initial contact with the potential participants was made entirely on the Internet. As stated above, the survey was distributed via email with a link to Google Forms to the University sample and via a Facebook post with a link to Google Forms to the Facebook sample.
	Advertising the survey	As previously described, the survey was distributed using the university's graduate student mailing list (University sample) and using a Facebook post in the Hospital de Clínicas de Porto Alegre official Facebook page (Facebook sample).

Survey administration	Web/Email	This is a web survey. Although the method of distribution to the University sample was via email, the email contained a link which led to a Google Forms website. All responses were captured automatically through Google Forms.
	Context	The mailing list (University sample) is composed of emails of students of all level graduate programs. The Facebook page of Hospital de Clínicas de Porto Alegre (Facebook sample) is the official Facebook page of the hospital, which posts updates about the hospital's actions in public health matters, innovations in research made by the hospital, calls for actions of the community, and health promotion campaigns.
	Mandatory/voluntary	It was a voluntary survey.
	Incentives	We offered to provide the survey results to those who requested it by checking an item on the questionnaire.
	Time/Date	Responses were collected between May and June 2020.
	Randomization of items or questionnaires	The items on the questionnaire were not randomized to follow a logical sequential order.

	Adaptive questioning	Participants who answered “yes” to the question “Have you ever used coconut oil” answered all the 10 questions, while participants who answered “no” to the same question answered only 7 questions.
	Number of Items	There was one questionnaire item per page, comprising a total of 11 items (10 questions and email item).
	Number of screens (pages)	The full survey was distributed in 12 pages (home page, 10 questions, email page).
	Completeness check	In each question, the participants had the option of clicking on the “back” button to change their answers. Respondents were unable to change their responses once the full questionnaire was submitted.
	Review step	In each question, respondents had the option of clicking on the “back” button to change their answers. Respondents were unable to change their responses once the full questionnaire was submitted.
Response rates	Unique site visitor	Not applicable. Respondents were invited through an external link. Unique respondents were determined using the provided valid email address, as described in the item “Registration” below.
	View rate (Ratio of unique survey visitors/unique site visitors)	Not applicable. Google forms does not provide the number of site or survey visitors.

	Participation rate (Ratio of unique visitors who agreed to participate/unique first survey page visitors)	For University sample, we determined the participation rate by dividing the number of entries (3582) by the number of emails registered in the mailing list (11753) resulting in a participation rate of 30.5%. Not applicable for Facebook sample as Google forms does not provide the number of survey visitors nor stores information of incomplete questionnaires.
	Completion rate (Ratio of users who finished the survey/users who agreed to participate)	Not applicable; Google Forms only stores complete surveys, after the respondent submitted it through the “send” button.
Preventing multiple entries from the same individual	Cookies used	Not used.
	IP check	Not used.
	Log file analysis	Not used.
	Registration	The questionnaire requested a valid email address which was stored together with the survey results to avoid multiple entries. In the case of duplicate entries, the first entry was kept and the last was excluded from the analysis.
Analysis	Handling of incomplete questionnaires	Not applicable; Google Forms only stores complete surveys, after the respondent submitted it through the “send” button.

	Questionnaires submitted with an atypical timestamp	No respondents were removed from the survey for completing the items too quickly.
	Statistical correction	Not used.

QUESTIONNAIRE DEVELOPMENT

Initial Questionnaire Construction

We constructed an online questionnaire using Google Forms to evaluate coconut oil consumption and the public opinion on its effects on metabolic and cardiovascular health. The initial questionnaire consisted of 13 objective questions. First, we submitted the questionnaire to major experts in the field of metabolism and mental health as well as clinical dieticians developing research projects in the field. After changes of the constructs composing the questionnaire, a second round of submission was conducted to graduate students in order to provide feedback changes for the final training version of the questionnaire, resulting in a new version with 12 questions.

Pilot Test

We tested the training questionnaire using an official Facebook page for the study, targeting adults (age ≥18 years). The training questionnaire consisted of 12 questions, originally formulated in Portuguese. An English version was translated, as, initially, the idea was to apply the same questionnaire in a country on each continent. The Portuguese version was shared in Brazil and an English version was shared in the United States and India (where coconut oil intake is considered significant) [5]. In all, 51 people answered the questionnaire, 26 in Brazil, 19 in India and 5 in the United States. The purpose of the pilot test was to assess understanding of the questions and answer items and to examine questions with invalid or unsatisfactory answers. From the analysis of the answers obtained with this pilot and the refinement of the questions and answers, the official version of the questionnaire was elaborated.

Questionnaire Refinement

Based on the pilot test, we performed new modifications in the wording and response items of the questionnaire in order to make it more comprehensive. The wording of questions with lower response rates was revised. The final questionnaire consisted of 10 sequential objective questions distributed in 12 pages (one question per page, home and email pages), with multiple-choice answers where the participant could mark more than one answer. In each question, the participants had the option of clicking on the “back” button to change their answers. Individuals not using coconut oil were redirected to questions on sample characteristics and the reason why they did not use it, answering only 7 questions. Questions were marked as forced responses whenever possible, to facilitate statistical analysis. For forced-response questions, the option "I prefer not to answer" was included to ensure participants the right not to answer. Since the Google Forms tool only stores information after the participant finishes the questionnaire, we only had access to entries in which the participant formally clicked the "send" button. All questions and responses were examined by the research team to ensure readability and face validity prior to survey administration.

FINAL QUESTIONNAIRE

1. Have you ever used coconut oil?
( ) Yes
( ) No
( ) I don't want to answer

Yes / I don't want to answer

2. Why do you use coconut oil (check as many alternatives as you want)?
( ) I like the flavor
( ) It's the oil most commonly used for cooking
( ) It's cheaper than other oils
( ) It takes longer to spoil
( ) It gives me more energy
( ) It's healthy
( ) I don't want to answer

3. What amount of coconut oil do you intake? Choose the alternative that comes closest.
( ) Less than a tablespoon/day
( ) 1 tablespoon/day
( ) 2 tablespoons/day
( ) 3 tablespoons/day
( ) 1-3×/week
( ) Once every 15 days
( ) 1×/month
( ) I don't want to answer

4. Which benefits did you observe by using coconut oil (check as many alternatives as you want)?
( ) I did not observe any improvements in my health or aesthetic
( ) Weight loss
( ) Reduced waist circumference
( ) Improvement of cholesterol levels
( ) Improvement of glycemic levels
( ) I don't want to answer

5. A current study has reviewed scientific articles and concluded that nutritional consumption of coconut oil does not improve bad cholesterol, reduces weight or blood sugar (glucose) levels. Do you still consider coconut oil good for your health?
( ) Yes
( ) No
( ) I don't want to answer

No

2. Why don't you use coconut oil (check as many alternatives as you want)?
( ) I don't like the flavor
( ) I don't think it is a healthy oil
( ) It's expensive
( ) It's hard to find where I live
( ) I don't want to answer
( ) Other: ____

6. You are:
( ) Female
( ) Male
( ) I don't want to declare

7. Where do you live?
( ) Brazil
( ) United States
( ) United Kingdom
( ) India
( ) Australia
( ) South Africa
( ) Other
( ) I don't want to answer

8. How old are you?
( ) Less than 18 years old
( ) Between 18 and 20 years old
( ) Between 20 and 29 years old
( ) Between 30 and 39 years old
( ) Between 40 and 49 years old
( ) Between 50 and 59 years old
( ) Over 60 years old
( ) I don't want to answer

9. What is your educational level?
( ) Incomplete elementary school
( ) Complete elementary school
( ) Incomplete high school
( ) Complete high school
( ) Incomplete undergraduate
( ) Undergraduate degree
( ) Graduate
( ) I don't want to answer

10. Do you want to receive the results of this survey? If you accept, we will forward the results to the email you inform us.
( ) Yes, I would like to receive the results of this survey
( ) I'm not interested in receiving the results of this survey

11. What is your email address?
____