CH-01 Diabetes after the Fall: type 1's honest prognosis, type 2's diet cure and the pre-insulin regimens

HEALING after the FALL, research library, CH-01

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How to read this report

Two diseases share one name. One kills a child in weeks and cannot be cured by anything a village can make. The other is often reversed by food alone. A book that blurs them will kill the first group with false hope and fail the second with fatalism.

ME-10 holds the clock for type 1 without insulin, the verdict on making insulin, the Allen mortality table and Galega officinalis. ME-09 holds insulin storage and the salvage ranking, FO-08 the rule for the last dose, HY-09 refeeding, and TR-10 the Mowanjum and Wai'anae studies (THRIVING library, ME-09, ME-10, FO-08, HY-09, TR-10). New here: diagnosis without a laboratory, the ketoacidosis nursing record, the arithmetic of stretching a vial, the plants and the complications. [VERIFY] marks what is unconfirmed or my own arithmetic.

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1. Two diseases under one name, and how to tell them apart without a laboratory

Type 1 is the destruction of the insulin-making cells, so the body makes none. It appears in children and young adults over days to weeks, the patient is thin and getting thinner, and the end is acidosis and coma. Type 2 is the loss of response to the body's own insulin, with fat in the liver and pancreas at the centre of it. It appears in middle age over years, the patient is usually heavy, and the end is a slow ruin of nerves, eyes, kidneys and feet. With no antibody test the village has age, body, speed and ketones, plus acanthosis nigricans, the velvety dark thickening in neck folds and armpits, which belongs to insulin resistance and never to type 1 (Smiley, Chandra and Umpierrez 2011).

The confusing middle. A large minority of adults arriving in ketoacidosis are not type 1. In series reviewed by Smiley, Chandra and Umpierrez (2011) the ketosis-prone type 2 share ran from 22 to 63 per cent in African American, Peruvian and Pakistani cohorts and under 10 per cent in people of European and East Asian descent. They are obese, have no autoantibodies, and recover insulin function, 42 per cent in remission at a mean of 83 days in one cohort and about 70 per cent by nine weeks in another. An overweight adult in ketoacidosis with dark skin folds may need insulin only for a season. A thin child is not that case.

Finding the disease. Thirst, large volumes of urine, weight loss and exhaustion are the classic four, and sugar in the urine confirms them. The kidney spills glucose only above the renal threshold, about 10 millimoles per litre in an adult, under about 8.3 in pregnancy, and up to about 16.7 once the kidney is damaged, so a person with kidney disease can be severely hyperglycaemic with a clear urine (NCBI Bookshelf, Clinical Methods, and Altmeyer's entry on the renal threshold). Urine testing finds diabetes well and steers treatment badly.

Benedict's test. Sugar reduces copper held in solution by citrate in alkaline carbonate, and blue turns green, yellow, then brick red. Two independent sources give the same proportions, set out with the colour bands in Protocol 2 (HiMedia technical data sheet R002, and the Benedict's reagent entry at Wikipedia, both tracing to Benedict 1909, which I did not read, the colour bands from a teaching source, therefore [VERIFY]). It keeps for years stoppered, and it is not specific for glucose: lactose, galactose and large doses of vitamin C also reduce it.

Ketones. Before any test, the smell was the sign. In 1857 Wilhelm Petters of Prague isolated acetone from the urine of a woman of 30 whose room smelled so strongly of chloroform that his assistant thought she had been anaesthetised in the night (Tattersall 2012). A fifth to a third of people cannot smell acetone at all, so a village should find out early which of its people can, and name them. Smell screens, and never rules out.

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2. Type 1 after the Fall: the untreated course and the honest prognosis

ME-10's clock stands, six hours from stopped insulin to ketosis in a rested adult (THRIVING library, ME-10). The course is thirst and polyuria for days to weeks, weight falling off visibly, vomiting and abdominal pain, then the deep sighing breathing Adolf Kussmaul described in 1874, then drowsiness, stupor and coma. In 1884 Oskar Minkowski named the acid responsible as beta-hydroxybutyric (Tattersall 2012).

How long. Weeks to months in a child, months to a year or two in an adult with some pancreas left. No trial exists, because no one withholds insulin. The evidence is what happens where insulin is unaffordable, and Beran, Yudkin and de Courten's Rapid Assessment studies in Mozambique and Zambia are the primary work (Beran, Yudkin and de Courten 2005, abstract read). Secondary accounts put life expectancy after diagnosis in rural Mozambique at about seven months and about four years for a child in a town with a hospital (search summaries, [VERIFY], primary figures not reached). ME-10's citation of Yudkin 2000 gives the same order.

Written plainly. A child of eight with type 1 diabetes and no insulin will die, and will probably die within a year of the day the thirst began, often within a few months. A district of 3,000 cannot make insulin. No diet, no fast, no plant and no technique in this book will change that. The one serious attempt to extend such a life by diet bought about a year, in children, at the cost described in section 3. Say that early, in those words, then say what can be done, which is a great deal: the time can be made longer and far less frightening, and the death need not be agonised.

The one honest piece of good news. Adults diagnosed after about 30, not emaciated and not ketotic at diagnosis, often have residual insulin for years, and for them a low-carbohydrate diet with enough calories is worth doing. Look not at the label but at the ketones and the wasting (THRIVING library, ME-10).

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3. The pre-insulin regimens: Bouchardat, Naunyn, Allen, Joslin

What the tradition said

Carbohydrate restriction is older than germ theory. John Rollo prescribed meat and fat in 1797. Apollinaire Bouchardat, a Paris pharmacist, built the first clinical system in the mid-nineteenth century: starch and sugar forbidden, the patient testing his own urine, vigorous exercise, and occasional fast days. He is also credited with noticing that sugar vanished from the urine of the hungry (Lennerz and colleagues 2021, and the Bouchardat entries at Wikipedia and historyofmedicine.com, all search summaries). The story that he saw this during the siege of Paris in 1870 to 1871 is repeated everywhere and I found it in no source I read, so the book should not print it as fact [VERIFY]. Bernhard Naunyn restricted protein too and let fat carry the energy, publishing Der Diabetes mellitus in 1898. Frederick Allen at the Rockefeller Institute took the logic to its end: if the pancreas is failing, rest it. Fast until the urine is sugar-free, then feed the smallest diet that holds that state. He began in humans in 1913 or 1914, and Elliott Joslin adopted the method in 1916.

What the record showed

Allen's 1919 monograph holds the records of 76 of 100 diabetic patients at the Rockefeller Hospital. ME-10 reports Mazur's reading: 43 per cent dead by late 1917, four of inanition, with mortality by February 1918 of 75 per cent at ages 0 to 9, 71 per cent at 10 to 19, 64 per cent at 20 to 29 and 18 per cent at 50 and over (THRIVING library, ME-10). A secondary essay gives 46 per cent dead among 41 patients treated from 1914 to 1917 and 78 per cent among those under 20 (CrossFit Essentials). There were no untreated controls, and Allen's preface says the method "has never been proposed as such a cure".

The case records matter more than the tables, because they say what the deaths were like. Case 1 was kept alive about three years and died of "simple wasting and asthenia". Case 4, a boy of 12, lost 5.8 kilograms during four months of treatment and his death "may be attributed to inanition". Case 30, a woman of 45, died on 9 February 1915 of diabetic coma during a fast, the first case at the Institute to develop fatal acidosis on fasting. Case 26, a girl of 14, died in February 1918 after her parents tried to build her up at home (Allen, Stillman and Fitz 1919, case records read). Those four are the whole harm test in the author's own words.

Deaths among Joslin's patients within ten years of onset ran at 824 per 1,000 in the Naunyn era of 1897 to 1914, 386 in the Allen era of 1914 to 1922, and 61 in 1922 to 1926 once insulin arrived (NIDDK, Diabetes in America, search summary, and THRIVING library, ME-10). He had lost 62 patients under 15 by 1916, every one to coma, and of 169 children treated between 1914 and 1922 a third were still living, which he called not a cure but "coma deferred". Three of his patients starved to death in his first year using the method (CrossFit Essentials).

Elizabeth Hughes, daughter of the United States Secretary of State, was diagnosed in 1919 aged 11 at about 75 pounds. Allen fasted her a week, then held her near 500 kilocalories a day with a weekly fast day. She reached Toronto in August 1922 weighing about 45 pounds and barely able to walk, gained ten pounds in five weeks on insulin, and lived to 73. Sources give her allowance variously as 300, 400, 500 and 800 kilocalories (Wikipedia, University of Toronto insulin collection, search summaries). The spread is the lesson: the regimen was improvised around a starving child.

What modern science infers

The diet worked by removing the substrate, so the small residual insulin went further. In mild older cases, whom we would now call type 2, it worked well, and those patients inflate every pre-insulin survival figure. In children with no insulin it delayed the coma and substituted wasting. **Grade B** for adults with residual insulin, on uncontrolled clinical series. **D** as treatment for absolute insulin deficiency and **X** as a regimen imposed on a child, because the record shows deaths from inanition and from fasting-induced acidosis with no convincing survival gain against them.

The descendant worth more. Lennerz and colleagues surveyed 316 people with type 1 eating very low carbohydrate: mean intake 36 plus or minus 15 grams a day, mean HbA1c 5.67 plus or minus 0.66 per cent against a general type 1 average near 8.2, with insulin doses below average (Lennerz and colleagues 2018, search summaries, full text not read). The respondents were self-selected enthusiasts and the authors said so. What matters here is the dose, and that unlike Allen's regimen this holds the calories up with fat and protein.

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4. Salvaged insulin: how long it keeps, how far it stretches, how a dose is judged

Heat

ME-09 gives the Cochrane summary and the storage plan (THRIVING library, ME-09). Kaufmann's primary paper adds one qualification that changes practice. Kaufmann and colleagues logged temperatures at Dagahaley camp in northern Kenya, found a 12-hour cycle between 25 and 37 degrees, and reproduced it in the laboratory. Under that oscillating cycle every insulin held potency: 98.4 to 103.3 per cent at four weeks and 99.4 to 102.7 per cent at twelve weeks, against a pharmacopoeia limit of 100 plus or minus 10. But held at a constant 31 degrees, about the median of

that cycle, the same insulins lost 5 to 13.5 per cent at four weeks and 12 to 19 per cent at eight, outside the limit, and at a constant 37 degrees a rapid-and-NPH suspension lost 15.4 per cent in a single week (Kaufmann and colleagues 2021, read in full).

Insulin survives a hot day with a cool night far better than a steady warm room. A shaded verandah swinging from 20 at night to 33 at noon beats a cupboard that sits at 30 day and night. The authors cite Gill 2000 for pit storage and Vimalavathini, who kept insulin in mud pots at a mean of 26 degrees with potency like 25-degree storage and advised a two-week in-use period where cool storage cannot be assured. ME-09's extrapolation to two or three years in a cool cellar remains unmeasured and stays [VERIFY].

Arithmetic

An adult on 40 units a day needs about 14,600 units a year, roughly fifteen 1,000-unit vials (THRIVING library, ME-09). A strict low-carbohydrate diet might cut the daily total by a quarter to a third, turning fifteen vials a year into ten or eleven [VERIFY, my inference from Lennerz 2018 and standard split regimens, not measured]. That is not a cure. It is the difference between three years and four.

Judging a dose without a meter

Benedict's reagent, modified, was the mainstay of urine monitoring for over fifty years, and Joslin's whole teaching was diet, exercise and frequent urine testing aimed at a sugar-free urine (Frontiers Partnerships reprint of the British Journal of Biomedical Science history article). The limits were known then. Urine glucose lags the blood by hours, cannot show a falling glucose and so cannot warn of hypoglycaemia, and is bounded below by the renal threshold. A persistently negative urine means only that blood glucose stayed under about 10 millimoles per litre, which could be good control or an imminent convulsion, and the one modern written protocol for dosing by urine that I found, the American Animal Hospital Association's feline guidance, calls it a last resort for that reason. **The rule for the book:** use urine sugar to titrate upward and never downward. Increase insulin when the urine is heavily positive before meals on several days running. Reduce insulin for symptoms, not for a negative urine. Keep sugar within arm's reach of the patient at all times.

Regimen. With only NPH and regular insulin, the split-mix is what the world used: two thirds of the daily dose in the morning and one third in the evening, NPH to regular about 2 to 1 in the morning and 1 to 1 in the evening, starting at 0.1 to 0.3 units per kilogram per day in children and 0.3 to 0.5 in adults (Texas Department of State Health Services insulin algorithm). It demands fixed meal times and fixed portions.

The concentration trap. Human insulin is U-100, veterinary insulin often U-40. A U-40 syringe filled to its 10-unit mark holds 0.25 millilitres, which is 25 units of U-100, a two-and-a-half-fold overdose [VERIFY, my arithmetic, since several web pages state the direction backwards]. One concentration per village store, the syringe matched to the vial, the vial read aloud before every draw.

Rationing

FO-08 gives the rule for the last dose and ME-09 names the allocation question as the owner's. Two numbers belong beside them. In Tigray under siege, paediatric diabetes admissions at Ayder hospital rose from 56 with no deaths before the war to 87 with 9 per cent case fatality, with running out of medicines cited in 24 per cent (THRIVING library, FO-08, citing Gebrekidan 2024). In a rich country with no siege at all, 25.5 per cent of 199 patients at the Yale Diabetes Center reported cost-related insulin underuse in one summer (Herkert and colleagues 2019). People ration insulin quietly and badly when the rule is not written down.

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5. Ketoacidosis without insulin: what the nursing record shows

Before insulin, coma was fatal. The period's treatments were purgation, alkaline enemas, intravenous bicarbonate and hypodermic strychnine. "Despite heroic measures ... coma was always fatal" (Tattersall 2012). None of it works. Do not give alkali, do not give strychnine, do not purge.

After insulin, nursing was still most of it. Joslin, Root and White reviewed their first 33 cases treated with insulin between 1 January 1923 and 1 April 1925. Thirty-one survived. They credited "promptly applied medical care. Rest in bed, special nursing attendance, warmth, evacuation of the bowels by enema, the introduction of liquids into the body, lavage of the stomach, cardiac stimulants, and above all the exclusion of alkalis" (Joslin, Root and White 1925, quoted in Tattersall 2012). Between 1930 and 1945 ketoacidosis mortality in many American hospitals ran at 20 to 40 per cent against under 5 per cent at Joslin's, and the difference was organisation: at the Massachusetts General, outcomes improved in 1944 when each case was made an emergency, a house officer stayed in constant attendance for the first 24 hours, and a coma sheet on the wall recorded pulse, fluid in and out, and urine sugar and acetone every 30 to 60 minutes (Harwood 1951, via Tattersall 2012).

The route for fluid. Joslin accepted these patients were profoundly dehydrated and still called intravenous rehydration "too risky. Too sudden a burden is thrown on a weak heart". Fluid went rectally, or more often subcutaneously, subpectorally or into the thighs (Tattersall 2012). A village that cannot find a vein can still give fluid. IN-04 gives hypodermoclysis at about 1 millilitre a minute per site, up to 3 litres in 24 hours over two sites, contraindicated in shock, and WO-08 gives proctoclysis at about 250 millilitres an hour (THRIVING library, IN-04, WO-08). These routes are for the dehydrated patient who still has a circulation, not for the collapsed one.

Potassium, and the second day. In 1946 Jacob Holler described a woman of 18 who had taken no insulin for five days, came round after four hours of treatment, and by 21 hours could hardly breathe or move. Potassium chloride intravenously restored her breathing within twenty minutes. Bellet and Dyer had already found electrocardiographic changes in all of 17 coma and six pre-coma cases in 1937, worst not at the height of the acidosis but 24 hours later, when the patient seemed recovered. Alexander Cooke in 1948 described a woman recovering from coma who "asked for a drink and on being sat up to receive it fell back dead", and proposed that such a patient be nursed as a cardiac case for at least a week (all via Tattersall 2012). That is the village rule and it costs nothing: **the second day is the dangerous day, and the recovering patient stays flat.**

Prevention beats everything. Joslin held that ketoacidosis was preventable and that when it happened it was someone's fault. He cited Alfred Gottschalck in Stettin, a city of 270,000, with one case of ketoacidosis in 1928 to 1930, 0.3 per cent, against 3.6 to 16 per cent in other major German cities, achieved by systematic education of patients and doctors, follow-up of everyone discharged, and social help with nursing, food and insulin (via Tattersall 2012). A village cannot make insulin. It can run Stettin's system, and a named person who knows every diabetic, visits them when they are sick, holds the stock and the log and is woken at night is worth more than most of the pharmacy. Where organisation is poor, case fatality stays high even with insulin: 21.7 per cent in a Cameroon series and 12 per cent in Shashemene, against 0.67 per cent in developed countries (search summaries).

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6. Type 2 after the Fall: the diet cure, with its numbers

Cuba. The crisis of 1991 to 1995 cut daily energy intake from 2,899 to 1,863 kilocalories a head and raised physical activity, and the population lost 4 to 5 kilograms. Diabetes incidence fell 53 per cent and diabetes mortality 50 per cent. As the economy recovered the weight returned, prevalence rose 116 per cent, incidence 140 per cent, and diabetes mortality rose 49 per cent (Franco and colleagues 2013, secondary summaries only, full text blocked, and Franco and colleagues 2007). The crisis also caused an outbreak of neuropathy affecting about 50,000 people in 1992 to 1993, probably from vitamin deficiency. The experiment was not benign. Under siege in Sarajevo, of 279

people with type 2 diabetes, 55 no longer needed their usual treatment, with mean weight loss of 11.7 kilograms (THRIVING library, FO-08).

Newcastle. Lim, Hollingsworth, Aribisala, Chen, Mathers and Taylor put eleven people with type 2 diabetes of up to four years' duration on about 600 kilocalories a day for eight weeks. Mean weight loss was 15.3 plus or minus 1.2 kilograms. Fasting glucose fell from 9.2 to 5.9 millimoles per litre in the first week and was 5.7 at eight weeks, HbA1c fell from 7.4 to 6.0 per cent, liver fat from 12.8 to 2.9 per cent, pancreatic fat from 8.0 to 6.2 per cent, and first-phase insulin response rose from 0.19 to 0.46 nanomoles per minute per square metre. Twelve weeks after the diet ended, seven of the ten retested remained non-diabetic (Lim and colleagues 2011, read in full). The authors list the limits: eleven people, short duration of diabetes, no long-term data.

DiRECT. Total diet replacement at about 825 to 853 kilocalories for three to five months in primary care, stepped food reintroduction, diabetes drugs withdrawn at the start. Remission off all drugs was 46 per cent at one year against 4 per cent of controls, and 36 per cent at two years, with weight the whole story: 86 per cent remission in those losing 15 kilograms or more, 57 per cent at 10 to 15, 34 per cent at 5 to 10, 7 per cent at 0 to 5 (Lean and colleagues 2018, via THRIVING library, ME-10). DiRECT-Extension followed 95 remaining participants to five years. Of those in remission at two years, about 23 to 26 per cent were still in remission at five, and across all participants with five-year data overall remission was 13 per cent (Glasgow and Newcastle press releases 2024, the discrepancy unresolved [VERIFY]).

What a village takes from it. The Fall will do to a village what the Special Period did to Cuba, and many type 2 diabetics will simply stop having diabetes within a year or two. The healer's job is not to achieve this but to manage it safely. Anyone on a sulfonylurea or insulin must come down as the food reduces or they will have hypoglycaemia, which is the commonest avoidable death in this report. HY-09's refeeding rules apply if the loss goes too far, and the micronutrient failures of the Cuban crisis are a real cost (THRIVING library, HY-09). TR-10 shows the same result achieved on better food, at Mowanjum and in Hawai'i, and those are the models to copy, because they are food systems rather than deprivation (THRIVING library, TR-10).

Exercise is a separate effect. The Da Qing trial randomised 33 clinics in China from 1986, giving 577 adults with impaired glucose tolerance diet, exercise or both for six years, and followed 540 of 576 for 30 years. The headline incidence reduction and life-expectancy gain I could not confirm from a source I read, so they are not quoted. A 2024 post hoc analysis found that those free of diabetes at year four had 26 per cent lower long-term all-cause mortality, hazard ratio 0.74 with interval 0.57 to 0.97.

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7. Salvaged tablets, and the hypoglycaemia they cause

Metformin. The one oral drug worth hoarding. Start 500 or 850 milligrams once daily with or after food, maximum 2 grams a day in two or three divided doses with meals (UK summary of product characteristics). Lactic acidosis is rare, about 3 to 10 cases per 100,000 person-years, with 6.3 per 100,000 patient-years in users against 7.8 in non-users in a pooled analysis of 206 studies. European labelling caps the dose by kidney function, contraindicated below an estimated filtration of 30. A village cannot measure filtration, so the proxies are age, known kidney disease and above all dehydration. **Stop metformin in anyone vomiting, with diarrhoea, feverish or unable to drink**, and restart when they are eating and drinking normally.

Sulfonylureas. Glibenclamide, glipizide and gliclazide squeeze insulin out of the remaining cells and cause hypoglycaemia, which kills. Glibenclamide was the worst in a Tennessee Medicaid cohort of people aged 65 and over, at 16.6 serious hypoglycaemic events per 1,000 person-years against 3.5 for tolbutamide (Shorr and colleagues, via secondary summaries). A meta-analysis of 21 studies found an 83 per cent higher risk with glibenclamide than other sulfonylureas, with age over 65 carrying a relative risk of 1.27, interval 1.06 to 1.51. These are the drugs most likely to kill their user after the Fall, because the food supply falls and the dose does not. Halve or stop at the first sign of weight

loss, missed meals or illness. Never in a thin old person, and never where food is uncertain, which after the Fall is everyone.

Treating hypoglycaemia. Fifteen grams of fast sugar, wait fifteen minutes, reassess, repeat if needed. Fifteen grams is about one tablespoon of sugar, honey or syrup, or about 120 millilitres of fruit juice. The rule is standard but weakly evidenced, and one hospital study found only 34.5 per cent of patients at about 2.2 millimoles per litre reached 3.9 after a fixed oral 15 grams [VERIFY as to the underlying trials]. For an unconscious patient, honey or sugar paste smeared inside the cheek is the village method, and nothing liquid goes into an unconscious mouth. A sulfonylurea hypoglycaemia relapses for 24 to 72 hours after the first recovery, so the patient must be fed repeatedly and watched [VERIFY, standard practice, not from a source I read].

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8. The plants claimed for sugar

Fenugreek, Trigonella foenum-graecum L., Fabaceae, seed. An annual to about 60 centimetres with trifoliate leaves and a sickle-shaped pod, cultivated from the Mediterranean to India. The seed is a kitchen spice and the tradition across Arabia, India and the Levant gives it for the sweet urine. Neelakantan and colleagues pooled ten trials of at least a week, 278 participants, and found fasting glucose down 0.96 millimoles per litre with 95 per cent confidence interval 1.52 to 0.40 and HbA1c down 0.85 per cent with interval 1.49 to 0.22. Dose mattered: below 5 grams a day the fasting effect was nil, at 5 to 10 grams it was 1.27 millimoles per litre and above 10 grams 2.07. The authors judged most trials of low quality, none describing randomisation or allocation concealment, with heterogeneity at 80 per cent and possible publication bias (Neelakantan, Narayanan, de Souza and van Dam 2014, read in full). **Grade B minus**, the only plant I would carry. It is a food, it grows easily, the dose is a kitchen measure and the harm is wind, but it is no substitute for losing weight and useless in type 1.

Bitter melon, Momordica charantia L., Cucurbitaceae, fruit. A climbing vine with deeply lobed leaves and a warty oblong fruit, eaten across South and East Asia and the Caribbean, where it is given for sugar on the traditional logic that bitterness cuts the sweet. The Cochrane review found four randomised trials of up to three months and 479 participants, no significant difference against placebo and none against metformin or glibenclamide, with risk of bias generally high and no two trials using the same preparation (Ooi, Yassin and Hamid 2012). **Grade C for the tradition, D as treatment.** The seeds and red aril are reported to cause vomiting in children and the plant is traditionally avoided in pregnancy [VERIFY, no primary source reached].

Gymnema sylvestre R.Br., Apocynaceae, leaf. A woody climber of India and Sri Lanka, called gurmar, the sugar destroyer, because chewing a leaf abolishes the taste of sweetness for an hour or more. That effect is real and is the basis of the tradition. The clinical evidence is not. A systematic review found ten controlled studies, 419 adults with type 2 diabetes, only two randomised, outcomes reported only against baseline, with pooled fasting glucose falling about 1.57 milligrams per decilitre, which is nothing (Drugs.com monograph summarising that review). **Grade D**, though a plant that makes sweet things taste of nothing might help someone give up sugar.

Cinnamon, Cinnamomum verum J.Presl and Cinnamomum cassia (L.) J.Presl, inner bark. The Cochrane review pooled ten randomised trials, 577 participants, risk of bias high or unclear in all but two, and found no significant difference in HbA1c, insulin or post-meal glucose (Leach and Kumar 2012). The harm is the reason to name it. Cassia is rich in coumarin, hepatotoxic in a dose-dependent and idiosyncratic way, and the European Food Safety Authority and the German Federal Institute for Risk Assessment both set a tolerable daily intake of 0.1 milligrams per kilogram, which is 6 milligrams a day for a 60-kilogram adult. The widely quoted cassia content of 1 to 12 milligrams of coumarin per gram, against about 0.004 for Ceylon, comes from a commercial source I could not confirm against a regulator [VERIFY]. **Grade X at therapeutic doses of cassia, D at any dose for benefit.**

Goat's rue, Galega officinalis L., Fabaceae. ME-10 covers it: grade D as medicine and X as a dose, metformin's ancestor with a far worse margin, lethal to sheep with no treatment, toxins active in dried hay, worst in flower and

fruit, confusable with the vetches (THRIVING library, ME-10). Carry it as history and as a poisonous plant to recognise.

One plant, fenugreek, reaches a weak B at 10 grams of seed a day. The rest are D or worse, and none touches type 1. **The real harm is not toxicity but delay**, the point TR-10 makes across the Pacific region (THRIVING library, TR-10).

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9. Complications: feet, eyes, kidneys, infection

Feet. Where the village can do most for least. Lifetime ulcer incidence in diabetes is estimated at 19 to 34 per cent, and about 40 per cent of ulcers recur within a year of healing (Armstrong, Boulton and Bus 2017, secondary summaries). The 2023 IWGDF guideline recommends annual screening for neuropathy and peripheral artery disease even at lowest risk, therapeutic footwear at moderate-to-high risk, treatment of every pre-ulcerative lesion, and teaching patients never to walk unprotected (Bus and colleagues 2023, abstract read, the practical guideline PDF not machine-readable). The village version costs only discipline, and is in Protocol 6. WO-14's wound rules govern the ulcer itself (THRIVING library, WO-14).

Eyes. In the Wisconsin study of 955 insulin-taking people with type 1 diagnosed before 30, the 25-year cumulative progression of retinopathy was 83 per cent and progression to proliferative retinopathy 42 per cent (Klein and colleagues, WESDR reports XXII and XXIII). There is no treatment after the Fall, only prevention, and what the village owes a person going blind.

Kidneys. Krolewski and colleagues followed 292 people with juvenile-onset type 1 for 20 to 40 years. Once persistent proteinuria appeared, renal failure almost always followed, and half reached it within ten years (Krolewski, Warram, Christlieb, Busick and Kahn 1985, secondary summaries). The village cannot measure albumin and cannot dialyse. It can see frothy urine, swelling of ankles and face and rising breathlessness, control blood pressure with salt restriction and weight, and tell the truth.

Infection. Diabetes roughly triples the risk of active tuberculosis. Jeon and Murray's meta-analysis of 13 observational studies, 1,786,212 participants and 17,698 tuberculosis cases, found a pooled relative risk of 3.11 with 95 per cent confidence interval 2.27 to 4.26 in cohort studies (Jeon and Murray 2008, with the published correction of August 2008). Any diabetic coughing for more than two or three weeks is a tuberculosis suspect. Infections run hotter and heal slower, and an infection will push a type 1 patient into ketoacidosis on a stable dose, which is why insulin requirements rise during illness rather than fall.

Evidence grades

Remedy or practice	For	Grade	Basis
Salvaged insulin, rationed	Type 1	A	Standard of care, Kaufmann 2021 for heat
Low-carbohydrate diet, calories held up	Stretching insulin	C	Lennerz 2018, survey
Allen fasting diet	Adults with residual insulin	B	Allen 1919, Joslin, no controls
Allen fasting diet	Type 1 children	X	Inanition and fasting acidosis, Allen cases 1, 4, 30

Remedy or practice	For	Grade	Basis
Joslin nursing package with insulin	Ketoacidosis	B	31 of 33 survived 1923 to 1925
Subcutaneous or rectal fluid	Dehydration in ketoacidosis	B	Joslin's practice, IN-04, WO-08
Alkali, strychnine, purging	Ketoacidosis	X	Tattersall 2012, all failed
Energy restriction to remission	Type 2	A	Lim 2011, Lean 2018, DiRECT-Extension
Metformin, salvaged	Type 2	A	Lactic acidosis rare
Sulfonylureas, salvaged	Type 2	A for glucose, X in famine	16.6 severe events per 1,000 person-years
Fenugreek seed, 10 g a day	Type 2	B minus	Neelakantan 2014, poor trial quality
Cassia cinnamon, therapeutic dose	Type 2	X	No benefit, coumarin liver risk
Daily foot inspection and footwear	Complications	A	IWGDF 2023
Urine sugar for dose titration	Type 1	C upward, X downward	Cannot see a falling glucose

Means ledger

Rung	Type 1	Type 2	What it changes
Hands and knowledge	The four symptoms, the acetone smell, keeping the patient flat and warm, the truth	Naming it, teaching walking and less food, stopping tablets	Nothing for type 1. Most of the type 2 cure
The hearth	Sugar for hypoglycaemia, salted fluid by mouth, a cool shaded place for a vial, fixed small meals	The whole diet cure, weighed on a home balance	Type 1 unchanged. Type 2 remission in about half at a year if 15 kg goes
Garden and still	Fenugreek grown and dried, a scale, boiled water, Benedict's reagent if copper sulfate can be found	Fenugreek 10 g a day as an adjunct	Diagnosis becomes objective. Marginal glucose effect
Village dispensary (300)	Insulin rationed by written rule, a log, a named keeper woken at night, syringes, urine testing, subcutaneous fluid	Metformin salvage, a weight programme, a foot clinic	Type 1 life in years rather than months while stock lasts. Type 2 remission made deliberate

Rung	Type 1	Type 2	What it changes
District infirmary (3,000)	Ten beds, continuous nursing of a coma case, a coma sheet, intravenous fluid, pooled stock, an allocator who is not the healer	The drug store, training of village healers	Ketoacidosis mortality from near-certain to perhaps the 20 to 40 per cent of 1930s America
Hospital before the Fall	Pumps, monitors, intensive care, dialysis, laser, revascularisation	Drugs, surgery, screening	Near-normal life expectancy for type 1. Remission still needs the diet

For type 1 the ledger has one lever, the number of vials. For type 2 the ledger is almost flat, because the treatment is food and walking.

Harm test

The Allen diet kills by starvation. Allen's case 4, a boy of 12, lost 5.8 kilograms in four months and died of inanition, case 1 died of wasting and asthenia, and three of Joslin's patients starved to death in his first year with the method. Never impose it on a child, a pregnant woman or anyone already thin. It is going wrong when weight keeps falling after the urine is clear, with loss of muscle in thighs and shoulders, cold, apathy and a slow pulse. Stop and feed, under HY-09's refeeding rules (THRIVING library, HY-09).

Fasting. Allen's case 30 died of ketoacidosis during a fast on 9 February 1915. Never fast anyone with ketones on the breath, anyone vomiting, or anyone thin and young.

Insulin kills by hypoglycaemia in minutes to hours. Risk rises with a new lot of uncertain potency, a missed meal, a hard day's work, a smaller body, and any change of concentration or syringe. The signs are sweating, trembling, hunger, confusion and aggression, then convulsion and coma, with no acetone and warm moist skin rather than the dry skin of acidosis. Treat with 15 grams of sugar, or paste inside the cheek if unconscious. Never give insulin to a drowsy patient without first excluding hypoglycaemia, because that mistake is irreversible in one direction and recoverable in the other.

Sulfonylureas kill the same way and for longer, at 16.6 serious events per 1,000 person-years for glibenclamide in the over-65s, and the recovered patient relapses for one to three days. Never in the old and thin, never where meals are uncertain, halve at the first missed meal. **Metformin** causes lactic acidosis rarely, at 3 to 10 per 100,000 person-years, triggered by dehydration and kidney failure, and looking like ketoacidosis without the acetone. Stop it for vomiting, diarrhoea, fever or inability to drink.

Metformin. Lactic acidosis, rare at 3 to 10 per 100,000 person-years, triggered by dehydration and kidney failure: deep breathing, vomiting, abdominal pain and cold mottled collapse, looking like ketoacidosis without the acetone. Stop for vomiting, diarrhoea, fever or inability to drink, and never give it in advanced kidney disease.

Benedict's reagent. Copper sulfate is a poison and boiling urine spits, so label the bottle POISON and keep it from children. **Cassia cinnamon** injures the liver above about 6 milligrams of coumarin a day in an adult, which a therapeutic spoonful may exceed several times over.

Cassia cinnamon. Liver injury from coumarin above about 6 milligrams a day in an adult, which a therapeutic spoonful may exceed several times over.

The deepest harm is delay. Any thin young person with thirst, weight loss and acetone breath goes straight to whoever holds the insulin, and no plant is tried first.

Red flags

1. **A child or young adult with thirst, weight loss, vomiting, deep sighing breathing and acetone breath, and no insulin.** Death in hours to days. Do anyway: salted fluid by mouth, or subcutaneously if they cannot swallow and still have a circulation. Flat, warm, no alkali. Tell the family what is coming and that the coma itself is not painful.
2. **The same child with insulin available.** The one case where the district's whole effort is justified, because it is reversible. Nurse as Joslin nursed, by Protocol 4.
3. **A patient who has come round from coma and is sat up.** Cooke's patient fell back dead. Flat for a week.
4. **A diabetic foot with a black toe, a foul smell or red streaks.** Gangrene, fatal without surgery. Amputation at the infirmary, or comfort care.
5. **A diabetic coughing for more than three weeks.** Tuberculosis, risk tripled.
6. **Frothy urine with swelling of face and ankles and breathlessness lying flat.** Kidney failure. Reduce salt, sit them up.
7. **Sudden loss of vision in one eye, or a diabetic who stops passing urine.** No treatment exists for the first. The second is either profound dehydration, which is treatable, or kidney shutdown, which is not.
8. **Anyone on a sulfonylurea found confused, sweating or convulsing.** Reversible in minutes with sugar, then watched for 72 hours.
9. **A known type 1 patient whose last vial is empty.** FO-08's rules for the not-chosen apply (THRIVING library, FO-08). Honest comfort means saying the date, not hiding it.

The case against

Against the type 1 prognosis. Critics would say pre-insulin series mix type 1 and type 2 and carry survivor bias, that the African life-expectancy figures measure the absence of a health system rather than of insulin, and that nobody has measured untreated type 1 survival in a well-nourished, well-nursed population. All true, and it cuts both ways. The direction is not in doubt: Joslin lost 62 children under 15 and every one died in coma.

Against the Allen diet's reputation. It rests on uncontrolled series by the method's inventors, and Allen himself said the results were below the ideal. The honest reading is that it bought months to a year in children at the cost of hunger and wasting, and that the good figures belong to older, milder patients who were probably type 2.

Against the remission story. The criticism of Lim 2011 came at once: eleven people, an extreme intervention, and the observation that not everyone can stick to it. DiRECT answers the second with a primary-care programme and DiRECT-Extension the first with the figure nobody quotes, 13 per cent still in remission at five years. After the Fall the criticism loses most of its force, because the thing DiRECT had to manufacture, a sustained large energy deficit, will be the condition of life.

Against the plants. Defenders argue that Cochrane's standardisation demands are unfair to whole-plant medicines and that convergent traditional use counts for something. Ooi and colleagues themselves say preparation variability is why they did not pool. The answer is that a plant which cannot be dosed cannot be prescribed, and the harm of a plant that does not work is measured in the hours it takes from a child who needed insulin.

Against urine testing. It lags, cannot detect a falling glucose, and everyone abandoned it the moment blood strips existed. The reply is that it is what the world used for fifty years and the alternative after the Fall is no measurement at all.

Against this chapter's optimism about type 2. The Cuban experiment also produced neuropathy in 50,000 people. Weight loss by hunger is not weight loss by design, and HY-09's warnings are the limit on how far this is good

news.

10-line summary

1. Type 1 diabetes without insulin is fatal, usually within months in a child, and nothing a village or district can make will change it.
2. Pre-insulin diet bought months to a year in children at the cost of starvation, and Allen's own records contain deaths from inanition and from fasting-induced acidosis.
3. Joslin's deaths within ten years of onset fell from 824 per 1,000 before 1914, to 386 on the Allen diet, to 61 once insulin arrived.
4. Unopened insulin held 98 to 103 per cent of potency through four weeks of a 25 to 37 degree daily cycle but lost 5 to 13.5 per cent at a constant 31 degrees, so swinging heat is kinder than steady warmth.
5. A low-carbohydrate diet with calories held up lowers the insulin requirement and is the only way to make a salvaged stock last longer.
6. Urine sugar can raise an insulin dose and must never lower one, because it cannot see a falling glucose.
7. Before insulin, diabetic coma was always fatal, and the 31 of 33 who survived at Joslin's in 1923 to 1925 owed it to nursing, warmth, fluid by any route, and the exclusion of alkalis.
8. The recovering patient stays flat for about a week, because the second day kills.
9. Type 2 diabetes remits in about 46 per cent at one year and 36 per cent at two on an 800-kilocalorie diet, and the whole effect is weight, 86 per cent remission in those losing 15 kilograms.
10. Of the plants claimed for sugar only fenugreek seed at about 10 grams a day reaches a weak grade B, and salvaged sulfonylureas in a hungry village are more dangerous than the disease.

Open questions

- How long does an untreated, well-nourished, well-nursed type 1 patient actually live? No source I read measured it.
- Does the oscillating-temperature advantage hold beyond twelve weeks, and what happens in a cellar at a steady 15 degrees over years? Nobody has measured insulin potency past six months.
- What fraction of a post-Fall village's type 2 diabetics will remit spontaneously, and how many will be harmed by the same hunger? Cuba gives both halves and no way to weigh them.
- Does ketosis-prone type 2 behave the same under famine, and what is the real cassia coumarin content, from a regulator rather than a spice seller?

Sources to read in full

1. **Tattersall 2012, Diabetic ketoacidosis 1850 to 1970.** The whole bedside record of treating this disease without modern means, and the most useful single document I found.
2. **Allen, Stillman and Fitz 1919.** The case records rather than the tables: cases 1, 4, 26 and 30, and the preface.
3. **Lim and colleagues 2011, Diabetologia.** The mechanism of the type 2 cure, week by week.
4. **Kaufmann and colleagues 2021, PLoS ONE.** Insulin in the heat, with the constant-temperature caveat the summaries leave out.
5. **Smiley, Chandra and Umpierrez 2011.** Ketosis-prone type 2, the group a village will misclassify.
6. **Franco and colleagues 2013, BMJ.** What happens to a population's diabetes when the food goes and returns.
7. **Bus and colleagues, IWGDF 2023.** For the foot, the one complication a village can prevent.

At the bedside after the Fall

A household knows which of its members cannot smell acetone, keeps sugar within reach of any diabetic day and night, looks at the feet every evening, keeps a vial in the coolest place that still swings with the day, and writes on the

vial the date it was opened and on the wall the date the stock runs out. *To start:* sugar, salt, clean water, a cool shaded place, eyes that look at feet, and a calendar. *First failure to expect:* someone stretching the insulin quietly to make it last, as a quarter of patients did in New Haven with only money as the pressure, and slipping into ketoacidosis without telling anyone.

A village of 300 holds one list of every diabetic by name, type, dose and run-out date, kept by one person who can be woken at night, and holds the insulin in one place under the written rule. It teaches every household the four symptoms and the acetone smell, runs a foot clinic once a month, grows fenugreek, withdraws sulfonylureas as the food falls, and runs a deliberate weight programme instead of waiting for hunger to do it badly. *To start:* a log book, a named keeper, Benedict's reagent or salvaged strips, syringes matched to one insulin concentration, a cool store, a fenugreek bed. *First failure to expect:* a hypoglycaemic death in an old person left on a salvaged sulfonylurea through a bad harvest.

A district of 3,000 holds the pooled stock and an allocator who is not the healer, under FO-08's rule, and keeps one room able to nurse a ketoacidosis case continuously for 72 hours with a coma sheet on the wall. It trains the village keepers, runs Stettin's system of follow-up, and accepts publicly that it cannot make insulin rather than letting a rumour do the work. *To start:* ten beds, one trained nurse, a store, a written allocation rule agreed before anyone needs it, and people who will sit up all night. *First failure to expect:* the second-day death of a patient who seemed to be recovering and was allowed to sit up.

Protocols

Protocol 1. Finding diabetes without a laboratory

- ☐ Ask about thirst, large frequent urine, weight loss and exhaustion. Any three of the four is a strong suspicion.
- ☐ Thin and young, over days to weeks, points to type 1. Heavy and middle-aged, over years, to type 2.
- ☐ Smell the breath for acetone, by someone known to be able to smell it, and look at neck folds and armpits for velvety dark thickening.
- ☐ Test the urine: 5 ml Benedict's reagent, 8 drops urine, boil 3 to 5 minutes, cool. Blue negative, green trace, yellow about 1 to 1.5 g/dl, orange 1.5 to 2, brick red over 2.
- ☐ Record result, date and weight in the village log, and re-weigh weekly for a month. Continued loss on adequate food is the worst sign there is.

Protocol 2. Making Benedict's reagent (bench with a scale)

- ☐ Dissolve 100 g anhydrous sodium carbonate and 173 g sodium citrate in about 600 ml hot water, stirring until clear.
- ☐ Dissolve 17.3 g copper sulfate pentahydrate separately in about 100 ml water.
- ☐ Add the copper solution slowly to the first, stirring constantly, and make up to 1 litre. Ten per cent either way still works [VERIFY, my inference]. Yield about 200 tests a litre.
- ☐ Store stoppered and dark, labelled POISON, out of the reach of children, and point the tube mouth away from faces when boiling.

Protocol 3. Stretching an insulin stock

- ☐ Count every vial and unit, divide by the daily dose, write the run-out date on the wall and tell the patient.
- ☐ Cut carbohydrate hard, under 50 g a day and towards 36 g, keeping calories up with fat, protein, eggs, fish, meat, nuts and green vegetables.
- ☐ Reduce the insulin as the food drops, not after: cut the mealtime dose by about a third on the first day of the new diet and adjust [VERIFY, my inference, not from a trial].

- ☐ Store below 25 degrees where possible. A place that swings cool at night beats one that sits warm. Never freeze. Use one vial per person at a time, dated on opening, for 28 days or the label period, whichever is shorter.
- ☐ One insulin concentration in the village. Match syringe to vial and read the vial aloud before every draw.
- ☐ Test urine before meals two or three days a week, more when ill. Raise the dose for persistent heavy sugar, never lower it because the urine is clear.
- ☐ Keep sugar within arm's reach. Increase the dose during fever or infection, and do not stop it because the patient is not eating.

Protocol 4. Nursing ketoacidosis, after Joslin 1925

- ☐ Decide first whether this is high sugar or low. Dry skin, acetone breath, deep sighing breathing and days of thirst mean high. Sweating, warm moist skin, hunger, trembling and minutes of onset mean low. If in doubt, give sugar first.
- ☐ Lie the patient flat, keep them warm, do not let them sit up, and name one person responsible continuously for the first 24 hours.
- ☐ Pin a sheet to the wall and record pulse, breathing, fluid in, urine out, urine sugar and acetone every 30 to 60 minutes.
- ☐ Give insulin if there is any. That is the treatment. Everything else is support.
- ☐ Fluid by mouth if they can swallow: one litre of clean water, six level teaspoons of sugar, half a level teaspoon of salt. If they cannot swallow and still have a pulse and warm skin, subcutaneously at about 1 ml a minute per site, up to 3 litres in 24 hours over two sites, or rectally at about 250 ml an hour (THRIVING library, HY-03, IN-04, WO-08).
- ☐ No alkali, no bicarbonate, no purgatives, no stimulants. Empty a loaded bowel by enema, as Joslin did.
- ☐ Once they come round, keep them flat and nursed as a heart case for at least one week.
- ☐ Watch 12 to 24 hours after recovery begins for sudden weakness of limbs and breathing, which is potassium loss. Give potassium-rich food by mouth: fruit, potato, broth.

Protocol 5. The type 2 diet programme

- ☐ Weigh the patient and write it down. This number is the whole treatment.
- ☐ Stop every sulfonylurea and all insulin on day one. Halve metformin, or stop it if food is short.
- ☐ Target about 800 kilocalories a day for 8 to 12 weeks, from clear broth, lean meat or fish, eggs, and three portions a day of non-starchy green vegetables. No bread, grain, potato, sugar, fruit juice or alcohol.
- ☐ Weigh weekly, expecting 1 to 1.5 kilograms a week at first, targeting 15 kilograms. Walk every day. Watch the first fortnight for hypoglycaemia in anyone who was on a drug.
- ☐ Reintroduce food in steps over 2 to 8 weeks, keeping carbohydrate low. Test urine monthly, and weigh monthly for life, because regain is what ends remission. A clear urine on no drugs is remission.
- ☐ Never run this on anyone already thin, pregnant, breastfeeding, under 18, or recovering from starvation. HY-09 governs those cases.

Protocol 6. The foot round

- ☐ Both feet every day in good light, between the toes and the whole sole, feeling for hot spots and numb areas.
- ☐ Never barefoot. Check inside shoes by hand first. Cut nails straight across, and never cut corns or calluses with a blade at home.
- ☐ No hot water bottles, no hot soaking, no sitting close to the fire, because neuropathic feet burn without feeling it.
- ☐ Any new blister, crack, callus or hot spot: all weight off that foot until it heals, and tell the healer the same day.

Protocol 7. Treating low blood sugar

- ☐ Awake and able to swallow: 15 grams of fast sugar, one level tablespoon of sugar or honey, or about 120 ml of fruit juice. Wait 15 minutes, repeat once if still unwell, then give a proper meal.
- ☐ Unconscious or fitting: nothing liquid by mouth. Smear honey or sugar paste inside the cheek, lie them on their side, keep the airway clear.
- ☐ After a sulfonylurea low, feed repeatedly and watch for 72 hours.

Numbers to carry

1. **Weeks to months.** How long a child with type 1 lives with no insulin at all.
2. **824, 386, 61.** Joslin's deaths per 1,000 within ten years of onset before 1914, on the Allen diet, after insulin.
3. **Hot and swinging beats hot and steady.** Unopened insulin keeps four weeks through a 25 to 37 degree daily cycle but loses 5 to 13.5 per cent at a constant 31 degrees.
4. **Fifteen kilograms.** The weight loss that puts 86 per cent of type 2 diabetics into remission.
5. **46, 36, 13.** Per cent in remission at one, two and five years after an 800-kilocalorie programme.
6. **Fifteen grams, fifteen minutes.** The sugar dose and recheck for hypoglycaemia, and 72 hours of watching after a sulfonylurea.
7. **Ten millimoles per litre.** Where sugar starts to appear in the urine, which is why a clear urine never proves a safe dose.
8. **Ten grams of fenugreek seed a day.** The only plant dose with a measured effect, about 2 millimoles per litre off the fasting glucose.
9. **One week flat.** How long a patient who has come round from diabetic coma stays lying down.
10. **Thirty-one of thirty-three.** Joslin's 1923 to 1925 coma survival: insulin plus continuous nursing, warmth, fluid by any route, and no alkali.

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