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World Development Vol. 74, pp. 15–24, 2015
0305-750X/
http://dx.doi.org/10.1016/j.worlddev.2015.04.007

Using Agriculture to Improve Child Health: Promoting Orange Sweet Potatoes Reduces Diarrhea

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Summary. — Vitamin A deficiency (VAD) is prevalent throughout the developing world, and causes night blindness and increases child morbidity and mortality. We studied the health benefits of biofortification in reducing VAD, using a cluster-randomized impact evaluation in 36 villages in northern Mozambique. Based on a sample of 1,321 observations of children under the age 5, biofortification reduced diarrhea prevalence by 11.4 percentage points (95% CI 2.0–20.8), and by 18.9 percentage points in children under the age three (95% CI 6.6–68.3). Diarrhea duration was also reduced. This is promising evidence that child health can be improved through agricultural interventions such as biofortification.

Key words — nutrition, child health, morbidity, micronutrients, vitamin A, diarrhea

1. INTRODUCTION

Vitamin A deficiency (VAD) is prevalent in young children throughout the developing world, afflicting 190 million children under the age five worldwide (WHO, 2009). VAD is rated as a severe public health problem in 73 countries, many located in Sub-Saharan Africa (WHO, 2009). The impacts of VAD in early childhood include not only night blindness, but increased risk of other morbidity, as well as mortality. In a systematic review covering 43 randomized controlled trials (RCT) or cluster-RCTs, Mayo-Wilson, Imdad, Herzer, Yakoob, and Bhutta (2011) found that vitamin A supplementation (VAS) reduced diarrhea incidence by 15% (based on 13 studies) and measles incidence by 50% (six studies) in children aged six months to five years. Further, they found that VAS reduced all-cause mortality for these children by 24% (43 studies); evidence that the authors rate as high quality. [1]

According to Villamor and Fawzi (2005), the impact of vitamin A on measles is due to increased lymphocyte proliferation, which increases short-term antibody production. In contrast, the reduction in severe diarrhea is likely due to the role of vitamin A in restoring and maintaining gut mucosal integrity, though there may be other immunological pathways. These impacts are believed to be the strongest among children who are undernourished or suffering from severe infection. While reductions in these morbidities contribute to the impact of vitamin A on all-cause mortality, that impact is likely also bolstered by the ability of vitamin A to increase T-cell counts, particularly of the CD4 population, in children infected with HIV.

Akachi and Canning (2010) argue that improving nutrition and reducing morbidity should be a key focus for development interventions, as child morbidity is a strong predictor of later-life outcomes. Indeed, individuals experiencing high levels of childhood morbidity have been shown to have reduced cognition, impaired adult stature, and be at increased risk of later-life morbidity and mortality. [2] In Sub-Saharan Africa in particular, achievements in reducing child mortality may have outpaced reductions in childhood morbidity. This hypothesis is based on the fact that, despite advances in reducing child mortality in Africa, today's adult Africans are shorter than their predecessors, which may be an indicator of increased childhood morbidity (Akachi & Canning, 2010). Reducing childhood morbidity may prove an effective and sustainable option for improving the productivity and longevity of the next generation of Africans.

However, indefinitely continuing high-frequency supplementation can be costly. Edejer *et al.* (2005) estimated that VAS costs $2.71 per recipient per dose. Relying on VAS alone, at least quarterly dosing is required for maximum impact, at a cost of $10.84/child/year. This implies that alleviating VAD worldwide through supplementation alone would cost at least $2.8 billion per year, implying a net present value of nearly $40 billion to alleviate VAD for the next 20 years. If other interventions can effectively complement VAS, these may be useful not only for reducing the cost of addressing VAD now, but also as more sustainable future alternatives to VAS, once the prevalence of VAD has been sufficiently reduced.

One complementary approach to reducing VAD and other micronutrient deficiencies is to encourage shifts toward more micronutrient-dense diets (Ruel, Alderman, & Maternal Child Nutrition Study Group, 2013). In rural areas, where diets often include a great deal of own-produced food, such interventions must occur through agriculture. Potential interventions include home gardening, various types of animal husbandry, aquaculture, and biofortification of staple crops (Masset, Haddad, Cornelius, & Isaza-Castro, 2012). Yet many of these strategies are still unproven in improving nutrition or health (Bhutta *et al.*, 2008; Ruel *et al.*, 2013). The primary goal of these programs is generally to improve dietary diversity, yet there is little existing evidence that such interventions actually affect dietary diversity, not to mention health. Bhutta *et al.* (2008) suggest that lack of evidence is partially due to a general paucity of rigorous evaluations of agricultural interventions to deal with micronutrient deficiencies.

One recent exception is the biofortification of staple crops (Bouis, Hotz, McClafferty, Meenakshi, & Pfieffer, 2011). Biofortification involves the breeding of micronutrients into staple

* We thank Dan Gilligan, Melissa Hidrobo, Mourad Moursi, and three anonymous referees for suggestions and contributions that have greatly improved this manuscript. Final revision accepted: April 17, 2015.

crops using conventional breeding practices to control vitamin A, iron, and zinc deficiencies (Nestel, Bouis, Meenakshi, & Pfieffer, 2006). Since biofortification works through improving the nutritional quality of the variety of staple crops that households are already consuming, it is a promising, potentially cost-effective way to reduce VAD or other micronutrient deficiencies (Gilligan, 2012). This approach is currently recommended as a complement to ongoing efforts of VAS, with the potential to act as a longer-term replacement once the prevalence and severity of VAD in Africa has been significantly reduced.

In this work we examined the impact on child health of a targeted biofortification intervention for improving vitamin A intakes among young children in northern Mozambique. At the time of the intervention, 69% of children under the age five in Mozambique were vitamin A deficient (Aguayo, Kahn, Ismael, & Meershoek, 2005). The intervention, called the HarvestPlus Reaching End Users (REU) program, promoted the cultivation of orange sweet potatoes (OSP) in home gardens, rather than the traditionally grown white or yellow varieties. The intervention was designed as a scaled up version of the prior Toward Sustainable Nutrition Improvement (TSNI) intervention that distributed OSP at a much higher cost per beneficiary (Low *et al.*, 2007). The intervention included modules on production, consumption, and exchange of OSP. The REU was successful in promoting OSP adoption, increasing dietary intake of vitamin A, and decreasing VAD prevalence among both women of child bearing age and children (Hotz, Loechl, de Brauw *et al.*, 2012; Hotz, Loechl, Lubowa *et al.*, 2012). In this paper, we study the impacts of the REU on childhood morbidity. Primary outcome indicators include prevalence and duration of diarrhea within the two weeks prior to the interview.

2. METHODS

(a) *Intervention*

The HarvestPlus REU project aimed to promote and distribute provitamin A-rich OSP in Mozambique with the goal of reducing vitamin A deficiency among children under the age five and women of childbearing age. The REU included three components: seed systems, demand creation, and marketing. In the seed systems component, vines for growing OSP were distributed to households in treatment villages and households were trained in planting OSP, how to disinfect vines, and vine preservation.[3] In the demand creation component, mothers in treatment households were offered training on the benefits of consuming OSP, methods of preparing OSP, and other general health messages. Finally, the project included a marketing component to increase visibility of and demand for OSP in local markets.[4] The project worked in Zambézia with 144 farmer groups, approximately corresponding to villages, and reached over 10,000 farmers in total. Villages were chosen for the study based on their agricultural potential for growing sweet potatoes and maintaining sweet potato vines over the dry season.

The impact evaluation included 36 villages spread over three strata; two strata were in the north of Zambézia (Milange district and Gurué district) and a third stratum in the south (combining Mopeia and Nicoadala districts). Within strata, 2/3 of villages were randomly selected to receive the program and 1/3 were reserved as controls, in which the program would be implemented following endline data collection.[5] Villages included in the impact evaluation sample were chosen to ensure that no other health or agriculture-health interventions were taking place simultaneously in those villages. The program, as described, was implemented in 2007, 2008, and early 2009 with baseline data collection in 2006 (October to December) and endline data collection in June 2009 for dietary intakes, nutrition, and morbidity, and in August and September of 2009 for socioeconomics and OSP production. Within study villages, households were selected for inclusion in the study based on the presence of a child under age three within the household.

We first compiled a list of households in the village with children under 36 months, and then took a random sample of 12 households from the list for the main nutrition study. We then added four households from the list to those twelve for measuring anthropometry, morbidity (diarrhea), and food frequency.

(b) *Data*

Information regarding health and the frequency of consumption of different food items was collected at the baseline for one child each from 553 households. At the endline, this information was collected for the same children, plus additional children within these households who were born since the baseline, as well as children in newly recruited households from the same study villages. In total at the endline, 866 children were included in this component from 673 households.

For this paper, OSP consumption is measured through the food frequency questionnaire. The food frequency questionnaire gathered information on patterns of consumption of a variety of foods and food groups, with an emphasis on vitamin A-rich foods. Respondent guardians were asked about the number of times children had consumed 26 specific foods during the past seven days. Diarrhea and other health conditions were measured using a set of relatively standard recall questions asked to a guardian regarding all children under the age 5 resident in targeted households who were included in the sample frame. The pertinent questions in this survey were whether or not the child had diarrhea in the past 14 days, and if so, how many days the diarrhea spell lasted.

Table 1 shows the sample size by age. The baseline recruitment for this study yielded a sample of 540 children observed in 2006 when under the age 3. In addition to following-up these children in 2009, new children under the age three in the same households were also enrolled at that time.[6] The 2009 sample included children aged zero to 5, so the mean age for the 2009 sample was significantly higher than in the 2006 sample. The final row of Table 1 indicates that when restricting the sample to those under the age of 36 months

Table 1. *Sample size by child age*

Age in years	2006	2009	Total
0	93	45	138
1	212	137	349
2	218	112	330
3	14	175	189
4	3	193	196
5	0	119	119
Total	540	781	1,321
Mean age	1.30	2.88	2.24
Total U3	523	294	817
Mean age of U3	1.24	1.23	1.24

(U36), the mean ages across 2006 and 2009 samples were virtually identical.

Baseline characteristics of households and children in the treatment *versus* control group are presented in Table 2. There were no significant differences in children's characteristics between treatment and control at the baseline, with the exception that children in treatment villages had a higher prevalence of "other" illness (that is, other than diarrhea, respiratory illness, or fever). We do find that households included in the study had higher levels of education of the household head in treatment villages relative to control villages. Therefore, we included this variable as a control in our preferred specification.

Attrition of households from the sample was 12%, and attrition of children was 15.6%. However, attrition did not vary by treatment status for either households or children. The only significant difference between those who were followed up and others was the age and gender of the child. As shown in Table 3, children who were younger at baseline, specifically those aged six to 11 months, had a 25% attrition rate, *versus* 13% for children aged 12–35 months. Differential mortality rates by age group partially explained this difference. In Zambézia province, infant mortality was 15%, while mortality of children aged 12–59 months was only 6.8% (MoH, 2009). Therefore, approximately half of the differential attrition was due to differential mortality; however, nonmortality attrition from the study was also higher for younger children. Additionally, attrition was 18% for girls and 13% for boys, a differential that is not at all explained by mortality differences. However, the difference was only significant at the 10% level, and given the number of characteristics tested, at least one significant difference was to be expected. There were no significant differences between those attritting from the control relative to the treatment group (columns four through eight of Table 2).

In previous work, Hotz, Loechl, de Brauw *et al.* (2012) estimated the impact of the REU on OSP consumption and dietary intake of vitamin A, using intensive 24-h dietary recalls, as well as the seven-day recall food frequencies. Children's consumption of OSP increased from 3% of the total sweet potato intake in the baseline (not statistically different across groups) to 22% in control villages and 55% in treatment villages at the endline. Average vitamin A consumption among children at the baseline was about 200 μg retinol activity equivalents (RAE)/day in all villages. At the endline, children were consuming 600 μg RAE/day on average in treatment villages and 350 μg RAE/day on average in control villages. The increase in all villages is likely due to the increased age and total consumption of the sample over time. Among children aged 12–35 months, the prevalence of inadequate intakes was 12% among the treatment group at the endline, and 63% among the control.[7] The increase in average intakes among children and the decrease in the prevalence of inadequate intakes demonstrates that the REU was successful at incorporating OSP into children's diets when they were available, and significantly increased the adequacy of vitamin A intakes among the target population in Mozambique. Here we test whether this intervention exerted downstream impacts on child morbidity.

Table 3. *Child attrition by age*

	Age at baseline	
	6–11 months (%)	12–35 months (%)
Survey attrition rate	24.5	13.4
Local Infant mortality rate (age 0–12 months)	14.7	
Local Child mortality rate (age 12–59 months)		6.8
Predicted mortality from base to endline	12.1	5.1
Nonmortality attrition	12.4	8.3
Of baseline sample, age group composes...	20	80

Table 2. *Baseline balance by treatment assignment and attrition status*

	Control	Treatment	Diff	*p*-Value	Control attritors	Treatment attritors	Diff	*p*-Value
HOUSEHOLDS	184	369			20	47		
Household size (members)	5.81	5.80	0.02	0.916	5.47	5.70	−0.23	0.671
Children U5	3.58	3.49	0.09	0.570	3.21	3.51	−0.30	0.527
Female headship	0.07	0.07	0.00	0.924	0.16	0.11	0.05	0.569
House area (sq m)	21.78	22.09	−0.32	0.697	23.56	21.01	2.55	0.393
Consumption index (quintile)	2.96	3.03	−0.07	0.509	2.83	2.98	−0.15	0.685
Head education (highest grade)	2.66	3.27	−0.62	0.005***	2.75	3.15	−0.40	0.593
Mother education (highest grade)	1.16	1.40	−0.24	0.163	1.45	1.45	0.00	0.996
Grows OSP	0.07	0.10	−0.03	0.305	0.00	0.15	−0.15	0.232
CHILDREN	184	369			27	58		
Age (mos)	21.56	21.62	−0.07	0.937	18.55	22.10	−3.55	0.176
Male	0.53	0.48	0.04	0.322	0.52	0.34	0.17	0.131
Ate OSP past week	0.05	0.06	−0.01	0.735	0.00	0.04	−0.04	0.364
HAZ	−2.24	−2.17	−0.08	0.601	−2.58	−2.26	−0.32	0.549
WAZ	−1.20	−1.17	−0.04	0.785	−1.47	−1.34	−0.13	0.795
Illness in past 2 weeks								
Diarrhea	0.38	0.39	−0.01	0.755	0.37	0.38	−0.01	0.938
Respiratory illness	0.27	0.26	0.00	0.931	0.11	0.24	−0.13	0.166
Fever	0.62	0.57	0.05	0.233	0.41	0.55	−0.14	0.22
Other illness	0.15	0.23	−0.08	0.038**	0.15	0.19	−0.04	0.645
Recent VAS	0.31	0.31	−0.00	0.94	0.38	0.35	0.03	0.77

** Statistical significance at the 5% level.
*** Statistical significance at the 1% level.

(c) *Analysis*

We first employed a linear probability model to estimate the impact of exposure to the program on childhood morbidity. Our outcome of interest was a binary indicator of whether the child had experienced diarrhea in the past 14 days.[8] We estimated

$$D_{ijt} = \alpha + \beta_1(T_j * v_t) + \beta_2 T_j + \beta_3 v_t + X_j'\phi + Z_{ijt}'\gamma + \varepsilon_{ijt}$$

where D_{ijt} was the outcome of interest for child i in household j at time t. T_j was an indicator variable that the household is in a village assigned to treatment and v_t indicated that the survey round was in 2009 after the treatment occurred. The coefficient β_1 on the interaction of T_j and v_t estimated the treatment effect. That is, the differential between the treatment and control group in the *change over time* in the outcome of interest. The estimation also controlled for X_j, a vector of household characteristics, and Z_{ijt}, child characteristics at time t. The variables included in X_j and Z_{ijt} were educational attainment of household head (highest grade completed), educational attainment of the child's mother, household's wealth quintile, size of home (m^2), child's gender, and child's age in months. The equation was estimated with and without these controls. α was a constant, representing diarrhea prevalence among the control group at the baseline (specific to the household and child characteristics that were the excluded categories of controls). ε_{ijt} was a mean-zero error term. Standard errors of the estimates were clustered at the village level.[9]

While the random assignment of treatment status provides the justification for estimating a causal effect, the hypothesis was that OSP consumption was the pathway by which assignment to treatment potentially impacted morbidity. Therefore we tested this hypothesis directly with a two-stage least squares (2SLS) estimation, using treatment assignment as an instrument for child's OSP consumption. That is, we used treatment and time indicators, along with household and child characteristics to predict whether a guardian reported that a child ate OSP in the seven days before the survey and then estimated the impact of predicted OSP consumption on the health outcome of interest, correcting the standard errors for the use of an estimated independent variable. We estimated

$$\widehat{OSP}_{ijt} = \alpha + \mu_1(T_j * v_t) + \mu_2 T_j + \mu_3 v_t + X_j'\phi + Z_{ijt}'\gamma + \varepsilon_{ijt}$$

where the independent variables were as described above. In the second stage we estimated

$$M_{ijt} = \alpha + \delta_1 \widehat{OSP}_{ijt} + X_j'\phi + Z_{ijt}'\gamma + \epsilon_{ijt}.$$

Further, to ensure that differences in morbidity across ages were not driving the results, we repeated these estimations with the sample restricted to observations when a child was under the age 3. In this case the mean ages in both the 2006 and 2009 samples were identical.

In addition to diarrhea occurrence as the outcome, we also estimated analogous models with the outcome as the length of the diarrhea (in days) for those with $D_{ijt} = 1$. Duration, in combination with dehydration, is the primary indicator for severity of diarrhea. Key risk factors for increased diarrhea duration are malnutrition, as evidenced by low weight-for-age, and decreased cell-mediated immunity (Baqui *et al.*, 1993; Bhutta *et al.*, 2000; Black, Brown, & Becker, 1984). Cell-mediated immunity is an immune response that does not involve antibodies but rather involves the activation of phagocytes, T-lymphocytes (T-cells), and cytokines. Given the ability of vitamin A to increase T-cells (Villamor & Fawzi, 2005), it likely also improves cell-mediated immunity.

3. RESULTS

The results for diarrhea occurrence within the past two weeks are presented in Table 4. The first three columns show estimations that employed the full sample. The basic linear probability model shown in column one suggests that both treatment and control study villages experienced a reduction in diarrhea prevalence between baseline and follow-up (likely an artifact of the increased mean age in the 2009 sample). However, the inclusion of household- and child-level controls in the estimates shown in column two indicated that any change in control villages was not distinguishable from zero. In contrast, treatment villages showed reductions in diarrhea prevalence of 11.4 percentage points, relative to the control group (95% CI 2.0–20.8). This represented a 39% reduction based on the mean indicator that 29% of children experienced diarrhea in the two weeks prior to the survey. This effect was robust to the inclusion of all controls and is significant at the 5% level.[10]

Residence in a treatment village was the primary indicator of exposure to the program, however, it is consumption of OSP that was expected to impact the health of children. Therefore, in column 3, we present the results of a two-stage least squares estimation where we estimated the recent occurrence of diarrhea as a function of whether the child regularly ate OSP. Consumption of OSP was proxied by guardian-reports of having eaten OSP in the week prior to the survey, and was instrumented with treatment assignment. The first stage of this estimation is shown in Appendix Table 7. The 2SLS results suggest slightly stronger and more precise impacts; that is, children eating OSP were less likely to experience diarrhea by 15.9 percentage points, an effect that is significant at the 1% level (95% CI 5.9–25.9).

In order to ensure that the results were not driven by significant age differences in the baseline and follow-up samples (as indicated in Table 1), the last three columns of Table 4 present the same set of results for a sample restricted to children under the age of 36 months (U36) at the time of the interview. In these estimations, the mean age of children was the same in both rounds of data collection. These results indicate a large and robust reduction in diarrhea prevalence among the U36 population of treatment villages relative to control villages. After including covariates, the reduction was estimated to be 18.9 percentage points on a mean of 36.6%, or a 52% reduction (95% CI 6.6–68.3). The 2SLS results provide a nearly identical estimate, though more precisely estimated, significant at the 1% level (95% CI 6.7–29.9).

Table 5 presents results of estimates analogous to those in Table 4, but that employed diarrhea duration as the outcome of interest. That is, for the subsample of children with diarrhea in the past two weeks, what was the duration of the illness (in days). The point estimates were consistently negative, though only the 2SLS estimates were statistically significant. The estimate in column three indicates that children eating OSP had diarrhea duration that was reduced by 13% (0.6 days on a mean of 4.7), significant at the 10% level. The effect was larger and more precise for the younger (and consistently aged) sub-sample. For these children, diarrhea duration was reduced by 1.3 days (or 27%), significant at the 1% level (95% CI 0.4–2.2 days).[11]

(a) *Heterogeneous results*

Table 6 presents heterogeneous results by gender of child, education of guardian, and recent VAS status, employing interaction terms. Column 1 shows a 16.6 percentage point

Table 4. *Diarrhea prevalence*

	Full Sample			Under 36 mos. sample		
	LPM (1)	LPM (2)	IV (3)	LPM (4)	LPM (5)	IV (6)
OSP consumption			−.159***			−.183***
			(.051)			(.059)
Treatment × Post	−.116**	−.114**		−.213***	−.189**	
	(.049)	(.048)		(.075)	(.078)	
Treatment	.014	.017		.009	.012	
	(.037)	(.036)		(.038)	(.038)	
Post	−.085**	.012		.085	.075	
	(.034)	(.041)		(.061)	(.066)	
Male		.048*	.041*		.067*	.053*
		(.024)	(.024)		(.033)	(.032)
Age		−.005***	−.005***		−.007***	−.007***
		(.001)	(.001)		(.002)	(.002)
Wealth quintile		−.018	−.021		−.020	−.024
		(.019)	(.018)		(.021)	(.021)
House area		−.002	−.001		−.002	−.002
		(.003)	(.003)		(.003)	(.004)
Head education		.008	.009		.007	.008
		(.006)	(.006)		(.008)	(.008)
Mother education		−.008	−.006		−.011	−.007
		(.009)	(.009)		(.011)	(.011)
Constant	.368***	.525***	.543***	.355***	.557***	.594***
	(.022)	(.046)	(.044)	(.027)	(.073)	(.069)
Observations	1321	1304	1264	817	800	768
R^2	.043	.078	.053	.023	.046	.025
Mean DV	.291	.291	.289	.364	.366	.365

Note: The dependent variable is a binary indicator for whether the guardian reported that the child had experienced any diarrhea in the two weeks prior to the interview. "OSP consumption" is a binary indicator for whether OSP was mentioned in a 7-day food frequency recall by the guardian (see text). Estimations include stratum fixed effects. Standard errors are clustered at the village level and presented in parentheses.
*Statistical significance at the 10% level.
**Statistical significance at the 5% level.
***Statistical significance at the 1% level.

effect for boy children. While we cannot reject that the effect is the same for girls (p-value = .274), the linear combination indicates that the point estimate for girl children is negative, though not precisely estimated. We thus explored potential pathways for any differential effect. We first considered whether there was differential feeding of OSP by gender. A t-test reveals that OSP consumption in 2009 was slightly higher for boys, but did not significantly differ from girls, either within the treatment group (p-value = 0.12) or overall (p-value = 0.16). [12] We next considered a biological pathway, whereby the same increase in vitamin A intake may have had differential effects by gender. We did find that baseline diarrhea was significantly higher among boys (43%) than girls (35%; p-value = 0.06). This suggests that impacts may have been greater for boys due to higher initial morbidity.

We also disaggregated the results both by education of the mother, and by education of the household head. [13] Across the 519 households in the sample, 27% of household heads had completed the first level of primary school (grade 5) or higher. Completed education was considerably lower for mothers. Only 54% of children had mothers reporting any completed schooling, and only 7% had completed grade five or higher. [14] Columns 2 and 3 of Table 6 show that for both mothers and heads of household, having higher education generated a higher point estimate of treatment impact.

In particular, for mothers, we found that for children of women who had completed the first level of primary school, there was a nearly 100% reduction in diarrhea resulting from the program (30 percentage points, significant at the 5% level; column 2). Children of women with less education also benefited, experiencing diarrhea reductions of 10 percentage points, which was also significant at the 5% level (linear combination). We were able to reject that these effects were the same at the 10% level of significance, based on the interaction term.

Diaggregating household heads into the same education groups, we could not reject that the effects are the same for both education groups based on the interaction term (column 3). However, children in households in which the household head had not completed primary school exhibited an effect of only six percentage points, and the coefficient estimate was not significantly different from zero (linear combination).

Finally, column 4 of Table 6 explores the differential impact of treatment for children who have received VAS within 6 months prior to the interview. We could not reject that the beneficial effect is the same for children regardless of VAS status (interaction term is not significant). However, the point estimates do suggest that the effects are less for children who have recently received VAS (linear combination is small and not significantly different from zero). We used the same set of coefficients to compare the correlation between recent VAS and diarrhea across children who are exposed to OSP and those who are not. Linear combinations suggested that for children without access to OSP, recent VAS was associated with a 14.4 percentage point reduction in diarrhea (p-value 0.22), while for those with access to OSP, the association was an increase of 0.5 percentage points. [15] While this was nonexperimental, it similarly suggested that children benefited less from either OSP or VAS when they were already receiving the other. Though in both cases we could not reject that the effects were the same regardless of other treatment.

Table 5. *Diarrhea duration*

	Full sample			Under 36 mos. sample		
	OLS (1)	OLS (2)	IV (3)	OLS (4)	OLS (5)	IV (6)
OSP consumption			−.612*			−1.319***
			(.371)			(.472)
Treatment × Post	−.332	−.384		−.771	−.842	
	(.655)	(.663)		(.650)	(.678)	
Treatment	−.307	−.208		−.194	−.081	
	(.535)	(.525)		(.553)	(.534)	
Post	−.365	−.022		−.084	.020	
	(.553)	(.491)		(.546)	(.530)	
Male		.512*	.456*		.510	.535*
		(.276)	(.276)		(.315)	(.321)
Age		−.020**	−.023**		−.057**	−.061**
		(.008)	(.009)		(.025)	(.025)
Wealth quintile		−.453	−.474*		−.357	−.426
		(.272)	(.255)		(.304)	(.291)
House area		.057	.061		.047	.051
		(.043)	(.040)		(.049)	(.046)
Head education		−.039	−.036		−.067	−.078
		(.057)	(.050)		(.063)	(.058)
Mother education		−.067	−.079		−.056	−.070
		(.085)	(.086)		(.101)	(.105)
Constant	4.912***	5.236***	5.257***	4.781***	5.849***	6.162***
	(.404)	(.440)	(.452)	(.400)	(.646)	(.536)
Observations	382	378	363	295	291	278
R^2	.025	.060	.070	.031	.078	.090
Mean DV	4.730	4.741	4.705	4.861	4.876	4.881

Note: The dependent variable is the duration (in days) of the diarrhea episode, as reported by the parent; the samples include only those reporting an episode in the two weeks prior to the interview. "OSP consumption" is a binary indicator for whether OSP was mentioned in a 7-day food frequency recall by the guardian (see text). Estimations include stratum fixed effects. Standard errors are clustered at the village level and presented in parentheses.
* Statistical significance at the 10% level.
** Statistical significance at the 5% level.
*** Statistical significance at the 1% level.

4. DISCUSSION

This work builds on existing studies of behavioral interventions for increasing children's micronutrient intake (for a partial review, see Gibson & Anderson, 2009). For example, Nana, Brouwer, Zagré, Kok, and Traoré (2006) found that promotion of mango and liver consumption increased VA intakes and serum retinol levels among children in Burkina Faso and Low *et al.* (2007) found that promoting orange sweet potatoes had similar effects in Mozambique. This work moves beyond the traditionally examined outcomes of intake, nutritional status, and growth to examine whether diet-based interventions have the potential to improve child health as measured by morbidity.

The intervention we evaluated was the promotion of biofortified orange sweet potatoes and we measured its impact on child diarrhea outcomes. We found that the HarvestPlus REU intervention reduced diarrhea prevalence and duration for children in treated villages. We did not find that the intervention affected other forms of child illness. However, the changes in diarrhea suggest that agriculture-based programs aimed at increasing micronutrient intake via diet change can significantly impact child health.

In earlier analyses of this intervention, de Brauw *et al.* (2013) employ causal mediation analysis to determine the primary pathways by which the REU achieved increases in adoption and estimated increases in vitamin A consumption. They found that vine distribution had the largest impact on OSP adoption and vitamin A intakes, and that the behavioral component of the intervention had very minimal effects on vitamin A intakes. These findings provide encouraging evidence that a program promoting micronutrient-dense alternatives to traditional varieties can be successful by employing broad messages of the new variety's healthful attributes, repeated provision of starter supplies, and agricultural extension to sustain the crop. However, consumer acceptance of biofortified crops may also be a necessary condition for a program's success. In this case, the program was accompanied by consumer acceptance research, demonstrating that people liked OSP at least as well as traditional white varieties (Chowdhury, Meenakshi, Tomlins, & Owori, 2011).

We found that the intervention had greater morbidity impacts for children with more educated mothers. It may be that mothers with higher education were better able to understand the potential health benefits of OSP, and also to change children's diets. Children in households where the head had more education also exhibited stronger impacts, though not significantly so. To the extent that men play a large role in growing OSP, this result may reflect a lower ability of less educated household heads to adopt and sustain OSP vines. Such heterogeneities suggest a complementary effect of education on the success of biofortification for health. The fact that this intervention exhibited some success in a population with such low levels of education implies it could be even more effective in populations where a majority have completed low levels of primary education or better, holding constant other factors such as preferences for orange and white sweet potatoes.

We also explored the interaction between exposure to OSP and recent supplementation of vitamin A. Both the effect of

Table 6. *Diarrhea prevalence: Heterogeneous results (full sample)*

	Testing for heterogeneity by...			
	Child gender (1)	Mother educ (2)	HH head educ (3)	Recent VAS (4)
Treatment × Post	−0.166**	−0.301***	−0.281**	−0.183**
	(0.079)	(0.105)	(0.126)	(0.072)
Treatment × Post × Girl	0.104			
	(0.093)			
Treatment × Post × Low Educ		0.200*	0.216	
		(0.104)	(0.154)	
Treatment × Post × VAS				
Treatment	0.031	0.272***	0.201*	0.051
	(0.057)	(0.099)	(0.106)	(0.051)
Post	0.022	0.185**	0.126	0.078
	(0.071)	(0.082)	(0.113)	(0.061)
Treatment × Girl	−0.027			
	(0.078)			
Post × Girl	−0.020			
	(0.079)			
Treatment × Low Educ		−0.274**	−0.238*	
		(0.106)	(0.126)	
Post × Low Educ		−0.184**	−0.139	
		(0.078)	(0.136)	
Treatment × VAS				
After × VAS				
Linear combination	−.0623	−.101**	−.0647	−.0338
	(.0514)	(.0486)	(.0618)	(.0901)
Observations	1304	1304	1304	1270
R^2	.08	.08	.082	.082

Note: The estimates shown are variations on model (2) from Table 4, employing the same dependent variable. "Girl" indicates that the child is a girl; "low educ" indicates that the guardian shown in column header did not complete grade 5; "VAS" indicates that the parent reported that the child received vitamin A supplementation no more than 6 months prior to the data collection regarding diarrhea. Linear combination is the sum of "treatment × post" and the relevant triple interaction. Estimations also include individual controls for Girl, Low Educ and VAS, as well as stratum fixed effects and the same controls shown in Table 4. Standard errors are clustered at the village level and presented in parentheses.
* Statistical significance at the 10% level.
** Statistical significance at the 5% level.
*** Statistical significance at the 1% level.

OSP and the association of VAS with diarrhea were greater for children not receiving the alternative (or complementary) treatment. However, in both cases the differences were not statistically significant. These strategies may be alternatives within-child, as each intervention has the ability to reduce VAD significantly. However, from a public health perspective they are complementary interventions, as neither has the capability of reaching every child with VAD. Indeed, children in more densely populated areas are easier to reach with VAS, while subsistence farming households which are more remote on average are the targets of biofortification, making them complementary strategies in public health policy.

A final caveat is that the severe prevalence of VAD at the baseline in Mozambique may have contributed to the significant impacts on morbidity in Mozambique. In the main intervention report, no impacts on diarrhea prevalence or severity were found in Uganda, where vitamin A deficiency was much lower and vitamin A capsule distribution was reasonably widespread (de Brauw *et al.*, 2010). In the Ugandan context, Meenakshi *et al.* (2010) estimated that biofortification and dissemination of sweet potatoes cost $9 to $30 per disability-life-year (DALY) gained. The cost per DALY would likely be lower in high VAD contexts such as Mozambique, due to the higher potential for impact. In comparison to biofortification, the estimated cost effectiveness of VAS is $73 per DALY gained (estimated in Zambia; Fiedler *et al.*, 2012). Given the lower cost and potential for spillovers of agriculture-based diet change across both geographical space and generations, diet change through biofortified crops seems to be a viable complement to current supplementation strategies and a sustainable solution in the longer term for increasing micronutrient intake and improving child health.

DISCLOSURE

The authors declare that they have no conflicts of interest in this work. The primary data collection and the REU program were conducted in collaboration with HarvestPlus. The European Commission funded researcher time used to complete this paper and had no role in study design, in the collection, analysis, or interpretation of data, in the writing of the report, or in the decision to submit the paper for publication. The REU project was approved as human subjects research by the Mozambique Ministry of Health's National Bioethics Committee for Health in 2006, with further approval in

2009. Kelly Jones was primarily responsible for the analysis, writing, revision, and preparation of the article for submission. Alan de Brauw was a principal investigator of the REU project and contributed to the analysis, writing, and revision of this article. Both authors have approved the final version of this article.

NOTES

1. This meta-analysis did not include the DEVTA trial in India, the largest randomized controlled trial ever conducted regarding VAS (Awasthi *et al.*, 2013). DEVTA did not find impacts on mortality, however, serious concerns have been raised regarding both the implementation of VAS in the DEVTA study and its analysis (Habicht & Victora, 2013; Sloan & Mitra, 2013; Sommer, West, & Martorell, 2013). Regardless, Mayo-Wilson *et al.* (2011) note that an update of their meta-analysis to include the DEVTA trial still estimates a significant reduction in all-cause mortality of 11%.

2. This literature is summarized by both Akachi and Canning (2010) and Aksan and Chakraborty (2013).

3. Vines were distributed annually to the treatment groups. In the first year, households received two kilograms of OSP for free, with the ability to purchase up to eight additional kilograms. In the second year, households only received vines if they could not maintain them; in the third year, households received up to six kilograms for free, with three kilograms going to the household head and three kilograms to the spouse.

4. Further detail regarding site selection and program implementation are provided by Hotz, Loechl, de Brauw *et al.* (2012).

5. There were in fact two treatment arms, each representing 1/3 of villages. One treatment was intended to be more intensive in terms of supervision and support of village-level promoters in the second year of the intervention. In practice, almost no difference was observed between the two treatment groups in terms of outcomes, including the outcomes used in this paper, and as such they are considered here as a single treatment group.

6. There are also an additional 85 children under the age three observed in 2006 who are lost to follow up in 2009, and children aged zero to five in 133 new households added in 2009.

7. Among reference children who were 12–35 months old at the baseline and were measured at follow up, the impact on the prevalence of inadequate intakes was a more modest 17 percentage points.

8. We find no significant impact of the intervention on respiratory illness or fever. Significant impacts were found for "other" illness, however the imbalance in this indicator at baseline calls the finding into question. The results presented here are restricted to diarrhea prevalence and duration.

9. While most households, and indeed some children, appear in both rounds of data, our estimates do not include household- or child-fixed effects. The outcome of interest represents household- or child-level health status only at an arbitrary, limited point in time, and therefore it is a very noisy indicator of diarrheal frequency for an individual household or child. A within-household or within-child estimate would be a poor representation of the change over time in household or child health status. An appropriate indicator for that type of analysis would be all diarrheal occurrences in the past year for the individual, which is not available and in fact would be exceptionally difficult and expensive to accurately collect. In contrast, the two-week measure is a clear indicator of village- or strata-level diarrhea prevalence, allowing estimates of changes in prevalence over time.

10. Marginal effects at the mean estimated with a Probit model produce a similar effect of 12.0 percentage points, significant at the 1% level.

11. Estimating Poisson models for this outcome, the marginal effects at the mean are comparable to coefficients from the OLS model.

12. Approximately 1/3 of households had data for more than one child in 2009. Relying on these we compared OSP consumption by gender across children within the same household, using household fixed effects. Again we found slightly higher consumption for boys (two percentage points on a mean of 60%) but failed to reject that consumption was the same across genders

13. In only 7% of cases was the mother the head of household.

14. These indicators are not collinear; 37% of children resided in households where either the head had completed some schooling and his/her mother had not (34%) or vice versa (3%).

15. Linear combination for VAS without OSP is "VAS + After*VAS + Treatment*VAS;" for VAS with OSP it combines these terms with the additional "Treatment*After*VAS."

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APPENDIX A

Table 7. *First Stage regressions from 2SLS estimations*

	Prevalence sample		Duration sample	
	All (1)	Under 36 months (2)	All (3)	Under 36 months (4)
Treatment × Post	.467***	.525***	.461***	.428***
	(.058)	(.071)	(.081)	(.136)
Treatment	.010	.009	.004	−.002
	(.020)	(.020)	(.029)	(.031)
Post	.187***	.132**	.239***	.242*
	(.056)	(.064)	(.088)	(.125)
Male	.022	−.000	−.015	.011
	(.023)	(.022)	(.029)	(.027)
Age	.002***	.003**	.002	−.001
	(.001)	(.001)	(.001)	(.003)
Wealth quintile	−.018	−.010	−.044	−.015
	(.017)	(.020)	(.032)	(.032)
House area	.000	−.002	.000	−.001
	(.002)	(.002)	(.004)	(.004)

(*continued on next page*)

Table 7 (*continued*)

	Prevalence sample		Duration sample	
	All (1)	Under 36 months (2)	All (3)	Under 36 months (4)
Head education	.004	.002	−.008	−.006
	(.005)	(.005)	(.007)	(.008)
Mother education	.005	.007	.003	−.006
	(.008)	(.008)	(.011)	(.013)
Constant	.042	.049	.129**	.132*
	(.035)	(.034)	(.049)	(.069)
Observations	1264	768	363	278
R^2	.430	.450	.494	.430
Mean DV	.376	.233	.295	.198

Note: The dependent variable is a binary indicator for whether the guardian reported that the child ate OSP in the week prior to the interview. Estimations include stratum fixed effects. Standard errors are clustered at the village level and presented in parentheses.
* Statistical significance at the 10% level.
** Statistical significance at the 5% level.
*** Statistical significance at the 1% level.