Modelling change in adolescent smoking behaviour: Stability of predictors across analytic models

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Objectives. The current paper examined the variability of predictors of changes in adolescent smoking across linear and nonlinear analytic models.

Design. Three analytic models typically used to model adolescent smoking behaviour were tested: one linear model of change (standard linear), one static linear model (pre-post linear) and one nonlinear model of change (cusp catastrophe). Variability in model composition was assessed by examining the pattern of variables achieving statistical significance and proportion of variance explained.

Methods. Model testing was conducted on data from Australian adolescents successfully tracked through a 12-month longitudinal study of smoking ($N = 779$). The survey measured demographics, self-reported smoking, smoking among friends and family, self-esteem, neuroticism, coping, stress and risk taking.

Results. The results indicated that while predictors of change were invariant across analytic models explanatory power varied markedly. Models of change in smoking that included simple, interacted or polynomial forms of initial conditions (past behaviour) explained more than four times the variance of models without.

Conclusions. These results justified confidence in the predictors of change in adolescent smoking across analytic models. A secondary implication was that more research into past behaviour's role in the context of dynamical models of adolescent smoking and other health behaviour is needed.

Longitudinal adolescent smoking research has demonstrated dynamical models such as the cusp catastrophe model (CCM) explain more change variance than comparable general linear models (GLM; Byrne, Mazanov, & Gregson, 2001). Byrne *et al.* report a CCM based analysis of change in adolescent smoking behaviour explained 14–20% more variance than a comparable GLM. If dynamical models prove a reliably better explanation of changes in adolescent smoking behaviour, the relationship of other

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explanatory variables with the dependent variable may change. That is, the variables that achieve statistical significance may vary across the type of analytic model. Evidence suggests the predictive strength of some variables (e.g. self-esteem) would be invariant (Byrne & Mazanov, 1999, 2002) and others variant (e.g. neuroticism; Byrne, Byrne, & Reinhart, 1993, *vs.* Wakefield, 1989). The aim of the current paper is to provide evidence on whether the predictors of changes in adolescent smoking are consistent or vary across analytic models.

The comparison focuses on similarities and differences in model composition between GLM and CCM analyses of change in smoking behaviour, after Guastello (1982, 1987, 1992). The CCM was selected for its success in explaining changes in adolescent smoking behaviour (Byrne *et al.*, 2001) and capacity to be approximated using least squares (Guastello, 1982, 1992; see Method). This latter point means comparison of model results can be conducted using regression coefficients and measures of effect size (adjusted- R^2 ; see Alexander Herbert, DeShon, & Hanges, 1992; Guastello, 1992; Ratkowsky, 1990, for further discussion).

A brief overview of the CCM is offered. The interested reader is directed to Byrne *et al.* (2001) and Clair (1998) for more detailed discussions of the CCM in the context of adolescent substance use.

Catastrophe Theory (Thom, 1975) seeks to explain how continuous inputs become discontinuous outputs (Parker, 1997) using a combination of Singularity and Bifurcation Theory (Arnol'd, 1991). More simply, catastrophe models describe sudden changes in otherwise stable behaviour (Scott, 1985). The cusp catastrophe is a class of catastrophe that describes changes between two areas of stability, such as radical changes in smoking behaviour (e.g. rapid deceleration when smokers quit).

The cusp catastrophe is defined by two control parameters (sy and a) and the target behaviour (y or the dependent variable; DV) (see Equation 1).

$$df(y)/dy = y^3 - sy - a \quad (1)$$

The term s from Equation 1 splits or bifurcates the DV at the 'divergence' point (see Figure 1). That is, it has the effect of changing the DV's distribution from unimodal at low values to bimodal at high values (Alexander *et al.*, 1992, p. 367). The term s is usually known as the splitting or bifurcating variable and sy the splitting or bifurcating parameter. The asymmetry parameter (a from Equation 1) behaves differently across the bifurcation term (see Figure 1). For example, (from Byrne *et al.*, 2001), attitude towards smoking stably predicts change in smoking behaviour (e.g. negative attitudes predict decreases, positive attitudes increases) when there is little or no peer pressure (e.g. very few friends smoke). As more friends smoke, smoking behaviour bifurcates and attitude predicts both increases and decreases. Thus, peer pressure can be said to have had a bifurcating effect on the prediction of changes in smoking behaviour using attitude.

The cubed and bifurcation terms have specific functions in the CCM. The cubed term defines the cusp and the bifurcation term the sudden change or the catastrophe (Guastello, Gershon, & Murphy, 1999). If either term is statistically non-significant, the CCM has failed. The bifurcation term also has to have the character of creating a divergence in the behaviour of interest. This is usually determined by looking for bimodality in the distribution of the DV as the bifurcation variable increases in magnitude (Clair, 1998). This forms an important precondition to a CCM based analysis.

The literature reports that two splitting variables have been considered in relation to adolescent substance use behaviour (alcohol and smoking). The first, used as an

Figure 1. Behaviour manifold of the hypothesized cusp catastrophe (adapted from Byrne et al., 2001).

example above, is peer pressure. Clair (1998) developed a strong theoretical case for peer pressure acting as a splitting variable in the context of adolescent substance use. Clair's test of this theoretical argument empirically demonstrated peer pressure as a viable splitting variable in an analysis of adolescent alcohol consumption. Byrne *et al.* (2001) replicated Clair's empirical support for peer pressure as a splitting variable in the context of adolescent smoking behaviour. The second, assessed by Mazanov and Byrne (2006) as an alternative splitting variable to peer pressure in the context of adolescent smoking, is behavioural intention. The results of this study indicated that while behavioural intention was no worse than peer pressure, it was certainly no better. On the weight of available theoretical and empirical evidence it seems that peer pressure is the better candidate for a splitting variable in the context of a CCM analysis of adolescent substance use behaviour. For the current analysis, then, the precondition of a bifurcatory relationship is met if bimodality in smoking behaviour is observed as peer pressure increases in magnitude.

Preconditions and selection of splitting variables aside, this study is most interested in the asymmetry parameter, a . The composition of a is the basis for understanding whether the analytic model used to describe the data changes the relationship the dependent variable has with other variables. The GLM analysis used for comparison can be expressed as

$$df(y)/dy = s - a \quad (2)$$

where s is the splitting/bifurcation variable and a the asymmetry parameter. See Methods for more detail and the least squares approximation of Equation 2.

The selection of variables to include in a is difficult given the volume of variables associated with adolescent smoking behaviour (McGee & Stanton, 1993; Tyas & Pederson, 1998). The suite chosen aimed to achieve a broad cross-section of variables associated with adolescent smoking. Using this approach, a is defined by Equation 3:

$$a = f(\delta, \omega, \psi, \epsilon) \quad (3)$$

where δ indicates demographic variables, ω the smoking of significant others, ψ represents the psychosocial (individual difference) variables and ϵ the error associated with being unable to tap all variables associated with smoking behaviour.

The dominant demographic variables usually chosen for this sort of analysis include age and gender. The pattern of smoking among adolescents changes with age (Choi, Gilpin, Farkas, & Pierce, 2001). Inclusion of age may only obscure the effect of developmentally relevant phenomena (e.g. risk taking behaviour; see below). Since the differences between the sexes have been established (e.g. Byrne & Mazanov, 2003), gender is included as a demographic variable by using different models for each sex rather than dummy coding gender into a single model.

The smoking behaviour of significant others is an established predictor of adolescent smoking, especially familial and peer smoking. Parental smoking has been shown to effect smoking both in terms of single time prediction (Biglan, Duncan, Ary, & Smolkowski, 1995) and uptake and quitting (Farkas, Distefan, Choi, Gilpin, & Pierce, 1999). Likewise, an elder sibling's smoking status has been shown to be a risk factor for the younger cross-sectionally (Wang, Fitzhugh, Westerfield, & Eddy, 1995) and longitudinally (Chassin, Presson, Sherman, Corty, & Olshavsky, 1984). Thus, the smoking of the immediate family demands inclusion in a model of adolescent smoking.

Peer smoking was included in the current analysis two ways. The first was the bifurcation variable (s from Equation 1), which measured the perceived proportion of 'friends who smoke' (Byrne *et al.*, 2001). The second was 'best friend' smoking, demonstrated as a non-trivial predictor of adolescent smoking behaviour longitudinally (Alexander, Piazza, Mekos, & Valente, 2001; De Vries, Engels, Kremers, Wetzels, & Mudde, 2003).

The psychosocial variables chosen were coping, neuroticism, risk taking, self-esteem and stress. Coping was included due to its theoretical (e.g. Protection Motivation Theory; Rogers, 1975) and empirical relationship with smoking behaviour (Dugan, Lloyd, & Lucas, 1999; Sussman, Dent, & Galaif, 1997; Vaccaro & Wills, 1998). Neuroticism has been included due to its variable significance as a predictor of smoking behaviour (Byrne *et al.*, 1993, *vs.* Wakefield, 1989). Risk taking behaviour was included as adolescence is characterized by experimentation with risky behaviours (Gonzalez, Field, Yando, Gonzalez, Lasko, & Bendell, 1994) and the result that smokers are less risk adverse (Greening & Dollinger, 1991). Byrne and Mazanov (1999, 2001, 2003) have demonstrated self-esteem has both cross-sectional and longitudinal impacts on adolescent smoking. Finally, psychosocial stress experienced by adolescents has been implicated as a descriptor and predictor of change in smoking behaviour (Byrne & Mazanov, 1999, 2003; Mazanov & Byrne, 2002).

Hypotheses

Three hypotheses were used to determine whether the composition of a varied across analytic models. The first hypothesis checked the preconditions of the CCM:

- (1a) The frequency plot of smoking behaviour became bimodal as peer pressure increased.
- (1b) The cubed and bifurcation terms were nonzero. The second hypothesis tested the viability of the CCM as an explanation of the data. If the CCM explained an equivalent or greater amount of variance then it was a viable alternative to the GLM as a way of analysing adolescent smoking behaviour.
- (2) The CCM of changes in adolescent smoking accounted for more variance than GLM counterparts. The third hypothesis tested whether a varied across analytic models using the null case.
- (3) There was no difference in the composition of the asymmetry parameter a across models.

The alternative to this hypothesis suggested some difference did exist.

Method

This analysis used data from a larger study on adolescent smoking by Mazanov (2003). Only measures relevant to the current paper are reported.

Sampling frame and sample

The sampling frame targeted Year 7-10 students attending public high schools (Years 7-10) or private/Catholic Colleges (Years 7-12) in the Australian Capital Territory (ACT). Ethics approval for the study was obtained from the Australian National University Human Research Ethics Committee (approval number 2001/4),

ACT Department of Education and Community Services and the Catholic Education Office. Out of the four private, three Catholic and six public schools approached, one all male private, three Catholic (one all female, two co-educational) and one co-educational public agreed to participate (38%).

Some 2,671 students responded to the T1 survey (March 2001) and 1,871 to the follow-up survey (T2; March 2002). A total of 779 (29.2%) observations were available for hypothesis testing, representing approximately 4.1% of the entire population of ACT high school students in 2002 (19,000; Dwyer, personal communication). Those unavailable included 1,693 (63.4%) lost to attrition and 199 (7.4%) observations successfully tracked but missing data requisite for testing the hypotheses.

Measures

Demographics

This part followed Byrne's surveys on adolescent smoking (Byrne *et al.*, 1993; Byrne & Mazanov, 1999, 2002) with questions on age, gender and year at school.

Smoking

Following Byrne *et al.* (1993), respondents answered questions on parental, sibling and personal smoking. Parental (0 = neither parent currently smoke, 1 = one or both parents currently smoke) and sibling smoking was dummy coded (0 = no sibling currently smokes, 1 = one or more siblings currently smoke). Smokers reported the number of cigarettes smoked over an average day or week. The average daily figure was converted to a weekly figure to establish common terms and then converted to one out of the seven categories for the analysis: 0 for non-smoking, 1-6 cigarettes per week for weekly smoking, and five categories for degrees of daily smoking (7-35, 36-70, 71-105, 106-140, 141 +).

Bifurcation variable

Peer pressure was operationalized by a question asking how many of the respondent's friends smoked (none, a few, half or most) used by Byrne *et al.* (2001).

Self-Esteem Inventory (SEI)

The 10-item (each item with a score of 1-4) self-esteem measure by Rosenberg (1989) was used. The items were combined according to instructions to give a single summary score ranged from 10 to 40.

Junior Eysenck Personality Inventory – Neuroticism Scale (JEPI-N)

The scale was extracted from Eysenck (1965) using the yes/no response format (23 items) and scored according to instructions. Neuroticism scores ranged from 0 to 23.

General Health Questionnaire (GHQ)

Coping was measured using Goldberg's (1972) GHQ-12 and scored using the 0/1 format.

Adolescent Stress Questionnaire (ASQ)

The original 40 items tested for this scale was used for T1 (see Byrne, Byrne, & Reinhart, 1995) and the 31-item version at T2 after confirmatory factor analysis (Byrne & Mazanov, 2002). The confirmed factor structure included seven correlated factors that describe adolescent psychosocial stress: (1) attending school, (2) family conflict, (3) parental control, (4) school performance, (5) future uncertainty, (6) perceived educational irrelevance and (7) interactions with the opposite sex. The responses were summed to give a single score 'stress' (range 31–155).

Adolescent Risk Taking Questionnaire (ARQ)

Gullone, Moore, Moss, and Boyd's (2000) questionnaire on the frequency and perception of risk behaviours was employed. The questionnaire has four correlated factors confirmed for frequency and perception: thrill seeking; rebellious; reckless and antisocial. Frequency was assessed using a five-point scale of 0 (never done) to 4 (done very often). Perception of risk was assessed with a similar scale of 0 (not at all risky) to 4 (extremely risky). The responses were summarized according to the instructions given by Gullone *et al.* by taking the average of each scale (range 0 to 4).

Design and procedure

The surveys were administered using a longitudinal design (12-month measurement interval). Surveys were matched over time on the basis of identifiers constructed from information within each survey: respondent school; gender; first two numbers of their address and their birthday (day and month). Other information was used to match the data when identical identifiers appeared (e.g. year at school). Respondents were protected from searches of school or governmental databases to reconstruct identifiers by the response of participating schools and the ACT public service to the *Australian Privacy Act* (Commonwealth of Australia, 2002), thus ensuring anonymity.

Respondents completed surveys during health or physical education classes, contingent upon consent from parents or guardians, and were able to withdraw at any time without qualification. Instructions were printed on the surveys and teachers permitted to clarify words as required. Completed schedules were placed in sealable envelopes and returned to a central point for collection by the research team.

Equations

The CCM was tested against linear expressions that use least squares regression to specify coefficient weights. The general form of the cusp catastrophe was translated into a form that made use of least squares estimation (Guastello, 1992). This translation necessitated parameterization of change in smoking over time (see below). The comparison models used included a standard linear combination and a 'pre-post' model (see Clair, 1998; Guastello, 1987, 1988).

Least squares estimation of a CCM

The variable y in Equation 1 was transformed into z_i by Equation 4 to correct for location and scale (Cobb, 1981; Guastello, 1992). This transformation established a rational zero point and hence comparisons were made on the same ratio scale (Clair, 1998, p. 228; see Guastello, 1988, p. 248 for a more technical account and rationale). This transformation

meant non-smokers were represented by the zero point and positive scores represent smokers. Each observation making up the DV was transformed using:

$$z_i = (y_i - \text{lower limit of } y_i) / \text{standard deviation of } y_i \quad (4)$$

The subscript i represented the administration involved.

The least squares version of a CCM (see Cobb, 1981; Guastello, 1992, p. 375; and also Alexander *et al.*, 1992) is:

$$z_i - z_{i-1} = b_0 + b_1 a + b_2 z_{i-1}^3 + b_3 s_{pp} z_{i-1} \quad (5)$$

The weights associated with each model (the b_j) are reset to zero for each new model; the constant is expressed as b_0 for each model and does not represent the same constant being used across all models. The asymmetry parameter a (from Equation 1 and Equation 3) represented the linear combination of the predictors of adolescent smoking. The bifurcation variable (peer pressure) was represented by s_{pp} in the context of the bifurcation term ($s_{pp} z_{i-1}$). To understand how the b_j relate to the DV it is important to note that with the DV being $z_i - z_{i-1}$, scores close to zero represented no change (e.g. continued non-smoking or smoking at the same level), a negative score a decrease in smoking (e.g. from smoking daily to weekly) and a positive score increase (e.g. either uptake or an increase in involvement).

Models for comparison

The first comparison model was the least squares version of Equation 2, termed the *standard linear model*. This model included the asymmetry parameter plus the bifurcation variable and represented the simplest way in which change might be modelled.

$$z_i - z_{i-1} = b_0 + b_1 a + b_2 s_{pp} \quad (6)$$

The second comparison model was the *pre-post linear model*. This model moved the z_{i-1} term to the right side of the equation, testing whether the best model used past behaviour (T1) to predict future behaviour (T2). This modification represented a second appropriately simple way to understand future behaviour (Clair, 1998).

$$z_i = b_0 + b_1 a + b_2 s_{pp} + b_3 z_{i-1} \quad (7)$$

The third model was the *CCM*, established by Equation 5.

Results

This section has three parts. The first looked at generalizability by comparing follow-up (analysis), attrition and 'missing data' groups in terms of age and smoking behaviour. The second part responded to the first hypothesis by assessing the bimodality of the bifurcation variable. The final part reported the explanatory power (second hypothesis) and composition (third hypothesis) of the models.

Demographics

Age

Respondents (both genders) were typically 14.5 years old (standard deviation of 1 year; see Table 1). This covered the range of ages expected from students in Years 7–11. The pattern of differences between the groups seen in males ($F_{2,1389} = 39.170, p < .001$) was also seen in females ($F_{2,1251} = 41.540, p < .001$). Respondents in the attrition group were typically older (by around 5 months) than those in the analysis group, who were in turn older than those in the 'missing data' group by 4–5 months. Seventeen males and eleven females failed to record useable age data.

Table 1. Sample size, average age and standard deviation for each gender by the analysis, missing data and attrition groups

	<i>N</i>	Average	<i>SD</i>
Males			
Analysis	340	14.22	0.88
Missing data	101	13.96	1.02
Attrition	951	14.63	0.97
Total	1392	14.48	0.98
Females			
Analysis	439	14.23	0.91
Missing data	94	13.80	1.03
Attrition	718	14.60	0.98
Total	1251	14.41	0.99

Smoking behaviour

The pattern of male smoking (see Table 2) demonstrated statistically significant differences across the groups ($\chi^2 = 10.275, df = 4, p < .037$). Notably, the analysis group displayed almost half the rate of T1 smoking as the attrition group and around two-thirds that of the 'missing data' group. The ex-smoker rates showed equivalence between the analysis and attrition groups, and a much lower rate in the 'missing data' group. This led to the analysis and 'missing data' groups having about the same rate of non-smoking and the lesser rate of T1 non-smoking in the attrition group. Smokers may be under-represented in the male data set. There were four missing data points from the 'missing data' and attrition groups.

Table 2. Smoking status at T1 by analysis, missing data and attrition groups

	Smoker	Ex-smoker	Non-smoker	Total
Males				
Analysis	26 (7.6%)	42 (12.3%)	274 (80.1%)	342 (100.0%)
Missing data	11 (10.8%)	9 (8.8%)	82 (80.4%)	102 (100.0%)
Attrition	133 (13.8%)	110 (11.4%)	718 (74.7%)	961 (100.0%)
Total	170 (12.1%)	161 (11.5%)	1074 (76.4%)	1405 (100.0%)
Females				
Analysis	33 (7.5%)	42 (9.6%)	364 (82.9%)	439 (100.0%)
Missing data	5 (5.3%)	9 (9.5%)	81 (85.3%)	95 (100.0%)
Attrition	117 (16.1%)	117 (16.1%)	494 (67.9%)	728 (100.0%)
Total	155 (12.3%)	168 (13.3%)	939 (74.4%)	1262 (100.0%)

Female smoking (see Table 2) also showed differences between the groups ($\chi^2 = 40.251$, $df = 4$, $p < .001$). The analysis group had about half the rate of T1 smoking as the attrition group and showed a higher rate of smoking than the 'missing data' group. Both the analysis and 'missing data' groups had a notably lower rate of ex-smoking, when compared with the attrition group (see Table 2). This meant the analysis group had a slightly lower rate of non-smoking than the 'missing data' group.

Summary

The differences observed between the analysis, attrition and 'missing data' groups indicated the data were more reflective of younger non-smokers. This may limit the opportunity to observe smokers reducing consumption and ample opportunity to observe non-smoker uptake. This pattern of demographics limiting generalizability is common in longitudinal field studies of adolescent smoking (e.g. Byrne & Mazanov, 2003; Engels, Knibbe, & Drop, 1999; Pirie, Murray, & Luepker, 1988), and formed a caveat to generalizing the results to changes in older adolescents who smoke.

Bimodality

The distributions of smoking behaviour by peer pressure categories for males and females are illustrated by Figures 2 and 3, respectively. Male smoking (see Figure 2) appeared to achieve a resemblance of bimodality in the manner reported by Byrne *et al.* (2001), although to a lesser degree. The female results (see Figure 3) showed a slightly different indication of potential emergent bimodality at the 7-35 and 36-70 categories. However, neither figure provided a clear indication of peer pressure having a bimodal relationship with smoking behaviour along its range. A closer examination of the transition matrices (see Table 3) indicated some variation in both directions, although the main diagonal indicated that most respondents were consistent in their behaviour over time. The CCM was pursued on the possibility a bifurcating effect undetectable by inspection of frequency plots may manifest in the context of the analysis. However, based on these histograms, the bifurcation term was expected to be non-significant and the CCM inappropriate for the data.

Figure 2. Category proportion of T1 personal smoking by peer pressure for males.

Figure 3. Category proportion of T1 personal smoking by peer pressure for females.

Model comparison

The signs associated with the regression weights for the standard linear model and CCM indicated the direction of change:

- a negative score on the DV represented a decrease in smoking behaviour;
 - no change was represented by scores close to zero; and
 - a positive score an increase in smoking behaviour.
- The pre-post linear model predicted level of smoking (e.g. 0 = non-smoking, 1 = weekly smoking, etc).

Males

In terms of the first hypothesis, the non-significance of the cubed-term meant the CCM was an inappropriate way of modelling the data (see Table 4). In terms of the second

Table 3. Changes in male and female analysis group smoking over the 12-month measurement interval

T1	0	1-7	8-35	36-70	71-105	106-140	141+	Total
Males								
T2								
0	288	16	9	4	1	1	0	319
1-7	5	2	3	1	0	0	0	11
8-35	2	1	4	2	0	0	0	9
36-70	1	0	0	0	0	0	0	1
71-105	0	0	0	0	0	0	0	0
106-140	1	0	0	0	0	0	0	1
141+	0	0	0	1	0	0	0	1
Total	297	19	16	8	1	1	0	342
Females								
T2								
0	379	17	8	4	0	1	0	409
1-7	5	7	5	2	0	1	0	20
8-35	1	0	3	0	0	1	0	5
36-70	0	1	1	2	0	0	0	4
71-105	0	1	0	0	0	0	0	1
106-140	0	0	0	0	0	0	0	0
141+	0	0	0	0	0	0	0	0
Total	385	26	17	8	0	3	0	439

Table 4. Regression coefficient confidence intervals (95%), beta scores, *F*-scores and adjusted-*R*² for the male standard linear, pre-post linear and cusp catastrophe models (statistical significance in italics)

Variable	Standard linear		Pre-post linear		Cusp catastrophe	
	95% CI	Beta	95% CI	Beta	95% CI	Beta
Constant	(0.051, 2.333)	—	(-0.537, 1.319)	—	(-0.627, 1.228)	—
Parental smoking	(-0.198, 0.338)	0.027	(-0.060, 0.373)	0.071	(-0.085, 0.350)	0.052
Sibling smoking	(-0.398, 0.257)	-0.024	(-0.090, 0.443)	0.068	(-0.130, 0.411)	0.047
Male best friend smoking	(-0.619, 0.182)	-0.066	(0.109, 0.783)	0.155	(0.111, 0.755)	0.133
Female best friend smoking	(-0.764, 0.107)	-0.088	(-0.398, 0.310)	-0.014	(-0.305, 0.400)	0.013
Self-esteem	(-0.066, -0.008)	-0.161	(-0.033, 0.0158)	-0.046	(-0.033, 0.015)	-0.038
Neuroticism	(-0.030, 0.029)	-0.002	(-0.014, 0.033)	0.048	(-0.010, 0.037)	0.059
Coping (GHQ)	(-0.162, -0.023)	-0.180	(-0.083, 0.031)	-0.058	(-0.083, 0.033)	-0.049
Stress	(-0.010, 0.003)	-0.074	(-0.010, 0.001)	-0.124	(-0.010, 0.001)	-0.116
Risk taking perception	(-0.004, 0.361)	0.111	(-0.147, 0.152)	0.002	(-0.109, 0.191)	0.025
Risk taking frequency	(0.017, 0.490)	0.143	(0.180, 0.563)	0.239	(0.167, 0.545)	0.198
Peer pressure	(-0.249, 0.106)	-0.052	(-0.140, 0.148)	0.004	—	—
T1 smoking	—	—	(0.101, 0.333)	0.219	—	—
Bifurcation term (PP)	—	—	—	—	(-0.315, -0.180)	-0.750
Z-cubed	—	—	—	—	(-0.002, 0.004)	0.083
<i>F</i>	2.343	—	7.369	—	17.939	—
<i>df</i>	11, 328	—	12, 327	—	12, 329	—
Adj- <i>R</i> ²	0.042	—	0.184	—	0.373	—

hypothesis, the data was best characterized by the failed CCM (37.3%), explaining almost nine times as much variance as the standard linear model (4.2%) and twice that of the pre-post linear model (18.4%).

Model composition varied only slightly (see Table 4). The standard linear model started all cases towards increased smoking, which amplified with involvement in risk taking. Higher self-esteem predicted decreased smoking. The pre-post linear model predicted T2 smoking on the basis of previous (T1) smoking, the frequency of risk taking behaviour and whether the male best friend also smoked. The absence of these predicted T2 non-smoking. The failed CCM predicted increases in smoking behaviour using the presence of a male best friend smoker and involvement in risk taking, balanced by the bifurcation term. The bifurcation term predicted decreases in smoking when high peer pressure was apparent, previous smoking or both. Where a respondent reported T1 non-smoking or few friends who smoked (low peer pressure), there was little influence on predicted smoking behaviour.

The expected commonality across the models predicting change in male smoking was found. Frequency of risk taking behaviour achieved statistical significance across all three models. The pre-post model and failed CCM both included best male friend smoking and the influence of previous smoking. The key difference between the latter models was the bifurcation parameter (β_7), which helped the failed CCM to explain twice the variance relative to the pre-post model.

Females

The CCM met the criteria to be a meaningful representation of the data (both the cubed and the bifurcation terms were non-zero) and explained the most variance (31.8%; see Table 5). The pre-post linear model explained marginally less variance (28.8%), while both the pre-post model and the CCM explained notably more than the standard linear model (4.4%).

The standard linear model used only two terms to explain changes in female smoking (see Table 5). At least one parent smoking predicted acceleration in smoking behaviour, while increased peer pressure to smoke decelerated smoking behaviour. The pre-post linear model adjusted all cases towards T2 weekly smoking and then predicted movement up the categories based on the presence of at least one parent who smoked and T respondent 1 smoking (see Table 5). The CCM terms both pushed cases towards decreased smoking behaviour with a greater level of smoking at T1 (the cubed-term) predicting a more substantial change, whereas T1 non-smoking predicted no change. The bifurcation term predicted that smoking would decrease in the presence of peer pressure, higher T1 smoking or both. T1 non-smoking or few friends who smoked again indicated behavioural maintenance. Finally, the effect of at least one parent smoking predicted an increase in smoking behaviour for the CCM (see Table 5).

The commonality across the models predicting change in female smoking centred on the presence of a parent who smoked and some combination of peer pressure, previous smoking or their combination in the bifurcation parameter.

Summary

In relation to the second hypothesis, there was no consistent evidence that the CCM was the better explanation of the data. The test of the third hypothesis indicated commonality in the composition of α across models. The results suggested previous

Table 5. Regression coefficient confidence intervals (95%), beta scores, *F*-scores and adjusted-*R*² for the female standard linear, pre-post linear and cusp catastrophe models (statistical significance in italics)

Variable	Standard linear		Pre-post linear		Cusp catastrophe	
	95% CI	Beta	95% CI	Beta	95% CI	Beta
Constant	(-0.124, 1.881)	-	(0.001, 1.752)	-	(-0.260, 1.375)	-
Parental smoking	(0.030, 0.446)	0.107	(0.054, 0.417)	0.105	(0.027, 0.378)	0.091
Sibling smoking	(-0.249, 0.225)	-0.005	(-0.149, 0.265)	0.023	(-0.099, 0.303)	0.042
Male best friend smoking	(-0.423, 0.152)	-0.049	(-0.144, 0.365)	0.039	(-0.162, 0.319)	0.028
Female best friend smoking	(-0.524, 0.088)	-0.074	(-0.260, 0.280)	0.003	(-0.458, 0.059)	-0.068
Self-esteem	(-0.036, 0.013)	-0.057	(-0.036, 0.007)	-0.068	(-0.032, 0.009)	-0.057
Neuroticism	(-0.028, 0.018)	-0.027	(-0.029, 0.011)	-0.047	(-0.029, 0.010)	-0.051
Coping (GHQ)	(-0.003, 0.079)	0.109	(-0.014, 0.058)	0.062	(-0.022, 0.047)	0.036
Stress	(-0.005, 0.005)	-0.007	(-0.002, 0.006)	0.055	(-0.002, 0.007)	0.064
Risk taking perception	(-0.199, 0.136)	-0.018	(-0.284, 0.010)	-0.076	(-0.256, 0.028)	-0.064
Risk taking frequency	(-0.179, 0.196)	0.005	(-0.029, 0.301)	0.071	(-0.057, 0.260)	0.054
Peer pressure	(-0.377, -0.086)	-0.172	(-0.255, 0.002)	-0.093	-	-
T1 smoking	-	-	(0.380, 0.560)	0.470	-	-
Bifurcation term (PP)	-	-	-	-	(-0.139, -0.024)	-0.266
Z-cubed	-	-	-	-	(-0.011, -0.003)	-0.335
<i>F</i>	2.833		15.786		18.034	
<i>df</i>	11, 427		12, 426		12, 426	
Adj- <i>R</i> ²	0.044		0.288		0.318	

smoking (i.e. initial conditions) and peer pressure have a role to play across the genders. The genders differed on the influence of significant others on respondent smoking, with males influenced by their male best friend smoking and females by parental smoking behaviour.

Discussion

This study sought to provide evidence on the variation in predictors of change in adolescent smoking behaviour across three analytic models. The results indicated little variability in model composition. The results also indicated that while the standard linear approach was relatively ineffective, the CCM failed to be unambiguously effective. This finding may have been influenced by the demographic characteristics of those analysed being younger non-smokers taking up smoking. For example, the power of the CCM may have been diminished by too little bidirectional change (e.g. uptake and quitting behaviour). Taking this into account, the implications of the general result included justified confidence in the predictors of change in adolescent smoking and that more work is needed to identify the mathematical description of the processes underlying change in adolescent smoking behaviour.

The striking outcome of the study was the stability in the composition of *a* across analytic models. For males, the frequency of risk taking behaviour was an effective predictor of change in smoking across all three models and a male best friend who was a smoker in the pre-post linear model and failed CCM analyses. For females, the key predictor was whether at least one parent was a smoker. These results suggested that the variables identified by any model used to analyse change in adolescent smoking were likely to be robust across models. Therefore, intervention and prevention programmes based on results from simple models of change in adolescent smoking are likely to be targeting psychological variables that have some influence on the phenomenon.

Where the models varied was in their explanatory power, examined as part of the second hypothesis. The standard linear model performed poorly relative to the other models. If model composition (at least in terms of *a*) is static across analytic method, this suggested that the improvement in explanatory power was due to the underlying analytic model. The implication of this result was that the standard linear model missed some aspect of the underlying variance more effectively captured by the other models. That is, the inclusion of some combination of peer pressure and simple or polynomial forms of T1 smoking significantly enhanced the prediction of change in adolescent smoking behaviour.

The common feature of the pre-post linear model and CCM was T1 smoking. That is, simple or polynomial forms of past smoking behaviour were central to explaining a relatively larger proportion of smoking behaviour variance. The predictive relationship between the past and the subsequent behaviour has been a sticking point in the literature for some time. While empirically robust (Norman & Conner, 1996; Steptoe, Sanderman, & Wardle, 1995), past behaviour has been declared theoretically suspect on the basis it can never 'cause' subsequent behaviour (Ajzen, 1991; Connor & Armitage, 1998; Ouellette & Wood, 1998). This can be resolved to an extent by reinterpreting past behaviour. Dynamical models such as the CCM use past behaviour as the point from which behaviour departs or remains consistent (e.g. Boker & Graham, 1998; Byrne *et al.*, 2001). That is, looking at past behaviour with an eye to causality may be the wrong way

to interpret its role in predicting later behaviour. Even though the CCM failed to be an unambiguously better explanation of the data (in terms of the first and second hypotheses), the principle of including simple, interaction (in this case the bifurcation parameter) or polynomial forms of past behaviour in analytic models may lead to more effective explanations of change in adolescent smoking and perhaps other health behaviours.

The failure of the CCM may have been due to the demographic characteristics of the data. The key result was that more non-smokers than smokers were available for analysis. Apart from the usual caveats of generalizability this result brings, there was a specific effect for models based on the description of bidirectional change between two stable states. According to Table 3, the data largely represented change in one direction (acceleration) representing the behaviour of non-smokers transiting to smokers. This may indicate fold degeneracy, where the threshold of change (catastrophe point) for non-smokers is more influential than that of smokers.

One way of addressing fold degeneracy is to include a quadratic term (z_{i-1}^2) as a correction for location (Guastello, 1982). *Post hoc* forward stepwise regression analysis (step 1 z_{i-1}^3 , $S_{PP}z_{i-1}$; step 2 z_{i-1}^2 ; step 3 *a*) on the male data saw the cubed term gain and the bifurcating term lose statistical significance (step 1 to step 2), which reversed on step 3. The quadratic term rendered all cusp-catastrophe terms non-significant for the female data, including the quadratic term. This is consistent with Byrne *et al.* (2001) report that the *exclusion* of the quadratic term saw either the cubed or bifurcating term become significant, resulting in the CCM being appropriate to explain the data. The quadratic term may be taking variance away from the more important cusp-catastrophe terms (see Darlington's, 1990, discussion on complementarity, pp. 57–58), making its inclusion inappropriate in the current context (Guastello, personal communication). At a broader level, it may be inappropriate to include a correction for fold degeneracy in CCMs of adolescent smoking. However, such a claim requires more in-depth mathematical and empirical investigation than can be achieved here.

Another way of approaching the fold degeneracy issue may be to analyse non-smokers and smokers separately. A basic fold model (Fararo, 1978) could be used for non-smokers to capture their acceleration and a CCM for smokers to capture movement in both the directions. Such a division in the current data would render the analysis meaningless for smokers given their relatively lower proportional representation in the sample. As such, using separate models for smokers and non-smokers represents a potentially useful extension to the current work.

The generalizability of the results was limited by the characteristics of the follow-up sample relative to those who attrited from the study. The follow-up rate is unusually low, indicating alternative tracking methods may be needed. Despite the low follow-up rate, the differences between the follow-up and attrition samples (in terms of age and smoking status) were of similar direction and magnitude seen in comparable longitudinal studies of adolescent smoking (see Results). It is important to note that older adolescent smokers still comprised a part of the sample and therefore reflected in the results. With this in mind, the results should be taken as indicative towards informing replication or extension of the current work rather than a robust statement about the nature of reality.

Future work in this area might do well to collect and model data based on smoking status to overcome the problems of demography experienced in the current study. The first step would be to test whether a basic fold model of non-smoker uptake of smoking was an effective representation of the data. The next step would be to collect enough

longitudinal data from smokers to analyse their transitions using the CCM to determine whether it was an effective representation of how smokers' behaviour changed. This may require concentrated research rather than a general survey of adolescent smoking behaviour where the emergent characteristics of the sample often mean smokers are under-represented (Mazanov & Byrne, 2002, p. 195). Following the methods outlined in this paper it would mean collecting and analysing data across gender and smoking status to yield four separate models.

The current paper provided indicative evidence that gives some confidence in the variables identified as having significant influence on changes in adolescent smoking behaviour. The consequence is improved confidence in intervention and prevention programmes based on such variables. While models using past behaviour appeared to be a better way of describing changes in adolescent smoking, more work needs to be done to ratify what sort of dynamical model is the most effective. With this ratification comes an understanding of the process underlying adolescent smoking by identifying the mathematical relationship between the variables. Such identification would then yield greater sensitivity to 'what to do when' rather than relying on trying to remove the (linear) influence of one variable to (hopefully) compel an adolescent non-smoker to remain so or a smoker to quit.

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References

- Ajzen, I. (1991). The theory of planned behaviour. *Organisational Behaviour and Human Decision Processes*, 50, 179-211.
- Alexander, C., Piazza, M., Mekos, D., & Valente, T. (2001). Peers, schools, and adolescent cigarette smoking. *Journal of Adolescent Health*, 29(1), 22-30.
- Alexander, R. A., Herbert, G. R., DeShon, R. P., & Hanges, P. J. (1992). An examination of least squares regression modelling of Catastrophe theory. *Psychological Bulletin*, 111(2), 366-374.
- Arnol'd, V. I. (1991). Catastrophe Theory. In V. I. Arnol'd (Ed.), *Dynamical Systems V*. Springer-Verlag: Berlin. Chapter II (pp. 207-259).
- Biglan, A., Duncan, T. E., Ary, D. A., & Smolkowski, K. (1995). Peer and parental influences on adolescent tobacco use. *Journal of Behavioural Medicine*, 18(4), 315-330.
- Boker, S. M., & Graham, J. (1998). A dynamical systems analysis of adolescent substance abuse. *Multivariate Behavioural Research*, 33(4), 479-507.
- Byrne, D. G., Byrne, A. E., & Reinhart, M. I. (1993). Psychosocial correlates of adolescent cigarette smoking: Personality or environment. *Australian Journal of Psychology*, 45(2), 87-95.
- Byrne, D. G., Byrne, A. E., & Reinhart, M. I. (1995). Personality, stress and the decision to commence cigarette smoking in adolescence. *Journal of Psychosomatic Research*, 39, 53-62.
- Byrne, D. G., & Mazanov, J. (1999). Sources of adolescent stress, smoking and the use of other drugs. *Stress Medicine*, 15, 215-227.
- Byrne, D. G., & Mazanov, J. (2001). Self-esteem, stress and cigarette smoking in adolescents. *Stress and Health*, 17, 105-110.
- Byrne, D. G., & Mazanov, J. (2002). Sources of stress in Australian adolescents: Factor structure and stability over time. *Stress and Health*, 18, 185-192.

- Byrne, D. G., & Mazanov, J. (2003). Adolescent stress and future smoking behaviour: A prospective investigation. *Journal of Psychosomatic Research*, 54, 313-321.
- Byrne, D. G., Mazanov, J., & Gregson, R. A. M. (2001). A cusp catastrophe model (CCM) analysis of changes to adolescent smoking behaviour in response to smoking prevention programs. *Nonlinear Dynamics, Psychology and Life Sciences*, 5(2), 115-137.
- Chassin, L., Presson, C. C., Sherman, S. J., Corty, E., & Olshavsky, R. W. (1984). Predicting the onset of cigarette smoking in adolescents: A longitudinal study. *Journal of Applied Social Psychology*, 14(3), 224-243.
- Choi, W. S., Gilpin, E. A., Farkas, A. J., & Pierce, J. P. (2001). Determining the probability of future smoking among adolescents. *Addiction*, 96, 313-323.
- Clair, S. (1998). A cusp catastrophe model for adolescent alcohol use: An empirical test. *Nonlinear Dynamics, Psychology and the Life Sciences*, 2, 217-241.
- Cobb, L. (1981). Parameter estimation for the cusp catastrophe model. *Behavioral Science*, 26(1), 75-78.
- Commonwealth of Australia (2002). *Australian federal privacy act (1988). Act No. 118 of 1988, incorporating the changes of Act No. 125 of 2002*. Canberra: Commonwealth Government Printers.
- Connor, M., & Armitage, C. J. (1998). Extending the theory of planned behaviour: A review and avenues for further research. *Journal of Applied Social Psychology*, 28(15), 1429-1464.
- Darlington, R. B. (1990). *Regression and linear models*. New York: McGraw-Hill.
- De Vries, H., Engels, R., Kremers, S., Wetzels, J., & Mudde, A. (2003). Parents' and friends' smoking status as predictors of smoking onset: Finding from six European countries. *Health Education Research*, 18(5), 627-636.
- Dugan, S., Lloyd, B., & Lucas, K. (1999). Stress and coping as determinants of adolescent smoking behaviour. *Journal of Applied Social Psychology*, 29(4), 870-888.
- Engels, R. C. M. E., Knibbe, R. A., & Drop, M. J. (1999). Predictability of smoking in adolescence: Between optimism and pessimism. *Addiction*, 94(1), 115-124.
- Eysenck, S. (1965). *Manual of the junior Eysenck personality questionnaire*. UK: Chigwell Press.
- Fararo, T. J. (1978). An introduction to catastrophes. *Behavioural Science*, 23, 291-317.
- Farkas, A. J., Distefan, J. M., Choi, W. S., Gilpin, E. A., & Pierce, J. P. (1999). Does parental smoking cessation discourage adolescent smoking? *Preventive Medicine*, 28, 213-218.
- Goldberg, D. P. (1972). *The Detection of Psychiatric Illness by Questionnaire*. Maudsley Monographs (no. 21). Oxford University Press: London, UK.
- Gonzalez, J., Field, T., Yando, R., Gonzalez, K., Lasko, D., & Bendel, D. (1994). Adolescents' perceptions of their risk-taking behaviour. *Adolescence*, 29(115), 701-709.
- Greening, L., & Dollinger, S. J. (1991). Adolescent smoking and perceived vulnerability to smoking related causes of death. *Journal of Paediatric Psychology*, 16(6), 687-699.
- Guastello, S. J. (1982). Moderator regression and the cusp catastrophe: Application of two-stage personnel selection, training, therapy and policy evaluation. *Behavioral Science*, 27(3), 259-272.
- Guastello, S. J. (1987). A butterfly catastrophe model of motivation in organisations: Academic performance. *Journal of Applied Psychology*, 72, 165-182.
- Guastello, S. J. (1988). Catastrophe modelling of the accident process: Organisational subunit size. *Psychological Bulletin*, 103(2), 246-255.
- Guastello, S. J. (1992). Clash of the paradigms: A critique of an examination of the polynomial regression technique for evaluating catastrophe theory hypotheses. *Psychological Bulletin*, 111(2), 375-379.
- Guastello, S. J., Gershon, R. R. M., & Murphy, L. R. (1999). Catastrophe model for the exposure to blood-borne pathogens and other accidents in health care settings. *Accident Analysis and Prevention*, 31, 739-749.
- Gullone, E., Moore, S., Moss, S., & Boyd, C. (2000). The Adolescent risk-taking questionnaire: Development and psychometric evaluation. *Journal of Adolescent Research*, 15(2), 231-250.

- Mazanov, J. (2003). *Linear versus nonlinear modelling of adolescent smoking behaviour: The role of behavioural intention*. Unpublished doctoral dissertation, Australian National University, Canberra.
- Mazanov, J., & Byrne, D. G. (2002). A comparison of predictors of the adolescent intention to smoke with adolescent current smoking using discriminant function analysis. *British Journal of Health Psychology*, 7(2), 185-201.
- Mazanov, J., & Byrne, D. G. (2006). A cusp catastrophe model analysis of changes in adolescent substance use: Assessment of behavioural intention as a bifurcation variable. *Nonlinear Dynamics, Psychology and the Life Sciences*, 10(4), 445-470.
- McGee, R., & Stanton, W. R. (1993). A longitudinal study of reasons for smoking in adolescence. *Addiction*, 88, 265-271.
- Norman, P., Conner, M. (1996). The role of social cognition models in predicting health behaviours: Future directions. In M. Conner and P. Norman (Eds.), *Predicting Health Behaviour: Research and Practice with Social Cognition Models* (Ch. 7). Buckingham, UK: Open University Press. (pp. 197-225).
- Ouellette, J. A., & Wood, W. (1998). Habit and intention in everyday life: The multiple processes by which past behaviour predicts future behaviour. *Psychological Bulletin*, 124(1), 54-74.
- Parker, S. B. (Ed.). (1997). *Dictionary of mathematics*. Sydney: McGraw-Hill.
- Pirie, P. L., Murray, D. M., & Luepker, R. V. (1988). Smoking prevalence in a cohort of adolescents, including absentees, dropouts and transfers. *American Journal of Public Health*, 78(2), 176-178.
- Ratkowsky, D. A. (1990). *Handbook of nonlinear regression models*. USA: Marcel Dekker Inc.
- Rogers, R. W. (1975). A protection motivation theory of fear appeals and attitude change. *Journal of Psychology*, 91, 93-114.
- Rosenberg, M. (1989). *Society and the adolescent self concept*. USA: Wesleyan University Press.
- Scott, D. W. (1985). Catastrophe theory applications in clinical psychology: A review. *Current Psychological Research and Reviews*, Spring, 69-86.
- Stephens, A., Sanderman, R., & Wardle, J. (1995). Stability and changes in health behaviour in young adults over a one-year period. *Psychology and Health*, 10, 155-169.
- Sussman, S., Dent, C. W., & Galaif, E. R. (1997). The correlates of substance abuse and dependence among adolescents at high risk for drug abuse. *Journal of Substance Abuse*, 9, 241-255.
- Thom, R. (1975). *Structural stability and morphogenesis*. New York: Benjamin-Addison-Wesley.
- Tyas, S. L., & Pederson, L. L. (1998). Psychosocial factors related to adolescent smoking: A critical review of the literature. *Tobacco Control*, 7, 409-420.
- Vaccaro, D., & Wills, T. A. (1998). Stress-coping factors in adolescent substance use: Test of ethnic and gender differences in samples of urban adolescents. *Journal of Drug Education*, 28(3), 257-280.
- Wakefield, J. A. (1989). Personality, health and cigarette smoking. *Personality and Individual Differences*, 10(5), 541-546.
- Wang, M. W., Fitzhugh, E. C., Westerfield, R. C., & Eddy, J. M. (1995). Family and peer influences on smoking behaviour among American adolescents: An age trend. *Journal of Adolescent Health*, 16, 200-203.