

Forward telescoping bias in reported age of onset: an example from cigarette smoking

ERIC O. JOHNSON,^{1,2} LONNI SCHULTZ³

1 Department of Psychiatry, Henry Ford Health Sciences Center, Detroit, USA

2 Department of Psychiatry and Behavioral Neurosciences, Wayne State University School of Medicine, Detroit, USA

3 Department of Biostatistics and Research Epidemiology, Henry Ford Health Sciences Center, Detroit, USA.

Abstract

Age at the onset of a disorder is one of its key descriptive characteristics. Early onset may indicate increased risk of a severe course and increased genetic liability. However, retrospectively reported onset in surveys is subject to forward telescoping, a bias in which respondents report events closer to the time of interview than is true. We examined the effect of this bias on age of onset for smoking initiation and daily smoking. Data came from the 1966–77 birth cohorts interviewed in the cross-sectional National Household Surveys on Drug Abuse (NHDSA) 1979–98 (N = 82,122). An association between age at onset and age at interview, within birth year, was found for experimenters and for daily smokers. This was indicative of forward telescoping. As age at interview increased from 12 to 25 the probability of reporting early onset dropped by half. An association was also found between early onset of daily smoking and age at interview. This response bias differed significantly by sex and race, created significant misclassification of smokers as late instead of early onset cases, and biased cohort comparisons from cross-sectional data.

These results suggest efforts need to be made to limit the effect of forward telescoping in epidemiological studies by survey question and sampling design. Copyright © 2005 John Wiley & Sons, Ltd.

Key words: forward telescoping, age at onset, cigarette smoking

The age at which the onset of symptoms or behaviours first occurs is a key descriptive characteristic of the natural history of a disease or disorder. Early onset may indicate increased risk of a severe course. It has been associated with increased genetic liability for some disorders (Ho et al., 2002; Engstrom et al., 2003; Hyttinen et al., 2003) and has been used to define high-risk phenotypes (Zubenko et al., 2002; Palmer et al., 2003). However, reported age of onset may be subject to forward telescoping – a bias in which respondents report events closer to the time of the interview than is true. If forward telescoping affects reports of age of onset then descriptions of the course of a disorder and the risk associated with early onset

may be biased due to misclassification. If forward telescoping influences individuals with different characteristics differently (for example, a greater effect for men than women) then comparisons of early onset across those groups may produce erroneous results.

Despite the importance of age of onset in epidemiology and evidence of forward telescoping in dating other events (such as last doctor's appointment), investigation of forward telescoping on age at onset has been limited (Raphael and Marbach, 1997; Prohaska, Brown and Belli, 1998; Gaskell, Wright and O'Muircheartaigh, 2000; Burt, Kemp and Conway, 2001). Two studies have examined forward telescoping and onset of substance use. Johnson et al. (1998)

examined bias in reported age at onset for alcohol and marijuana use finding that the longer the interval between the age of interest and the age at interview the lower the age-specific incidence of use within a birth cohort.

Golub et al. (2000) examined reported age at onset of alcohol, tobacco, marijuana, and hard drug use in the longitudinal Rutgers Health and Human Development Project and the NHSDA 1982–95. Based on visual inspection, Golub et al. (2000) concluded that the reductions that were observed in reported first use as age at interview increased, in both data sources, represented forward telescoping rather than attrition from the household population.

These studies suggest that forward telescoping influences reported age at onset of first substance use. However, focusing on initiation of any use, Johnson et al. (1998) and Golub et al. (2000) could not address two important questions:

- Is the influence of forward telescoping on age of initiation similar for experimental users (those who never go on to regular use) and for those who make the transition to regular use?
- Does forward telescoping influence reported age at onset of more advanced stages of substance use?

Age of initiation may be less salient for experimenters than for regular users and therefore more influenced by forward telescoping. If this occurs, the association often found between early onset and more advanced stages of substance use would be, in part, attributable to the differential impact of this response bias because experimenters would be more biased toward reporting later ages of onset than regular users. Similarly, onset of regular substance use may be more salient for individuals because of the relatively larger impact on their lives, suggesting lower likelihood of forward telescoping for advanced stages of use. If this were the case, forward telescoping may be of less concern for the onset of significant clusters of symptoms or of discrete diseases than for their earliest signs.

We focus on age of onset for smoking initiation and daily smoking. These descriptors of the course of smoking help to identify ages at which interventions are needed and what environmental or social risk factors may be involved, and they figure in the calculation of 'pack years' of exposure to cigarette smoke. Early age of smoking initiation and daily smoking are also risk fac-

tors for dependent smoking (Yamaguchi and Kandel, 1984; Lando et al., 1999), lower cessation rates (Breslau and Peterson, 1996), and higher risk of smoking-related disease (USDHHS, 1990). As part of a sequence of substance use, early onset of smoking indicates increased risk of progressing to use of other drugs (Kandel, 1975; Kandel et al., 1992). Early onset of smoking is also associated with psychiatric disorders (Wu and Anthony, 1999; Upadhyaya et al., 2002).

We used data from the National Household Survey on Drug Abuse (NHSDA – USDHHS, SAMHSA, 1997–2000) to estimate the magnitude of forward telescoping in reported age when the first cigarette was smoked, to test the hypotheses that forward telescoping will be greater for experimental smokers than for regular smokers, and to assess whether the magnitude of forward telescoping in reported age at first daily smoking was lower than that of initiation. We present examples of the impact of forward telescoping on the classification of respondents as early onset cases, and the comparison of birth cohorts from retrospective data.

Material and methods

Sample

Data come from the NHSDA (USDHHS, SAMHSA, 1997–2000), which was designed to estimate the prevalence of substance abuse in the non-institutionalized population of the US 12 years of age and older. Multistage probability sampling and face-to-face interview techniques were used. Surveys were conducted in 1979, 1982, 1985, 1988, and then yearly since 1990. The sample design of the survey was changed starting with the 1999 NHSDA, making these data incompatible with earlier NHSDA data for these analyses (USDHHS, SAMHSA, 2003). The overall response rates for surveys from 1979 to 1998 ranged from 77% to 83%.

Our analyses of forward telescoping were based on testing the degree to which age at interview was associated with the probability of early onset of 'ever smoking' or 'daily smoking' within (or adjusting for) birth-year cohort. To estimate the effect of age at interview independent of birth-year cohort based on multiple cross-sectional surveys of the same population represented by the NHSDA 1979–1998 samples, we selected those born between 1966 and 1977. By selecting these birth years we generated a subsample of

respondents, 12 to 32 years of age, which covered the period of risk for smoking initiation and initiation of daily smoking ($N = 82, 122$). This analytic sample is thus restricted and not representative of the non-institutionalized US population as a whole.

Measures

In order to estimate the degree of forward telescoping we focused on (a) the onset of smoking before age 12 and (b) daily smoking before age 15. These ages were chosen based on the age of onset distribution reported in the combined NHDSA from 1979 to 1998. Of those who reported 'ever smoking', 21.0% ($SE = 0.3$) reported having their first cigarette before age 12. Of those who reported 'ever smoking' daily, 22.5% ($SE = 0.5$) reported having started before age 15.

In 1994 the NHDSA was revised, changing questions and procedures for the interview (USDHHS, SAMHSA, 1998). The sample was split into those receiving the old and new versions of the interview. In the older version 1994 interviews, which will be referred to as the '1994a interviews', and in earlier interviews, having smoked and age at onset were combined by asking age at onset, but allowing for a 'never smoked cigarettes' response. The wordings of these questions were:

- 'About how old were you when you first tried a cigarette?'
- 'About how old were you when you first started smoking daily?'

For the 1994b interviews through to the 1998 interviews, separate questions were asked about having smoked cigarettes and the age at onset:

- 'Have you ever smoked a cigarette, even one or two puffs?'

Then:

- 'How old were you the first time you smoked a cigarette, even one or two puffs?'
- 'Has there ever been a period in your life when you smoked cigarettes every day?'

Then:

- 'How old were you when you first started smoking cigarettes every day?'

We tested for differences in the percentage of participants who reported 'ever smoking' cigarettes and

smoking daily between these subsamples and found no significant difference ($p = 0.12$ and $p = 0.30$). There was a mean difference of 0.38 years in age of first cigarette use between the 1994a and 1994b subsamples ($p = 0.05$) but no difference in age of first daily smoking ($p = 0.30$). With a minimal difference in cigarette smoking, combining the data across these survey forms appeared acceptable.

Analysis

Forward telescoping

Conducting multiple cross-sectional studies of the same population over time permits identification of individuals with the same birth year who were interviewed at different ages, creating independent samples of that birth-year cohort at different points in time. The incidence of cigarette smoking by some specific age prior to the interview should not be significantly different for a birth-year cohort sampled at different ages. For example, the true percentage of a birth-year cohort first smoking a cigarette before age 12 should be the same regardless of whether that cohort was interviewed at age 15 or age 25. To the degree the percentage of a birth-year cohort who report onset of smoking by a specific age is significantly associated with age at interview there is either a reporting or sampling bias.

Repeated sampling of a birth-year cohort across time may be biased if the rate of attrition from the sampling frame is influenced by early onset of smoking. We were unable to test this possibility but Golub et al. (2000) found no evidence of such a sampling bias for initiation of alcohol, tobacco or marijuana use. We thus concluded that any associations between age at interview and age-specific incidence of smoking are due to a reporting bias.

There are at least three potential reporting biases that may create an inverse association between age-specific incidence of smoking initiation and age at interview within birth cohorts: forward telescoping, recanting and recall decay. Study participants may remember age at first smoking cigarette as occurring closer to their age at interview than is true (forward telescoping). Participants at a later age may choose not to report behaviour that they would have reported at a younger age (recanting). Participants' ability to recall having smoked cigarettes may decrease with increased time since they smoked (recall decay). It should be

noted that backward telescoping, recalling events further back in time than is true, can bias reports of the timing of events (Lee and Brown, 2004) but this would produce a positive association between age specific incidence of smoking initiation and age at interview.

To estimate the degree of forward telescoping we focused on age-specific incidence. This included early onset of smoking initiation (before age 12), and daily smoking (before age 15). Thus our outcomes were dichotomous: 'onset of smoking initiation prior to age 12' (yes/no) and 'onset of daily smoking prior to age 15' (yes/no). We estimated the degree to which early onset of smoking and daily smoking were associated with age at interview within a birth year by using a logistic regression model. In this model cigarette smoking by age 12 or daily smoking by age 15 were predicted by age at interview, adjusting for birth year. A significant inverse association would indicate a decline in the age-specific incidence of 'ever smoking' or daily smoking as age at interview increased within birth year, attributable to forward telescoping, recanting, or recall decay. Only those who were 15 and older were included in the analysis of early onset daily smoking.

Treating 'age at interview' as a continuous variable in these logistic regression models assumes that age at interview was linearly associated with incidence of cigarette smoking before age 12 or daily smoking before age 15. We tested this assumption using the fractional polynomial method (Royston and Altman, 1994; Hosmer and Lemeshow, 2000). In this analytic method, successive combinations of the degree and power of polynomials are fitted to the data. The best fitting degree and power of polynomials, including a linear function, are determined by partial likelihood ratio tests for significantly increased model fit for each increasing degree of polynomial.

Sex and race were included in all logistic regression analyses. To test for differences in magnitude of forward telescoping of the age of first cigarette smoked between experimental and regular users we tested for an interaction between age at interview and 'ever becoming a daily smoker' in predicting onset of 'ever smoking' before age 12.

Recanting or recall decay

Recanting or recall decay could account for lower odds of early onset of smoking initiation and daily smoking associated with age at interview (which is taken here as evidence of forward telescoping) to the extent that

as respondents age they deny smoking or do not recall having smoked. Such recanting or recall decay would reduce the reported prevalence of early onset smoking by age within a birth-year cohort. To distinguish between forward telescoping and recanting or recall decay, Johnson et al. (1998) examined the extent to which lifetime rates of 'ever using alcohol or marijuana' declined as age at interview increased among adults of the same birth-year cohorts, who had passed through the period of risk for initiation. Johnson et al. (1988) did not find such an association, suggesting that recanting or recall decay were not contributing to the response bias observed. It is important for the analysis to include only those who were through the period of risk, so that any difference by age at interview within birth-year cohorts can be attributed to age rather than differences in the proportion of the period of risk through which respondents have lived. Consequently, we tested for differences in lifetime reports of 'ever smoking' and daily smoking for the same birth years (1967–77) when they were 26 to 32 years of age. The use of ages up to 25 as the period of risk was suggested by results from the National Comorbidity Survey where incidence of daily smoking largely ceased by age 25 (Breslau et al., 2001) and was confirmed in these NHSDA data; 99.8% of those who report 'ever smoking' and 99.4% of those who report 'ever smoking daily', reported having done so by age 25.

Impact of forward telescoping

We provide two illustrations of the impact of forward telescoping. The first examined misclassification of respondents as early versus late onset. The second examined bias in birth cohort comparisons from cross-sectional data. To estimate the degree of misclassification we compared the reported prevalence of ever and daily smoking before ages 12 and 15, respectively, to adjusted prevalence. The adjusted prevalence was calculated using the logistic regression equations that estimated the degree of forward telescoping, fixing age at interview to 12 or 15 for ever and daily smoking respectively and taking the mean of the predicted values from these modified equations.

To estimate the effect of forward telescoping on cohort comparisons from cross-sectional data we created four birth cohorts consisting of those born in three year blocks (1966–8, 1969–71, 1972–4, and 1975–7). The oldest age at interview within the most recent birth cohort was 21. Thus, the four cohorts

could be compared on incidence of 'ever smoking' and 'daily smoking' through age 20. Cases where there was a tie between age at interview and age at onset were censored one year prior to the age at interview. Cox proportional hazard models were used to test for differences in incidence of 'ever smoking' and 'daily smoking' by birth cohort adjusted for sex and race/ethnicity. A term for 'age at interview' was added to adjust these models for forward telescoping. This is possible without severe colinearity between 'age at interview' and cohort because participants with the same birth year were interviewed at different ages.

All analyses use NHSDA analytic weights to adjust for the probability of selection and to adjust to the US population. Standard errors were adjusted, the complex sampling design using STATA/SE 8.0 survey analysis functions (StataCorp, 2003). The Cox proportional hazard models were estimated using SUDAAN 7.5.6 to adjust for complex sampling and analytic weights (Shah et al., 1997).

Results

Table 1 shows the characteristics of the forward telescoping and recanting analysis samples. Among those 12 to 32 years of age from the 1966 through 1977 birth cohorts included in the 1979–98 NHSDA ($n = 82, 122$), prevalence of 'ever smoking' was 64.1% (SE = 0.4) and 'ever smoking daily' was 30.5% (SE = 0.4). Early onset of smoking, initiation before the age of 12 was reported by 13.4% (SE = 0.2) of this sample (21.0% of ever smokers, SE = 0.3) and 7.3% (SE = 0.2) reported early onset of daily smoking, initiation before the age of 15 (22.5% of ever daily smokers, SE = 0.5).

Test of forward telescoping

Early initiation of smoking

An inverse association was found between early onset of smoking and age at interview (OR = 0.93 for ever one year increase in age, 95% CI: 0.92, 0.94), adjusting for birth year, sex, and race/ethnicity. This was indicative of forward telescoping. However, a second degree polynomial was found to fit the association significantly better than a linear model ($p = 0.003$). Also, marginally significant interactions were found between fractional polynomial transformed age at interview and sex ($p = 0.06$ and $p = 0.05$ for each transformed age term respectively) but not for race/ethnicity ($p > 0.63$). Figure 1 shows the estimated

probability of early onset of smoking as a function of age at interview adjusted for birth year and race/ethnicity, separately for males and females.

Although the absolute levels of early onset of smoking were higher for males than females, both showed a significant reduction in reported early onset of smoking as age at interview increased, adjusting for birth year. From age 12 to age 25 at interview the probability of reporting early onset of smoking decreased by over half for both sexes, with this effect lessening after age 25.

Experimental versus daily smokers

To test the hypothesis that daily smokers would be less affected by forward telescoping than experimental smokers (those who have 'ever' smoked but who never smoked daily), interaction terms between daily smoking status and fractional polynomial transformed age at interview were added to the logistic regression models for males and females. Significant interactions were found for both males ($p < 0.02$) and females ($p < 0.001$) – see Figure 2.

The higher absolute probability of early onset of smoking among daily smokers compared to experimental smokers, across all ages at interview, was expected. However, contrary to our hypothesis, the probability function of early onset smoking by age at interview suggests that daily smokers were equally or more likely to be affected by forward telescoping than experimental smokers.

Early onset of daily smoking

An inverse association was also found between age at interview and reported onset of daily smoking before the age of 15 (early onset daily smoking), adjusting for birth year, sex, and race (OR = 0.95 for ever one year increase in age, 95% CI: 0.93, 0.97). Fractional polynomial analysis did not find any higher degree polynomials fit the data better than the linear model ($p > 0.32$). However there was a significant interaction between age at interview and race ($p < 0.001$). The association between early onset of daily smoking and age at interview was only evident for Caucasian, Hispanic and other racial/ethnic groups (OR = 0.95 95% CI: 0.93, 0.97). No association was found among African Americans (OR = 1.00 95% CI: 0.96, 1.03).

Test for recall decay or recanting

The inverse association found between age at interview and onset of smoking may be attributable to

Table 1. Demographic characteristics of the 1966–77 birth cohort from the NHSDA 1979–98

		Forward telescoping sample			Recall decay / recanting subsample		
		Unweighted N	Est. %	SE of %	Unweighted N	Est. %	SE of %
Total		82122			15407		
Sex	Male	37391	49.8	0.3	6523	49.2	0.6
	Female	44731	50.2	0.3	8884	50.8	0.6
Age at interview	12	1352	2.6	0.1	–	–	–
	13	1797	3.2	0.1	–	–	–
	14	2912	3.5	0.1	–	–	–
	15	4014	4.1	0.1	–	–	–
	16	5397	4.8	0.1	–	–	–
	17	5755	5.3	0.1	–	–	–
	18	4805	6.4	0.2	–	–	–
	19	4855	6.7	0.2	–	–	–
	20	5225	6.6	0.2	–	–	–
	21	5965	7.2	0.2	–	–	–
	22	5962	7.2	0.2	–	–	–
	23	6084	6.6	0.1	–	–	–
	24	6288	6.8	0.2	–	–	–
	25	6304	6.1	0.2	–	–	–
	26	4385	6	0.2	4385	26.5	0.5
	27	3403	4.9	0.1	3403	21.7	0.5
	28	2675	4.3	0.1	2675	19.2	0.5
29	2004	2.9	0.1	2004	13	0.4	
30	1545	2.3	0.1	1545	10.2	0.4	
31	889	1.4	0.1	889	6.1	0.2	
32	506	0.7	0.1	506	3.2	0.2	
Race/ethnicity	White	37929	70.2	0.6	6898	68.6	0.9
	Black	19134	13.8	0.4	3698	13	0.6
	Hispanic	22098	12	0.3	4306	13.5	0.5
	Other	2961	4	0.2	505	4.9	0.4
Birth year	1966	6745	10	0.2	4389	26.1	0.5
	1967	6539	9.8	0.2	3349	22.1	0.5
	1968	6203	8.8	0.2	2581	18.8	0.5
	1969	6256	9.4	0.2	2027	13.3	0.4
	1970	6353	9.2	0.2	1554	10.2	0.3
	1971	6687	8.6	0.2	968	6.3	0.3
	1972	6635	8.1	0.2	539	3.2	0.2
	1973	7145	7.8	0.2	–	–	–
	1974	6941	7.1	0.2	–	–	–
	1975	7447	7.3	0.2	–	–	–
	1976	7599	7	0.2	–	–	–
	1977	7572	6.6	0.2	–	–	–
Ever smoked a cigarette		48989	64.1	0.4	10799	72.4	0.6
Ever smoked daily		22120	30.5	0.4	5865	37.8	0.7

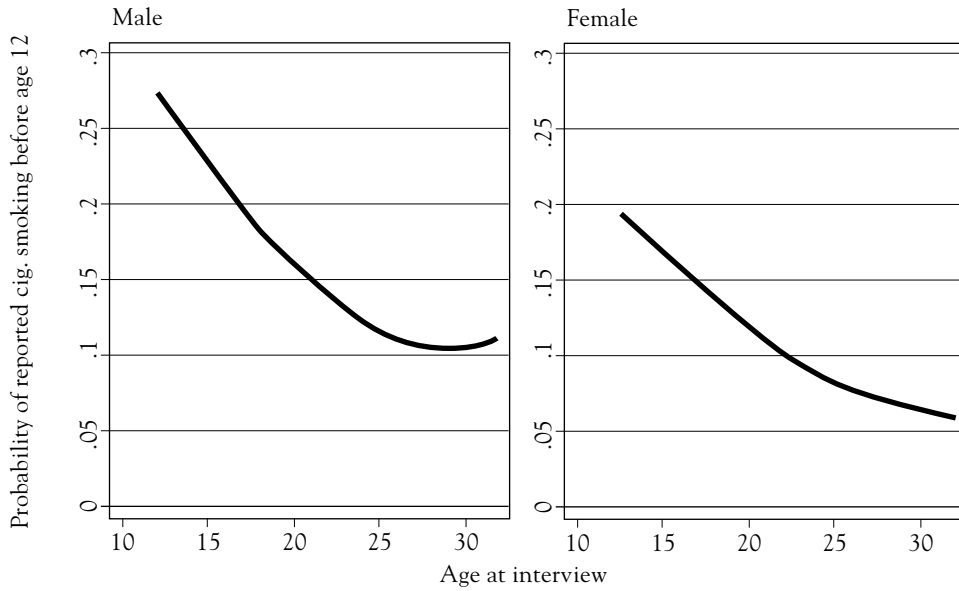


Figure 1. Probability of reported early onset by age at interview.

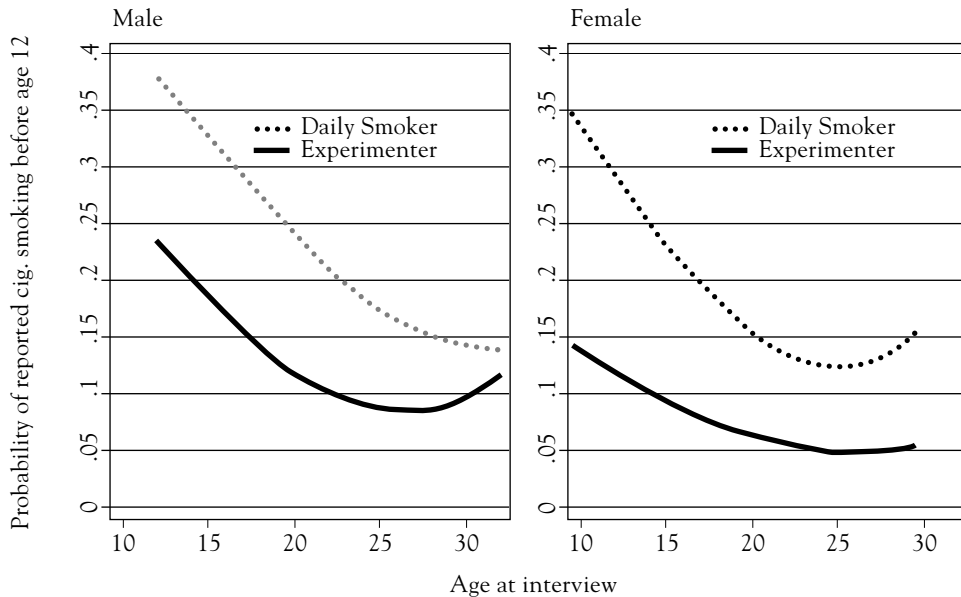


Figure 2. Probability of reported early onset by age at interview: experimenters versus daily smokers.

recanting or recall decay as participants age. Evidence of recanting or recall decay would be found if the likelihood of reporting ever smoking or daily smoking decreased as age at interview increased, adjusting for birth year, among adults older than the period of risk for onset of ever and daily smoking. This was not found for 'ever' smoking (OR = 1.01, 95% CI: 0.98,1.05). Age at interview was marginally associated with daily smoking, but in the opposite direction to

recanting (OR = 1.04, 95% CI: 1.00, 1.08, p = 0.06).

Examples of the effects of forward telescoping

Self-reported age of onset is commonly used in substance-use and psychiatric epidemiology to classify research participants as early onset cases. The forward telescoping found here could significantly influence estimates of early onset and its association with increased risk of adverse outcomes.

Table 2 shows the reported and forward-telescoping adjusted prevalence of early onset smoking and daily smoking. The adjusted prevalence of early onset smoking was approximately 1.7 to 1.8 times greater than the reported prevalence, whereas the adjusted prevalence of early onset daily smoking was 1.4 times greater than the reported prevalence. Approximately 10% of an adolescent and young adult population may be misclassified as late onset 'ever' smokers and 3% of this population misclassified as late onset daily smokers due to forward telescoping.

We also examined the effect of forward telescoping on cohort comparisons in cross-sectional studies by estimating differences in age specific incidence of 'ever' smoking and daily smoking, among ever smokers,

across four birth cohorts without and with adjustment for forward telescoping (see Tables 3 and 4).

In the unadjusted Cox proportional hazards analyses of ever smoking, each more recent birth cohort was significantly less likely 'ever' to smoke than the earliest birth cohort. Adjusting for forward telescoping increased these estimated cohort differences. Such a result would be expected in that forward telescoping disproportionately influences earlier birth cohort members to report later ages of onset, which, when comparing cohorts over the same age range (for example, 0 to 20 years of age) means that they disproportionately report ages of onset that are older than the comparison age range and are thereby counted as censored cases.

Table 2. Reported and forward telescoping adjusted prevalence of early onset

Early onset of:	n	Reported prevalence	95% CI	Adjusted prevalence	95% CI
Ever smoking (male)	37,323	16.1	15.5, 16.6	27.7	27.1, 28.3
Ever smoking (female)	44,654	11.0	10.4, 11.5	19.9	19.4, 20.4
Daily smoking (non-African Americans)	56,494	8.0	7.6, 8.4	10.9	10.5, 11.3

Table 3. Risk of ever smoking across birth cohorts: Cox proportional hazards unadjusted and adjusted for forward telescoping

	Hazard Ratio*	95% CI
Unadjusted for forward telescoping		
Birth cohort		
1966–8	1.0	
1969–71	0.95	0.91, 0.99
1972–4	0.92	0.88, 0.96
1975–7	0.85	0.81, 0.89
Adjusted for forward telescoping		
Birth cohort		
1966–8	1.0	
1969–71	0.91	0.87, 0.95
1972–4	0.85	0.81, 0.89
1975–7	0.75	0.71, 0.80
Age at interview	0.98	0.98, 0.99

*Sex and race/ethnicity were included as covariates in the proportion hazards models to adjust for their effects.

Table 4. Risk of daily smoking across birth cohorts: Cox proportional hazards unadjusted and adjusted for forward telescoping among non-African Americans

	Hazard Ratio*	95% CI
Unadjusted for forward telescoping		
Birth cohort		
1966–8	1.0	
1969–71	1.06	0.99, 1.13
1972–4	1.10	1.03, 1.17
1975–7	1.19	1.10, 1.28
Adjusted for forward telescoping		
Birth cohort		
1966–8	1.0	
1969–71	1.00	0.93, 1.08
1972–4	0.99	0.91, 1.08
1975–7	1.03	0.93, 1.15
Age at interview	0.98	0.98, 0.99

*Sex and race/ethnicity were included as covariates in the proportion hazards models to adjust for their effects.

The results of the unadjusted model for the incidence of daily smoking, among 'ever' smoking non-African Americans indicate that more recent cohorts are significantly more likely to make the transition from ever to daily smoking than earlier cohorts. However, once the model is adjusted for forward telescoping there were no differences in the likelihood of becoming a daily smoker, 'among ever smokers', by birth cohort.

Adjusting for forward telescoping corrected for members of earlier cohorts disproportionately reporting onset of daily smoking as occurring later than the comparison age range. It thereby increased the likelihood of daily smoking disproportionately among earlier cohorts and diminished the cohort differences.

Discussion

We found significant evidence of forward telescoping in reported age at onset of initiation of smoking and of daily smoking. Among 12- to 32-year-old participants in the 1979–98 NHSDA, we found that age at interview, adjusting for birth year, was inversely associated with early initiation of smoking. The probability of reporting early initiation when interviewed at age 25 was approximately half that when interviewed at age 12 for both sexes. Contrary to our hypothesis, forward telescoping of age at initiation appeared to influence both experimental smokers and daily smokers. There was no evidence that this apparent forward telescoping was due to recanting or recall decay.

These results are consistent with the findings reported by Johnson et al. (1998) and Golub et al. (2000), but extend their findings, showing that forward telescoping is equally present among those who experimented with cigarettes and those who became daily smokers. We also found that age at onset of a more advanced stage of substance use, daily smoking, was subject to significant forward telescoping among non-African American participants. These data suggest that greater impact of behaviours or symptoms on study participants' lives may not increase the accuracy of recalled age of onset. This implication contrasts with what might be expected based on studies of the test-retest reliability of age at onset in common psychiatric disorders, in which age at onset showed fair to high reliability (Farrer et al., 1989; Wittchen et al., 1989; Barkow et al., 2002). However, the periods over which reliability was assessed ranged from 1 to 2 years. This may be too short a time frame to detect forward

telescoping. We found a 5% reduction in odds of reporting early onset of daily smoking with each increased year of age at interview, which might not be enough to substantially affect test-retest reliability over a single year if a similar forward telescoping effect was present for onset of psychiatric disorders. Consequently, forward telescoping may still be of concern for the onset of significant clusters of symptoms or of discrete diseases as much as for their earliest signs.

It is also important to note that the magnitude of forward telescoping was not uniform across groups. Males and females significantly differed in the degree of forward telescoping of age at initiation of smoking. We also found no evidence of forward telescoping of age at onset of daily smoking among African Americans but significant forward telescoping among Caucasian, Hispanic, and 'other' Americans. These group differences indicate that forward telescoping may significantly bias group comparisons and magnitudes of association between early onset and outcomes of interest.

We also found that forward telescoping appears to cause underestimation of early onset of initiation as well as daily smoking, and can influence incidence comparisons between birth cohorts based on cross-sectional data. For example, in the unadjusted analysis, 'ever' smokers in the most recent birth cohort (1975–77) were 20% more likely to become daily smokers than 'ever' smokers in the earliest birth cohort (1966–8). However, once one adjusted for forward telescoping there were no significant differences between birth cohorts in risk, among 'ever' smokers, of becoming a daily smoker.

Limitations

There were several limitations to this study:

- First, these analyses were based on cross-sectional data in which the association of age at interview within birth-year cohorts was estimated using different individuals within those cohorts interviewed at different ages. Stronger conclusions could be drawn from analyses of longitudinal data in which the same individuals were interviewed at least twice, over a fairly long follow-up interval, and where age at onset was assessed at each interview (rather than onset since the most recent interview, which is usually the case). To our knowledge such data do not yet exist.

- Second, data from the NHSDA collected after 1998 could not be included with data from prior NHSDA years due to changes in sample design and variable reporting in more recent years of the survey.
- Third, we could only examine differences in forward telescoping by sex and race/ethnicity due to variation in how other covariates were collected over different years of the NHSDA.

Implications for question and study design

The data set for this study was constructed from multiple cross-sectional samples of the same population over time, thus it was possible to adjust for age at interview within a birth year. Most epidemiological studies do not have this data structure. However, there are question and sampling design techniques that might limit forward telescoping. Questions that require greater reconstruction, making the participant work harder, might generate more accurate responses (Prohaska et al., 1998); the use of event history calendars or landmark events can reduce recall bias (Axinn et al., 1999; Belli et al., 2001) and within the limits of the hypotheses, the age range of a study sample could be kept narrow to limit forward telescoping.

Conclusion

Evidence of forward telescoping in reported age at onset of smoking was found among experimental smokers and daily smokers, as well as for age at onset of daily smoking. This bias differed significantly by sex and race, suggesting that comparisons between these groups based on age at onset could lead to erroneous conclusions. We also demonstrated that forward telescoping might cause participants to be misclassified as having a late onset of smoking and daily smoking, as well as erroneous cohort comparisons from cross-sectional data. These results question the assumption that forward telescoping is a bias that can be largely ignored and suggests that attention needs to be paid to limiting it by question and sampling design.

References

Axinn WG, Pearce LD, Ghimire D. Innovations in life history calendar applications. *Social Science Research* 1999; 28: 243–64.

Barkow K, Heun R, Ustun TB, Gansicke M, Wittchen H-U, Maier W. Test-retest reliability of self-reported age at onset of selected psychiatric diagnoses in general health care. *Acta Psychiatr Scand* Aug 2002; 106(2):117–25.

Belli RF, Shay WL, Stafford FP. Event history calendars and question list surveys. *Public Opinion Quarterly* 2001; 65: 45–74.

Breslau N, Peterson EL. Smoking cessation in young adults: age at initiation of cigarette smoking and other suspected influences. *Am J Public Health* 1996; 86(2): 214–20.

Breslau N, Johnson EO, Hiripi E, Kessler R. Nicotine dependence in the United States: prevalence, trends, and smoking persistence. *Arch Gen Psych* 2001; 58: 810–16.

Burt CD, Kemp S, Conway M. What happens if you retest autobiographical memory 10 years on? *Memory and Cog* 2001; 29(1):127–36.

Engstrom C, Brandstrom S, Sigvardsson S, Cloninger R, Nylander PO. Bipolar disorder. II: personality and age of onset. *Bipolar Disorders* 2003; 5(5):340–8.

Farrer LA, Florio LP, Bruce ML, Leaf PJ, Weissman MM. Reliability of self-reported age at onset of major depression. *J Psychiatr Res* 1989; 23(1): 35–47.

Gaskell GD, Wright DB, O'Muircheartaigh CA. Telescoping of landmark events: implications for survey research. *Pub Op Quarterly* 2000; 64:77–89.

Golub A, Johnson BD, Labouvie E. On correcting bias in self-reports of age at first substance use with repeated cross-section analysis. *J of Quant Crim* 2000; 16(1): 45–68.

Ho GJ, Hansen LA, Alford MF, Foster K, Salmon DP, Galasko D, Thal LJ, Masliah E. Age at onset is associated with disease severity in Lewy body variant and Alzheimer's disease. *Neuroreport* 2002; 13(14):1825–8.

Hosmer DW, Lemeshow S. *Applied Logistic Regression*. 2 edn. New York: John Wiley & Sons, 2000, pp. 91–141.

Hyttinen V, Kaprio J, Kinnunen L, Koskenvuo M, Tuomilehto J. Genetic liability of type 1 diabetes and the onset age among 22,650 young Finnish twin pairs: a nationwide follow-up study. *Diabetes* 2003; 52(4):1052–5.

Johnson RA, Gerstein DR, Rasinski KA. Adjusting survey estimates for response bias: an application to trends in alcohol and marijuana use. *Pub Op Quart* 1988; 62: 354–77.

Kandel DB. Stages in adolescent involvement in drug use. *Science* 1975; 190: 912–14.

Kandel DB, Yamaguchi K, Chen K. Stages of progression in drug involvement from adolescence to adulthood: further evidence for the gateway theory. *J Stud Alcohol* 1992; 53: 4547–57.

Lando HA, Thai D, Murray DM, Robinson LA, Jeffery RW, Sherwood NE, Hennrikus DJ. Age of initiation, smoking patterns, and risk in a population of working adults. *Prev Med* 1999; 29: 590–8.

Lee PJ, Brown NR. The role of guessing and boundaries on date estimation biases. *Psychon Bull Rev* 2004; 11(4): 748–54.

Palmer LJ, Celedon JC, Chapman HA, Speizer FE, Weiss

- ST, Silverman EK. Genome-wide linkage analysis of bronchodilator responsiveness and post-bronchodilator spirometric phenotypes in chronic obstructive pulmonary disease. *Human M Gen* 2003; 12(10): 1199–210.
- Prohaska V, Brown NR, Belli RF. Forward telescoping: the question matters. *Memory* 1998; 6(4): 455–65.
- Raphael KG, Marbach JJ. When did your pain start? Reliability of self-reported age of onset of facial pain. *Clin J Pain* 1997; 13(4): 352–9.
- Royston P, Altman DG. Regression using fractional polynomials of continuous covariates: parsimonious parametric modeling (with discussion). *Applied Statistics* 1994; 43: 429–67.
- Shah BV, Barnwell BG, Bieler GS. SUDAAN User's Manual. Release 7.5. Research Triangle Park NC: Research Triangle Institute, 1997.
- StataCorp. Stata Statistical Software: Release 8.0. College Station TX: Stata Corporation, 2003.
- Upadhyaya HP, Deas D, Brady KT, Kruesi M. Cigarette smoking and psychiatric comorbidity in children and adolescents. *J Am Acad Adolesc Psychiatry* 2002; 41(11):1294–305.
- US Department of Health and Human Services. The health benefits of smoking cessation. USDHHS, Public Health Service, Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. Publication No. (CDC) 90-8416. Rockville MA: DHHS, 1990.
- US Department of Health and Human Services, Substance Abuse and Mental Health Services Administration (SAMHSA), Office of Applied Studies. National Household Survey on Drug Abuse, 1994. Computer file. ICPSR version. Research Triangle Park NC: Research Triangle Institute/Chicago IL: National Opinion Research Center (producers), 1997. Ann Arbor, MI: Inter-university Consortium for Political and Social Research (distributor), 1998.
- US Department of Health and Human Services, Substance Abuse and Mental Health Services Administration (SAMHSA), Office of Applied Studies. National Household Survey on Drug Abuse, 1979–98. Computer file. ICPSR versions. Research Triangle Park NC: Research Triangle Institute (producer), 1995–2000. Ann Arbor MI: Inter-university Consortium for Political and Social Research (distributor), 1997–2000.
- US Department of Health and Human Services, Substance Abuse and Mental Health Services Administration (SAMHSA), Office of Applied Studies. National Household Survey on Drug Abuse, 2001 (Computer file). ICPSR version. Research Triangle Park NC: Research Triangle Institute (producer), 2002. Ann Arbor MI: Inter-university Consortium for Political and Social Research (distributor), 2003.
- Wittchen HU, Burke JD, Semler G, Pfister H, Von Cranach M, Zaudig M. Recall and dating of psychiatric symptoms. Test-retest reliability of time-related symptoms questions in a standardized psychiatric interview. *Arch Gen Psychiatry* 1989; 46(5): 437–43.
- Wu LT, Anthony JC. Tobacco smoking and depressed mood in late childhood and early adolescence. *Am J Public Health* 1999; 89: 1837–40.
- Yamaguchi K, Kandel D. Patterns of drug use in adolescence to young adulthood: III predictors of progression. *Am J Public Health* 1984; 74: 673–81.
- Zubenko GS, Hughes HB 3rd, Maher BS, Stiffler JS, Zubenko WN, Marazita ML. Genetic linkage of region containing the CREB 1 gene to depressive disorders in women from families with recurrent, early-onset, major depression. *Am J Med Gen* 2002; 114(8): 980–7.

Correspondence: Eric O Johnson, Substance Abuse Epi, Prevention, and Risk Behavior, Division of Health, Social and Economic Research, Research Triangle Institute International, PO Box 12194, 3040 Cornwallis Road, Research Triangle Park NC 27709-2194, USA.
Telephone (+1) 919 990 8347.
Fax (+1) 919 485 5555.
Email: ejohnson@rti.org.