



Food Insecurity and Glycaemic Control: The Clinic Visit That Cannot Fix the Kitchen

Alina Craciun^{1*}, Joao Vasconcelos² and Duncan Sinclair³

¹Department of Public Health, Banat University Hospital, Timisoara, Romania

²Department of Clinical Nutrition, Algarve Institute of Clinical Nutrition, Faro, Portugal

³Centre for Health Inequalities, Clyde Valley University Hospital, Glasgow, United Kingdom

*Correspondence to: Alina Craciun, Department of Public Health, Banat University Hospital, Timisoara, Romania

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Abstract

Food insecurity is consistently associated with worse glycaemic control, and the association survives adjustment. In a safety-net population where 46 per cent of patients were food insecure, 42 per cent of those patients had an HbA1c of 8.5 per cent or above against 33 per cent of food-secure patients, an adjusted odds ratio of 1.48. In a longitudinal cohort followed for a mean of 37 months, food insecurity was associated with an HbA1c 0.6 percentage points higher, and the gap did not narrow over time. The more instructive finding is what was not associated. In the same cohort, living in an area of low physical food access made no significant difference, and in a separate health system of 5,500 patients, area-level food insecurity alone did not predict control while self-reported unmet food need did. Intervention evidence is weaker than the observational literature: results come largely from before-and-after studies with substantial attrition, and a randomised evaluation found no effect on HbA1c except within a self-selected fully engaged subgroup. This review sets out what is established, what is measured badly, and why the geographic framing misleads.

Keywords: Food insecurity; Glycaemic control; Social determinants; Food is medicine; Health system screening

Introduction

Food insecurity is defined as limited or uncertain access to adequate food, and it is measured at the household level by validated survey instruments rather than by income or geography [1,2]. It is common among people with diabetes, and considerably more common in populations attending safety-net and publicly funded services [3,4,19]. Metabolic associations have been reported in national survey data [20] and in children with diabetes [29].

The mechanism by which it might worsen diabetes outcomes is not mysterious. Managing the condition requires predictable access to food, and unpredictability interacts badly with insulin and sulfonylurea therapy, with the cost of medication competing for the same limited budget [5,6,21]. Self-care behaviours and pantry-based cohorts show the same pattern [26,27]. What is less obvious is how much of the observed association is explained by these pathways and how much by everything else that accompanies poverty [7,28].

Distress has been proposed as one mediating route [30], and cardiovascular risk tracks food security in the longer term [31,32].

This review examines the strength and consistency of the association, the distinction between household food insecurity and geographic food access, and the considerably thinner evidence that intervening changes anything.

Material and Methods

Search strategy: PubMed and the archives of the major diabetes, nutrition, public health and primary care journals were searched for studies of food insecurity in diabetes. Search terms combined food insecurity and food access with glycaemic control, HbA1c, diabetes, food desert, produce prescription and medically tailored meals. Reference lists of overview articles [8,9] were screened by hand for primary sources.

Eligibility: Studies were included if they measured food insecurity with a defined instrument and related it to a glycaemic

or utilisation outcome, or evaluated an intervention supplying food. Studies using area-level proxies are included and identified as such, because the distinction between individual and area measurement is central to this review.

Data extraction and handling: Design, setting, sample size, the instrument used and the reported estimate were extracted as published. Whether food insecurity was measured at household or area level is stated alongside every estimate. This review reports measured associations and trial outcomes and makes no dietary, caloric or exercise recommendations. No pooling or re-analysis was performed. Figures 1 to 6 and Tables 1 to 3 reproduce published values only. This is a narrative synthesis and was not registered as a systematic review.

Results

The core association: In 711 patients with type 2 diabetes attending safety-net clinics, the prevalence of food insecurity was 46 per cent (Figure 2). Poor glycaemic control, defined as HbA1c of 8.5 per cent or above, was present in 42 per cent of food-insecure patients against 33 per cent of food-secure patients, an adjusted odds ratio of 1.48 (95% CI 1.07 to 2.04), and the relationship persisted after adjustment for age, sex, income, education, body mass index, insulin use, tobacco use and medication adherence (Figure 1) [3].

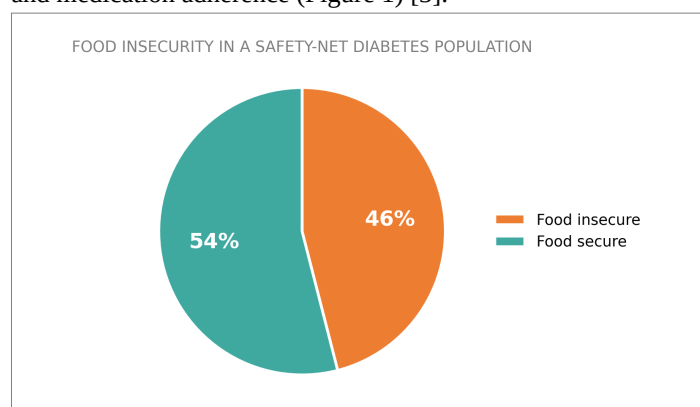


Figure 2: Prevalence of food insecurity in a safety-net diabetes population [3]

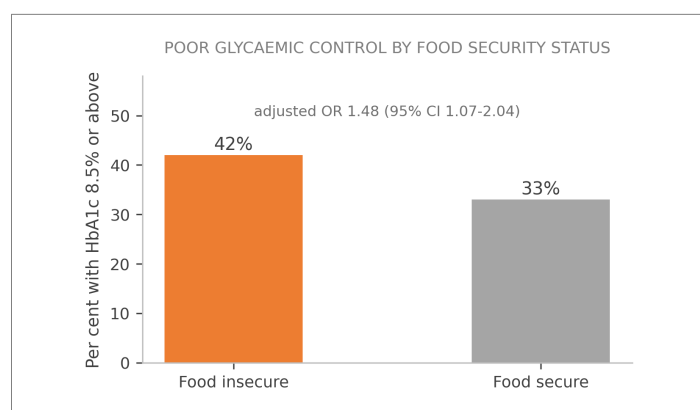


Figure 1: Poor glycaemic control by food security status [3]

The proposed pathway: Food-insecure participants more often reported difficulty affording a suitable diet, at 64 against

49 per cent ($p < 0.001$), and reported lower diabetes-specific self-efficacy and higher diabetes-related emotional distress (both $p < 0.001$). Difficulty following a suitable diet and emotional distress partially mediated the association, but the direct association persisted, so mediation was incomplete (Figure 6) [3,10].

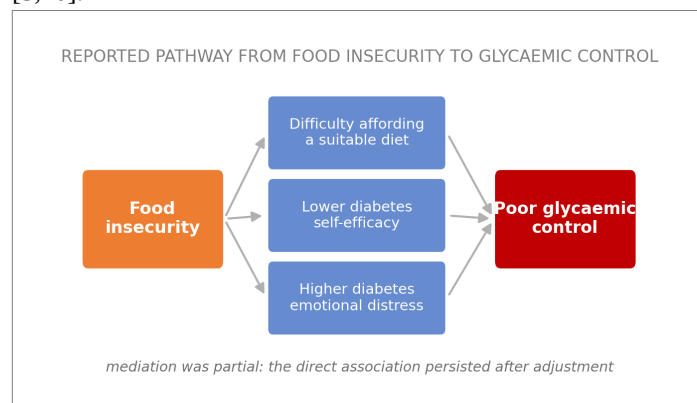


Figure 6: Reported pathway from food insecurity to glycaemic control [3,10]

Longitudinal confirmation, and a negative result beside it: A prospective cohort of 391 patients followed for a mean of 37 months found 20 per cent food insecure and 31 per cent resident in an area of low physical food access. Food insecurity was associated with an HbA1c 0.6 percentage points higher (95% CI 0.4 to 0.8, $p < 0.0001$), a difference that did not narrow over time ($p = 0.50$). Living in an area of low physical food access was not associated with any difference in HbA1c (0.2 percentage points, 95% CI -0.2 to 0.5, $p = 0.33$) (Figure 4) [11].

Individual need against area measurement: A health system analysis of 5,500 patients with type 2 diabetes screened for health-related social needs found 7.1 per cent reporting an unmet food need. Self-reported food need was associated with poor control (adjusted OR 1.59, 95% CI 1.26 to 2.00), while area-level food insecurity alone was not (adjusted OR 1.15, 95% CI 0.96 to 1.39) (Figure 3). Patients reporting food need had raised odds whether they lived in food-secure areas (OR 1.83) or food-insecure areas (OR 1.72) [12].

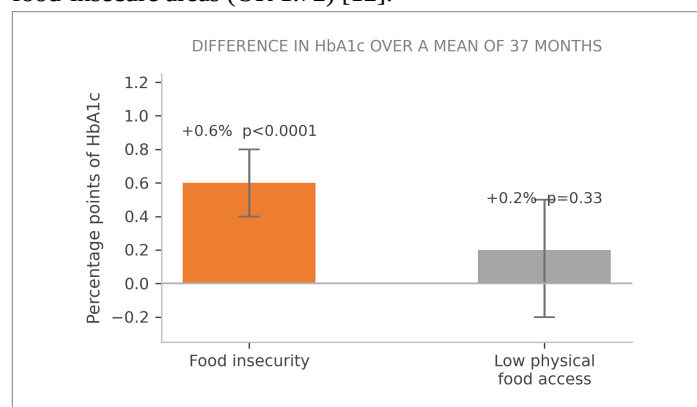


Figure 4: Difference in HbA1c associated with food insecurity and with low physical food access [11]

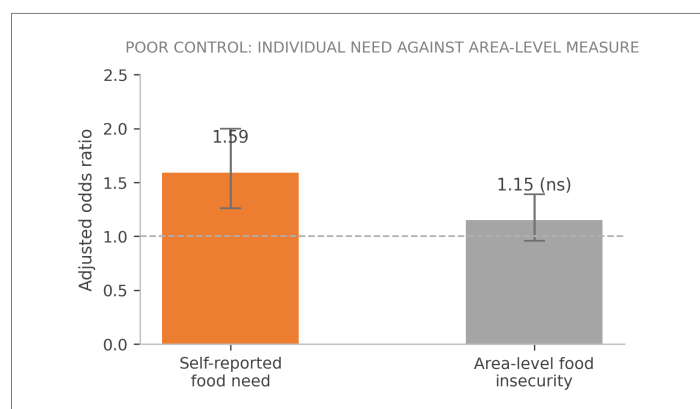


Figure 3: Poor glycaemic control by individual need and by area-level measure [12]

In type 1 diabetes and in younger patients: Among youth and young adults with type 1 diabetes, food insecurity prevalence was 19.5 per cent, and those from food-insecure households had 2.37 times the adjusted odds of an HbA1c above 9 per cent (95% CI 1.10 to 5.09) and an adjusted emergency department visit rate 2.95 times as great (95% CI 1.17 to 7.45) (Figure 5) [13].

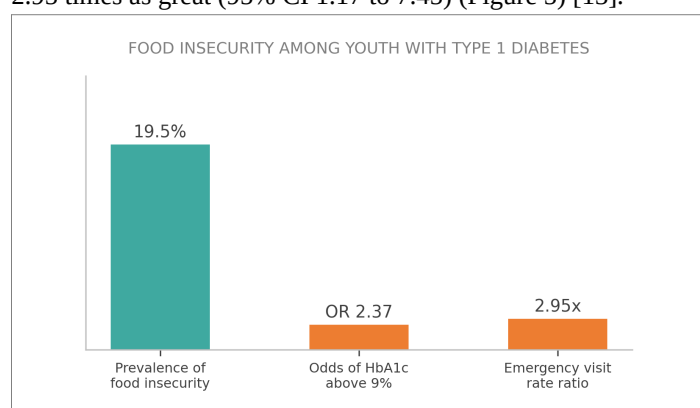


Figure 5: Food insecurity among youth and young adults with type 1 diabetes [13]

Study	Measure used	Principal reported finding
Seligman et al. 2012 [3]	Household survey, n = 711	46% food insecure; adjusted OR 1.48 for HbA1c 8.5% or above
Berkowitz et al. 2018 [11]	Household survey plus area access, n = 391	HbA1c +0.6% with insecurity; no effect of low physical access
Health system analysis [12]	Self-report plus area measure, n = 5,500	Individual need OR 1.59; area-level alone not significant
SEARCH analysis [13]	Household survey, youth with type 1	OR 2.37 for HbA1c above 9%; 2.95x emergency visits
Dietary quality analysis [14]	Survey, diet quality and insecurity	Both factors together carried the highest odds
Produce prescription study [15]	Before-and-after, n = 97	HbA1c fell 1.3 percentage points
Rural health centre study [9]	Before-and-after, n = 333	Uncontrolled diabetes fell 46%, with 48% attrition

Table 1: Principal studies reviewed, with the finding as reported by the original investigators

What interventions have shown: Before-and-after evaluations report meaningful improvements. A produce

prescription programme combined with self-management education in 97 adults reported a fall in HbA1c of 1.3 percentage points, with larger reductions among those redeeming more prescriptions [9,15]. A study of 333 adults at rural community health centres reported a 46 per cent fall in the number with an HbA1c above 9 per cent, but lost 48 per cent of participants to follow-up [9]. Systematic reviews of food-is-medicine interventions describe improvements alongside substantial heterogeneity and variable methodological quality [8,9,16]. Meal delivery has been associated with reduced costly health care use [22], and food-bank and medically appropriate food programmes report glycaemic improvement in uncontrolled designs [23,24,25]. A randomised evaluation found no effect on HbA1c except among participants who were fully engaged with the intervention, which is a non-random subgroup [9,17].

Design	What it can support	What it cannot
Cross-sectional survey	Association and prevalence	Direction of effect [3,12]
Longitudinal cohort	Persistence over time	Causation; unmeasured confounding [11]
Before-and-after study	Change in a treated group	Attribution, when attrition is high [9]
Randomised evaluation	Effect of assignment	Subgroup claims made post hoc [9,17]

Table 2: What each study design in this literature is able to establish

Comparison	Individual measure	Area-level measure
Longitudinal HbA1c	+0.6% (p < 0.0001)	+0.2% (p = 0.33)
Odds of poor control	OR 1.59 (1.26-2.00)	OR 1.15 (0.96-1.39)
Conclusion	Consistently associated	Not independently predictive

Table 3: Household food insecurity against geographic food access as predictors of glycaemic control [11,12]

Cost-related medication underuse: Food insecurity rarely occurs alone. Patients facing constrained budgets report trading off medication against food, and cost-related medication underuse is more common among the food insecure, which offers a route to worse control that has nothing to do with what is eaten [5,21]. Analyses reporting associations with self-care behaviours and with sleep quality describe a similar picture of competing demands rather than of dietary choice [26,30].

Where the strongest intervention evidence sits: Reviews judge the evidence most robust for medically tailored meals delivered to people with complex or severe disease, rather than for produce prescriptions or food bank referral [9,22]. Meal delivery has been associated with reduced use of costly health care in dually eligible populations [22], and food bank programmes supplying diabetes-appropriate food reported glycaemic improvement across three states, though again without randomisation [24,25]. Closer integration with the health system has been proposed as the factor distinguishing programmes that work from those that do not [9].

Discussion

The association is about as well established as an observational finding in this field can be. It appears in safety-net clinics, in a longitudinal cohort where it did not attenuate over three years, in a large health system, and in young people with type 1 diabetes, with effect sizes that are modest but consistent in direction [3,11,12,13].

The finding that should change how services respond is the negative one. Living in an area with poor physical access to a supermarket was not associated with worse control, and area-level food insecurity did not independently predict it, while individual reported need did in both cohorts that tested the comparison [11,12]. The popular framing of the problem as a matter of geography, of food deserts and the distance to the nearest shop, is not supported by these data. People who cannot afford food have worse glycaemic control whether or not there is a supermarket nearby, and people living near no supermarket but able to afford food largely do not.

That distinction has a direct operational consequence. Targeting resources by neighbourhood deprivation index will misallocate them in both directions, because a substantial share of food-insecure patients live in food-secure areas [12]. The measure that predicted the outcome was a question asked of the patient. That is cheap, and it is the step most health systems have not taken.

Where the literature is genuinely weak is intervention. The headline results come from before-and-after studies, and a 46 per cent improvement in a cohort that lost 48 per cent of its participants tells us more about who remained than about what the programme did [9]. The randomised evidence is less encouraging: no effect on HbA1c except within a fully engaged subgroup, which is precisely the comparison that cannot support a causal claim, because engagement is a choice made by participants and tracks everything else that predicts good control [9,17].

None of that argues against supplying food to people who need it, which has a justification independent of HbA1c. It argues against presenting these programmes as though their glycaemic benefit were established, and against clinicians concluding that a referral has solved the problem. A clinic visit can identify food insecurity reliably with a two-question screen; what it can do about it afterwards is a good deal more limited than the enthusiasm in this literature suggests [12,18]. Unmet social needs are unevenly distributed across clinic populations [33], and screening for them carries its own unintended consequences if nothing follows the question [34].

There is a screening question underneath all of this that the literature raises and does not settle. Asking about food insecurity in a clinic identifies a real and modifiable determinant, and validated two-item instruments make it quick [2,12]. But asking creates an expectation, and where no effective referral pathway exists the question may do more for documentation than for the

patient [34]. Commentary on social needs screening has warned specifically about this, and the warning applies with force here, because the intervention evidence is weaker than the screening evidence [33,34].

The honest position is therefore split. That food insecurity is associated with worse glycaemic control is established across designs, populations and both types of diabetes [3,11,12,13,20]. That identifying it in clinic and referring onward improves glycaemic control is not established, and the studies most often cited in support of it are uncontrolled [9,15,17]. Those two statements are compatible, and conflating them produces the common but unsupported claim that screening improves outcomes.

The distinction between measuring need and meeting it also shapes what these data can tell a clinician. The screening instruments perform well, they are brief, and they identify a group whose glycaemic control is measurably worse and whose emergency use is higher [2,13,33]. What the same literature does not supply is a referral endpoint with demonstrated glycaemic benefit, which means the clinician who asks the question is better informed but not necessarily better equipped [9,17,34].

A last observation concerns type 1 diabetes specifically. The strongest effect sizes in this review come from young people with type 1 diabetes, where food insecurity carried more than double the odds of an HbA1c above 9 per cent and nearly three times the emergency department visit rate [13]. Insulin dosing is matched to carbohydrate intake, so unpredictable access to food is not merely a dietary problem in this group but a dosing one, with hypoglycaemia the immediate risk rather than the eventual complication [13,29]. That mechanism is specific, plausible and largely unexamined in the intervention literature, which has concentrated on type 2 diabetes [9,16].

Study limitations

The limitations belong to the underlying literature. The principal associations are cross-sectional or observational, so reverse causation and residual confounding cannot be excluded, and food insecurity is entangled with income, housing, employment and health literacy [3,7,12]. Food insecurity is self-reported and subject to stigma, which plausibly produces under-reporting [1,2]. Definitions of poor glycaemic control differ between studies, at thresholds of 8.5 and 9 per cent, which affects the reported odds [3,13]. Intervention studies are predominantly uncontrolled, with attrition reaching 48 per cent in one case, and reviews describe substantial heterogeneity in quality [9,16]. The single randomised finding of benefit rests on a non-random engaged subgroup [9,17]. And almost all of this evidence comes from the United States, where the relationship between insurance, income and access to both food and medication differs from other high-income settings [8,12].

Conclusion

Household food insecurity is consistently associated with worse glycaemic control, by roughly half a percentage point of HbA1c in longitudinal follow-up, and the gap does not close over time. Geographic measures of food access are not associated with it, which means the problem is affordability rather than distance and that targeting by neighbourhood will miss a large share of affected patients. Asking the patient is the step that identifies them, and it is inexpensive. What follows the question is the weaker part: the evidence that supplying food improves glycaemic control comes mostly from uncontrolled studies with heavy attrition, and the one randomised look found nothing except in a subgroup that selected itself.

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Conflict of Interest

The authors declare no competing interests.

Ethics Statement

This article reviews previously published studies only. No new patient data were collected and no participants were recruited, so ethical approval was not required.

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