Proceedings of the Nutrition Society

cambridge.org/pns

The 57th Nutrition Society of New Zealand and 47th Nutrition Society of Australia Joint Annual Scientific Meeting was held at Massey University on 5th–6th December 2023

Conference on 'Nutrition & Wellbeing in Oceania'

Plenary Lecture 5

Review Article

Cite this article: Yap JM, Wall CL, Schultz M, Meredith-Jones K, and Osborne H (2025). The efficacy and feasibility of lifestyle interventions on modifiable cardiovascular disease risk factors among people with inflammatory bowel disease. *Proceedings of the Nutrition Society*, page 1 of 8. doi: 10.1017/S0029665124007596

Received: 11 June 2024
Revised: 17 November 2024
Accepted: 19 November 2024

Keywords:
Inflammatory bowel disease; Diet; Physical activity; Cardiovascular disease

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The efficacy and feasibility of lifestyle interventions on modifiable cardiovascular disease risk factors among people with inflammatory bowel disease

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Abstract

This review aims to highlight the relative importance of cardiovascular disease (CVD) lifestyle-associated risk factors among individuals with inflammatory bowel disease (IBD) and examine the effectiveness of lifestyle interventions to improve these CVD risk factors. Adults with IBD are at higher risk of CVD due to systemic and gut inflammation. Besides that, tobacco smoking, dyslipidaemia, hypertension, obesity, physical inactivity and poor diet can also increase CVD risk. Typical IBD behavioural modification including food avoidance and reduced physical activity, as well as frequent corticosteroid use, can further increase CVD risk. We reviewed seven studies and found that there is insufficient evidence to conclude the effects of diet and/or physical activity interventions on CVD risk outcomes among populations with IBD. However, the limited findings suggest that people with IBD can adhere to a healthy diet or Mediterranean diet (for which there is most evidence) and safely participate in moderately intense aerobic and resistance training to potentially improve anthropometric risk factors. This review highlights the need for more robust controlled trials with larger sample sizes to assess and confirm the effects of lifestyle interventions to mitigate modifiable CVD risk factors among the IBD population.

Introduction

Cardiovascular diseases (CVD) are the leading causes of morbidity and mortality worldwide with ischaemic heart disease and stroke contributing the largest number of deaths[(1)]. It is well known that systemic inflammation plays a role in the pathogenesis and progression of CVD[(2)]. In this context, studies have reported a higher prevalence of CVD among people with inflammatory conditions such as rheumatoid arthritis, systemic lupus erythematosus[(3)] and more recently inflammatory bowel disease (IBD). A meta-analysis of 10 IBD cohort studies found that individuals with IBD have an increased risk of ischaemic heart disease compared to the general population (relative risk (RR): 1.24 (95% confidence interval (CI) 1.14, 1.35)). This was more prominent in female (RR: 1.35; 95% CI: 1.20, 1.51) and younger patients (RR: 1.35; 95% CI: 1.05, 1.73)[(4)]. A subsequent large retrospective study also showed that individuals with IBD are 1.61 (95% CI: 1.34, 1.94) times more likely to experience premature atherosclerotic CVD, diagnosed before the age of 40, than those without IBD[(5)].

IBD is mainly represented by Crohn's disease (CD) and ulcerative colitis (UC). It is a chronic disease characterised by gut and systemic inflammation, with periods of quiescent and active disease. The link between IBD and atherosclerotic CVD is complex as it involves systemic inflammation, vascular endothelial dysfunction, disruption of the intestinal microbial population, increased thrombosis susceptibility and lipid abnormalities[(6)]. Alongside the inflammation, there are a myriad of generic lifestyle-associated risk factors that contribute to the development of CVD (Fig. 1). These include tobacco smoking, dyslipidaemia, hypertension, obesity, physical inactivity and poor diet[(7)]. Typical IBD behavioural modifications to manage symptoms such as restrictive eating and reducing physical activity, might present a barrier to achieving an optimal lifestyle to manage CVD risks[(8–11)]. In addition, the frequent use of IBD medical therapies like corticosteroids may negatively influence lipid profiles, blood pressure, body composition and metabolic regulations that further increase the risk of CVD[(12–17)].

This review aims to summarise the relative importance of CVD lifestyle-associated risk factors among individuals with IBD and evaluate the effectiveness of lifestyle interventions in mitigating these CVD risk factors.

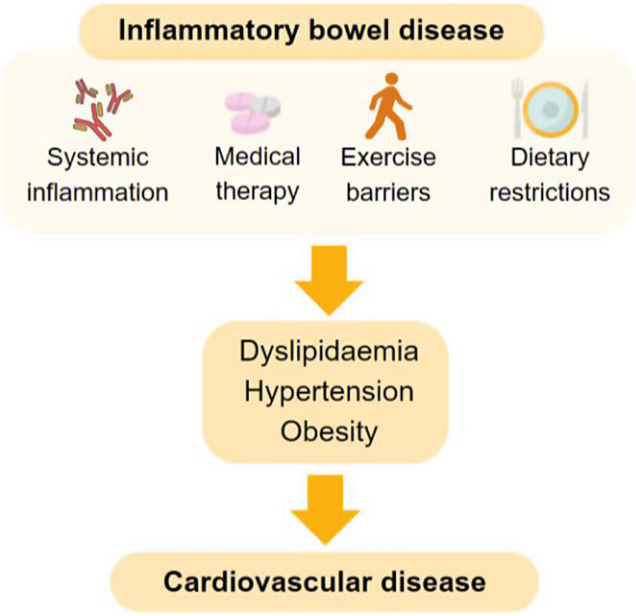

Figure 1. Aspects of inflammatory bowel disease associated with cardiovascular disease.

Modifiable risk factors for CVD

This section describes the existing literature on modifiable CVD risk factors in non-IBD populations and, where available, in IBD cohorts. The influence of diet and physical activity interventions on these risk factors is subsequently reviewed.

Tobacco smoking

Tobacco smoking increases the risk of developing all major CVD events (hazard ratio (HR): 1.63; 95% CI: 1.56, 1.71), especially atrial myocardial infarction (HR 2.45 (95% CI: 2.22, 2.70) and cerebrovascular disease (HR: 2.16; 95% CI: 1.93, 2.42) compared to non-smokers[18]. Passive smoking is also associated with a higher risk of CVD (relative risk (RR): 1.23; 95% CI: 1.16, 1.31)[19] and females are 25% more likely to develop coronary heart disease than males with the same smoking exposure[20]. The mechanism of tobacco smoking and CVD is complex, but it is proposed that smoking induces oxidative processes, impairs platelet function, fibrinolysis and vasomotor function which are all pro-atherogenic[21].

For individuals with IBD, smoking is also detrimental to both the clinical course and long-term prognosis, particularly in CD. Epidemiological studies have shown that smokers with CD have higher rates of hospitalisation, relapses, surgery and immunosuppressant prescriptions[22,23]. For instance, smokers with CD treated with biologics were 37% and 25% less likely to achieve a clinical response and remission respectively[24]. On the contrary, smoking cessation beyond a year can lower the risk of relapse by 65% and results in a more benign course of CD compared to active smokers[25]. Smoking has no clinical benefits in CD and instead may potentially increase CVD risk due to inflammatory burden.

In contrast to CD, smokers with UC have a milder disease course and significantly lower rates of hospitalisation, relapses, colectomy and high-potency drug prescriptions than non-smokers with UC[22,23]. A meta-analysis showed that smokers had lower odds of developing UC (odds ratio: 0.58; 95% CI: 0.45, 0.75) than non-smokers, suggesting that smoking was protective against developing UC[26]. However, this protective effect ceases following smoking cessation, increasing the odds of developing UC by 64% compared to those who have never smoked[27]. Despite the seeming advantages of smoking for those with UC, smoking cessation should still be encouraged as the detrimental systemic effects of tobacco on cardiovascular health outweigh the temporary benefits.

Dyslipidaemia

Low-density lipoproteins (LDL) transport cholesterol to the peripheral tissue while high-density lipoproteins (HDL) reverse cholesterol transportation to the liver for hormone synthesis and excretion[28]. This process is regulated through a negative feedback loop between exogenous (diet) and endogenous (body) synthesis[29]. In some cases, dyslipidaemia, a condition characterised by high levels of total cholesterol (TC), LDL, triglycerides and low levels of HDL can manifest[28]. When there is an imbalance between the levels of LDL and HDL circulating in the blood, excess LDL may accumulate in the arterial wall, forming an atherosclerotic plaque over time[28]. This eventually thickens and narrows the blood vessels leading to atherosclerosis, a hallmark of CVD[7].

The known contributing factors of dyslipidaemia include genetic predisposition, lifestyle habits such as a diet rich in saturated and trans-fat, physical inactivity, smoking and other medical conditions[28]. A classic example of this is the high prevalence of dyslipidaemia among those with systemic lupus erythematosus[30]. It is deduced that the immune and inflammatory mechanisms of lupus alter lipoprotein metabolism which suppresses HDL and increases triglycerides[30]. Given that IBD demonstrates a similar inflammatory cytokine profile as lupus, people with IBD may be at a higher risk of dyslipidaemia. According to a large-scale cohort study of 1326 patients with IBD, the RR of dyslipidaemia was 2.18 times (95% CI: 2.03, 2.34) higher compared to the general population[31]. A previous cohort study also showed that people with IBD have a significantly higher LDL and lower HDL levels compared to healthy peers of the same age and BMI[32].

Besides inflammation, some medications prescribed to suppress inflammation in IBD, particularly corticosteroids, alter lipid profiles. A study reported that after 10 weeks of prednisone, TC, LDL and HDL significantly increased by 26%, 12% and 31% respectively[12]. Although prednisone may seem to improve HDL levels, it is offset by the increase in LDL levels. In n a cross-sectional study examining lipid profiles, individuals prescribed corticosteroids were found to have reduced risk of dyslipidaemia (HR: 0.45; 95% CI: 0.42, 0.49) compared to those not taking any medications[31]. Furthermore, immunomodulators and aminosalicylate therapy have also been shown to significantly decrease the risk of dyslipidaemia by 29% and 43%. These effects are potentially achieved through downregulation of lipogenic genes[31].

Hypertension

Genetics, aging and gender can influence blood pressure but hypertension (defined as 140/90 mmHg for systolic/diastolic blood pressure) can manifest prematurely due to smoking, excess intake of sodium and alcohol, being overweight or physically inactive[33]. Chronically elevated blood pressure damages arteries causing them to stiffen and narrow. This can happen simultaneously with the build-up of atherosclerotic plaques narrowing the arterial walls, thereby reducing the blood flow to vital organs such as the heart

https://doi.org/10.1017/S0029665124007596 Published online by Cambridge University Press

and brain. Hypertension is thus the leading cause of CVD and premature death worldwide[33].

Hypertension is frequently observed in people with immune-mediated diseases such as rheumatoid arthritis and psoriasis[34]. This could be due to increased proinflammatory cytokines and chemokines from circulating immune cells, promoting oxidative stress and causing vascular dysfunction[34]. However, there is little data on the prevalence of hypertension among IBD populations. Based on the United Kingdom biobank database analysis, people with IBD have a higher cumulative risk of hypertension than the general population, though the association was small (10.9% in UC and 7.7% in CD)[35]. In this population, CD was not a predictor of hypertension but those with UC were at higher risk of subsequent hypertension (HR: 1.30; 95% CI: 1.11, 1.52)[35].

It is hypothesised that anti-inflammatory medications may help to mitigate hypertension associated with systemic inflammation. Some studies have shown that methotrexate and anti-tumour necrosis factor therapies were correlated with lower blood pressure among people with rheumatoid arthritis, but this is not well studied in IBD. On the other hand, steroid use was found to associate with an increased risk of hypertension among those with chronic inflammatory conditions[13]. Steroid users with IBD had a 33% greater risk of developing hypertension[35]. Likewise, using thiopurines, an immunosuppressant, was also associated with higher rates of subsequent hypertension but only in CD (HR: 1.34; 95% CI: 1.02, 1.77)[35].

Body composition

Overweight and obesity (defined as a body mass index (BMI) ≥25 kg/m2) is a worldwide epidemic with increasing prevalence since the 1980s[36]. Although BMI correlates with body fat at a population level, BMI is unable to provide an accurate estimate of one's adiposity which varies by sex, age, ethnicity and physical activity level[36]. BMI is also a poor estimate of muscle mass and can be misleading for individuals with high muscle mass. Conventionally, people with IBD were more likely to be underweight and therefore weight gain was seen as a desirable outcome. However, recent literature has shown that the prevalence of obesity among this population has been increasing at a staggering rate that mirrors the obesity phenomenon[37].

Healthy eating and being active can favourably influence body weight and composition. However, adopting these lifestyle habits can be challenging for people with IBD due to disease symptoms such as nausea, diarrhoea, fatigue and abdominal pain, as well as medical therapy side effects[8,10,17]. Prolonged corticosteroid use to treat IBD can induce metabolic changes and may lead to a 2–5 kg weight gain[14]. Likewise, initiating biological treatments for IBD can also result in significant weight gain (1.7 kg with infliximab[16] and 2.18 kg with adalimumab[15]). This is potentially due to treatment-related metabolic alterations or reduced inflammatory burden that improves appetite and food digestion.

It is estimated that 20–40% of this population is overweight and 15–40% are obese according to their BMI[37]. In parallel with the excess weight gain, there is a notable disturbance in body composition among those with IBD, characterised by decreased muscle mass and increased fat mass including visceral adipose tissue (VAT) [38]. High-fat mass is not only strongly associated with increased cardiometabolic disease risk[36], but it is also associated with an increased risk of surgical complications and suboptimal response to weight-based biologic regimens, possibly due to increased adipose tissue sequestering the biologic agents[37]. However, VAT is more strongly correlated with increased odds of metabolic syndrome and CVD mortality than subcutaneous adiposity[39,40]. For those with CD, VAT is also uniquely deposited around areas of the inflamed bowel. These mesenteric depots are thought to be more immunologically active than other VAT depots as they promote mucosal inflammation[37].

Other than body fat, muscle mass is also a strong independent predictor of cardiovascular health. A large-scale multinational study showed that reduced muscle strength (per 5 kg measured by handgrip strength) was associated with increased CVD mortality (HR: 1.17; 95% CI: 1.11, 1.24)[41]. A subsequent large prospective study showed that the risk of CVD morbidity and mortality were lower in those with high muscle mass compared to those with low muscle mass irrespective of fat mass[42]. For individuals with IBD, low muscle mass is also associated with an increased need for surgery, poor surgical outcomes and osteopenia[43].

Physical inactivity

Physical activity (PA) refers to all bodily movements such as walking, cycling, sports and active recreation during leisure time[44]. Regular PA improves cardiorespiratory fitness (measured as maximum oxygen uptake, VO_{2max}) which correlates with lower CVD risk[45]. Among the general population, a high fitness level was associated with lower mortality rates from CVD regardless of sex, age, and other relevant risk factors[46]. Studies have also shown that PA helps to maintain a healthier weight and body composition (reducing fat mass and increasing muscle mass), better mental health and improved quality of life[44,47]. Despite the abundant evidence around PA and health, globally 27.5% of adults are still inactive[44]. This means that more than a quarter of the world's adult population does not meet the World Health Organisation PA guidelines to participate in at least 150 minutes of moderate-intensity or 75 minutes of vigorous-intensity aerobic PA per week[44].

It is known that environmental and social factors highly influence an individual's ability to engage in PA[44]. The shift to using mainly screen-based devices for work, daily tasks and leisure time also contributes to a more inactive and sedentary lifestyle[48]. For those with IBD, additional barriers such as abdominal pain, joint pain, fatigue, symptom flares, bowel motion urgency, lack of toilet access, and anxiety make it more challenging to be active[10,11,49]. PA can also be further limited due to side effects of medical therapy including headache, fever, arthralgia, myalgia, oedema, rashes, GI upset, and weakened immunity, which can cause higher infection rates[17]. Therefore, following an IBD diagnosis, it is common for PA levels to drastically decrease compared to the general population[11].

The current evidence on PA and CVD risk is robust but its effect on the course of IBD is limited. Some suggestive benefits of PA for those with IBD are improved quality of life and reduced anxiety, fatigue and disease symptoms[50]. However, some may still refrain from PA in fear of worsening their symptoms. In an Aotearoa, New Zealand study of 77 participants with IBD, it was found that 66.0% of participants were active despite experiencing IBD-related barriers to PA, which was much higher than the general population (34.8%) in 2016/17[49]. Furthermore, a multicentre survey of 158 participants with IBD from European countries and Israel showed that 87.5% of the cohort considered PA important and 46.1% believed that PA should be given more importance by their healthcare professionals[11]. These findings suggest that people with IBD may be receptive to participate in PA with appropriate support.

https://doi.org/10.1017/S0029665124007596 Published online by Cambridge University Press

Poor diet

The 2021 European Society of Cardiology recommends a healthy eating pattern like the Mediterranean diet or the Dietary Approach to Stop Hypertension (DASH) diet to prevent CVD[7]. Such a diet is high in plant-based foods that are rich in fibre (fruit, vegetables, wholegrains, legumes) and low in red meat, saturated fat, sugar, salt and heavily processed food. Meta-analyses and systematic reviews have demonstrated that a Mediterranean diet and a DASH diet improve lipid profile, blood pressure and body weight among adult populations[51,52]. While most people are familiar with the healthy eating principles, these may not translate well into daily practice[53], especially for those with IBD.

The standard dietary advice for patients to manage active disease and stricturing or penetrating CD is to adopt a modified fibre diet for reducing symptoms and the risk of bowel obstructions[54]. In some cases, people with IBD may also be instructed or choose to follow specialised diets that are designed to treat active inflammation or manage symptoms. These diets focus on restriction and modification of carbohydrate-rich foods (dairy, grains, legumes, lentils, fruits and vegetables). For example, the Autoimmune Protocol diet progressively eliminates grains, legumes and nightshades[55], while the Specific Carbohydrate Diet excludes starchy vegetables, grains and some legumes[56]. The low Fermentable, Oligo-, Di-, Mono- saccharides and Polyols (FODMAP) diet may restrict carbohydrate foods like wheat, legumes and specific vegetables and fruits that are high in FODMAP[57]. The IBD anti-inflammatory diet only allows specific carbohydrates, fruits, vegetables, and nuts[58], while the Crohn's Disease Exclusion Diet consists of selected carbohydrate foods with specific dietary fibres[59]. Although these diets may help to alleviate symptoms[55–59], some individuals with IBD may inadvertently restrict these fibre-rich foods longer than intended.

According to literature, 53% to 83% of people with IBD believe that modifying their diet can help control symptoms and prevent future disease relapses[8,9,60,61]. Many people consequently restrict consumption of certain food or drinks that they perceive to cause abdominal pain, diarrhoea, bloating and flatulence[8]. While these foods vary between individuals, common trigger food and drinks include spicy, strongly seasoned or fatty foods, dairy products and carbonated or caffeinated drinks[8,9,60]. It is also not uncommon to restrict eating grains, legumes, vegetables and fruits due to the belief that fibre aggravates the gut and contributes to a relapse[62]. For instance, a study of 82 participants with IBD believed that raw fruits (35.8%), raw vegetables (28.4%), cruciferous vegetables (47.8%) and legumes (59.7%) should be eliminated from their diet. Some participants completely avoid eating raw fruits (20.8%), raw vegetables (20.8%), and high-fibre products (43.1%) even in periods of remission[8]. Another survey also found that 19% of the 223 participating IBD healthcare providers recommend avoiding or limiting fibre-rich foods to reduce the risk of relapse[62] despite IBD consensus guidelines only recommending a modified fibre diet for patients with symptomatic stricturing CD[54].

Interventions for the prevention of CVD among populations with IBD

Physical activity interventions

PA has been shown to improve CVD risk factors including cardiorespiratory fitness, body composition and blood pressure in adult populations[47], and it is assumed that these benefits can be translated to those with IBD. Five studies have provided suggestive evidence of the impact of physical activity on CVD risk among physically inactive adults with IBD[63–67]. Among them, two controlled trials and one pilot study recommended aerobic and resistance training[63,65,67], one pilot study investigated light-intensity PA[66], and the other pilot study explored high- and moderate-intensity PA[64]. These studies primarily reported changes in anthropometry, body composition, blood pressure and VO_{2max} within groups[64–67], with the exception of Jones *et al.*, which was a randomised controlled trial[63].

Aerobic and resistance training (combination PA)

Cronin *et al.* investigated changes in body composition (Dual Energy X-ray Absorptiometry) after 8 weeks of combination PA in a cross-over trial[65]. Participants in the control phase maintained their usual PA while those in the intervention phase were prescribed a moderately intense exercise programme with three weekly sessions supervised by the study investigator or gym[65]. The intervention group experienced a 2.1% (lower quartile (LQ), upper quartile (UQ): 0.45%, 2.15%) decrease in median body fat percentage from baseline, while the control group had a slight increase of 0.1% (LQ, UQ: 0.1%, 1.0%)[65]. Additionally, the intervention group gained 1.59 kg (LQ, UQ: 0.68, 2.69) in muscle mass whereas the control group lost 1.38 kg (LQ, UQ: 0.26, 2.45)[65]. These changes in body composition were observed irrespective of BMI as the intervention group showed no changes in BMI (0 kg/m^2; LQ, UQ: −0.39, 0.48), while the control group had a slight decrease in BMI (−0.58 kg/m^2; LQ, UQ: −0.74, −0.54). No significant blood pressure changes were observed[65].

Jones *et al.* have measured the effects of combination PA on bone mineral density and muscle function in a 6-month randomised controlled trial[63]. Participants in the control group did not receive any advice while those in the intervention group completed 60-minute training sessions on three non-consecutive days each week for 6 months[63]. The session included a short warmup followed by moderate-high impact (skipping or jumps) and resistance training (bodyweight or resistance bands)[63]. Two sessions were supervised in the first two weeks, one in weeks 3 and 4, that eventually tapered to once a month from week 8 onwards[63]. Although not a direct measure of body composition, Jones *et al.* observed that the intervention group had superior handgrip strength, which correlates with overall muscle mass, with a mean difference of 8.3 kg (95% CI: 6.2, 10.5) compared to the control group[63]. Blood pressure was not significantly different between the groups[63].

van Erp *et al.* have conducted an uncontrolled, 12-week pilot study that examined the effect of a personalised combination PA on fatigue, quality of life, VO_{2max} and body fat percentage (measured with skinfold thickness)[67]. The programme was developed by a sports medicine physician and study physiotherapist[67]. Sessions were supervised and included 30 minutes of aerobic training (65–80% of maximum heart rate on indoor bicycle, cross-trainer, or treadmill) followed by 30 minutes of progressive resistance training (circuit training with machines)[67]. van Erp *et al.* reported a 1.8% (95% CI: 0.8%, 2.8%) body fat reduction without any significant changes in BMI, and VO_{2max} improved slightly from baseline following the intervention[67].

Aerobic PA at different intensities

Tew *et al.* determined the acceptability of high- or moderate-intensity training compared to a control group with no training advice for 12 weeks[64]. The sessions were supervised and carried out on a cycle ergometer at different peak power outputs (W_{peak}). Patients in the high-intensity group were asked to carry out

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ten 1-minute bouts at 90% W_{peak} interspersed with 1-min bouts at 15% W_{peak} while the moderate-intensity group were asked to carry out 30 minutes of training at 35% W_{peak}[64]. There were small changes in body weight and blood pressure in both intensity groups[64]. The moderate-intensity group experienced a larger mean reduction in waist circumference (−2.7 cm) from baseline compared to the high-intensity group (−0.8 cm)[64]. In contrast, the control group gained 1.7cm. Both moderate and high-intensity groups experienced improvement in $VO_{2max,}$ 2.2 ml/kg/min vs. 0.3 ml/kg/min, while VO_{2max} decreased in the control group[64].

Loudon *et al.* piloted a 12-week structured group walking programme using an uncontrolled study protocol to investigate the feasibility and the effects of the intervention on IBD symptoms, perceived stress and physical ability[66]. The walking programme included three weekly sessions (one supervised session per week) starting at 20 minutes and eventually progressing to 35 minutes per session[66]. The authors reported a reduction of BMI from 24.3 kg/m2 (standard deviation (SD): 5.3) to 23.9 kg/m^2 (SD: 5.3) in conjunction with a significant increase in VO_{2max} from 30.6 ml/kg/min (SD: 4.7) to 32.4 ml/kg/min (SD: 4.8) after 12 weeks of walking[66].

Collectively, these studies showed that all PA at various intensities were associated with increase cardiorespiratory fitness[64,66,67]. A combined PA of moderately intense aerobic and resistance training resulted in significant improvements in body fat percentage and/or muscle mass while maintaining remission[65,67]. These findings suggest that regular PA, particularly combined resistance, and aerobic activity, can improve cardiorespiratory fitness and body composition without aggravating symptoms. However, none of the PA interventions had any noticeable impact on blood pressure[63–65]. The lack of an effect on blood pressure was not surprising as none of these studies aimed to improve blood pressure and most participants were not hypertensive. Further research is required to ascertain the effects of PA interventions to reduce blood pressure among hypertensive populations with IBD.

It is common for people with IBD to experience additional PA barriers other than low motivation and time constraints. A survey showed that 78.8% of 859 participants with IBD felt that their PA was limited by abdominal/joint pain (70%), fatigue (69%), flare-ups (63%), toilet urgencies (61%) and even anxiety[10]. Overcoming these barriers are crucial factors in determining the practicality and effectiveness of PA interventions in the IBD population. While there are some concerns around higher PA intensities potentially aggravating gut symptoms, this was not observed in the literature[63–67]. In fact, studies suggest it may be safe and feasible for people with IBD to engage in PA without exacerbating symptoms[63–67]. However, as with most PA interventions, compliance tends to be higher in supervised settings as demonstrated in Jones *et al.*'s study where adherence rate was 81% for supervised sessions compared to 58% for independent sessions[63]. Other factors contributing to withdrawal or missed sessions included lack of motivation, previous injuries, time constraints, work-related fatigue and commitments, holiday and mainly illness unrelated to IBD[63–67]. Furthermore, adherence to high-intensity training was notably lower than for other forms of PA, likely due to the challenging nature of high-intensity PA rather than a worsening of IBD symptoms[64]. These findings reflect the real-world challenges for people with, and without, IBD to engage in PA. Overall, these results indicate that light or moderately intense PA is more likely to have better adherence and potentially improved long-term outcomes.

Dietary intervention

Diet and IBD are inseparable topics which have been extensively researched. There are numerous dietary trials investigating specific diets[55–59] but very few explicitly target CVD risk factors[68]. Only one uncontrolled, dietary prospective trial has reported on CVD risk factor outcomes in a cohort of adults with IBD who had been diagnosed for more than 6 months, had limited alcohol consumption, and did not have chronic liver disease[68]. Chicco *et al.* have measured the effects of a 6- 6-month Mediterranean diet on nutritional state, disease activity, and quality of life[68]. A nutritionist prescribed an isocaloric diet to participants with a BMI <30 kgm^2 while those with a BMI ≥30 kgm^2 received a hypocaloric diet. Participants were advised to consume ≥2 serves of vegetables, 1–2 serves of fruits, 1–2 serves of bread/cereals and olive oil in the form of 3 main meals and 2 light snacks per day[68]. They were also asked to include ≥2 serves of legumes/fish/seafood, ≥2 serves of poultry and dairy, 2–4 serves of eggs each week and limit their consumption of red meat and sweets to <2 serves per week[68]. Anthropometric parameters (BMI, waist circumference), body composition (bioimpedance analysis), and lipid profile were extracted from this study and reported as within-group differences between means pre- and post-intervention [68].

After six months, participants with UC and CD lost weight, BMI reduced by 0.42 kg/m^2 (SD: 1.22) and 0.48 kg/m^2 (SD: 1.57), and waist circumference reduced by 1.25 cm (SD: 5.39) and 1.35 cm (SD: 5.03) respectively[68]. However, there were no significant changes in body composition[68]. These findings were expected since 85.3% of participants had a BMI below 30 kg/m^2 and were prescribed an isocaloric diet[68]. There were also no significant changes in lipid profile which was unexpected, given that a meta-analysis of 57 controlled trials of non-IBD adult populations with metabolic risk factors such as hyperlipidaemia, metabolic syndrome and/or its associated conditions, prescribed a Mediterranean diet found improvements in total cholesterol (mean difference: −5.70; 95% CI: −9.96, −1.43) mg/dL, LDL cholesterol (mean difference: −8.24; 95% CI: −13.50, −2.99) mg/dL and HDL cholesterol (mean difference: 1.30; 95% CI: 0.38, 2.21) mg/dL[52]. Though, it is important to note that the baseline cholesterol profile of participants in Chicco *et al.'s* study did not show characteristics of dyslipidaemia and thus, improving these parameters would be difficult[68]. Perhaps a Mediterranean diet would result in favourable lipid profile changes in an IBD cohort with dyslipidaemia and excess adiposity.

Dietary and physical activity interventions (combined intervention)

Combining both diet and PA interventions may result in greater CVD risk factor improvements[69]. Lamers *et al.* have investigated the effects of a combined intervention (dietary and PA) on disease activity, fatigue and quality of life of those who had been diagnosed with IBD for more than 2 years and have poor diet quality[70]. This was an uncontrolled study of 6 months where BMI was reported as a secondary outcome[70]. A dietitian provided personalised dietary recommendations following the Dutch dietary guidelines with some alterations[70]. Participants were encouraged to consume more vegetables (>300 g/day), fruits, wholegrains and nuts while limiting their intakes of red meats (<100 g/week) and processed meats, processed foods and soft drinks[70]. A physiotherapist provided tailored advice to assist participants in meeting the Dutch PA guideline of 150 minutes of moderate PA per week, incorporating both aerobic and resistance training[70]. A booklet

https://doi.org/10.1017/S0029665124007596 Published online by Cambridge University Press

with the dietary and PA information was provided and participants could access an application for recipes[70]. Five consults were provided in the first 3 months and a further consult in the last 3 months[70]. However, the authors only reported a significant reduction in BMI from 26.4 kg/m^2 (SD: 3.8) at baseline to 25.0 kg/m^2 (SD: 3.0) at endpoint, which provides limited evidence for improved CVD risk outcome[70]. Thus, the potential implications of a combined intervention on anthropometric risk factors are not yet determined and require further investigation.

That said, adherence is crucial for the effectiveness of interventions, which can be particularly challenging for those with IBD. It is estimated that 32.5% of people with IBD have gut symptoms even when in disease remission[71]. People with IBD and healthcare professionals commonly perceive fibre as a trigger food for symptoms[8,62] despite the lack of scientific evidence. However, this was not apparent in the two studies reviewed that recommended a Mediterranean diet[68] and a healthy eating pattern[70]. Both IBD cohorts adhered to the recommended diet, with one study demonstrating a significant improvement in diet quality[70], while the other reported an adherence rate of up to 86.1%[68]. Although the authors did not speculate on the reasons for non-adherence, Lamers *et al.*[70] noted that dining out and celebrating often made it difficult to stick to the dietary guidelines. Nevertheless, these findings indicate that people with IBD can consume more fibre-rich foods such as fruits, vegetables, wholegrains and legumes while maintaining stable disease symptoms and activity[68,70]. It is presumed that these dietary changes were partly attributed to the education being delivered by a dietitian[70] or nutritionist[68], thereby increasing their confidence to include more dietary fibre. Taken altogether, these findings suggest that adhering to a Mediterranean diet or a healthy eating pattern is feasible for the IBD population without exacerbating symptoms but its potential for improving anthropometrics and lipid profile risk factors needs further confirmation with future research.

Summary

In summary, emerging literature suggests that people with IBD are more likely to be diagnosed with premature atherosclerotic CVD (RR: 1.61; 95% CI: 1.34, 1.94)[5] and develop ischaemic heart disease (RR: 1.24, 95% CI: 1.14, 1.35) than the general population[4]. While numerous lifestyle intervention studies have shown to improve modifiable CVD risk factors among the general population[7,47,51,52], it is not known whether similar interventions are as effective among those with IBD. In light of the increased CVD prevalence and associated conditions (obesity, hypertension and dyslipidaemia) among IBD populations, this review aims to discuss the relative importance of modifiable CVD risk factors and evaluate whether lifestyle interventions can effectively target these risk factors.

Contrary to existing evidence regarding the impact of diet, PA, or combined interventions on CVD risk factors in the general population[7,47,51,52,69], these were inconclusive among populations with IBD. This discrepancy is attributed several limitations in the literature. Firstly, most studies did not include a control group and therefore it is unknown whether the effects are exclusive to the intervention[64,66–68,70]. Furthermore, not adjusting for confounders can also cause bias in the estimate of the effects, especially for diet and PA interventions. Additionally, only two studies (one on the Mediterranean diet[68] and another on combined training[65]) reported the CVD risk factors as a primary outcome, whereas the others reported them as a secondary outcome[63,64,66,67,70]. Most studies were also of short duration (8–12 weeks)[64–67] and small sample sizes (<30 participants)[65–67,70], rendering them underpowered to detect meaningful changes. Despite these limitations, available data to date suggests that following a Mediterranean or healthy diet as well as participating in moderately intense aerobic and resistance PA, may be a feasible method to improve anthropometric risk factors for CVD. To confirm the effects of lifestyle interventions on these CVD risk factors among people with IBD, larger studies with independent control groups and longer durations are necessary.

Acknowledgements. The authors thank the Organising Committee of the Nutrition Societies of Australia and New Zealand Conference 2023 for the review invitation.

Author contributions. JMY, MS and CW contributed to the outline of the manuscript. JMY completed the literature retrieval and drafted the manuscript. MS, CW, KJ and HO supervised and reviewed the content. JMY finalised the manuscript and all authors provided final approval for submission.

Financial support. This research received no specific grant from any funding agency, commercial or not-for-profit sectors.

Competing interests. The authors declare none.

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