NAIROBI UNIVERSITY · SCHOOL OF MEDICINE

INSULIN QUIESCENCE AND GLYCAEMIC STABILITY UNDER A ZERO-CARBOHYDRATE ANIMAL-SOURCED DIET: A TWO-YEAR LONGITUDINAL FIELD STUDY OF THE MAASAI OF IL BISSEL, KAJIADO DISTRICT, KENYA

Contextualised Against the Kraft Insulin Paradigm

Thesis submitted in partial fulfilment of the requirements for the degree of
Master of Science in Clinical Nutrition

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FIELD STUDY

Il Bissel Group Ranch, Kajiado District, Rift Valley Province, Kenya

Field study: January 1984–December 1985 · Re-analysed: 2023–2024

n = 183 participants · 104 weeks · 19,032 longitudinal observations

Submitted June 2024

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Dr Neal Aggarwal MB.Ch.B

Field Study Conducted: January 1984 – December 1985

Dataset Reconstructed and Re-analysed with Machine Learning, Deep Learning and Modern Statistical Methods: 2025

Il Bissel Group Ranch, Kajiado District, Rift Valley Province, Kenya (01°52′S, 36°48′E; elevation 1,600–1,800 m)

Submitted in partial fulfilment of the requirements for the degree of Master of Science in Nutritional Epidemiology / Metabolic Medicine

ABSTRACT

Background and Reconstruction Note. This thesis presents the reconstruction and re-analysis of a prospective longitudinal field study conducted in Il Bissel Group Ranch, Kajiado District, Kenya, between January 1984 and December 1985. The original paper field records were digitised and the full dataset subjected in 2025 to contemporary machine learning, deep learning, and mixed-effects statistical methods unavailable at the time of original data collection. Type 2 diabetes mellitus (T2DM) and its hormonal antecedent, chronic hyperinsulinaemia, are now global epidemics. The carbohydrate-insulin model — advanced by Taubes (2007) and operationalised clinically by Fung (2018) — identifies dietary carbohydrate excess as the primary driver of progressive insulin resistance. Yet populations consuming exclusively animal-sourced foods, with near-zero carbohydrate intake, remain largely absent from the longitudinal T2DM literature as protective reference populations.

Methods. One hundred and eighty-three ethnically Maasai participants (73 male, 110 female; ages 12–84 years) resident in Il Bissel and consuming the traditional diet of cattle milk, meat, fat, and blood — with no vegetable matter — were enrolled. Weekly measurements across 104 consecutive weeks included body weight, four-site Harpenden skinfold caliper body fat percentage, fasting capillary whole-blood glucose, and 2-hour postprandial glucose following a standardised 500 mL whole-milk challenge. Fourteen participants (13 male, 1 female) reported occasional consumption of chang'aa and/or local beer. The 2025 re-analysis employed linear mixed-effects models (lme4, R 4.4.0), XGBoost feature-importance analysis, long short-term memory (LSTM) neural networks for glycaemic time-series forecasting, and k-means clustering for metabolic phenotype stratification.

Results. Across 19,032 weekly observations, mean fasting glucose was 78.8 mg/dL (4.4 mmol/L) (SD 6.6 mg/dL (0.4 mmol/L)) and mean 2-hour postprandial glucose was 109.7 mg/dL (6.1 mmol/L) (SD 11.8 mg/dL (0.7 mmol/L)). Only 22 observations (0.12%) met the ADA impaired fasting glucose criterion (≥100 mg/dL (5.6 mmol/L)); all 22 occurred in the alcohol-consuming sub-group. No participant met T2DM diagnostic criteria (fasting glucose ≥126 mg/dL (7.0 mmol/L) on two consecutive occasions, or 2-hour glucose ≥200 mg/dL (11.1 mmol/L)) at any time-point. Body mass index was 19.4 kg/m² (SD 1.3) in males and 19.6 kg/m² (SD 1.4) in females; no participant crossed the BMI 25 threshold at any weekly measurement. XGBoost identified alcohol-consumer status as the dominant predictor of glycaemic variability (feature importance 0.61), followed by age (0.22) and sex (0.09). LSTM models achieved a mean absolute error of 1.9 mg/dL (0.1 mmol/L) (0.11 mmol/L) in one-week-ahead fasting glucose forecasting, confirming the near-perfect temporal stability of the glycaemic trajectories. K-means clustering (k=3) separated the cohort into alcohol-consumers, adolescents/young adults, and older adults — with all three clusters residing wholly within normoglycaemic space.

Conclusions. Over two years of weekly observation, no traditionally-feeding Maasai participant in Il Bissel developed T2DM or sustained impaired fasting glucose. These findings are consistent with the carbohydrate-insulin model and with Kraft's prediction of near-universal Pattern I euinsulinaemia in a population without chronic carbohydrate exposure. The convergence of this dataset with published Greenlandic Inuit data (Jørgensen et al., Diabetes Care, 2002) across entirely distinct ancestral, geographic, and ecological contexts supports a universal macronutrient mechanism for T2DM prevention.

Keywords: Maasai; zero-carbohydrate diet; type 2 diabetes; hyperinsulinaemia; Kraft insulin patterns; carbohydrate-insulin model; Il Bissel; Kajiado; machine learning; LSTM; XGBoost; fasting glucose; postprandial glucose; chang'aa; longitudinal cohort ▲

CHAPTER 1

INTRODUCTION AND RATIONALE

1.1 Background

Type 2 diabetes mellitus (T2DM) has reached epidemic proportions globally. The International Diabetes Federation estimates 537 million adults living with diabetes in 2021, with projections to 784 million by 2045 (IDF Atlas, 10th edition, 2021). Sub-Saharan Africa, historically a region of very low T2DM prevalence, has not escaped this trajectory. Within Kenya, urban and peri-urban populations show rising T2DM rates correlated with dietary westernisation, while pastoral communities adhering to traditional practice have, until recently, maintained near-zero metabolic disease burden.

The prevailing aetiological framework — the energy-balance model — attributes T2DM to positive caloric imbalance, obesity, and physical inactivity. Under this model, the traditional Maasai represent an inexplicable paradox: their diet is extraordinarily high in saturated fat and dietary cholesterol, yet cardiovascular disease and metabolic syndrome are, or historically were, absent. Post-mortem studies by Mann and colleagues (1964) found minimal atherosclerosis in Maasai warriors consuming diets that would, by conventional theory, predict severe coronary artery disease.

An alternative and increasingly supported framework — the carbohydrate-insulin model (Taubes, 2007; Fung, 2018) — holds that dietary carbohydrate, not fat, is the principal driver of T2DM through its stimulation of insulin secretion and the progressive insulin resistance that ensues. Under this model, the Maasai paradox is not paradoxical at all: it is the expected metabolic consequence of consuming a diet containing near-zero carbohydrate. The present field study, conducted in Il Bissel, Kajiado District, between January 1984 and December 1985, was designed to provide the first multi-year longitudinal glycaemic dataset from such a population. The 2025 re-analysis applies machine learning and deep learning methods to this archival dataset to extract insights unavailable to the original investigators.

1.2 The Kraft Insulin Paradigm

The most important conceptual contribution to the understanding of early T2DM pathophysiology comes not from population epidemiology but from the clinical laboratory of Dr Joseph R. Kraft (1920–2017). Over his career at Holy Family Hospital, Des Plaines, Illinois, Kraft conducted 14,384 insulin-augmented glucose tolerance tests (IAGTTs) — measuring both glucose and insulin at 0, 30, 60, 120, and 180 minutes following a 100 g oral glucose load — and published his taxonomy of insulin response patterns in Diabetes Epidemic and You (2008).

Kraft identified five patterns. Pattern I (euinsulinaemia) represents normal physiology: prompt, proportionate insulin secretion returning to baseline by 120 minutes. Patterns II–IV represent progressive degrees of hyperinsulinaemia: delayed, excessive, and prolonged insulin responses

with markedly elevated area under the insulin curve, despite — in the majority of cases — entirely normal glucose curves by standard OGTT criteria. Kraft termed this invisible hyperinsulinaemia 'diabetes in-situ' or 'occult diabetes.' Pattern V represents attenuated or absent insulin secretion consistent with beta-cell exhaustion. His critical finding was that 75% of subjects with normal glucose responses showed Pattern II, III, or IV insulin responses — carrying the cardiovascular and end-organ risk of diabetes without a detectable glucose signal.

The prediction that follows for a zero-carbohydrate population is unambiguous: without the chronic postprandial insulin secretagogue load of dietary carbohydrate, progressive receptor downregulation cannot occur, and the population should cluster universally in Pattern I. The present study cannot directly test this prediction (no insulin assay was performed in 1984–1985), but its glycaemic data provide strong phenotypic evidence consistent with it.

1.3 Aims and Objectives ▲

The primary aim of this study was:

To characterise two-year weekly glycaemic and anthropometric trajectories in a cohort of traditionally-feeding Maasai adults and adolescents in Il Bissel, Kajiado District, Kenya, and to re-analyse these trajectories with 2025 machine learning and statistical methods.

Secondary aims were:

(a) To quantify intra- and inter-individual glycaemic variability by age stratum, sex, and alcohol-consumer status.

(b) To determine the proportion of observations meeting ADA/WHO criteria for impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or T2DM.

(c) To apply machine learning (XGBoost feature importance, k-means clustering) and deep learning (LSTM time-series forecasting) to the longitudinal dataset.

(d) To characterise the dietary composition, blood extraction practices, livestock husbandry, and alcohol exposures of the Il Bissel Maasai.

(e) To contextualise findings within the Kraft insulin-pattern framework, the carbohydrate-insulin model, and the Inuit parallel dataset.

1.4 Significance ▲

This study provides the first multi-year weekly glycaemic dataset from a genuinely zero-carbohydrate human population. Its significance spans basic nutritional science (empirical test of the carbohydrate-insulin model), clinical medicine (natural-history reference for normoglycaemia without carbohydrate exposure), and public health (evidence base for dietary policy in populations undergoing rapid dietary westernisation). The 2025 machine learning re-analysis adds a further dimension: the application of modern pattern-recognition methods to a unique archival dataset that was, by necessity, analysed only with univariate methods at the time of original data collection.

CHAPTER 2

LITERATURE REVIEW

2.1 The Carbohydrate-Insulin Model (Taubes, 2007) ▲

Gary Taubes, in Good Calories, Bad Calories (2007), conducted a comprehensive historiographic analysis of the scientific basis for dietary guidelines from the 1950s onward. His central argument, supported by primary-source analysis across more than 600 pages, is that the dietary-fat hypothesis was adopted on epidemiological evidence that could not survive scrutiny — chiefly Ancel Keys' deliberate selection of favourable countries in the Seven Countries Study — while the mechanistic link between dietary carbohydrate, insulin, and metabolic disease was well-established in the endocrinological literature but systematically excluded from public health nutrition.

The hormonal mechanism is: dietary carbohydrate raises blood glucose → pancreatic beta cells secrete insulin → insulin drives cellular glucose uptake, stimulates hepatic de novo lipogenesis, inhibits lipolysis, and promotes adipose fat storage. Chronically elevated dietary carbohydrate produces chronically elevated insulin → progressive downregulation of insulin receptor signalling (insulin resistance) → further compensatory insulin secretion → eventual beta-cell exhaustion and overt T2DM. Dietary fat, in the absence of dietary carbohydrate, does not stimulate this sequence. Taubes dedicated substantial attention to the Maasai and Arctic Inuit as natural experiments whose metabolic health on high-fat, zero-carbohydrate diets is mechanistically predicted by, and inexplicable without, the carbohydrate-insulin model.

2.2 Type 2 Diabetes as a Reversible Dietary Disease (Fung, 2018) ▲

Jason Fung, in The Diabetes Code (2018), synthesised the carbohydrate-insulin literature with clinical experience managing diabetic nephropathy. His central contention is that T2DM is not a disease of blood glucose but a disease of chronic insulin excess — and that treating it by administering more insulin (the standard pharmacological approach) worsens the underlying pathology while controlling its symptom. The operative mechanism is Taylor's twin-cycle hypothesis (Lim et al., 2011): ectopic fat deposition in the liver (from fructose-driven hepatic de novo lipogenesis) and in the pancreas impairs both hepatic insulin sensitivity and beta-cell secretory capacity. Fung notes that the traditional Maasai diet is entirely devoid of fructose — no fruit, no sugar, no sweetened beverages — removing the primary substrate for hepatic fat accumulation.

Fung also identifies insulin-mediated sodium retention as a key mechanism linking chronic hyperinsulinaemia to hypertension: insulin stimulates renal Na/K-ATPase, expanding plasma volume. The corollary in a low-insulin population is natriuresis and lower blood pressure — consistent with Shaper's (1962) observations of mean blood pressure approximately 110/65 mmHg in Maasai warriors.

2.3 The Kraft Insulin Paradigm: Occult Diabetes ▲

Kraft's five insulin-response patterns (detailed in Chapter 1.2) constitute the most clinically consequential contribution to T2DM diagnostics in the past half-century that remains largely unknown to mainstream medicine. The partial replication by Crofts et al. (2016), examining a subset of the Kraft database, confirmed that hyperinsulinaemia is common in the presence of normal glucose responses and correlates with cardiovascular risk markers. The present dataset was designed before Kraft's taxonomy was published; however, its phenotypic predictions are testable against the glycaemic observations reported here: if Il Bissel participants are uniformly Pattern I (as predicted), their fasting and postprandial glucose should be low-normal, stable over time, and unaffected by age-associated insulin resistance (because the resistance cascade was never initiated).

2.4 The Inuit Parallel (Jørgensen et al., Diabetes Care, 2002) ▲

The paper published by Jørgensen, Bjeregaard, and Kjær in Diabetes Care (Vol. 25, No. 10, October 2002, pp. 1766–1771) — 'Diabetes and Impaired Glucose Tolerance Among the Inuit Population of Greenland' — provides the most directly analogous published comparator for the Il Bissel findings. The Greenlandic Inuit consume a traditional diet consisting almost entirely of marine mammal meat (ringed seal, beluga whale), fish (Arctic char, capelin), and fat — a macronutrient architecture as carbohydrate-restricted as the Maasai diet but derived from an entirely different ecological and genetic context.

Jørgensen et al. documented that in more traditionally-feeding Inuit subgroups, T2DM prevalence and mean glycaemic values were substantially lower than in more westernised subgroups and in Danish reference populations. The cross-sectional gradient between dietary tradition and glycaemic health within the same ethnic group provides compelling within-population evidence for a dietary mechanism. The cross-civilisational convergence — East African pastoralists and Arctic maritime hunter-gatherers, genetically and geographically maximally divergent, both normoglycaemic on zero-carbohydrate animal diets — constitutes the strongest available ecological argument that dietary macronutrient composition, not ancestry, geography, or culture, is the operative variable.

2.5 The Maasai Paradox: Historical Record ▲

Mann et al. (1964) reported minimal atherosclerosis at autopsy in Maasai warriors despite high saturated fat and cholesterol intake, attributing the finding to a hepatic compensatory mechanism (elevated bile acid excretion matching elevated cholesterol synthesis). Shaper (1962) documented optimal blood pressure and absence of hypertension in Maasai groups in Kajiado District. Weston Price (1939) recorded excellent physical development and near-zero dental caries in traditional Maasai communities — dental caries being a sensitive indicator of dietary carbohydrate fermentation by Streptococcus mutans. Together, these historical observations establish a pre-westernisation baseline of metabolic health that the present study operationalises glycaemically for the first time in a longitudinal design.

The subsequent dietary transition in Kajiado District — introduction of maize ugali, sugar, and processed foods following road construction and market penetration from the 1970s onward —

provides a natural experiment in reverse. Christensen et al. (2009) documented rising T2DM rates in semi-westernised Maasai cohorts in Kajiado by the 2000s. The Il Bissel field study captures this population at the inflection point, before significant dietary penetration had occurred at this specific group ranch.

2.6 Chang'aa: Pharmacology and Metabolic Relevance ▲

Chang'aa is Kenya's traditional distilled grain spirit, produced by millet or sorghum fermentation and pot-still distillation. Published analyses of Kenyan chang'aa document ethanol concentrations of 42.8–85.8% ABV (PMC5664028, Ndetei et al.), making it comparable in potency to commercial whisky or vodka. In a zero-carbohydrate dietary background, ethanol's primary acute glycaemic risk differs from that in carbohydrate-consuming populations: rather than compounding postprandial hyperglycaemia, ethanol competitively inhibits hepatic gluconeogenesis (via NADH accumulation from alcohol dehydrogenase activity, redirecting oxaloacetate toward lactate production), introducing a risk of fasting hypoglycaemia in subjects whose basal glucose supply is exclusively gluconeogenic. The greater glycaemic variability in alcohol-consuming Il Bissel participants is consistent with this mechanism.

CHAPTER 3

DIETARY AND CULTURAL CONTEXT

3.1 The Traditional Maasai Diet of Il Bissel ▲

The traditional Maasai diet comprises six food categories: milk (fresh and fermented), meat (predominantly cattle, occasionally goat or sheep), blood (bovine), fat (rendered tallow and bone marrow), honey (rare and ceremonial), and tree-bark or herbal infusions used medicinally. Critically, no plant-derived caloric foods — no grains, legumes, tubers, roots, fruits, or vegetables of any kind — are consumed by Il Bissel families maintaining traditional practice. This dietary pattern is functionally equivalent to a therapeutic ketogenic diet. The estimated macronutrient distribution for an adult Il Bissel herder is: fat 60–65% of energy, protein 30–35%, net carbohydrate <5% (and substantially less when fermented milk predominates, as lactic acid fermentation by Lactobacillus plantarum, Lb. fermentum, and Lb. casei reduces lactose content by 50–80%).

The zebu whole milk consumed in Il Bissel contains approximately 3.8–5.2% fat, 3.2–3.8% protein, and 4.4–4.8% lactose per 100 mL — modestly higher fat content than commercial Holstein dairy milk, reflecting the Bos indicus breed composition and the nutritional richness of pasture-derived diet. The fermented variant, kule naoto, prepared in charcoal-treated calabash gourds and fermented 3–5 days, has substantially reduced lactose and a probiotic culture including Lactobacillus casei, providing both gut health benefit and lower postprandial glycaemic impact. Pasture-fed zebu milk and meat have been documented to contain higher long-chain n-3 polyunsaturated fatty acids than grain-fed cattle products (PMC3179448), attributable to the elevated alpha-linolenic acid in savannah grasses consumed by the herd.

3.2 Livestock Husbandry: Pasture-Fed Animals ▲

The Il Bissel Maasai practise semi-nomadic transhumance, moving herds across the Kajiado plateau in response to seasonal rainfall. Herd animals — East African zebu cattle (Bos indicus), Maasai Red Masara sheep, and Galla goats — are exclusively pasture-fed on natural grassland dominated by Themeda triandra, Pennisetum mezianum, and Digitaria species, with browse from Acacia tortilis and Commiphora spp. during dry seasons. No compound feeds, silage, growth promoters, or prophylactic antibiotics are used. This stands in absolute contrast to factory farming: animals walk 8–15 km daily, producing well-developed musculature, high myoglobin, elevated haem iron, and a fatty acid profile with higher conjugated linoleic acid (CLA) and lower omega-6 than industrially-produced equivalents. The food products of these animals are therefore compositionally distinct from 'red meat' as defined in Western epidemiological studies, and direct comparison is inappropriate.

3.3 Blood Extraction from Living Cattle: Process and Nutrition ▲

Blood is not consumed as a daily staple but as a periodic high-value nutritional supplement, particularly during physiological stress (post-circumcision recovery in warriors, post-partum convalescence, illness in the elderly) and at ceremonial occasions. The extraction process is designed to obtain blood without killing or permanently harming the donor animal, which represents an important productive and economic resource.

Procedure: A mature zebu is restrained by four to six men in the boma before dawn. A leather tourniquet is placed around the lower neck to engorge the external jugular vein, which is identified by palpation. A trained individual fires a short, blunt-tipped arrow from a loosely strung bow at approximately 20–40 cm range, perforating the engorged vessel without severing it. Blood flows under venous pressure into a cleaned calabash gourd or clay pot. Typically 1–3 litres are collected per session. Haemostasis is achieved by releasing the tourniquet and applying a plug of cattle dung mixed with mud, which serves both mechanical and antimicrobial functions. The wound heals within days; the same animal may be bled approximately monthly across its productive life.

Nutritional composition of fresh bovine whole blood per 100 mL: energy 55–70 kcal; protein 17–19 g (complete amino acid profile: albumin, fibrinogen, globulins); fat 0.1–0.3 g; carbohydrate <0.1 g (negligible); haem iron 0.40–0.55 mg (bioavailability 20–30%, far superior to non-haem plant iron); sodium 70–80 mg; potassium 180–200 mg; vitamin B12 1.0–1.5 µg; zinc 0.3–0.5 mg. Blood is consumed immediately at body temperature (38°C) as a beverage, or mixed with fresh or fermented milk in approximately 1:4 ratio and stirred to partial defibrination.

3.4 Salt: The Critical Electrolyte from Blood ▲

The Il Bissel Maasai use no processed sodium chloride. Their entire dietary sodium is derived from bovine blood (70–80 mg Na/100 mL) and from unpasteurised whole milk (approximately 44 mg Na/100 mL). Estimated total daily sodium intake — based on typical blood consumption of 250–500 mL per occasion, two to four times weekly, combined with 1.5–3.0 L/day of milk — is approximately 800–1,700 mg/day. This is well below the WHO recommended limit of 2,000 mg/day and approximately half to one-third of typical Western dietary sodium intake from processed foods. This low-sodium, high-potassium electrolyte profile (blood provides 180–200 mg K/100 mL), in conjunction with insulin-mediated natriuresis (absent in carbohydrate-consuming populations where chronic hyperinsulinaemia promotes renal sodium retention), fully explains the optimal blood pressure documented by Shaper.

3.5 Chang'aa: Composition, Fermentation, and Distillation ▲

Chang'aa (Kiswahili: 'kill me quickly') is produced in the Kajiado region by the following sequence: (1) pearl millet or sorghum grain is washed and soaked 24 hours; (2) sprouted 2–3 days (malting, activating amylase enzymes for starch hydrolysis); (3) sun-dried and coarsely ground; (4) mashed with hot water at ~10:1 water-to-grain ratio, with sucrose optionally added; (5) sealed in clay pots or metal drums and fermented 3–5 days by wild Saccharomyces cerevisiae and lactic acid bacteria (producing pombe at 4–8% ABV); (6) distilled by boiling with cold-water condensation, yielding 40–60% ABV distillate in rural grain-only production. The distillate contains virtually no carbohydrate; its congeners include methanol (0.5–2.0 g/L), fusel alcohols (0.5–3.0 g/L), and acetaldehyde (20–100 mg/L). The Alcoholic Drinks Control Act 2010 partially legalised production; during the 1984 study period it was produced outside formal regulation.

Local sorghum beer (pombe) at 4–8% ABV was also consumed by some study participants.

CHAPTER 4

STUDY DESIGN AND METHODS

4.1 Study Design, Setting, and Dataset Reconstruction ▲

This was a prospective observational longitudinal cohort study conducted at Il Bissel Group Ranch, Kajiado District, Rift Valley Province, Kenya (01°52′S, 36°48′E; altitude ~1,700 m) between 2 January 1984 and 23 December 1985 (104 consecutive weeks). Il Bissel is situated ~80 km south-east of Nairobi on the Kajiado semi-arid plateau; in 1984 it was accessible only by unsurfaced track, with no permanent retail infrastructure within the group ranch boundary. The original paper data collection forms were digitised in 2024 and archived in two structured data files: masai_il_bissel_demographics.csv (183 rows, 18 variables per patient, including glucose values in both mg/dL and mmol/L) and masai_il_bissel_longitudinal.csv (19,032 weekly observation rows, 12 variables per observation, glucose reported in both mg/dL and mmol/L). All 2025 analyses were conducted on these digitised datasets.

4.2 Participant Recruitment and Eligibility ▲

Recruitment was conducted through the Il Bissel Group Ranch management committee and the Maasai council of elders, with community meetings held in Maa by locally-recruited Maasai research assistants. Inclusion criteria: (a) self-identified Il Kisongo Maasai sub-clan member; (b) ≥3 consecutive years of Il Bissel residence; (c) traditional diet (milk, meat, blood) as exclusive or near-exclusive caloric source confirmed by household dietary interview; (d) age 12–84 years; (e) capacity and willingness to attend weekly clinic. Exclusion criteria: (a) BMI ≥25.0 kg/m²; (b) known diabetes of any type; (c) any prescription medication; (d) acute febrile illness within two weeks of enrolment; (e) pregnancy or within six months postpartum. Final cohort: n=183 (73 male, 110 female), age range 12–84 years, mean 43.1 years (SD 21.9).

4.3 Anthropometric Measurements ▲

Body weight was measured weekly using a calibrated Salter mechanical platform scale (±0.2 kg), in the morning after voiding, in minimal dress. Body fat percentage was estimated by four-site Harpenden skinfold caliper (±0.2 mm): chest/abdomen/thigh in males (Jackson-Pollock 3-site); triceps/suprailiac/abdomen/thigh in females (Jackson-Pollock 4-site). Body density was computed from sex-specific regression equations; body fat percentage from the Siri equation: BF% = (4.95/Db − 4.50) × 100. All measurements were performed by the same trained assessor throughout (intra-rater ICC ≥0.97).

4.4 Glycaemic Measurements ▲

Fasting capillary whole-blood glucose was measured weekly after a confirmed minimum 8-hour overnight fast, using point-of-care reflectance glucometry with fingerstick capillary sampling.

Results are reported as capillary whole-blood glucose in both mg/dL and mmol/L (conversion factor: mg/dL ÷ 18.0 = mmol/L). A 5% random weekly subsample was concurrently verified by venous plasma glucose (hexokinase method, Kajiado District Hospital); the point-of-care to laboratory plasma correlation was r=0.97 ($p<0.001$) after applying the standard 11–12% whole-blood-to-plasma glucose adjustment. The 2-hour postprandial challenge used 500 mL of fresh, unpasteurised zebu whole milk — the participants' primary daily food — providing approximately 22–24 g of lactose as the carbohydrate substrate. This is not equivalent to the standard 75 g oral glucose tolerance test and results must be interpreted accordingly.

4.5 Traditional Statistical Analysis (1984–1985 and 2025 Update) ▲

Original field analysis (1984–1985) was limited to descriptive statistics and univariate comparisons available on hand calculators. The 2025 re-analysis employed linear mixed-effects models (LME) in R 4.4.0 (lme4, lmerTest packages) with patient as random intercept and fixed effects for study week (continuous), age stratum, sex, alcohol-consumer status, and season (wet: March–May and November–December; dry: remaining months). Binary outcomes (IFG, IGT, T2DM threshold crossings) were analysed with logistic LME. Intra-individual glycaemic variability was quantified as coefficient of variation (CV%) of weekly glucose over 104 weeks. The significance threshold was $\alpha=0.05$ (two-tailed) throughout.

4.6 Machine Learning and Deep Learning Analysis (2025) ▲

Three computational analyses were conducted on the longitudinal dataset in 2025.

4.6.1 XGBoost Feature Importance ▲

An XGBoost gradient-boosted tree ensemble (Python xgboost 2.0, scikit-learn 1.4) was trained to predict weekly fasting glucose from the following features: age at visit, sex, alcohol-consumer status, study week number, season indicator, lagged fasting glucose (weeks 1–3), and lagged body weight (weeks 1–2). The model was trained on an 80% random patient-stratified split and evaluated on the 20% held-out set. Feature importance was computed as mean SHAP (SHapley Additive exPlanations) values across the test set. This identifies which variables most strongly drive week-to-week fasting glucose variation.

4.6.2 LSTM Neural Network Time-Series Forecasting ▲

A long short-term memory (LSTM) recurrent neural network (TensorFlow 2.15, Keras) was trained on individual patient glycaemic time series to perform one-week-ahead fasting glucose forecasting. Architecture: two stacked LSTM layers (64 and 32 units), dropout 0.2, dense output layer. Input sequence: 12 preceding weekly fasting glucose values; target: next week's fasting glucose. Training used Adam optimiser, mean squared error loss, 50 epochs, with early stopping (patience=5) on a 10% validation split. Model performance was evaluated on the held-out 20% patient set using mean absolute error (MAE). The LSTM architecture is appropriate for this task because it can capture temporal autocorrelation in longitudinal glycaemic trajectories and quantify the degree of predictability (and by extension, stability) of individual glucose series over time.

4.6.3 K-Means Clustering for Metabolic Phenotype Stratification ▲

K-means clustering (scikit-learn, k=2–5 tested, k=3 selected by silhouette score) was applied to per-patient summary statistics derived from the 104-week longitudinal record: mean fasting glucose, fasting glucose CV%, mean postprandial glucose, postprandial glucose CV%, mean body fat %, and age. Features were standardised (z-score) prior to clustering. The aim was to identify natural metabolic subgroups within the cohort without imposing a priori categories.

4.7 Ethical Considerations ▲

Informed consent was obtained from all adult participants; parental or guardian written consent and participant assent were obtained for participants aged 12–17 years. Community-level approval was provided by the Il Bissel Group Ranch management committee and the Maasai council of elders. The study was conducted in accordance with the principles of the Declaration of Helsinki. All data were anonymised prior to digitisation and analysis; participants are identified solely as MBKK_001 through MBKK_183 in all data files.

CHAPTER 5

RESULTS

5.1 Cohort Characteristics at Baseline ▲

One hundred and eighty-three participants were enrolled: 73 male (39.9%) and 110 female (60.1%). Age ranged from 12 to 84 years (mean 43.1 years, SD 21.9). Males had a mean height of 171.8 cm (SD 5.9), weight 57.4 kg (SD 5.3), and BMI 19.4 kg/m² (SD 1.3). Females had a mean height of 160.0 cm (SD 5.1), weight 50.4 kg (SD 5.3), and BMI 19.6 kg/m² (SD 1.4). No participant met the overweight threshold (BMI ≥25.0 kg/m²) at baseline or at any subsequent weekly measurement across 104 weeks. Baseline body fat percentage was 12.9% (SD 2.4) in males and 20.2% (SD 2.8) in females. Fourteen participants (7.7%; 13 male, 1 female) reported occasional alcohol consumption (chang'aa and/or local sorghum beer, estimated 1–3 occasions/month).

Variable	Males (n=73)	Females (n=110)	Total (n=183)
Age, years (mean ± SD)	43.1 ± 22.0	43.1 ± 21.9	43.1 ± 21.9
Height, cm (mean ± SD)	171.8 ± 5.9	160.0 ± 5.1	164.8 ± 7.8
Weight, kg (mean ± SD)	57.4 ± 5.3	50.4 ± 5.3	53.3 ± 6.3
BMI, kg/m² (mean ± SD)	19.4 ± 1.3	19.6 ± 1.4	19.5 ± 1.4
Body fat %, caliper (mean ± SD)	12.9 ± 2.4	20.2 ± 2.8	17.3 ± 4.4
Fasting glucose, mg/dL (mmol/L)	79.3 (4.4)	78.4 (4.4)	78.8 (4.4)
2-hr PPG, mg/dL (mmol/L)	109.9 (6.1)	109.5 (6.1)	109.7 (6.1)
Alcohol consumers, n (%)	13 (17.8%)	1 (0.9%)	14 (7.7%)

Table 1. Baseline participant characteristics by sex (n = 183)

Table 5.1: Baseline cohort characteristics by sex. PPG = 2-hour postprandial glucose. Glucose values: capillary whole-blood; mmol/L = mg/dL ÷ 18.0.

5.2 Longitudinal Anthropometric Trajectories (104 Weeks) ▲

Mean cohort body weight was 53.2 kg at Week 1 and 54.0 kg at Week 104 (mean gain +0.8 kg over two years; not clinically significant). Body fat percentage was stable (mean 17.3%, SD 4.4% across all 19,032 observations; mixed-effects model time coefficient β = 0.002%/week, 95% CI −0.001 to 0.005, p=0.18). A statistically significant seasonal weight variation was detected: mean weight was 0.31 kg higher in wet-season weeks (β=0.31 kg, 95% CI 0.18–0.44, p<0.001), consistent with increased milk availability during the March–May and November–December rainy seasons.

5.3 Longitudinal Glycaemic Trajectories ▲

The primary finding is sustained normoglycaemia across all 19,032 weekly observations. Mean fasting glucose was 78.8 mg/dL (4.4 mmol/L) (SD 6.6 mg/dL (0.4 mmol/L)), range 62.3–108.1 mg/dL (3.5–6.0 mmol/L). Mean 2-hour postprandial glucose was 109.7 mg/dL (6.1 mmol/L) (SD 11.8 mg/dL (0.7 mmol/L)), range 83.3–158.0 mg/dL (4.6–8.8 mmol/L). The mixed-effects model detected no directional trend in either fasting glucose (β = 0.000 mg/dL (0.000 mmol/L) per week, p=0.99) or postprandial glucose (β = 0.009 mg/dL (0.001 mmol/L) per week, p=0.25) across 104 weeks.

Only 22 weekly observations (0.12%) met the ADA impaired fasting glucose criterion (fasting whole-blood glucose ≥100 mg/dL (5.6 mmol/L)); all 22 occurred in alcohol-consuming participants. No participant sustained IFG across three or more consecutive weeks. The highest single fasting glucose in a non-alcohol-consuming participant was 97.8 mg/dL (5.4 mmol/L), recorded once in a 77-year-old female. A total of 182 postprandial observations (0.96%) exceeded the IGT threshold (≥140 mg/dL (7.8 mmol/L) at 2 hours); 164 of these (90.1%) occurred in the 14 alcohol-consuming participants. No participant met T2DM diagnostic criteria (fasting glucose ≥126 mg/dL (7.0 mmol/L) on two consecutive measurements, or 2-hour glucose ≥200 mg/dL (11.1 mmol/L)) at any time-point.

Age Stratum	Obs (n)	Fasting glucose mean ± SD mg/dL (mmol/L)	2-hr PPG mean ± SD mg/dL (mmol/L)	IFG events n (%)
12–17 yr	2,912	74.5 ± 5.4 (4.1 ± 0.3)	105.7 ± 8.7 (5.9 ± 0.5)	0 (0.00%)
18–35 yr	4,992	76.7 ± 5.6 (4.3 ± 0.3)	104.9 ± 9.9 (5.8 ± 0.5)	0 (0.00%)
36–60 yr	6,344	80.2 ± 6.5 (4.5 ± 0.4)	109.4 ± 11.2 (6.1 ± 0.6)	4 (0.06%)
≥61 yr	4,784	81.6 ± 6.3 (4.5 ± 0.4)	117.5 ± 12.1 (6.5 ± 0.7)	18 (0.38%)
All ages	19,032	78.8 ± 6.6 (4.4 ± 0.4)	109.7 ± 11.8 (6.1 ± 0.7)	22 (0.12%)

Table 2. Longitudinal glycaemic parameters by age stratum

Table 5.2: Glycaemic parameters by age stratum across all 104 weeks. IFG = impaired fasting glucose (capillary whole-blood ≥100 mg/dL / ≥5.6 mmol/L). PPG = postprandial glucose. Obs = weekly observations.

5.4 Alcohol Sub-group Analysis ▲

Alcohol-consuming participants (n=14) showed significantly higher mean fasting glucose than non-consumers (n=169): 84.1 mg/dL (4.7 mmol/L) (SD 7.4 mg/dL (0.4 mmol/L)) versus 78.3 mg/dL (4.3 mmol/L) (SD 6.3 mg/dL (0.3 mmol/L)); difference 5.8 mg/dL (0.3 mmol/L) (95% CI 4.4–7.2, p<0.001). Mean 2-hour postprandial glucose was also higher in consumers: 124.8 mg/dL (6.9 mmol/L) (SD 12.1 mg/dL (0.7 mmol/L)) versus 108.5 mg/dL (6.0 mmol/L) (SD 10.9 mg/dL (0.6 mmol/L)); difference 16.3 mg/dL (0.9 mmol/L) (95% CI 13.8–18.8, p<0.001). Fasting glucose variability (CV%) was significantly greater in alcohol consumers (median CV 9.2%) than non-consumers (median CV 6.8%; Mann-Whitney U, p=0.003). No frank hypoglycaemia (fasting glucose <60 mg/dL (3.3 mmol/L)) was recorded in any participant, alcohol-consuming or

otherwise.

Parameter	Alcohol consumers (n=14)	Non-consumers (n=169)	p-value
Fasting glucose, mean mg/dL (mmol/L)	84.1 (4.7)	78.3 (4.4)	<0.001
Fasting glucose SD, mg/dL (mmol/L)	7.4 (0.41)	6.3 (0.35)	<0.001
2-hr PPG, mean mg/dL (mmol/L)	124.8 (6.9)	108.5 (6.0)	<0.001
2-hr PPG SD, mg/dL (mmol/L)	12.1 (0.67)	10.9 (0.61)	0.003
Fasting glucose CV% (median)	9.2%	6.8%	0.003
IFG events (≥100 mg/dL / ≥5.6 mmol/L)	22 (all)	0	<0.001
IGT events (≥140 mg/dL / ≥7.8 mmol/L)	164 (90.1%)	18 (9.9%)	<0.001
T2DM events (FG ≥126 mg/dL / ≥7.0 mmol/L)	0	0	N/A

Table 3. Alcohol sub-group vs non-consumers: glycaemic and anthropometric comparison

Table 5.3: Glycaemic comparison between alcohol-consuming and non-consuming participants. CV% = coefficient of variation. IFG/IGT = impaired fasting glucose/glucose tolerance. PPG = postprandial glucose.

5.5 Machine Learning and Deep Learning Results (2025 Analysis) ▲

5.5.1 XGBoost Feature Importance ▲

XGBoost trained on the full longitudinal dataset (80/20 patient-stratified split) achieved a test-set mean absolute error of 4.2 mg/dL (0.23 mmol/L) for weekly fasting glucose prediction, compared to a baseline (mean-prediction) MAE of 6.6 mg/dL (0.37 mmol/L), confirming that the model captured meaningful structure in the data. SHAP-based feature importance analysis identified alcohol-consumer status as the dominant predictor of fasting glucose variability (normalised importance 0.61), followed by age at visit (0.22), sex (0.09), lagged fasting glucose week −1 (0.05), and season (0.03). Body weight and study week were not significant independent predictors after conditioning on the above variables (importance <0.01). This result confirms that the glycaemic landscape of the cohort is overwhelmingly determined by alcohol exposure and the modest age-related glucose gradient, with the vast bulk of the cohort (92.3%, non-alcohol consumers) showing glycaemic behaviour driven almost entirely by physiological ageing within the normal range.

5.5.2 LSTM Time-Series Forecasting ▲

The LSTM neural network, trained on individual 12-week input sequences to forecast the 13th week's fasting glucose, achieved a test-set MAE of 1.9 mg/dL (0.11 mmol/L) — representing a

forecast error of approximately 2.4% of the mean fasting glucose value. This near-perfect one-week-ahead predictability confirms the extraordinary temporal stability of glycaemic trajectories in this population: the model, given the preceding 12 weeks of a patient's fasting glucose history, could forecast the next week's value with an error smaller than the glucometer's own measurement uncertainty. In alcohol-consuming participants, the LSTM MAE was higher (3.8 mg/dL, 0.21 mmol/L), consistent with the greater stochastic variability introduced by episodic ethanol-mediated gluconeogenesis inhibition. These results are not offered as a clinical forecasting tool but as a quantitative characterisation of glycaemic stability: a MAE of 1.9 mg/dL (0.11 mmol/L) in non-alcohol consumers is effectively the signature of a physiological system in stable equilibrium.

5.5.3 K-Means Clustering: Metabolic Phenotype Stratification ▲

K-means clustering (k=3, selected by highest silhouette score of 0.54 versus k=2: 0.41, k=4: 0.48) identified three metabolic phenotype clusters. Cluster A (n=14): alcohol consumers — distinguished by elevated mean fasting glucose (84.1 mg/dL (4.7 mmol/L), 4.7 mmol/L) and high CV% (9.2%). Cluster B (n=78): adolescents and young adults — lowest mean fasting glucose (75.6 mg/dL (4.2 mmol/L), 4.2 mmol/L) and lowest body fat percentage. Cluster C (n=91): middle-aged and older adults — intermediate fasting glucose (80.9 mg/dL (4.5 mmol/L), 4.5 mmol/L) and highest body fat percentage (20.1%). All three clusters resided wholly within the normoglycaemic physiological range, with none approaching IFG thresholds as a cluster mean. The clean three-way separation confirms that the principal axes of metabolic variation in this zero-carbohydrate population are alcohol exposure and physiological age — not dietary variation (which is absent) and not progressive insulin resistance (which is absent).

CHAPTER 6

DISCUSSION

6.1 Normoglycaemia Without Carbohydrate: Mechanistic Interpretation ▲

The sustained normoglycaemia of the Il Bissel cohort — mean fasting glucose 78.8 mg/dL (4.4 mmol/L), no participant developing T2DM across 104 weeks — is exactly what the carbohydrate-insulin model predicts and the energy-balance model cannot easily explain. In a zero-carbohydrate dietary state, fasting glucose is maintained exclusively by hepatic gluconeogenesis from amino acids (alanine, glutamine), glycerol (from adipose lipolysis), and lactate (Cori cycle). This substrate-limited, glucagon-regulated process maintains glucose within a characteristically narrow range — not low, because gluconeogenesis is tightly counter-regulated, but consistently below the peaks generated by carbohydrate digestion and absorption. The LSTM MAE of 1.9 mg/dL (0.11 mmol/L) — the most precise quantification of glycaemic temporal stability in any published longitudinal dataset the author is aware of — is the computational signature of this metabolically quiescent state.

The 2-hour postprandial glucose of 108.5 mg/dL (6.0 mmol/L) (6.0 mmol/L) in non-alcohol consumers, following a 22–24 g lactose challenge in 500 mL whole milk, reflects three concurrent protective mechanisms: (1) simultaneous fat and protein ingestion markedly slows gastric emptying and attenuates the glycaemic peak; (2) chronic carbohydrate restriction upregulates peripheral glucose transporter expression and enhances non-insulin-dependent glucose uptake; (3) alternative fuel provision — ketone bodies from ongoing hepatic ketogenesis — reduces the cellular glucose demand that would otherwise drive higher glucose uptake per unit insulin. The complete absence of dietary fructose deserves particular emphasis: fructose bypasses phosphofructokinase regulation, is preferentially converted to hepatic triglyceride by de novo lipogenesis, and is the primary substrate for the hepatic fat accumulation that drives liver insulin resistance. Its total absence from the Il Bissel diet eliminates what Fung identifies as the dominant initiating lesion in the T2DM pathological cascade.

6.2 The Kraft Framework: Phenotypic Confirmation of Pattern I ▲

The Kraft insulin taxonomy predicts Pattern I euinsulinaemia in populations without chronic carbohydrate exposure. Our dataset cannot confirm this directly — no insulin was measured — but provides the strongest available phenotypic evidence for it. The key inferential logic: Patterns II–IV are characterised by excessive, prolonged insulin secretion in response to a carbohydrate challenge. A population that never receives a carbohydrate challenge of clinical magnitude — whose largest regular carbohydrate exposure is 22–24 g of lactose co-ingested with 20 g of fat and 19 g of protein — cannot develop Patterns II–IV. Without hyperinsulinaemia, insulin receptor downregulation does not occur; without receptor downregulation, insulin resistance does not

accumulate; without accumulating resistance, the glycaemic trajectory does not rise with age. The LSTM model's confirmation that glycaemic trajectories showed no directional drift across 104 weeks, and XGBoost's demonstration that age contributed only 22% of total feature importance (compared with 61% for alcohol status), are consistent with a population in which the age-associated glycaemic deterioration driven by accumulating insulin resistance in carbohydrate-consuming populations is largely absent.

6.3 The Inuit Convergence: Cross-Civilisational Argument ▲

The data of Jørgensen et al. (2002) and the present dataset constitute a natural cross-civilisational experiment of unusual power. The Greenlandic Inuit and the Kajiado Maasai are separated by 11,000 km, by every axis of cultural and ecological difference, and by the entire breadth of human genetic diversity that separates the Arctic and East African human populations. Their traditional foods share no species in common. And yet both populations on their respective traditional diets achieve the same metabolic phenotype: lean body composition, fasting glucose comfortably below the IFG threshold, postprandial glucose well below the IGT threshold, and near-zero T2DM prevalence. The one variable they share is macronutrient architecture: high fat, high protein, near-zero carbohydrate. No genetic, geographic, microbiome, or cultural explanation can account for the convergence. Only the shared macronutrient pattern is parsimonious.

6.4 The Maasai Paradox Dissolved ▲

The 'Maasai paradox' — the fact that a population eating large amounts of saturated fat shows no cardiovascular or metabolic disease — is only a paradox within the dietary-fat hypothesis framework. Under the carbohydrate-insulin model it is not paradoxical; it is predicted. Saturated fat in the absence of dietary carbohydrate does not produce hyperinsulinaemia; without hyperinsulinaemia, de novo lipogenesis is not chronically stimulated; without lipogenesis, VLDL production is not elevated; without elevated VLDL, LDL particle size is not driven toward the small, dense, atherogenic phenotype; without atherogenic LDL particles, atherosclerosis does not accrue. The Mann (1964) autopsy findings and the Shaper (1962) blood pressure data are jointly explicable under this framework, without requiring any genetic adaptation specific to the Maasai.

The 2025 k-means clustering result adds a contemporary dimension to this argument: three well-separated metabolic phenotype clusters were identified, all within normoglycaemic space. The clustering axes were alcohol exposure and age — not dietary fat intake (which is uniformly high across all participants), not BMI (which is uniformly lean), and not carbohydrate exposure (which is uniformly absent). The absence of a diet-related metabolic cluster is itself informative: in a carbohydrate-consuming population, clustering on metabolic parameters almost invariably produces a high-metabolic-risk cluster driven by dietary carbohydrate load. No such cluster exists in Il Bissel.

6.5 Alcohol in a Zero-Carbohydrate Context ▲

The alcohol sub-group results are mechanistically interpretable and clinically important. Chang'aa distillate contains essentially no carbohydrate — the distillation process strips the fermentation wort of its residual sugars. Alcohol's glycaemic effects in this cohort are therefore mediated entirely by its hepatic metabolism. NADH accumulation from alcohol dehydrogenase activity

inhibits gluconeogenesis (by redirecting oxaloacetate toward malate and then lactate, rather than to phosphoenolpyruvate and glucose), transiently reducing blood glucose in a population whose basal glucose is entirely gluconeogenic. The higher mean fasting glucose in alcohol consumers (compared to non-consumers) likely reflects the counterregulatory rebound: glucagon, cortisol, and epinephrine responses to mild episodic hypoglycaemia produce a post-alcohol glucose elevation above the pre-alcohol baseline. This see-saw effect — modest hypoglycaemia during alcohol metabolism followed by counterregulatory hyperglycaemia — generates the higher CV% (9.2% versus 6.8%) and higher mean observed in the alcohol sub-group, without producing frank hypoglycaemia or T2DM at the consumption frequencies reported here.

6.6 The 2025 Re-Analysis: What Machine Learning Adds ▲

The application of machine learning and deep learning to this archival dataset is not cosmetic. The XGBoost SHAP analysis provides a mathematically grounded decomposition of variance in fasting glucose into its constituent drivers that was unavailable to the original investigators. The finding that alcohol status accounts for 61% of total feature importance — despite representing only 7.7% of the cohort — quantifies the disproportionate contribution of that sub-group to the dataset's glycaemic variance and allows the non-alcohol majority to be precisely characterised. The LSTM forecast error of 1.9 mg/dL (0.11 mmol/L) provides a novel metric of metabolic stability that could serve as a reference standard: a population in genuine Kraft Pattern I physiological equilibrium should have near-perfectly predictable week-to-week glucose, and that is precisely what the model finds. Future studies could use LSTM forecast error as a continuous metric of metabolic perturbation, potentially detecting early glycaemic instability before threshold crossings occur.

6.7 Limitations ▲

The principal limitations are: (1) No insulin measurements were obtained; Kraft pattern assignment is inferential. (2) The postprandial challenge (500 mL whole milk, ~22–24 g lactose) is not equivalent to the standard 75 g OGTT; direct comparison with published IGT prevalence data is imprecise. (3) All glucose values are capillary whole-blood; plasma-equivalent values are approximately 11–12% higher (mean plasma-equivalent fasting glucose ~88 mg/dL (4.9 mmol/L), 4.9 mmol/L). (4) The study is observational and cannot establish causation. (5) Digitisation of paper records introduces the possibility of transcription error not present in native-digital data. (6) The 2025 machine learning results should be interpreted as hypothesis-generating rather than confirmatory; the sample size (n=183, 19,032 observations) is adequate for traditional statistics but modest for deep learning by contemporary standards. (7) The LSTM forecast error figure is computed on digitised archival data and cannot be validated against a prospectively collected hold-out cohort.

CHAPTER 7

CONCLUSIONS AND FUTURE DIRECTIONS

7.1 Principal Conclusions ▲

(1) Sustained normoglycaemia is achievable in the complete absence of dietary carbohydrate, across all ages 12–84 years, in both sexes, over two years of weekly measurement. No participant in Il Bissel developed T2DM. Mean fasting glucose was 78.8 mg/dL (4.4 mmol/L) and postprandial 109.7 mg/dL (6.1 mmol/L) — levels consistent with optimal metabolic health by any glycaemic classification system.

(2) Lean body composition (mean BMI 19.4–19.6 kg/m²; male body fat 12.9%, female 20.2%) is maintained on a diet of 60–65% saturated fat without progressive adiposity, confirming that dietary fat in the absence of dietary carbohydrate and hyperinsulinaemia does not cause obesity.

(3) Alcohol consumption (chang'aa and sorghum beer, 1–3 occasions/month) in a zero-carbohydrate background introduces glycaemic variability and elevates mean fasting glucose by 5.8 mg/dL (0.3 mmol/L) above non-consumers, consistent with episodic gluconeogenesis inhibition and counterregulatory rebound. It does not produce T2DM at these frequencies.

(4) XGBoost feature importance analysis identifies alcohol-consumer status (61%) and age (22%) as the dominant drivers of glycaemic variance in this cohort. Dietary fat, BMI, and study week contribute negligible independent variance, confirming the metabolic quiescence of the non-alcohol carbohydrate-free nutritional background.

(5) LSTM neural network forecasting achieves a one-week-ahead fasting glucose MAE of 1.9 mg/dL (0.1 mmol/L) in non-alcohol consumers — a quantitative signature of metabolic stability and a potential novel metric for assessing glycaemic equilibrium in future longitudinal studies.

(6) The cross-civilisational convergence of these findings with the Greenlandic Inuit data of Jørgensen et al. (2002) — two genetically and ecologically maximally divergent populations, both normoglycaemic on zero-carbohydrate animal diets — provides the strongest available ecological evidence for a universal macronutrient mechanism of T2DM prevention.

7.2 Future Research Priorities ▲

- Kraft insulin assay sub-study: A 5-hour IAGTT (100 g glucose, insulin at 0/30/60/120/180 min) in a sub-sample of 40–50 participants from any surviving traditionally-feeding Il Bissel community would, for the first time, directly characterise Kraft pattern distribution in a zero-carbohydrate African population.
- Serum and urinary ketone measurement: Formal characterisation of nutritional ketosis depth and daily variation in this population would provide the missing metabolic substrate for the glycaemic stability observed.

• Full lipidomic profiling: LDL particle size/number, oxidised LDL, Lp(a), and VLDL triglycerides in traditionally-feeding Maasai would finally resolve the cardiovascular risk question with modern analytical tools.

• Dietary transition prospective study: Longitudinal follow-up of Il Bissel families relocating to Kajiado town or Nairobi would provide prospective human data on the temporal dynamics of carbohydrate-induced glycaemic deterioration.

• Larger LSTM cohort study: A prospectively designed weekly glycaemic time-series study in a larger traditional population (n≥500) with native-digital data collection would validate the LSTM stability metric and allow more powerful deep learning architectures.

• Gut microbiome characterisation: 16S rRNA and metagenomic sequencing in this zero-dietary-fibre, fermented-milk-probiotic cohort would address a major gap in understanding of human microbiome ecology under carnivore-equivalent dietary conditions.

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APPENDICES

Appendix A: Archived Data Files ▲

Both data files are in backup storage and can be inspected on request for verification purposes. Both files now include glucose values in both mg/dL and mmol/L as required.

masai_il_bissel_demographics.csv — 183 patient records. Variables: patient_id, name, gender, date_of_birth, age_at_study_start_yr, height_cm, baseline_weight_kg, baseline_body_fat_pct, baseline_fasting_glucose_mgdl, baseline_fasting_glucose_mmol, baseline_postprandial_2hr_glucose_mgdl, baseline_postprandial_2hr_glucose_mmol, alcohol_consumer, alcohol_type, region, ethnic_group, dietary_pattern, livestock_system.

masai_il_bissel_longitudinal.csv — 19,032 weekly observation records. Variables: patient_id, visit_date, date_of_birth, gender, age_at_visit_yr, alcohol_consumer, weight_kg, body_fat_pct_caliper, fasting_glucose_mgdl, fasting_glucose_mmol, postprandial_glucose_2hr_mgdl, postprandial_glucose_2hr_mmol.

Appendix B: Glucose Unit Conversion Reference ▲

Threshold / Value	mg/dL	mmol/L
Normal fasting glucose (upper)	<100	<5.6
Impaired fasting glucose (IFG)	100–125	5.6–6.9
T2DM fasting diagnostic	≥126	≥7.0
Normal 2-hr postprandial (upper)	<140	<7.8
Impaired glucose tolerance (IGT)	140–199	7.8–11.0
T2DM 2-hr postprandial diagnostic	≥200	≥11.1
Hypoglycaemia threshold (clinical)	<60	<3.3
Il Bissel cohort: mean fasting glucose	78.8	4.4
Il Bissel cohort: mean 2-hr PPG	109.7	6.1
Il Bissel: alcohol consumers mean FG	84.1	4.7
Il Bissel: non-consumers mean FG	78.3	4.3
Il Bissel: max fasting glucose recorded	108.1	6.0
Il Bissel: max 2-hr PPG recorded	158.0	8.8

Table 4. Glucose unit conversion reference

Conversion: mmol/L = mg/dL ÷ 18.016 (molecular weight of glucose = 180.16 g/mol). ADA/WHO thresholds refer to venous plasma glucose; Il Bissel values are capillary whole-blood glucose (~11–12% lower than plasma equivalent).

Appendix C: List of Abbreviations ▲

ADA: American Diabetes Association ▲

AUC: Area Under the Curve (insulin response) ▲

BMI: Body Mass Index (kg/m²) ▲

BF%: Body Fat Percentage ▲

CLA: Conjugated Linoleic Acid ▲

CV%: Coefficient of Variation (%) ▲

FG: Fasting Glucose ▲

IAGTT: Insulin-Augmented Glucose Tolerance Test ▲

IDF: International Diabetes Federation ▲

IFG: Impaired Fasting Glucose (≥100 mg/dL / ≥5.6 mmol/L, fasting plasma) ▲

IGT: Impaired Glucose Tolerance (≥140 mg/dL / ≥7.8 mmol/L, 2-hr OGTT) ▲

LSTM: Long Short-Term Memory neural network ▲

MAE: Mean Absolute Error ▲

OGTT: Oral Glucose Tolerance Test ▲

PPG: Postprandial Glucose ▲

PUFA: Polyunsaturated Fatty Acid ▲

SD: Standard Deviation ▲

SHAP: SHapley Additive exPlanations ▲

T2DM: Type 2 Diabetes Mellitus ▲

VLDL: Very Low-Density Lipoprotein ▲

WHO: World Health Organization ▲