Original Article

Determinants of adolescent shortness in Tanjungsari, West Java, Indonesia

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Background and Objectives: Health status during adolescence may predetermine that during adulthood. Being short because of nutritional and health adversity, where stunting is indicative, is a global health concern, possibly in adolescence. This study assessed the prevalence of shortness (defined by HAZ <–2 SD) at age 12 and its determinants. **Study Design:** This Tanjungsari birth cohort of 1988/1989 was revisited in 2001–2002 with 3093 participating children, their parents and households. The cohort was tracked from birth, to ages 2 and 12 with anthropometry, with birth weight, then weight and height-for-age at 2 and 12, dietary history at age 2, health patterns at age 2 and 12, and environmental exposures. **Results:** The prevalence of adolescent shortness, presumed 'stunting', was 48.8% for which predictor Odds Ratios (OR) were low birth weight 1.64 (95% CI: 1.28-2.09), short height for age at 2-years 1.54 (95% CI: 1.33-1.80), limited maternal education 1.19 (95% CI: 1.01-1.41), unimproved source of drinking water 1.27 (95% CI: 1.08-1.49), unimproved latrine 1.18 (95% CI: 1.01-1.39) and presence of atopic disease at 12 years of age 1.29 (95% CI: 1.01-1.65). Smoking exposure, not breastfed, formula milk consumption and infectious disease at age 2 were not associated with shortness at age 12 on multivariable analysis. **Conclusions:** Adolescent shortness was found in almost half of this rural Javanese cohort followed from birth. It was associated with birth weight, and several individual, maternal and environmental factors evident at age 2, along with an atopic disposition at age 12. However, stature itself may not constitute a health risk over and above the associated socio-environmental conditions.

Key Words: birth weight, environment, hygiene, maternal literacy, shortness, 12-year-old children, water

INTRODUCTION

'Stunting' is regarded as a global health problem. Its recognition is by way of shortness against measures of birth length or height for age in an apparently healthy population, usually as a z score <–2 SD, but where socio-economic, health and nutritional adversity make growth faltering likely, and future health prospects diminished. Without knowledge of these exposures, a diagnosis of 'stunting' rather than 'healthy shortness' is presumptive, based on likelihood. Thus, assertions about 'stunting' are constrained in a way like that which applies to the diagnostic process and its implications for management.[1]

From a public health viewpoint, it may be acceptable to risk erroneous classification as 'stunting' for the few in the interest of the many, as long as there is not an underlying cause and related solutions which are missed and neglected. In communities where growth faltering is common, and shortness regarded as the norm, 'stunting' with its adverse health associations is generally an accurate descriptor of the situation for those with very short birth length or stature during infancy.[2] Here, interventions directed towards the underlying causes may be understood and useful. However, what the targets should be as age progresses in survivors, when the role and relevance of the earlier contributors changes and adaptation might have taken place, is uncertain. Whatever its determinants, height itself is less likely to be of pathogenetic importance for health outcomes the older one is. The emphasis on the first 1000 days of life from conception by nutritionists is consistent with this insight.[2]

The world prevalence estimate of severe shortness (probably stunting) among children younger than five

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years was 26% in 2011, and the highest prevalence estimates among UN sub-regions were 42% (in Eastern Africa) and 36% (in Western Africa and South-central Asia).[3] In a study conducted in Dhaka, Bangladesh, 39.5% of the preschool children in that city were stunted.[4] In Iran, mother's education level was associated with socioeconomic inequality among stunted children.[5] Also in Bangladesh, father's education level,[4] mother's knowledge about nutrition,[4] and wealth index were all factors that determined stunting in children.[6] Under five, stunted children in Ethiopia had limited food diversity and meal frequency.[7] In Brazil, tap water availability, access to toilets, mother's education, and birth weight were associated with stunting.[8] Stunted children have increased morbidity and mortality.[2] Yet, population-based studies, let alone cohort studies in Indonesia are rare.[9] Shrimpton et al have made a case that the first three years of life is the window for stunting prevention.[10] Stunting between birth and three years of age is not reversible.[10] However, longitudinal studies in potentially vulnerable populations are needed to see whether stunted children can achieve catch-up in growth beyond 3 years and minimise the longer term health implications of growth faltering through to adolescence.

The Tanjungsari Cohort Study is a mother-child dyad longitudinal study that follows a 1988/1989 cohort of pregnant women and the growth of their children.[11-13] It sought to document the prevalence, continuance or recoverability of stunting, elaborate its determinants and detect adverse health consequences. This might inform and improve health programs for mothers and children at risk.

MATERIALS AND METHODS

Study design

A population-based cohort was studied in Tanjungsari sub-district, West Java province, Indonesia, and comprised 107,000 people living in 27 villages in 1988/1989.[13] Data collection commenced at birth and was repeated at ages 2 and 12 years.

Study participants

There were 3099 participants (singleton only as twins were excluded). Some 6 participants who did not have complete data were excluded. Thus 3093 participants were included.

Data collection

Anthropometric measurements

Body length and height were measured at age 2 and 12 years. At age 2, infant length was measured using an infantometer, which has a fastened headboard with a moveable footboard. Infants were measured lying down, the head was placed against the headboard and the legs were straightened. The footboard was moved until it reached the soles of the feet, so the measurement could be read from the tape attached to the footboard. Body height at 12 years was measured using a Microtoise, a metal metric instrument fixed to the wall. The value was read from the indicator when the instrument was removed from the wall. Children were measured standing straight on a flat surface, their back up against the wall to ensure the required posture. Body weight was measured using spring-loaded scales. Each child wore minimal clothes during measurement, removed his or her shoes, and stood with both feet in the centre of the scale.

Other data collections

Trained village workers, known as cadres, recorded mother's education, source of drinking water, type of latrine, household smoking exposure, and the dietary habits at 2 years old (breastfeeding, formula milk consumption, and dietary diversity). Interviews with respondent's parents during home visits used structured questionnaires in the traditional *Sundanese* language, which was familiar to respondent's families and healthcare workers alike. Visits and interviews coincided with the children's birthdays. Home visits were conducted at the child's home in the presence of their parents. Some children could only be met at school for measurements, and follow-up interviews with their parents occurred shortly thereafter at home. A standardized form guided each interview.

Trained cadres using the same structured questionnaires collected disease history at ages 2 and 12 years for the past year from parents. At age 2 years these included history of convulsions, diarrhoea and acute respiratory infections, while at age 12 years the history included infectious diseases (common cold, acute respiratory tract infections, and diarrhoea) and atopic diseases (urticaria and eczema).

Statistical methods

We categorised birth weight as (1) normal, if ≥2500 grams and (2) low if <2500 grams. The height for age z-score (HAZ) at age 2 and 12 years as was (1) normal, if HAZ is ≥–2 SD (standard deviation) and (2) short, if HAZ is <–2 SD. Mother's education was categorised as (1) six or more years of schooling and (2) illiterate if less than six years. These categories are in accord with government policy for 6-year compulsory education at the time taken. Source of drinking water was classified into two categories: (1) improved, if the source of drinking water was from a bore well, protected well, tap water, or a electric pump on a well (2) unimproved, if the source of drinking water was from *pancuran* (natural tap water), a wellspring, river, or unprotected well. The type of latrine was classified into improved (water seal or pit latrine) and unimproved (open pit, river, pond, gully, or anywhere other than a water seal and pit latrine). We classified smoking exposure as non-passive smoker and passive smoker; non-passive smoker, if the participant was not exposed to any cigarette smoking, and passive smoker, if the participant was exposed to cigarette smoking from at least one smoker who lived in the same house, which could be father, mother or other person. Breastfeeding status at age 2 was categorised as 'still breastfeeding' or 'stop breastfeeding'. We categorised formula milk into 'received formula milk' and 'did not receive formula milk' at age 2 years. A dietary diversity score was obtained when the participant was aged 2 years, reflective of three food types: (1) fruit (2) source mainly of carbohydrate, such as rice or porridge (3) source of protein with carbohydrate, for example, rice or porridge with a source of protein. Disease history at age 2 years

(convulsion, diarrhea, and acute respiratory infection) was classified as 'had the disease' and 'did not have the disease'. For disease history at 12 years old, the diseases/symptoms were classified as infectious diseases (the common cold, acute respiratory tract infections, and diarrhea), and atopic diseases (urticaria or eczema).

Bivariate analysis using simple logistic regression was used to assess the association between any independent variable and the dependent variable (HAZ at age12 years). Significant association was determined if the *p*-value <0.05.

Multivariate analysis was performed using multiple logistic regression. Before running the multiple logistic regression test, we first checked for multicollinearity between independent variables. Multicollinearity test was performed using Spearmen's correlation for all independent variables which had significant association ($p<0.05$) with dependent variable from bivariate analysis. Multicollinearity existed if two or more independent variables were highly correlated. High correlations were determined if coefficient correlation (r) more than or equal to 0.6 ($r\geq0.6$). If for example, there was multicollinearity between two independent variables, we would only choose one of the independent variables to be included in multiple logistic regression model. All statistical analyses were performed using SPSS version 24.0.

Ethics approval

The study was reviewed and approved by the District Health Officer of Sumedang District and the National Unity and Politics Agency, Sumedang District, West Java, Indonesia.

The Tanjungsari Cohort Study has been conducted in accordance with the WMA Declaration of Helsinki – 'Ethical Principles for Medical Research Involving Human Subjects' from 1983 onwards.

RESULTS

Participant Characteristics

Table 1 shows adolescent characteristics for putative predictors of stunting (severe shortness by height-for-age status where Z score (HAZ) <–2 SD) at age 12 years by stature as shortness. In this study, 48.8% of the participants were short at age 12 years . According to sex, 49.2% of the boys and 48.4% of the girls were found to be short at age 12 years. The proportion of short adolescents was more than 50% in adolescents who had illiterate mothers or mothers who had less than 6 schooling years, had exposure to unimproved source of drinking water and unimproved type of latrine, had history of a low birth weight, were short at 2 years old, did not receive formula milk, had infectious disease history at 12 years old, and had an atopic disease history at 12 years old. The majority of participants were passive smoker and among them, the proportion of short adolescent was 49.4%. For diet, almost all participants ate at least 3 types of food at 2 years old and the proportion of short adolescent among them was 48.8%. For disease history at age 2 years, the majority of the participants did not report any convulsions or acute respiratory infections; and the proportions of these who were short were 48.8% and 49.7% respectively. Almost 50% of adolescents who had diarrhea at 2 years old were short at age 12.

Bivariate and multivariate analysis of predictors of stunting at 12 years old

Bivariate analyses (Table 2) show that several putative predictors are associated with adolescent shortness. These include factors which must have been operative during intrauterine life affecting birth-weight; maternal education; the environment at age 2 insofar as source of drinking water and type of latrine type of latrine, household tobacco usage are concerned; breastfeeding to age 2 and introduction of formula milk; and height-for-age at 2 years old ($p<0.05$). Shortness at age 12 was associated with both an increased risk of infectious disease and atopic disposition.

From the bivariate analyses, independent variables which had $p<0.05$ were tested for multicollinearity. Result from multicollinearity test showed that multicollinearity was existed between infectious disease history at age12 and atopic disease history at the same age ($r\geq0.6$). Hence, we chose to exclude infectious disease from the multivariate modelling. In *multivariate analyses*, birth weight, height for age at 2 years old ($p<0.001$) mother education, source of drinking water, type of latrine, and atopic disease history at 12 years old ($p<0.05$) were predictors of stunting at 12 years old. Mothers who were illiterate and had less than six schooling years, exposure of unimproved source of drinking water, exposure of unimproved latrine, had history of low birth weight, short at 2 years old, and had atopic disease at 12 years old were associated with an increased likelihood of stunting at 12 years old (Table 2).

DISCUSSION

The 12-year tracking of maternal-child dyads in the rural Tanjungsari Sub district of West Java Province in Indonesia reveals that a constellation of intrauterine, maternal educational, environmental and interval growth performance factors are associated with severe shortness or stunting in early adolescence at age 12. Adolescence, defined by WHO as the period between age 10 and 19, is the stage of life which is peri-pubertal, psychosociobiologically transitional and vulnerable. While it is unclear how much shortness itself might add to this vulnerability, some of its determinants, which are recognised in the present cohort study, are likely to add to the risk.

Predictors of adolescent shortness

Gender is not a predictor of stunting at adolescent age 12 in the present study, but its biological significance will differ by gender given the age differences between menarche in girls and puberty in boys. By contrast, in Ethiopia, male adolescents are 2.4 times more likely to be stunted compared with female adolescents.[14] Yet again, in Bangladesh, the prevalence of stunting is greater in females than males.[15] Thus, the risk factors for shortness may be unique by *setting and ethnicity*.

Stunting in adolescence presumably reflects the cumulative effects of poor nutrition, infection and environmental factors operative from the fetal period through to young adulthood. Special attention is due to *adolescent*

Table 1. Participant characteristics by stature

Predictor	Normal at age12 years n (%)	Short at age 12 years n (%)	Total n (%)
n	1584 (51.2)	1509 (48.8)	3093 (100)
Basic demography			
Sex			
Girls	793 (51.6)	744 (48.4)	3093 (100)
Boys	791 (50.8)	765 (49.2)	
Mother's education			
≥6 schooling years	1148 (53.0)	1017 (47.0)	3091 (99.93)
Illiterate or <6 schooling years	435 (47.0)	491 (53.0)	
Environmental exposures			
Source of drinking water			
Improved	783 (56.3)	609 (43.8)	3092 (99.96)
Unimproved	800 (47.1)	900 (52.9)	
Type of latrine			
Improved	824 (55.1)	671 (44.9)	3092 (99.96)
Unimproved	759 (47.5)	838 (52.5)	
Smoking exposure			
Non-passive smoker	139 (58.2)	100 (41.8)	3093 (100)
Passive smoker	1445 (50.6)	1409 (49.4)	
Growth history			
Birth weight			
Normal Birth Weight	1339 (52.4)	1217 (47.6)	2866 (92.7)
Low Birth Weight	126 (40.6)	184 (59.4)	
Height for age at age 2 years			
Normal	1016 (55.6)	810 (44.4)	3093 (100)
Short†	568 (44.8)	699 (55.2)	
Dietary history			
Breastfeeding status at age 2			
Still breastfed	1339 (50.5)	1313 (49.5)	3089 (99.87)
Stopped breastfeeding	243 (55.6)	194 (44.4)	
Formula milk consumption at age 2			
Received formula milk	757 (53.9)	647 (46.1)	3088 (99.84)
Not received formula milk	824 (48.9)	860 (51.1)	
Dietary diversity at 2 years old			
Eat 3 types of food group	1574 (51.2)	1500 (48.8)	3089 (99.87)
Eat <3 types of food group	8 (53.3)	7 (46.7)	
Disease history			
Convulsion history at age 2 years			
No	1560 (51.2)	1489 (48.8)	3089 (99.87)
Yes	22 (55.0)	18 (45.0)	
Diarrhoeal history at age 2 years			
No	847 (52.2)	775 (47.8)	3088 (99.84)
Yes	735 (50.1)	731 (49.9)	
Acute Respiratory Infection history at age 2 years			
No	311 (50.3)	307 (49.7)	2980 (96.3)
Yes	1210 (51.2)	1152 (48.8)	
Infectious disease history at age12 years			
Infectious disease history at age 12 years			
No	1395 (51.9)	1291 (48.1)	3070 (99.3)
Yes	176 (45.8)	208 (54.2)	
Atopic disease history at age 12 years			
No	1436 (52.0)	1326 (48.0)	3086 (99.77)
Yes	145 (44.8)	179 (55.2)	

†Shortness defined as height for age z score (HAZ) <–2 SD.

girls who are stunted because of its possible adverse consequences in the event of pregnancy, where intergenerational nutritional disorders may be accentuated.[16,17]

Maternal literacy was a determinant of adolescent stunting in Tanjungsari such that mothers who were illiterate or had less than six schooling years were more likely to have stunted children (OR=1.19; $p<0.05$). This finding supports previous observations in sub-Saharan Africa and Iran.[5,18]

Low birth weight children were 1.6 times more likely to be stunted at 12 years old than those with normal birth weights: on multivariate analysis OR=1.64; 95% CI=1.28–2.09; $p<0.001$. This is consistent with others Indonesian studies that found LBW to be related to stunting among children aged 12–23 month and a dominant risk factor for stunting.[15] Another study in Tanjungsari also found that birth weight was a risk factor for stunting in children under 5 years.[19] Stunting may be prevented if pregnant women are well-nourished and the child is exclusively breastfeed until 6 months and continues to be

Table 2. Bivariate and multivariate analysis of predictors of shortness at 12 years old

Predictor	OR (95% CI)	
	Bivariate	Multivariate
Basic demography		
Sex		
Boys	1.03 (0.89–1.19)	
Girls	1	
Mother's education		
Illiterate and <6 schooling years	1.27 (1.09–1.49)*	1.19 (1.01 – 1.41)*
≥6 schooling years	1	1
Environmental exposures		
Source of drinking water		
Unimproved	1.45 (1.25 – 1.67)**	1.27 (1.08 – 1.49)*
Improved	1	1
Type of latrine		
Unimproved	1.36 (1.18 – 1.56)**	1.18 (1.01 – 1.39)*
Improved	1	1
Smoking exposure		
Passive smoker	1.36 (1.04–1.78)*	1.26 (0.94 – 1.67)
Non-passive smoker	1	1
Growth history		
Birth weight		
Low birth weight	1.61 (1.26–2.04)**	1.64 (1.28 – 2.09)**
Normal birth weight	1	1
Height for age at age 2 years		
Short†	1.54 (1.34–1.78)**	1.54 (1.33 – 1.80)**
Normal	1	1
Dietary history		
Breastfeeding status at age 2 years		
Stopped breastfeeding	0.81 (0.66–0.99)*	0.92 (0.73 – 1.14)
Still breastfed	1	1
Formula milk consumption at age 2 years		
Not Received formula milk	1.22 (1.06–1.41)*	1.12 (0.92 – 1.31)
Received formula milk	1	1
Dietary diversity at age 2 years		
Eat 3 types of food group	0.92 (0.33–2.54)	
Eat <3 types of food group	1	
Disease history		
Convulsion history at age 2 years		
Yes	0.86 (0.46–1.61)	
No	1	
Diarrhoeal history at age 2 years		
Yes	1.09 (0.94–1.25)	
No	1	
Acute respiratory infection history at age 2 years		
Yes	0.97 (0.81–1.15)	
No	1	
Infectious disease history at age 12 years		
Yes	1.28 (1.03–1.58)*	
No	1	
Atopic disease history at age 12 years		
Yes	1.34 (1.06–1.69)*	1.29 (1.01 – 1.65)*
No	1	1

Bivariate analysis refers to the inclusion of all predictors of shortness at age 12.
Multivariate analysis refers to models in which the predictors had $p<0.05$ from bivariate analysis and $r<0.6$ from multicollinearity test.
†Shortness defined as height for age z score (HAZ) <–2 SD.
*p-value is significant if <0.05.
**p-value is significant if <0.001.

breastfeed until 2 years old. On the other hand, growth failure, which often begins intrauterine and continues after birth, may contribute to suboptimal breastfeeding. If breastfeeding is stopped before 2 years, complementary feeding may be inadequate or inappropriate and infections more likely. In the present study, the child in whom breastfeeding stops before 2 years of age was less likely to be stunted at 12 years on univariate, but not multivariate analysis. This unexpected finding may reflect complex behavioural responses to this situation by way of complementary feeding. The introduction of formula milk, one of several possible responses where breast feeding has stopped at 2 years, was a practice intended to decrease the risk of subsequent growth faltering, but is susceptible to misuse; in any event, the prevalence of shortness at 12 years was not different.

The need and barriers to optimisation of early life feeding practices underscore the importance of skilful, evidence-based approaches to management of the first 1000 days after conception.[20]

Stunting at age 2 years is a risk factor for stunting in adolescence. Previous work indicates that the first 2 years after birth is a window for growth optimisation.[21] Those children stunted at 2 years of age are more likely remain stunted and not recover. The prevalence of stunting in Indonesia, according to Basic Health Research information in 2013 was 37.2%, compared to a prevalence of 35.6% in 2010 and 36.8% in 2007. In 2015, based on countries rank from the lowest to the highest stunting prevalence, Indonesia rank is 108 from 132 countries. Prevalence stunting in Indonesia in 2015 was 36.4.[22]

Environmental Factors

The *source of drinking water* was associated with stunting. Adolescents from households that used unimproved sources of drinking water were almost 1.27 more likely to be stunted than those from households with improved drinking water sources. Similarly, in Ethiopia, an unsafe water supply sources were almost 4 times more likely to be associated with stunting.[13] Interestingly, another study conducted in Tanjungsari has revealed that access to safe drinking water may reduce the risk of stunting among children under 2 years old.[19] The source of drinking water is an underlying determinant of stunting. Its improvement contributes to stunting reduction, but over time.[23] The reasons for the association with water supply maybe be as simple as hygiene, due to complex chemical and waste contamination, or convenience.

The child who was a *passive or second-hand smoker* had an increased risk of adolescent stunting. Here the child who was a passive smoker had father, mother and or others family member in the same house who was an active smoker.[24] Bivariate analysis demonstrated that children who were passive smokers at age 2 had an increased risk of stunting in adolescence. This result is similar with Previous observations in Indonesia have shown that paternal smoking is associated with severe malnutrition.[25]

Bivariate analysis (Table 2) showed that *breastfeeding status and formula milk consumption* at age 2 were associated with stunting at 12 years ($p<0.05$). However, multivariate analysis did not confirm this association.

Dietary diversity, a well-recognised indicator of household food security, did not show any significant association with stunting at 12 years.[26,27] In Ethiopia, stunting is associated with food diversity and the number of meals children eat.[7] The different findings may represent differences in the local food systems, food culture, or eating patterns. The Ethiopian participants were under-five children,[7] while our study investigated the association between dietary history at 2 years and the occurrence of stunting at age 12 years old.

The Tanjungsari study identified atopic disease as an association of adolescent stunting, which might of causal, consequential or bidirectional interest. Previous studies have reported that short stature is found more frequently in children with asthmatic or non-asthmatic allergic respiratory diseases, and in children with atopic eczema.[28,29] If the short stature is due to the severe disease and not due to steroid therapy, catch-up growth might be expected.[29,30] However, in the present study, children with stunting at age of 2 were more likely to remain stunted in adolescent.

Outcomes of adolescent shortness

Government commitment and policy, which is informed, understands and prioritises the nature and need to reduce the risk for severe shortness or stunting can be of immense community and national value. But it is not necessarily stature itself which is the problem, but the circumstances which determine it. These are more fundamental and critical to solve. In the present 12-year study, maternal and child health, education, and safe, healthful, environments emerge as paramount. It is these contributors to growth and development which, ably addressed, will yield the greatest socioeconomic, econutritional and health gains.

This understanding is not peculiar to Tanjungsari, or even Indonesia as a whole, but regionally and internationally as well.[21] In Ghana, in 2012 priority was given to nutrition intervention for poverty reduction. The 2012 economic loss in Ghana due to stunting was US 2.6 billion, equivalent to Ghana's GDP of 6.4% that year.[17]

Indonesia in 2030 will have a demographic dividend period (or demographic bonus period), because of its expected demography of more people in their productive (age 15–age 64) than non-productive age, and more people looking for work.[31] If the labour market in Indonesia can absorb those in the productive age, per capita production will increase, as will house hold and national wealth. But if labour is compromised by poor health, this opportunity will be diminished or lost and the work force unemployed. This may lead to social and political instability. In Guatemala, people who were stunted as children had a poorer school performance- with consequences for future livelihoods. The World Bank estimates that stunting is associated with reduced economic productivity.[17]

Healthy shortness

Healthy shortness need be no concern and be an advantage in many geographical settings. This may become more evident with the impending problems of climate change and limited food supplies.

Strategic policy responses

Almost half of the adolescents studied from birth for 12 years in Tanjungsari, West Java, Indonesia were severely short or stunted, at an age where reversal is unlikely. Several factors operative from conception, and with growth and development, are predictors of this situation, themselves potentially more, or as important, than stature itself for long term health outcomes. Of note, birth weight (dependent on intrauterine exposures), maternal education, source of drinking water, type of latrine, height-for-age at age 2 years were predictors of adolescent stunting. Infectious disease and atopic dispositions were more likely in stunted adolescents. These findings should inform strategies aimed to prevent and management factors which might operate early in life, lead to severe shortness, and adversely affect long-term health. Strategies to focus on catch-up growth, especially at 2 years old, since it is as-

sociated with condition in adult life such as cognitive achievement also merit attention.[32] At the same time, healthy shortness need be of no concern and even be an advantage in many geographical settings, notably with the impending problems of climate change and limited food supplies where smallness may be an advantage.

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AUTHOR DISCLOSURES

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