# A personal account of treating Myalgic Encephalomyelitis with Faecal Microbiome Transplant

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December 2020.

## Abstract

This is a report of my personal experience of the use of Faecal Microbiome Transplant to treat moderate ME, an illness for which treatment is not otherwise available. The outcome, recorded three months after treatment is very positive: nearly all symptoms have completely receded and no complications or side effects have occurred. The mechanism by which this phenomenon has occurred is unknown.

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## Foreword

***Unproven Interventions in Clinical Practice***

*In the treatment of an individual patient, where proven interventions do not exist or other known interventions have been ineffective, the physician, after seeking expert advice, with informed consent from the patient or a legally authorised representative, may use an unproven intervention if in the physician's judgement it offers hope of saving life, re-establishing health or alleviating suffering. This intervention should subsequently be made the object of research, designed to evaluate its safety and efficacy. In all cases, new information must be recorded and, where appropriate, made publicly available.*

Paragraph 37 from the World Medical Association Declaration of Helsinki on Ethical Principles for Medical Research[i].

## Introduction

This document is a personal account of my efforts to treat Myalgic Encephalomyelitis (ME) using Faecal Microbiome Transplant (FMT), written with the hope of helping others with ME. I feel two ethical obligations in creating this account: that I should be careful not to exaggerate the details, consequences and implications of my treatment BUT if my experience offers potential benefit to others then I have an obligation to disclose it freely.

I have tried to be objective but clearly it is not possible to be completely dispassionate about my own experience. Also, this is only a form of case report, albeit a personal one, and we are constantly reminded that "case reports do not constitute data". Finally, I must disclose that this report has not been peer-reviewed in any way. I fully accept these limitation and it is therefore both my duty as the author and your duty as the reader to show an appropriate scepticism to what is reported here.

The target for this report is physicians, scientists and influencers with an interest in ME, either clinically or in research.

## Disclosure

I have paid from my own income for the treatments described in this account. I have not received any incentives to undertake these treatments nor do I expect to do so.

## Competence

Given that I have encouraged the reader to show healthy scepticism about what I have reported here it is reasonable to ask what competence I have to write this report and critically analyse the findings. I have a Master's Degree in Biomedical Engineering Science (University of Dundee, 1997) and a PhD concerning medical robotics (University of Dundee, 2000). I worked as a research engineer in the University of Dundee's Department of Surgery and Molecular Oncology from 2000 until my retirement in 2012. Since then I have run the Fife ME Support Group and campaigned with #MEAction Scotland and other charities in the sector.

## Health prior to ME, pre-existing conditions and co-morbidities

I am a white married British male aged 53 at time of writing.

Since my mid-teens I have suffered from depression which is usually mild but occasionally moderate. For the last twelve or fifteen years my depression has been treated with prescription drugs which are generally very effective and I regard myself as free from symptoms of depression. I am currently using duloxetine and had been for a considerable time prior to my FMT treatment. I have never noticed any effect of antidepressant drugs on my ME symptoms. A component of my depression is Seasonal Affective Disorder for which reason I take Vitamin D supplements and employ a light box. SAD tends to make me inclined to sleepiness and this has been particularly evident this year.

For several years prior to the development of my ME I experienced IBS, characterised by a sudden urgent need to open my bowels. I have noted that this symptom can be worsened by alcohol, gluten, refined sugar and caffeine but eliminating these items from my diet does not consistently alleviate the condition and I continue to experience urgency approximately one day in three. After reporting my IBS symptoms to my General Practitioner (GP) I had a precautionary colonoscopy which achieved complete examination to the caecum and was negative for any evidence of colonic disease: this occurred sometime before the onset of my ME. I do not take any drugs to treat my IBS symptoms but I occasionally consult with a nutritionist in an attempt to optimise my diet and limit symptom severity – this effort has been of limited success. Note that under the direction of my nutritionist I eliminated refined sugar from my diet over a period approximately coincidental with the period of my FMT treatment. I have eliminated refined sugar from my diet several times in the past and any effect on

my ME symptoms has been very minimal or non-existent. As a result of my experience with IBS I have made great efforts to ensure a good diet: I do not eat gluten nor consume alcoholic or caffeinated beverages. My consumption of refined sugars is minimal and my diet is high in vegetables, nuts, fruit, cereals and pulses. Most of my meals are prepared from scratch. I consume some meat and fish but not every day.

Approximately four years ago I was identified with hypertension during a routine GP visit, with the greater problem being with diastolic pressure. This is now successfully managed with a combination of amlodipine and ramipril. In the period prior to my FMT treatment my resting heart rate was in the high 80s - low 90s BPM.

I have experienced problems with urination for about the last decade and benign prostatic hyperplasia was diagnosed approximately four years ago. This is treated with a combination of tamsulosin and oxybutynin.

The depression, hypertension and prostatic hyperplasia do not appear to have any connection with my ME. The IBS seems to have a weak link with ME in that changes to my diet occasionally had a weak influence on my ME symptoms during the early years of my illness (but not latterly). IBS is a common comorbidity in ME[ii].

## History of ME

My experience of ME symptoms had a sudden onset in September 2010 but without any obvious preceding viral infection. Prior to onset I had been fit and well (other than the issues mentioned above), was of normal BMI, did not smoke (and had never smoked) consumed only moderate alcohol and enjoyed strenuous exercise on a daily basis. The initial course of the illness was highly variable, presenting in cycles which started with severe symptoms and slowly moderated to mild symptoms. After approximately eighteen months of this cyclical presentation the disease became more consistently disabling and I retired from full time employment. At approximately this time I obtained a diagnosis of ME from a physician at Ninewells Hospital Dundee who was involved in running a trial treating ME with vitamin D. The disease proceeded for approximately four more years with mild to moderate symptoms but thereafter became more consistently moderate and more disabling. My symptoms have been largely stable for the last four years. My main symptoms throughout have been disabling fatigue which was severe during relapses, post exertional malaise, prolonged and unrefreshing sleep, cognitive impairment (particularly anomic aphasia) and breathlessness during severe episodes. I have never experienced Postural Orthostatic Tachycardia Syndrome or pain/fibromyalgia although these can be common in ME[iii] [iv]. I did not experience any particular worsening of depressive symptoms during the course of the illness.

## Description of treatment

Having noted an FMT study by Borody[v] which, although of poor quality claimed to be capable of delivering total remission of symptoms in some ME patients, I applied for treatment at the Taymount Clinic, Hertfordshire, England and was screened by telephone to ensure my suitability for the procedure. Two conditions were attached to the agreement to proceed: that I should inform my GP - with which I readily complied - and that I should undertake a suitable bowel preparation beforehand. Taymount Clinic offer three alternative protocols for bowel preparation: (i) a combination of laxative use and colonic irrigation; (ii) a combination of Moviprep and colonic irrigation; (iii) three consecutive daily colonic irrigations immediately prior to attending the clinic. Option (iii) was the most suitable for my circumstances and in early September 2020 I attended a nearby facility for three colonic lavages (colonic irrigation) each for a duration of thirty-five minutes, prior to travelling to Taymount. During my first appointment at the clinic I received a further and more thorough colonic lavage prior to delivery of the first implant. I attended the clinic for a further eight treatments (*i.e.* transplants 2 to 9) over a fortnight and administered the tenth transplant myself after returning home with it.

The donations were obtained from healthy adults who were screened for a range of infectious diseases. Each transplant was one of a set of ten from ten different donors to maximise the variety of flora over the program. The transplants were treated to remove mucus, dead cells and waste product so that the only content remaining was viable microbiota which was screened again for infectious agents then placed into liquid nitrogen for a three-month quarantine. When this had elapsed the transplants were thawed, re-tested for infectious agents and prepared for delivery. Delivery of the transplant was by a 300mm anal catheter, lubricated with natural oils and inserted so that it extended approximately 250mm into the sigmoid/descending colon and was followed by an injection of room-temperature sterile saline to wash the contents of the catheter into the bowel. After delivery the therapists gave the abdomen an external massage to encourage flow from the descending into the transverse colon and I was moved into a number of different positions on the couch for a period of ~60 minutes to encourage further progress of the transplant into the proximal colon. I did not experience any complications during delivery of the transplants and was able to retain all ten in my bowel for at least six hours each.

Note that I did not have any tests of any biomarkers in either bloods or stool before, during or after treatment.

## Outcome measures

This report is being written in December 2020 and thus provides a three-month review of the treatment. The outcome measures are as follows and are available for the three months preceding and following the FMT treatment:

1. Subjective wellbeing rating. Each day since the onset of my illness I have recorded my wellbeing three times a day (morning, afternoon and evening) on a self-devised scale in which "1" represents very unwell and confined to bed and "6" represents completely normal. I thus have a subjective record of severity for the entire duration of my illness and considerable experience in applying my own subjective judgements;
2. The number of steps taken each day, as recorded by my "Fitbit" wrist-worn pedometer. Where I know false results have accrued (*i.e.* as a result of cycling on a rough path) I have discarded the result;
3. Number of hours out of bed each day. This provides a simple but objective measure of health each day.

## Results

Table 1 records the average (mean) results of FMT treatment, comparing the three-month period preceding the treatment with the three months following treatment.

| | **3 months pre-treatment** | **3 months post-treatment** |
|---|---|---|
| **Self-reported wellbeing** | 3.1 | 4.8 |
| **Steps taken per day** | 5511 | 10540 |
| **Hours out of bed per day** | 7.2 | 11.9 |

*Table 1: Results of treatment averaged over three months.*

There was no measurable effect of the treatment on my depression, hypertension or prostatic hyperplasia but this is to be expected. Within 28 days of treatment I noted that my resting heartbeat had reduced from 80/90 BPM to approximately 60 BPM. Curiously, the FMT did not change my IBS symptoms although the introduction of healthy microbiota into the gut might reasonably be expected to do so. I have not experienced any side effect from the FMT and this is consistent with the reported experiences of others.

The only additional results to report are my personal observations. Prior to the treatment I was, on average, moderately ill although my symptoms fluctuated. Subsequent to the treatment I am almost

entirely and consistently symptom free: I do not suffer any abnormal fatigue, post exertional malaise or disturbed sleep. Although obviously unfit from prolonged illness I am able to exercise normally and my sleep is refreshing. I do not experience any breathlessness. The only remaining symptom is mild memory deficit and anomic aphasia which is irritating but not disabling. All other ME symptoms have entirely resolved.

## Confounding variables/placebo effect

Several factors probably partially confound the results in this case history:

1. Midway through November I experienced a brief respiratory viral illness for which I was obliged to take bedrest. This slightly depresses the results of the post-treatment results. The course of the illness was normal and I recovered without any prolonged issues: there was certainly no post-viral fatigue or other presage of ME;
2. The pre-treatment phase extended through the summer months while the post-treatment phase extended through autumn which happened to have been very wet in my area. This may have skewed the step count – I may have been more inclined to walk for exercise in the pre-treatment period and less inclined to walk in the post-treatment period;
3. As I noted above, my experience of SAD this year has been of excessive sleepiness.

All the above factors tend towards depressing the post-treatment outcomes and thus make the treatment appear less favourable than is otherwise the case.

There is an inevitable wish when expending one's personal income on treatment to view the treatment as a success: one does not wish to admit to wasting money. Thus there is psychological potential for bias towards viewing this treatment as a success (*i.e.* inflating post-treatment scores) and I cannot consider myself immune to such bias.

There is a long history of ascribing the symptoms of ME to a psychological disorder and it may be tempting to conclude that what I describe as ME and my subsequent recovery are all entirely psychological: this interpretation may be reinforced in the minds of some by the fact that I have experienced mental illness (depression) all my life. In general, the psychological model of ME has now been thoroughly rejected by progress on the research of organic causation. This is not the place to address this historical controversy but simply to note that the US Centre for Disease Control states "ME/CFS is a biological illness, not a psychologic disorder. Patients with ME/CFS are neither malingering nor seeking secondary gain"[vi]. It may be also accepted that I have suffered from a disease with organic causation but enjoyed a recovery which is primarily accounted for by a placebo effect and in the absence of a full medical investigation this cannot be discounted, however unlikely. The following are offered as reasons to believe that the placebo effect does not explain the recovery:

1. Although I am obviously very positive about the results I did not enter the treatment with a high expectation of success as I knew that Borody's paper, on which I had justified the treatment, was of poor quality. Thus my predisposition to suggestibility was low;
2. I have experienced what I regard as placebo effects previously in my efforts to manage my ME through supplements and dietary changes: changes I have made with the expectation of some success (*i.e.* high predisposition to suggestibility). The results have always been negligible and unsustained: any previous experience I have had of placebo effects has been inconsequential in terms of disease severity;
3. The change in my resting heart rate was an unexpected and unlooked-for result. There is some evidence that this change can be attributed to ME and thus unlikely to be a placebo effect but the evidence is very weak and the issue is under-researched[vii].

I find it highly unlikely that a change of the magnitude and consistency I have experienced is attributable to a placebo effect but it cannot be discounted.

## Discussion

Much of what I have reported here may be contested: this is a single case-study about the author in which at least some of the results are qualitative and all the results are very open to bias. The text has not been peer-reviewed nor could it be in any independent way. The mechanism of the primary pathology is not fully understood and the mechanism of the treatment is a complete mystery. I assert that only two facts are incontestable: I was once seriously ill and I am now in normal health.

ME is an illness in which symptoms must be managed and accommodated as best as possible but no competent authority on the disease would be reckless enough to suggest that it can be cured. Previous apparent breakthroughs in treatment, most recently the Rituximab trials in Norway[viii], have demonstrated that the road to success in this stigmatised and sparsely-funded disease is fraught with disappointing results.

Total remission of ME symptoms is very rarely reported in the literature: the ME Association estimate a recovery rate of 5% [ix (p104)]. The study by Borody is notable for identifying a 58% success rate 15-20 years post-treatment but it should be noted that the quality of this paper is poor[x]. A further paper by Kenyon also describes success in treating ME patients in qualitative terms[xi]. Nevertheless, given the rarity of any papers reporting significant success in treating ME, the Borody and Kenyon reports are highly unusual. Total remission of symptoms subsequent to treatment is unheard of. An attraction of FMT is that the treatment is accessible to the general public in the United Kingdom (unlike, say, Rituximab), is relatively cheap, carries no known side effects and the only associated risk is of infection from donated material (which is admittedly potentially fatal[xii]). Given that quality of life for ME patients is poor[xiii] and the drawbacks are minimal the attractions of FMT treatment for ME patients are self-evident, assuming the risk of infection is suitably managed by, for example, using a commercial clinic's transplant service.

I report persistent although very mild cognitive deficit especially anomic aphasia continuing after treatment. It is notable that a number of small studies have identified grey matter loss in ME patients[xiv] and I hypothesise that this may account for my cognitive issues.

This report does not - and should not - tackle two important questions: what is the pathology of ME and what is the mechanism by which the gut biome modifies the disease? I am not competent to address either.

It is important to note that in private correspondence I have had with two other individuals who have undergone FMT for ME both reported a brief remission of symptoms followed by a decline to previous levels.

## Conclusion and further action

The central personal conclusion is that after ten years of debilitating illness I am once again in more-or-less normal health. This is of course valuable to me but does little to advance the science.

A consistent message from any review of ME literature is that trials are small and of limited quality, not necessarily through any fault of the authors but on account of the paucity of funding. Gaps in knowledge are extensive. ME research needs more investment.

The attractions of FMT - its accessibility, low cost, absence of side effects and risks (which can be easily managed) make it attractive as a private treatment, especially given that the claims made by Borody, Kenyon and here, if valid, represent a very significant step in the treatment of ME. These attractions should, however, be equally appealing to research funders. There is much about a potential FMT trial which should commend itself to the scientific and funding communities.

[i] https://www.wma.net/policies-post/wma-declaration-of-helsinki-ethical-principles-for-medical-research-involving-human-subjects/

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[iv] http://sacfs.asn.au/download/consensus_overview_me_cfs.pdf

[v] Borody, Thomas J; Nowak, Anna; Finlayson, Sarah (Dec 2012). "The GI microbiome and its role in Chronic Fatigue Syndrome: A summary of bacteriotherapy". Journal of the Australasian College of Nutritional and Environmental Medicine. 31 (3): 3–8

[vi] https://www.cdc.gov/me-cfs/healthcare-providers/presentation-clinical-course/etiology-pathophysiology.html

[vii] https://www.meresearch.org.uk/research/reduced-cardiac-volumes/

[viii] Fluge, Øystein; Bruland, Ove; Risa, Kristin; Storstein, Anette; Kristoffersen, Einar K.; Sapkota, Dipak; Næss, Halvor; Dahl, Olav; Nyland, Harald (2011). "Benefit from B-lymphocyte depletion using the anti-CD20 antibody rituximab in chronic fatigue syndrome. A double-blind and placebo-controlled study". PloS One. 6 (10): e26358. doi:10.1371/journal.pone.0026358. ISSN 1932-6203. PMC 3198463 Freely accessible. PMID 22039471

[ix] ME/CFS/PVFS: An Exploration of the Key Clinical Issues; 12th Edition. Shepherd and Chaudhuri, The ME Association 2020. ISBN 978-0-95504909-8-8

[x] https://me-pedia.org/wiki/Fecal_matter_transplant#Critique_of_Borody_study_using_FMT_for_chronic_fatigue_syndrome accessed 11 December 2020.

[xi] J.N. Kenyon, Shelly Coe, Hooshang Izadi,A retrospective outcome study of 42 patients with Chronic Fatigue Syndrome, 30 of whom had Irritable Bowel Syndrome. Half were treated with oral approaches, and half were treated with Faecal Microbiome Transplantation, Human Microbiome Journal, Volume 13, 2019, https://doi.org/10.1016/j.humic.2019.100061.

[xii] FDA: Infections, 1 Death After Fecal Transplants: https://www.webmd.com/digestive-disorders/news/20190614/fda--infections-1-death-after-fecal-transplants#1 accessed 11 December 2020.

[xiii] Saarni, S.I., Härkänen, T., Sintonen, H. et al. The Impact of 29 Chronic Conditions on Health-related Quality of Life: A General Population Survey in Finland Using 15D and EQ-5D. Qual Life Res 15, 1403–1414 (2006). https://doi.org/10.1007/s11136-006-0020-1

[xiv] Puri, BK; Jakeman, PM; Agour, M; Gunatilake, KDR; Fernando, KAC; Gurusinghe, AI; Treasaden, IH; Waldman, AD; Gishen, P (2012), "Regional grey and white matter volumetric changes in myalgic encephalomyelitis (chronic fatigue syndrome): a voxel-based morphometry 3 T MRI study", British Journal of Radiology, 85 (1015): e270-3, doi:10.1259/bjr/93889091