INTERNAL MEDICINE JOURNAL

RACP Specialists. Together

doi:10.1111/imj.15941

ORIGINAL ARTICLE

High incidence of inflammatory bowel disease in Northern Australia: a prospective community population-based Australian incidence study in the Mackay-Isaac-Whitsunday region

Amanda Flanagan,[1] Susan M. Allsopp,[1,2,3] Sam A. O'Connor,[1,2,8] Jacinta Tobin,[3] Casper Pretorius,[1,3,9] Ian S. Brown,[5] Sally Bell [6] and A. James M. Daveson [1,2,4,7,8,9]

[1]EndosQ Gastroenterologists, [2]Mater Misericordiae Hospital, Hospital Mackay, [3]Mackay Base Hospital, [4]College of Medicine and Dentistry, James Cook University, and [9]Coral Sea Clinical Research Institute, Mackay, [5]Envoi Specialists Pathologists, [7]Coeliac and Immune Health Research Program, Wesley Medical Research, and [8]Faculty of Medicine, University of Queensland, Brisbane, Queensland, and [6]Department of Gastroenterology, St. Vincent's Hospital, Melbourne, Victoria, Australia

Key words
Crohn's disease, Ulcerative Colitis, Inflammatory Bowel Disease, Incidence, Australasia, North–South gradient.

Correspondence
A. James M. Daveson, Suite 16 Mater Medical Suites, 76 Willett's Road, Mackay, Qld. 4740, Australia.
Email: james.daveson@endosq.com

Received 10 January 2022; accepted 27 September 2022.

Abstract

Background and Aims: To determine the incidence of inflammatory bowel disease (IBD) in the Mackay-Isaac-Whitsunday region in Northern Queensland (−21.14° S) and to allow a comparison with Southern Australian and New Zealand data (Geelong, Australia −38.14° S; Tasmania −41.43° S and −42.88° S (Launceston and Hobart) and Canterbury, New Zealand −43.46 °S).
Design: A prospective observational community population-based IBD study was conducted between 1 June 2017 and 31 May 2018.
Outcome measures: Primary includes the crude annual incidence rate of IBD, Crohn's disease (CD), ulcerative colitis (UC) and inflammatory bowel disease-unclassified (IBDU), while secondary includes disease phenotype and behaviour.
Results: Fifty-six new cases of IBD were identified. Twenty-three were CD, 30 were UC and 3 were IBDU. The crude annual incidence rate per 100 000 for IBD, CD, UC and IBDU were 32.2 (95% confidence interval (CI): 24.78–41.84), 13.23 (95% CI: 8.79–19.90), 17.25 (95% CI: 12.06–24.67) and 1.73 (95% CI: 0.56–5.35). When directly age-standardised to the World Health Organisation Standard Population Distribution, the overall CD, UC and IBDU incidence were 13.19, 17.34 and 1.85 per 100 000, with an overall age-standardised IBD incidence of 32.38.
Conclusions: This is the first study to define the incidence of IBD in a Northern Australian cohort and to allow a comparison between North and Southern Australia. The IBD crude is the highest reported in Australia. Like others, we found a high and low incidence of upper gastrointestinal Crohn's disease and complicated disease at diagnosis respectively, likely reflective of the increased availability and early uptake of endoscopic procedures.

Introduction

Inflammatory bowel diseases (IBDs), including Crohn's disease (CD), ulcerative colitis (UC) and inflammatory bowel disease-unclassified (IBDU), are chronic disabling idiopathic conditions of unknown aetiology associated with significant individual and community disease burden.[1,2] Australian health costs attributed to IBD exceed AU$2.7 billion *per annum* with national productivity losses of AU$380 million.[3] Global data suggest the incidence of IBD has increased substantially over the past five decades, with previously low incidence areas, such as Asia, increasing rapidly.[4] Population-based studies in Southern Australia (Victoria and Tasmania) and New Zealand (Canterbury) have demonstrated high incidence rates,[5–7] though to date there are no epidemiological data on the incidence of IBD in Northern Australia. Prior studies in Europe and the United States (US) have also suggested a North–South gradient, with

Conflict of interest: None.

disease incidence decreasing the closer the location to the equator in both the Northern and Southern Hemispheres.[8,9]

Given Australia's large geographical land size, the Mackay-Isaac-Whitsunday region located above the tropic of Capricorn at −21.14° S provides a unique opportunity to evaluate whether a North–South gradient exists between it and those Southern Australian and New Zealand regions where epidemiological data have been defined (Geelong, Australia at −38.14° S; Launceston and Hobart, Tasmania at −41.43° S and − 42.88° S respectively; and Canterbury, New Zealand at −43.46°S; see Fig. 1). We aimed to perform a prospective observational community population-based study of IBD incidence in the Mackay-Isaac-Whitsunday region in Northern Queensland and to allow a comparison with southern Australian and New Zealand data.

Methods

Study population

The Mackay-Isaac-Whitsunday region (Fig. 1A) was defined using the Australian Statistical Geography Standard (ACGS)[10] statistical division (Fig. 1A,B). The region lies on the eastern coast of North Queensland, approximately 1000 km (621 miles) north of the state's capital

Figure 1 (A) SA4 geographic boundaries and population catchment area of the Mackay-Isaac-Whitsunday region and (B) in relation to Australia and New Zealand and (C) identification of Mackay (yellow dot) above the tropic of Capricorn. Geelong (black dot), Tasmania (red dot) and Christchurch (white dot).

city Brisbane. Bordered to the east by the Coral Sea and Great Barrier Reef and to the west by the Bowen and Galilee Basin coal deposits, most of the population resides by the ocean, with the population extending inland. The region is represented by a broad range of industries though predominantly sugarcane, beef and mining.

According to the 2016 census, the population of the Mackay-Isaac-Whitsunday region was 169 688 (51.4% M/48.6% F), with a median age of 37.[11] Comparably, the total Australian population was 23 401 892 with a median age of 38 years. 26.7% of the population was aged <20 with 18% >65 years of age. The population density was 1.9 persons/km^2. The indigenous population was 4.9%, 1.75 times higher than the national average of 2.8%. 10.3% had earned a university bachelor's degree level or above compared to 18.3% for Queensland and 22% for Australia. 12.7% were employed in the mining industry (1.2% for Queensland and 0.4% for Australia). 58.1% identified as couple or single-parent families compared to 59% for Queensland and 60.5% for Australia.[11]

Case identification and ascertainment

In Australia, healthcare is divided into a nationally subsidised 'private' fee for service model or 'public' state government-provided free health service, with subsequent care fragmented between healthcare providers and facilities, hindering epidemiological studies (in 2014–2015, 57.1% of Australians aged ≥18 years had private health insurance).[12] The location of >90% of specialists within metropolitan Australia further fragments IBD cases into multiple metropolitan centres.[13] There is also no national diagnostic or therapeutic registry, and to accurately identify cases for epidemiological studies, patients attending both public and private doctors, clinics and hospitals need to be identified.

The regional IBD clinic in the Mackay-Isaac-Whitsunday (Queensland, Australia) region provides high-quality endoscopy, radiology and pathology in IBD care and universal healthcare (both public and private) for IBD patients. Equally, there are only two large hospitals (one public and one private), both of which the clinic services (as compared to >30 private and >20 public hospitals within Queensland's capital of Brisbane). Given one clinic and two main hospitals, many of the obstacles traditionally inherent in prospectively determining disease incidence within the Australian healthcare system are removed. Incident community and admitted IBD patients are exclusively directed to the single clinic on the back of established mature referral pathways. Community referrals to the clinic are by the region's general practitioners, local general surgeons or other specialists. Admitted patients to either hospital are referred to the clinic while an in-patient or post-discharge.

The region is geographically well defined and isolated, with limited migration out of the region for healthcare. No other gastroenterological care is available for 393 km (244 miles; 8 h round car trip) to the north, while the nearest tertiary-level care is 959 km (596 miles; 24 h round car trip) to the south. Established in 2014, the clinic has a mature system of referrals from primary healthcare and services a geographical area of 400 km^2 (Fig. 1). As per the existing standard of care at the time, all endoscopy and histopathology reports during the period of the study generated from a colonoscopy either within the clinic or the public hospital were reviewed for evidence of IBD. General practitioners were advised of the study via their newsletters within their primary healthcare network as well as through meetings.

Case definition and participants

All new cases of IBD from 1 June 2017 to 31 May 2018 were prospectively defined using rigorously recognised and accepted criteria and categorised using the Montreal Classification.[14] Any suspected new case of IBD underwent a repeat colonoscopy and, if clinically indicated, an oesophagogastroduodenoscopy (OGD) by a board-certified gastroenterologist within the clinic to confirm the diagnosis prior to treatment. Cases were only included if the diagnosis of IBD was histologically confirmed. All cases of identified Crohn's disease involving the upper gastrointestinal (GI) tract were reviewed by a second histopathologist expert in upper GI pathology and only included in the final analysis if confirmed. Patients were excluded if there was any doubt as to the diagnosis, the case could not be confidently determined or if they lived outside of the catchment area at the time of diagnosis.

Consent and information storage

All information involving patients was handled in accordance with the Australian Code for the Responsible Conduct of Research and the National Statement on Ethical Conduct in Human Research and with any relevant legislative requirements or regulations which govern the collection and storage of health-related data. The study received ethical approval from Uniting Care Health HREC ref.: 2017.10.224 and the Townsville Hospital and Health Service ref.: HREC/17/QTHS/133.

Data analysis

Crude incidence rates in the 12-month period (1 June 2017 to 31 May 2018) were calculated for IBD (CD, UC and IBDU) by calculating newly diagnosed cases as compared to the total population at risk. Population data and demographics for the Mackay-Isaac-Whitsunday region were obtained from the 2016 census.[11] The crude rate was age-standardised to the World Health Organisation Standard Population Distribution[15] to enable other regional comparisons. Crude incidence rates were reported with a 95% confidence interval (CI), assuming a normal approximation to the Poisson distribution.

Results

IBD diagnosis and incidence

Between 1 June 2017 and 31 May 2018, 56 new cases of IBD were identified in the Mackay-Isaac-Whitsunday region. Of these, 23 were CD (52% F), 30 were UC (50% F) and three (3) were identified as IBDU (33% F) (ee Table 1). The crude annual incidence rate per 100 000 for IBD, CD, UC and IBDU was 32.2 (95% CI: 24.78–41.84), 13.23 (95% CI: 8.79–19.90), 17.25 (95% CI: 12.06–24.67) and 1.73 (95% CI: 0.56–5.35). When directly age-standardised (to the World Health Organisation Standard Population Distribution), the overall CD, UC and IBDU incidence was 13.19, 17.34 and 1.85 per 100 000, with an overall IBD incidence when directly age-standardised to the World Health Organisation Standard Population of 32.38 (compared with 39.5 in 2014 in Canterbury, New Zealand,[5] 29.5 in 2013 in Tasmania, Australia[7] and 24.7 in 2010 in Victoria, Australia[6]).

Of the 23 with CD, 20 (87%) remained in follow-up and on therapy within the clinic 2 years later. One (1) (4%) had relocated to care in NSW, while two (2) (8.7%) were lost to follow-up. Of the 30 patients with UC, 24 (80%) of the patients remained in follow-up and on therapy 2 years later. Three (3) (10%) had been transferred to care in Brisbane and three (3) (10%) had been lost to follow-up.

Referral indication

For IBD, 35.7% (20) were referred for rectal bleeding, 26.8% (15) for abdominal pain, 17.9% (10) for diarrhoea, 7.1% (4) for a positive FOBT and 3.5% (2) for reflux symptoms. Iron depletion, small bowel obstruction, acute severe colitis, surveillance and screening colonoscopies all accounted for one referral each (Table 2). For CD, the referral indication was abdominal pain in 52.2% (12), rectal bleeding 17.4% (4), positive FOBT in 8.7% (2), reflux symptoms 8.7% (2), diarrhoea 4.3% (1), a family history of bowel cancer in 4.3% (1) and a small bowel obstruction in 4.3% (1). For UC, the referral indication was rectal bleeding for 50% (15), diarrhoea 23% (7), abdominal pain 10% (3), positive FOBT 6.7% (2), iron depletion 3.3% (1), acute severe colitis 3.3% (1) and colorectal polyp surveillance 3.3% (1). In the three IBDU cases, the primary indication for referral was diarrhoea in 67% (2) and rectal bleeding in 33% (1).

Mode of referral

For IBD, 46.4% (26) were referred by their general practitioner, 39.7% (22) were referred by the public hospital, 12.5% (7) were referred by private general surgeons, and 1.8% (1) were referred by a private paediatrician. For CD, the referral source was a general practitioner in 52.2% (12), the public hospital in 34.8% (8), a private general surgeon in 8.7% (2) and a private paediatrician in 4.3% (1). For UC, the referral source was the public hospital in 43% (13), a general practitioner in 40% (12) and a private surgeon in 17% (5). For the two IBDU

Table 1 Comparison of IBD incidence in Australasia

Site	Canterbury, New Zealand[2]	Geelong, Victoria, Australia[16]	Geelong, Victoria, Australia[6]	Tasmania, Australia[7]	Canterbury, New Zealand[5]	Mackay, Australia
Dates	1 January 2004 to 31 December 2004	April 2007 to March 2008	July 2010 to June 2011	June 2013 to May 2014	January 2014 to December 2014	June 2017 to May 2018
Crude rate IBD	25.2	29.3	24.2	29.0	39.8	32.20
(Age-standardised rate)	(24.9)	(29.6)	(24.7)	(29.5)	(39.5)	(32.38)
Crude rate CD	16.5	17.4.	14.7	14.4	26.0	13.23
(Age-standardised Rate)	(16.3)	(N/A)	(N/A)	(15.4)	(26.4)	(13.19)
Crude rate UC	7.6	11.2	7.5	12.3	13.4	17.25
(Age-standardised rate)	(7.5)	(N/A)	(N/A)	(12.4)	(12.6)	(17.34)
Crude rate IBDU	1.1	0.8	2.0	2.4	0.39	1.73
(Age-standardised rate)	(0.3)	(N/A)	(N/A)	(1.7)	(0.17)	(1.85)

CD, Crohn disease; IBD, inflammatory bowel disease; IBDU, inflammatory bowel disease-unclassified; UC, ulcerative colitis.

Table 2 Referral indications for Crohn disease (CD), ulcerative colitis (UC), IBD-U and IBD

Indication	CD	UC	IBD-U	Total
Abdominal pain	52.2% (12)	10% (3)	0% (0)	26.8% (15)
Rectal bleeding	17.4% (4)	50% (15)	33% (1)	35.7% (20)
Diarrhoea	4.3% (1)	23% (7)	67% (2)	17.9% (10)
Positive FOBT	8.7% (2)	6.7% (2)	0% (0)	7.1% (4)
Reflux symptoms	8.7% (2)	0% (0)	0% (0)	3.5% (2)
Other: (one each of iron depletion, small bowel obstruction, acute severe colitis, surveillance colonoscopy for colorectal polyps, and colonoscopy for strong family history of colon cancer)	8.6% (2)	10% (3)	0% (0)	9% (5)

IBD, inflammatory bowel disease; IBDU, inflammatory bowel disease-unclassified.

cases, two were referred by their general practitioner, while one was referred by the public hospital.

Demographics

The genders of the cases were evenly distributed, with 50% female. The age of diagnosis ranged between 13 and 76, though with most diagnosed between the ages of 15 and 40 (Fig. 2). The incidence peaked between the ages of 15 and 40, though then again with a smaller peak between 55 and 64. There was no significant difference in median or mean age of diagnosis between IBD, CD and UC (36, 36 and 35 respectively) or mean for IBD, CD, UC and IBDU (38, 39, 38 and 32 respectively).

Disease phenotype

Of the CD patients, five (5) patients (21.7%) were ≤16 years of age, 10 patients (43.5%) were aged between 17 and 39 and eight (8) patients (34.78%) were aged >40 years (see Table 3). Nine (9) patients (39.1%) had isolated ileal disease (L1), three (3) patients (13%) had disease limited to the colon (L2), while 11 patients (47.8%) had ileocolonic disease (L3). Nine patients (39.1%) had concomitant upper GI disease (L4). In terms of disease behaviour, most patients (n = 18; 78.2%) had non-stricturing, non-penetrative disease (B1). Three (3) patients (13%) had stricturing disease (B2) and four (4) (17.4%) had penetrating disease (B3). Two (2) (8.7%) had perianal disease.

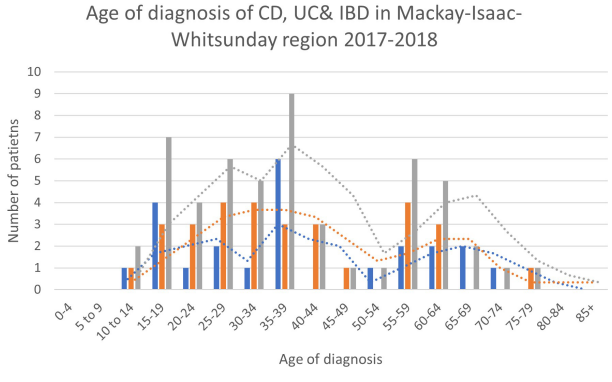

Figure 2 Age of diagnosis of CD, UC and IBD in the Mackay-Isaac-Whitsunday region. (■) Crohn's disease, (■) ulcerative colitis and (■) IBD.

Table 3 Montreal classification of Crohn disease (CD) and ulcerative colitis (UC) patients

Montreal classification of CD	*n* (%)
Age at diagnosis, years	
A1 < 16	5 (21.7)
A2 17–40	10 (43.5)
A3 > 40	8 (34.8)
Location	
L1 terminal ileum	9 (39.1)
L2 colonic	3 (13.0)
L3 ileocolonic	11(47.8)
L4 upper GI	9 (39.1)
Isolated L4 upper GI	
Behaviour	
B1 non-stricturing, non-penetrating	18 (78.2)
B2 stricturing	3 (13)
B3 penetrating	4 (17.4)
P perianal	2 (8.7)
Montreal classification UC	***n* (%)**
Age at diagnosis, years	
A1 < 16	2 (6.7)
A2 17–40	19 (63.3)
A3 > 40	9 (30)
Extent	
E1 ulcerative colitis	9 (30)
E2 left-sided UC (distal UC)	5 (16.7)
E3 extensive UC (pancolitis)	16 (53.3)

GI, gastrointestinal.

Of the UC patients, two (2) (6.7%) were ≤16 years of age, 19 (63.3%) were between 17 and 39 years of age and nine (9) (30%) were aged >40 years. Nine (9) patients (30%) had disease limited to the rectum (E1), five (5) patients (16.7%) had disease extending to the splenic flexure (E2), while 16 patients (53.3%) had extensive colitis (E3).

Primary sclerosing cholangitis

Of the 30 patients with UC, the alkaline phosphatase was elevated in 13.3% (4). 10% (3) were diagnosed with primary sclerosing cholangitis (PSC) based on characteristic findings at MRCP. In the three with PSC, antimitochondrial antibodies were absent in all, the IgM level was elevated in 1 (33%), and 66% (2/3) were female. PSC was not identified in any cases of CD or IBD-U.

Discussion

This is the first study to define the incidence of IBD in a Northern Australian cohort and the first to allow a comparison between Northern and Southern Australia. The IBD crude annual incidence rate of 32.2 in the Mackay-Isaac-Whitsunday region (located at −21.14°S and above the Tropic of Capricorn) is to date the highest reported in Australia. The most recent comparator from Tasmania (located at −41.43°S Launceston and −42.88°S Hobart) in 2014, while having a lower crude annual incidence rate at 29.0, their CIs (24.3–33.7) compared with ours (24.78–41.84), suggests no significant difference. Our rates were higher than the incidence rate of 24.2 (95% CI: 19.9–30.5) found in Geelong Australia located at −38.14°S in 2010–2011. There was no increase in incidence in Geelong from a study performed 3 years prior in 2007–2008, unlike the 1.6-fold increase found in Canterbury over 10 years between 2004 and 2014. Although a higher annual crude rate of 39.8 was found in Canterbury, New Zealand in 2014 (located at −43.46°S), again there was no difference in CIs (95% CI: 34.4–45.3).

Although the NZ authors suggest their high rate may be due to increased colonoscopy uptake, the areas of Mackay-Isaac-Whitsunday and Geelong equally have higher rates of colonoscopy uptake than the Australian national average.[17] Australia has entered the third epidemiological stage of compounding prevalence in its evolution of IBD,[18] and it is possible the uniformity of incidence rates reflects this. The incidence numbers for CD in the Mackay-Isaac-Whitsunday region of 13.23/100 000 and 17.25/100 000 for UC lie close to but above coalescing incidence ranges for the Western World of 6–11/100 000 for CD and 6–15/100 000 for UC.[19]

Of the six incidence studies now performed in Australia and New Zealand, the Mackay-Isaac-Whitsunday study was the only one to show a higher incidence of UC than CD. This could be due to a delay in diagnosis of CD. Combined, rectal bleeding and diarrhoea accounted for 73% of the referral indications for those diagnosed with UC, as opposed to 21.7% for CD, where abdominal pain and reflux symptoms accounted for 60.9% (compared to 10% for UC). As suggested by others, profuse diarrhoea or rectal bleeding could have led to an earlier referral for a colonoscopy in the young, as opposed to abdominal pain which may be attributed to IBS and, hence, a consequent delay in diagnosis.[20] The higher rate of UC as compared to CD may also reflect an 'unmasking of incidence'. An analogy of this is as IBD emerges in a population, UC cases are usually initially higher than CD, though with time, CD becomes more commonly diagnosed.[18] Although there is broad uniformity in funding to provide healthcare within Australia, there is marked heterogeneity in its provision, with health outcomes in regional and remote Australia inequitable compared to metropolitan Australia.[21] The arrival of a dedicated GE specialist in 2008 (AD) and the formal provision of an IBD clinic in 2014 allowing region-wide access to IBD care in the Mackay-Isaac-Whitsunday region likely resulted in increased disease awareness, improved diagnoses and a diagnostic bias still evident in 2017.

Why a North–South gradient was not evident is unclear. A large study from the US utilised data from two Nurses' Health Studies and examined the correlation between the development of IBD and latitude of residence (and unlike our study) at different ages in life. It found that a cohort of predominantly caucasian women residing at southern latitudes, particularly in later life (age 30), had a consistently lower risk of developing UC and CD.[9] Reduced exposure to sunlight or UVB radiation in the northern latitudes with consequent lower levels of plasma vitamin D (cholecalciferol) with its anti-inflammatory property was suggested as a reason.[9] In 2017, the average monthly global solar exposure was 20.76 MJ/m^2 in Mackay compared to 14.81 MJ/m^2 in Geelong and 13.21 MJ/m^2 in Hobart,[22] which argues against this as a cause in our study. The US study relied on medical record reviews rather than real-time prospective patient assessments as in our study. Another large European study identified 2201 new cases of IBD between 1991 and 1993. The authors concluded a modest excess of IBD incidence in Northern Europe, though, which they felt may have reflected a stabilising incidence of IBD in Northern Europe with an increasing incidence in Southern Europe.[8]

As in Canterbury, we also found a high rate of upper GI (L4) disease, with Su *et al*. suggesting this may be

secondary to the increased availability of endoscopic procedures such as capsule endoscopy.[5] 83% of those undergoing a colonoscopy for CD also had an OGD at the time of entry into our study, likely leading to the increased finding of L4 disease. A second GI histopathologist expert in upper GI disease independently reviewed and validated every case of identified upper GI disease. There are limited studies uniformly examining the upper GI manifestations in treatment-naïve patients with CD at first diagnosis. One study identified upper GI inflammatory changes in 68% of gastric biopsies, 60% of oesophageal biopsies and 43% of duodenal biopsies from a total of 111 consecutive cases of CD.[23] Hence, although our incidence of upper GI disease is higher than that reported in other studies, it likely reflects the performance of OGDs at the time of performing the diagnostic colonoscopy.

Our finding of a low incidence of complicated disease behaviour at diagnosis (B2 and B3) was also similar to that found in the 2014 Canterbury study, and others,[5,24,25] again suggesting the increased uptake of endoscopic procedures may have increased the chance of those with milder disease being detected earlier. Although the rate of perianal disease seemed low at 8.7%, 82.6% (19) of the patients with CD underwent imaging of the abdomen/pelvis to assess for either co-existent small bowel disease or invasive disease. 21% (4) had a CT, while 79% (15) underwent an MRI.

Our data of equivalent male and female incidence were in keeping with international literature, as was the observed second peak of UC in the sixth decade.[26] The proportions of UC disease location (L1–L3 and E1–E3) in our study are similar to other Australian data. The 10% incidence of PSC was similar to other reported rates of PSC in UC.[27]

The strengths of this study include its prospective recruitment with histological confirmation in all cases, a lack of tertiary referral bias with established public and private referral pathways into a single established community clinic, rigorous case definition with 98% of participants undergoing clinical review and endoscopy at study entry by college-certified gastroenterologists, all histopathology reported by a single group of dedicated GI histopathologists, 92% of participants remaining in IBD clinics 2 years after diagnosis (the remaining four lost to follow-up) and the geographical isolation of the study area. Leakage out of the area was unlikely and would only have resulted in a higher incidence had such gone undetected. Although it is possible a hospitalised patient may have been referred for tertiary-level care, 80% of IBD patients are never hospitalised,[28] and after discharge these patients would usually have their care shared with the local IBD clinic (as was the case of acute severe colitis). Importantly, a lack of tertiary referral bias may also be a factor attributable to the high IBD incidence seen in this study.

Global data suggest the incidence of IBD has increased substantially over the past five decades,[16] with previously low-incidence areas such as Asia increasing rapidly.[4] Although the reasons for this are yet to be elucidated, the similarly high incidence of IBD in the Mackay-Isaac-Whitsunday region compared to Southern regions supports the notion that relatively homogenous genetic, dietary and socioeconomic factors are playing a role in aetiology given the lower incidence in other countries of similar tropical latitude in the Asia-Pacific region.[25]

Conclusion

IBD often occurs in young people and, although disabling without therapy, is not universally fatal. The social and economic burdens, however, on patients, their families, the healthcare system and the community are significant. Healthcare in Australia is not equitable, with those in regional and remote Australia at higher risk of poor health outcomes compared to those in metropolitan Australia, as a result of geographical isolation, shortage of healthcare providers, socioeconomic disadvantage, greater difficulties in transport and communication and sparsely distributed populations, leading to diseconomies of scale.[21] The described high regional incidence of IBD in Northern Queensland, a state with a particularly disparate population, should challenge clinicians and health policymakers with respect to health economic modelling when predicting healthcare expenditure and healthcare delivery in Australia. Further studies to examine the change in incidence of IBD over time are warranted, as are studies examining innovative models of healthcare delivery for these regions.

Acknowledgement

The authors would like to acknowledge Dr. Mohamad Alshurafa for his contribution to the statistical analysis of the results. Open access publishing facilitated by James Cook University, as part of the Wiley - James Cook University agreement via the Council of Australian University Librarians.

References

1 Ng S, Tang W, Ching J, Wong M, Chow C, Hui AJ *et al*. Incidence and phenotype of inflammatory bowel disease based on results from the Asia-Pacific Crohn's and colitis epidemiology study. *Gastroenterology* 2013; **145**: 158–65.e2.

2 Gearry RB, Richardson A, Frampton CM, Collett JA, Burt MJ, Chapman BA *et al*. High incidence of Crohn's disease in Canterbury, New Zealand: results of an epidemiologic study. *Inflamm Bowel Dis* 2006; **12**: 936–43.

3 Price Waterhouse Coopers. *Improving Inflammatory Bowel Disease Care across Australia*. 2013.

4 Zhao J, Ng S, Lei Y, Yi F, Li J, Yu L *et al*. First prospective, population-based inflammatory bowel disease incidence study in mainland of China: the emergence of "western" disease. *Inflamm Bowel Dis* 2013; **19**: 1839–45.

5 Su H, Gupta V, Day A, Gearry R. Rising incidence of inflammatory bowel disease in Canterbury, New Zealand. *Inflamm Bowel Dis* 2016; **22**: 2238–44.

6 Studd C, Cameron G, Beswick L, Knight R, Hair C, McNeil J *et al*. Never underestimate inflammatory bowel disease: high prevalence rates and confirmation of high incidence rates in Australia. *J Gastroenterol Hepatol* 2016; **31**: 81–6.

7 Bhatia R, Yeoh S, Vaz K, Studd C, Wilson J, Bell S *et al*. Inflammatory bowel disease incidence, prevalence and 12-month initial disease course in Tasmania, Australia. *Intern Med J* 2019; **49**: 622–30.

8 Shivananda S, Lennard-Jones J, Logan R, Fear N, Price A, Carpenter L *et al*. Incidence of inflammatory bowel disease across Europe: is there a difference between north and south? Results of the European collaborative study on inflammatory bowel disease (EC-IBD). *Gut* 1996; **39**: 690–7.

9 Khalili H, Huang ES, Ananthakrishnan AN, Higuchi L, Richter JM, Fuchs CS *et al*. Geographical variation and incidence of inflammatory bowel disease among US women. *Gut* 2012; **61**: 1686–92.

10 Australian Bureau Of Statistics. Australian Statistical Geography Standard (ASGS): Volume 1 – Main Structure and Greater Capital City Statistical Areas; July 2016. Available from URL: https://www.abs.gov.au/ausstats/abs@.nsf/mf/1270.0.55.001

11 Australian Bureau Of Statistics. Mackay-Isaac-Whitsunday (SA4) (312) 2016–2019. Available from URL: https://itt.abs.gov.au/itt/r.jsp?RegionSummary&region=312&dataset=ABS_REGIONAL_ASGS2016&geoconcept=ASGS_2016&datasetASGS=ABS_REGIONAL_ASGS2016&datasetLGA=ABS_REGIONAL_LGA2017&regionLGA=LGA_2017&regionASGS=ASGS_2016

12 Australian Bureau Of Statistics. Health Service Usage and Health Related Actions, Australia, 2014–15; 2017. Available from URL: https://www.abs.gov.au/ausstats/abs@.nsf/Lookup/by%20Subject/4364.0.55.002~2014-15~Main%20Features~Private%20health%20insurance~5#:~:text=In%202014%2D15%20there%20were,2007%2D08%20(52.7%25

13 Health AGDo. NWHDS Medical Practitioner 2016. Australian Bureau of Statistics, 3218.0 Regional Population Growth, Australia: Australian Bureau of Statistics; 2016.

14 Lennard-Jones J. Classification of inflammatory bowel disease. *Scand J Gastroenterol* 1989; **170**: 2–6.

15 Ahmad O, Boschi-Pinto C, Lopez A, Murray C, Lozano R, Inoue M *et al*. *Age Standardization of Rates: A New WHO Standard (GPE Discussion Paper Series No. 31)*. Geneva: World Health Organization; 2001.

16 Wilson J, Hair C, Knight R, Catto-Smith A, Bell S, Kamm M *et al*. High incidence of inflammatory bowel disease in Australia: a prospective population-based Australian incidence study. *Inflamm Bowel Dis* 2010; **16**: 1550–6.

17 Australian Commision On Safety And Quality In Healthcare. 2.1 Colonoscopy Hospitalisations, All Ages; 2018. Available from URL: https://www.safetyandquality.gov.au/our-work/healthcare-variation/atlas-2018/atlas-2018-2-gastrointestinal-investigations-and-treatments/21-colonoscopy-hospitalisations-all-ages

18 Kaplan GG, Windsor JW. The four epidemiological stages in the global evolution of inflammatory bowel disease. *Nat Rev Gastroenterol Hepatol* 2021; **18**: 56–66.

19 Ng SC, Shi HY, Hamidi N, Underwood FE, Tang W, Benchimol EI *et al*. Worlwide incidence and prevalence of inflammatory bowel disease in the 21st century: a systematic review of population-based studies. *Lancet* 2017; **390**: 2769–78.

20 Vavricka SR, Spigaglia SM, Rogler G, Pittet V, Michetti P, Felley C *et al*. Systematic evaluation of risk factors for diagnostic delay in inflammatory bowel disease. *Inflamm Bowel Dis* 2012; **18**: 496–505.

21 Australian Institute of Health and Welfare 2016. Australia's Health 2016. Australia's Health Series No. 15. Canberra: AIHW.

22 Australian Government Bureau of Meteorology. Canberra. 2017. Available from URL: www.bom.gov.au

23 Brown IS, Miller GC, Bettington ML, Rosty C. Histopathological findings of extra-ileal manifestations at initial diagnosis of Crohn's disease-related ileitis. *Virchows Arch* 2016; **469**: 515–22.

24 Kaplan G. The global burden of IBD: from 2015 to 2025. *Nat Rev Gastroenterol Hepatol* 2015; **12**: 720–7.

25 Burisch J, Munkholm P. The epidemiology of inflammatory bowel disease. *Scand J Gastroenterol* 2015; **50**: 942–51.

26 Vind I, Riis L, Jess T, Knudsen E, Pedersen N, Elkjær M *et al*. Increasing incidences of inflammatory bowel disease and decreasing surgery rates in Copenhagen City and county, 2003-2005: a population-based study from the Danish Crohn colitis database. *Am J Gastroenterol* 2006; **101**: 1274–82.

27 Molodecky NA, Kareemi H, Parab R, Barkema HW, Quan H, Myers RP *et al*. Incidence of primary sclerosing cholangitis: asystematic review and meta-analysis. *Hepatology* 2011; **53**: 1590–9.

28 Stowe S, Redmond S, Stormont J, Shah A, Chessin L, Segal H *et al*. An epidemiologic study of inflammatory bowel disease in Rochester, New York Hospital incidence. *Gastroenterology* 1990; **98**: 104–10.