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A Prospective Study of Perception in Adolescent Smoking

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Abstract

Purpose—This prospective study examined how environmental smoking affects the perception of lifetime smoking prevalence and thereby the likelihood of subsequent regular smoking.

Methods—A longitudinal design (N = 6769) with three waves was used to test our research questions. Exposure to smoking by peers, best friends, and parents were assessed at T1. Perception of lifetime smoking prevalence was calculated at T2. Adolescent smoking was assessed at three waves.

Results—Overestimation of lifetime smoking prevalence was predicted by having a predominantly smoking peer group, having a best friend who smokes, and by having at least one parent who smokes. In consistency with a false consensus effect, smokers were more susceptible to overestimate lifetime smoking prevalence than nonsmokers. Subsequently, while controlling for smoking at T2, overestimating lifetime smoking prevalence was predictive of regular smoking at T3 (in accordance with the conformity hypothesis). Specifically, overestimation of lifetime smoking appeared to mediate the effects of environmental smoking (peers, best friends, and parents) on adolescent smoking. No support was found for a moderation effect of exposure to environmental smoking on the link between misperception of lifetime smoking prevalence and regular smoking.

Conclusions—The study offers a rare and needed theoretical and empirical research examining environmental and individual predictors of regular smoking. Besides direct prevention of exposure to smoking, cognitions that are a product of exposure to smoking need to be addressed in prevention campaigns.

Keywords

Adolescent smoking; Environmental smoking; Perception

Both environmental and individual predictors have been examined in past research to understand adolescents' risk for smoking. Yet, past work rarely has considered models integrating these two sets of predictors. Among environmental predictors, research has suggested that smoking among adolescents' parents [1,2] and peers [3,4] is associated with

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an overwhelming amount of stimuli [8]. However, the same cognitive constructs that provide us with a quick and simple way of dealing with large amounts of information also contribute to cognitive biases that lead to a misperception of reality [8]. Cognitive biases pertaining to smoking likely are developed based on exposure to smoking among others, and may interact with these environmental experiences in producing risk. Yet, this idea rarely has been considered in past work.

The present study focused on a specific cognitive bias identified in past research as relevant for understanding health risk behaviors that holds the idea that people tend to overestimate the degree to which our own behavior is shared by other people (i.e., false consensus). The easiness by which an example comes to mind increases the risk for overestimation. We followed two approaches to study false consensus and adolescent smoking. First, we focused on factors that predict individuals' likelihood to overestimate lifetime smoking, and subsequently we determined whether this bias may affect the likelihood for young adolescents to start regular smoking (i.e., a mediational model, Figure 1a). Second, we examined how exposure to environmental smoking (i.e., smoking by parents and peers) might alter the relation between overestimating lifetime smoking and adolescent regular smoking (i.e., a moderational model, Figure 1b). In doing so, this study offers a rare and needed theoretical and empirical integration of research examining environmental and individual predictors of adolescent regular smoking.

Cognitive biases

Ross et al [9] initiated a line of research demonstrating that individuals who engage in, or are frequently exposed to, a specific behavior are likely to overestimate the prevalence of this same behavior among others [10,11]. Ample studies have found evidence for this so-called false consensus effect among smokers, illustrating that people who smoke are more likely to overestimate the prevalence of smoking, including various age groups such as adolescents [12–14] and for middle-aged daily smokers [15].

It is unclear why individuals overestimate the prevalence of the same behavior they engage in among others. One theory posits that this overestimation may be the result of individuals' selective exposure [14,16,17]. According to this theory, people choose to interact with others who exhibit behavior that is similar to their own behavior. Hence, adolescents who smoke may predominantly select affiliation with others who smoke [18,19], and these selective observations lead to misestimations of smoking prevalence in the population. Other theories suggest that overestimation could be instigated by an "availability heuristic" or an overgeneralization effect [20]. In other words, individuals' who are exposed to a high prevalence of smoking among close others may be more likely to assume that these perceptions are representative of the general public, thus leading to an overestimate of smoking prevalence. Each of these explanations suggests that exposure to others who smoke

likely is a key predictor of adolescents' false consensus bias. Adolescents may be more likely to overestimate the general prevalence of adolescent smoking in accordance with their own behavior or with behavior prominently shown in their environment (i.e., among parents and peers) [8,11]. Unfortunately, these theories rarely have been examined empirically among adolescents.

Given the importance of misperceptions of smoking prevalence and associated health risks in the maintenance and escalation of smoking behavior, it is paramount to better understand why individuals develop such cognitive biases. This issue is relatively underexplored, however, particularly in research involving youth or in research using longitudinal designs. We hypothesized a mediational model, suggesting that exposure to parents and peers who smoke may be associated with the development of cognitive biases, and these biases subsequently are associated with increases in adolescents' smoking behavior. The use of data from three time points offers an excellent opportunity to stringently test this hypothesis.

In addition, it was hypothesized that both exposure to parents'/peers' smoking and cognitive biases might synergistically increase risks for adolescents' smoking. In other words, it was anticipated that person–environment transactions would be associated with greater risk for later smoking than either predictor in isolation. Specifically, it is likely that the link between cognitive biases and smoking behavior is stronger for adolescents who live in an environment where smoking is dominant behavior.

Present study

We used a longitudinal design to examine smoking in a nationwide large sample of Dutch adolescents. Analyses first considered the role of overestimation of lifetime smoking prevalence in adolescent smoking. We expected that exposure to environmental smoking (i.e., among parents and peers) would affect the perception of lifetime smoking prevalence (i.e., any engagement in smoking). Subsequently, we expected that overestimation of lifetime smoking would predict adolescents' future regular smoking [14]. We have chosen for regular smoking while individuals in this stage are most likely to develop a full-fledged smoking habit. Finally, we examined whether the potential associations between cognitive biases and adolescent regular smoking are stronger for adolescents who are exposed to higher levels of environmental smoking (i.e., parental smoking, smoking by peers) than for those who are not, suggesting moderation.

Methods

Participants

A total of 6769 students were included in this study. At T1, all respondents were 11-16 years of age (mean = 12.93 years, SD = .78 years), of whom 51.8% were female. With respect to education level, three categories were constructed; lower education level (38.3%), intermediate or general education level (19.7%), and highest level of secondary education (preparatory college and university education) (42%). Most of the respondents (95.8%) were born in the Netherlands, although 283 respondents (4.2%) were born elsewhere, including Turkey, Morocco, and Surinam.

Data were collected in the three waves as part of a broader longitudinal study on adolescent smoking that was conducted in November 2002 (T1) with the approval of the Central Committee on Research Involving Human Subjects (for more details with respect to the procedure, see [21,22]). We started approaching schools from the total pool of 692 secondary schools in the Netherlands. After having called the first 55 Dutch schools, 33 school boards agreed to participate, providing us with a sample that was large enough for the study purpose. Participation in other studies and lack of time were main reasons to refuse cooperation. Moreover, not in all cases we were able to speak to the right person to decide for participation. All classes of the first (55.8%) and second year (44.2%) of secondary school were selected from each school. Nonresponse was due mainly to absence on the day of assessment; only 15 explicit refusals from adolescents were recorded. The second measurement took place 6-8 months after the first and followed the same procedure, and the third wave took place 12-14 months after the second. Like all longitudinal studies, this study was limited by a dropout in the 22 months between the waves (2239), eventually leading to a sample of 6769 students who participated in all three waves (75.1%). The baseline measurements took place in the first 2 years of secondary school. A few of the participating schools are large organizations with different departments in different villages. Participants that were in school department A during the first measurement could be in department B during the second measurement. Although not all separate departments were approached for participation it was hard to trace and include everybody in these particular schools. A logistic regression analysis was conducted including all variables to test for differences between participants in all three times of assessments and dropouts. Dropouts were older (OR = 1.23, p < .001, CI = 1.15-1.32), they were more likely to have at least one parent who smoked (OR = 1.39, CI = 1.02-1.27, p < .05,) and more likely to be involved in a peer group dominated by smokers (OR = 1.38, CI = 1.22-1.56, p < .001). All variables together only explained 2.6% of the variance in dropout, suggesting that potential selective attrition was rather limited. Students from the same schools and classes are likely to produce common sources of variance (nesting), violating the accuracy of the effects. The intraclass correlation coefficient for lifetime smoking was .036, indicating that 3.6% of the variance could be explained by a school effect. For current smoking this was .031. This is in line with those estimates that were found in prior studies on school effects in adolescent smoking [23]. Analyses in which the ICC was accounted for showed virtually the same results.

Measures

Smoking behavior—Smoking behavior was assessed at all three measurements using a well-established measure [24–26]. The participants were asked to indicate their smoking status on a nine-point scale (e.g., 1 ="I have never smoked"; 9 ="I smoke every day"). Because results suggested a positively skewed distribution (mean > median) with the bulk of the cases falling into the lower part of the range of scores, we decided to dichotomize the smoking variable. Respondents who reported tobacco use once a month or more were defined as "regular smokers" (vs "nonregular smokers"). Dichotomized smoking measures have been used repeatedly in previous studies focusing on adolescent smoking [21,24–26].

Perception—At T2, respondents were asked to estimate the prevalence of lifetime smoking or any engagement with smoking among adolescents in their age group (between

the age of 12 and 14 years in the Netherlands; at the time of measurement of perception of the smoking prevalence (T2), 95% of the respondents were aged between 12 and 14 years old. The mean estimated prevalence of lifetime smoking by adolescents was 43.94 (SD = 20.24). According to an annual monitor in the Netherlands among a large national representative sample of adolescents, the prevalence of lifetime smoking in this age group at the year of smoking assessment for this study was 40.67% [27]. For every respondent we calculated a score implying over/underestimation by subtracting the annual monitor number from the respondents' estimate. In the present study we focused on overestimation of smoking by smokers, which would be indicated by a positive difference between the two measures.

Parental smoking behavior—At T1, respondents were asked: "Does your father/mother smoke? (yes/no)"[28,29]. By combining responses on smoking status of both parents, two levels were constructed (0 = parents do not smoke, 1 = at least one parent is a smoker). In total, 86.5% of the participating adolescents lived with both parents, 1.4% with their father, and 9.7% with their mother, whereas 2.4% of the participating adolescents lived with caregivers other than their parents. Although we used adolescent report to assess parental smoking, previous studies have shown reliable results, indicating that children are quite capable of estimating their parents' smoking behavior [30,31].

Best friend smoking—At T1, each respondent was asked: "Is your best friend a smoker or a nonsmoker," value 0 indicated best friend is a nonsmoker, value 1 indicated best friend was a smoker [32]. Although we were not able to use friends reports for friends smoking behavior, recent studies have shown that adolescents are rather adequate in estimating their best friends' smoking behavior [33].

Peer smoking—At T1, each respondent was asked to estimate the proportion of friends who smoke on a 5-point scale, ranging from 1 "None of my friends smoke," 2 "Less than 50% of my friends smoke," 3 "50% of my friends smoke," 4 "More than 50% of my friends smoke," to 5 "All of my friends smoke." [34]. Our aim was to distinguish between a nonsmoking environment and a predominantly smoking environment, therefore, responses were recoded to a variable differentiating two levels (0 =less than 50% of my friends smoke, and 1 = 50% or more of my friends smoke).

Data analyses

Logistic regression analyses were used 1) to predict overestimation of lifetime smoking prevalence, and 2) to predict adolescent regular smoking over time by overestimation of lifetime smoking prevalence. Gender, age, education level were included as covariates. Because there are strong indications that smokers overestimate the prevalence of smoking, we controlled for adolescent lifetime smoking at time of first measurement. Mediation of overestimation of smoking prevalence was tested using a Sobel test [35]. To test for possible moderation of environmental smoking on the link between overestimation of smoking prevalence and adolescent smoking, we included interaction terms in the final step of the equations [36,37].

Results

Descriptive statistics

At T1, 402 respondents (5.9%) reported regular smoking (once a month). At T2, 510 respondents (7.5%) of the total sample reported regular smoking while this number increased to 1,031 respondents (15.2%) at the third assessment.

A total of 54.3% of the children had two nonsmoking parents, while the remaining reported either having at least one parent who smokes. In total, 10.5% of the adolescents reported to be involved in a peer group in which smoking was dominant behavior (>50%) at T1. Finally, 11.5% reported their best friend was a smoker.

Perception of lifetime smoking prevalence

To determine perception, adolescents had to estimate the prevalence of lifetime smoking on a continuous scale. To be sure that we dealt with misperception and not just with accuracy in estimation (it is unlikely that the prevalence is estimated precisely), we considered perception of smoking prevalence as misperception when estimations were at least 1 SD above the mean prevalence of smoking determined by data of a national representative sample [18]. Analyses with data that also included respondents who estimated the prevalence of lifetime smoking <1SD above the precise prevalence of lifetime smoking and analyses with a weighting factor to account for nonresponse did not lead to significantly different results.

At T2, 55.4% of the regular smokers overestimated the prevalence of lifetime smoking, which is substantially higher than proportion of nonsmokers that overestimated the lifetime smoking prevalence (36.5%; χ^2 (1, N = 5472) = 56.04, *p* < .001), which is in accordance with a false consensus.

Table 1 shows the results of the logistic regression model including environmental smoking predicting overestimation of lifetime smoking (in all analyses we controlled for age, gender, and education level). On the first step of the logistic regression, we included age, gender, education level, and history of smoking as covariates. In the second step, we added proportion of smoking friends, smoking behavior of the best friends, and parental smoking. Respondents with a history of smoking were more likely to overestimate the prevalence of smokers than respondents without a history of smoking (OR = 1.26, CI = 1.09-1.46) and female respondents were more likely to overestimate the prevalence of smoking than male respondents (OR = 1.73, CI = 1.52-1.96). Moreover, respondents in the higher education levels were less likely to overestimate the prevalence of smoking.

Having a predominantly smoking peer group (OR = 1.36, CI = 1.07-1.72), smoking best friends (OR = 1.33, CI = 1.07-1.66), and smoking parents (OR = 1.29, CI = 1.14-1.47) significantly increased the risk for overestimating the prevalence of smoking.

After having determined antecedents of overestimation of lifetime smoking prevalence in terms of exposure to environmental smoking, we tested the predictive value of overestimation assessed at T2 for adolescent smoking at T3 (Table 2), while controlling for

gender, age, education level, and regular smoking at T2. A main effect was found showing that overestimating the prevalence of smoking at T2 was associated with an increased risk to smoke at a regular basis at T3 (OR = 1.43, CI = 1.19–1.72, p < .000) Additional analyses were conducted with lifetime smoking as outcome variable. Although environmental smoking was predictive of overestimation of lifetime smoking prevalence, overestimation was not predictive of lifetime smoking.

The significant links between environmental smoking, overestimation of lifetime smoking prevalence and adolescent smoking allowed us to do some additional mediation and moderation tests. (Note that main effects were also found of exposure to environmental smoking at T1 to adolescent smoking at T3. More specifically, parental smoking (OR =1.62, p < .000, CI = 1.38 - 1.91; best friend smoking (OR = 2.35, CI = 1.91 - 2.90, p < .000); and proportion of smoking friends (OR = 2.93, CI = 2.32 - 3.69, p < .000) at T2 were associated with increased risks to start smoking at T3.) A Sobel test, conducted to test mediation, showed that overestimation of the prevalence of lifetime smoking partially mediated the link between peers' smoking and adolescent regular smoking (estimate = 2.48, p = .01), smoking by best friends and adolescent regular smoking (estimate = 2.53, p = .01), and parental smoking and adolescent regular smoking (estimate = 2.61, p = .01). Hence, part of the effect of environmental smoking on own smoking can be explained by the mediating effect of overestimation of smoking prevalence (i.e., false consensus effect). Moderation was tested by including interaction terms. To reduce capitalisation on chance, all possible interactions between environmental smoking exposure and overestimation were checked by including them separately into the model (e.g., smoking by best friends * overestimation). Environmental smoke exposure did not moderate the link between overestimation and eventually smoking.

Discussion

Reducing the prevalence of smoking is one of the main objectives of health organizations worldwide [38]. Individual factors such as perception and environmental factors (i.e., smoking by parents, peers, and best friends) are strongly linked to adolescent smoking onset. This study provided support for a theoretical model that integrates both environmental and individual factors in predicting adolescent smoking. Moreover, support was found for the idea that exposure to environmental smoking functions as a mechanism underlying overestimation of lifetime smoking (i.e., false consensus effect) [14]. In turn, this overestimation appeared to predict regular smoking.

Overestimation of lifetime smoking

An initial goal of the present study was to examine the extent to which exposure to smoking of parents and peers contributed to the development of overestimation of lifetime smoking. In line with earlier studies we found that those adolescents that were engaged in smoking were more likely to overestimate the number of adolescents engaged in some sort of smoking (lifetime smoking), indicating a false consensus [12–15]. Moreover, we found support for a *conformity hypothesis* that proposes that perception of the commonness of a behavior leads to later adoption of this behavior [14]. The significant links between

environmental smoking exposure and overestimation of lifetime smoking, overestimation of lifetime smoking and regular smoking, and between environmental smoking exposure and regular smoking allowed us to test for mediation: the overestimation of lifetime smoking appeared to mediate the link between environmental smoking exposure and adolescent regular smoking.

Results also showed that adolescents who overestimated the prevalence of lifetime smoking were more likely to smoke regularly later in time (even after controlling for exposure to environmental smoking and engagement in some level of smoking at baseline measurement (lifetime smoking), which is in line with results shown by Botvin et al [39] showing that the expectation of peers' normative behavior predicted later smoking. However, in the Botvin et al study, participants were asked to estimate the proportion of smokers—the greater the estimated proportion, the higher the likelihood for future smoking—whereas we compared the perceived prevalence with the actual prevalence (i.e., overestimation). Moreover, Botvin et al did not test whether this overestimation mediated the link between environmental smoking exposure and future adolescent smoking.

The findings suggest that interventions that focus on norms may be useful to consider in addition to other more conventional interventions for adolescents. In addition, it may be useful to consider educating parents on the effects of their smoking on the perceptions of smoking in their children.

Implications for future research

Future studies should focus on underlying processes such as moderation and mediation that might influence the accuracy of the perception of smoking and other unhealthy behaviors and subsequent development of cognitive biases that may be caused by inaccurate perception of the commonness of the particular behavior. In a recent study by Prinstein and Wang [11], peer rejection and aggression were associated with greater overestimation of deviant and health risk behavior, while being partially mediated by adolescents' own behavior indicating a false consensus effect (the phenomenon that individuals who engage in or are frequently exposed to a specific behavior are likely to overestimate the prevalence of this same behavior among others [10, 11]). These findings emphasize the necessity to include aspects of friendships in future studies. Moreover, behavior shown by important group members may be more salient and may therefore have a larger impact than behavior shown by group members that have a lower social acceptance score. Furthermore, in this study we focused on environmental smoking exposure as a process leading to overestimating of smoking prevalence; however it is likely that other factors such as genes, personality or parenting affect cognitions and the development of cognitive biases in order to increase the likelihood of adolescent smoking over time.

Future research should address some of the limitations of this study. We used adolescent report to assess adolescent smoking and smoking by peers, best friends, and parents. Although studies have shown that adolescent report on parental smoking [33,34] and smoking by a best friend is quite reliable [33]; additional self reports by peers and parents would have been desirable.

A second limitation refers to the point of measurement of each of the study constructs. Having data about environmental smoking at T1, estimation of smoking prevalence at T2 and ultimate regular smoking at T3 allowed us to test mediation. Unfortunately, we did not have data on these constructs at each time of measurement. Therefore, caution is warranted in interpreting the results, while we could not control for prior estimation of smoking prevalence and exposure. For instance, it could be that overestimation of lifetime smoking between T2 and T3 increased. If this is the case, the link between overestimation at T2 and use at T3 may be different.

Because the distribution of regular smoking was skewed and we were interested in those individuals that were about to start regular smoking, we decided to dichotomize the smoking variable. Due to this decision we ended up with a distribution that shows less variance than a variable with a continuous distribution. A similar argument may be given for our choice to dichotomize the estimation of lifetime smoking prevalence. In this study we were interested in distinguishing individuals who overestimated lifetime smoking prevalence from those who did not. Future studies could test whether results are different for individuals who largely overestimate the prevalence.

Finally, overestimation was calculated by subtracting the annual monitor number from the respondents' estimate. Another possibility would be to compare respondents' estimates with school smoking rates. However, our aim was to demonstrate a general misperception independently from local smoking prevalence and focusing on subcultures (determined by school) was beyond the scope of this study. Future studies should concentrate on misperceptions of smoking prevalence while taking into account local smoking prevalence.

Conclusions

Within the context of these limitations, our findings have a number of potentially important implications. The results of this study support the idea that exposure to environmental smoking increases the risk for overestimating the prevalence of lifetime smoking. Subsequently, this overestimation increases the risk for future regular smoking. This study emphasizes the need for research that integrates environmental and individual factors, among which perception and cognitive factors. It is important for studies to consider both types of predictors in an integrative way. Moreover, interventions should address both factors since these factors seem to interact. Besides the direct prevention of exposure to parent/peers smoking, addressing cognitive biases that may be consequences of overestimation or misperception are products of this exposure may be a good way to mitigate the affects of parent/peer influence.

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Figure 1.

(a) Mediation model illustrating that the effect of environmental smoking on adolescent smoking runs/via/perception. (b) Moderation model illustrating that the effect of perception of smoking on adolescent smoking is different for different levels of exposure to environmental smoking.

Table 1

Logistic regression analyses predicting overestimation of lifetime smoking

			Overestimation at T2
T1		OR	95% CI
Covariate			
Age (y)	12.97	1.01	.93–1.10
Gender	Boys	1.00	
	Girls	1.73***	1.52–1.96
Education	Lower education level	1.00	
	Intermediate education level	.53***	4562
	High education level	.30***	.26–.35
Lifetime smoking	No history of smoking	1.00	
	Lifetime smoking	1.26**	1.09–1.46
Predictor variables Smoking friends	Less than 50% smoking	1.00	
	At least 50% smoking	1.36*	1.07–1.72
Best friend smoking	Best friend a nonsmoker	1.00	
	Best friend a smoker	1.33*	1.07–1.66
Parental smoking	No smoking parents	1.00	
	One or two parents who smoke	1.29***	1.14–1.47

CI = confidence interval; OR = odds ratio.

 R^2 for overestimation of lifetime smoking prevalence was .14.

* p <.05

** ¯p < .01

p < .001: two-tailed tests.

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			Overestim	ation of life	time smoking	R	egular smol	king	
			β (SE)	OR	95% CI	β (SE)	OR	95% CI	Mediation estimates
Aain effects	Estimation of lifetime smoking	Accurate perception					1.00		
		Overestimation perception				.36 (.09)	1.43^{***}	1.19–1.72	
Aediation 1									
tep 1 $X - M$ (a)	Smoking friends	Less than 50% smoking		1.00					
		At least 50% smoking	.45 (.11)	1.57^{***}	1.26 - 1.94				
tep 2 M $-Y$ (b + c')	Estimation of lifetime smoking	Accurate perception					1.00		
		Overestimation perception				.30 (.09)	1.35^{**}	1.13-1.61	2.48,
	Smoking friends	Less than 50% smoking					1.00		<i>p</i> =013
		At least 50% smoking				1.07 (.12)	2.90^{***}	2.29–3.68	
fediation 2									
tep 1 $X - M$ (a)	Best friend smoking	Best friend a nonsmoker		1.00				ı	
		Best friend a smoker	.37 (.10)	1.44^{***}	1.19-1.75				
tep 2 M $-Y$ (b + c')	Estimation of lifetime smoking	Accurate perception					1.00		
		Overestimation perception				.33 (.09)	1.38^{**}	1.15 - 1.67	2.53
	Best friend smoking	Best friend a nonsmoker					1.00		<i>p</i> =.011
		Best friend a smoker				84 (.12)	2.32***	1.84–2.94	
fediation 3									
tep 1 $X - M$ (a)	Parents smoking	No parents smoking		1.00					
		One or two parents smoking	.26 (.06)	1.30^{***}	1.15-1.47				
tep 2 M $-Y$ (b + c')	Estimation of lifetime smoking	Accurate perception					1.00		
		Overestimation perception				.32 (.09)	1.37^{**}	1.14–1.65	2.61
	Parents smoking	No parents smoking					1.00		<i>p</i> =.009
		One or two parents smoking				.42 (.09)	1.52^{***}	1.27-1.82	

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Table 2

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