Assessing the Dynamics of Smoking and Smoking-Related Factors:
A Comparison between Cusp Catastrophe and Linear Models.

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Abstract

Although many health risk behaviours, such as smoking, might have fundamentally non-linear relationships, most theory and statistical models traditionally assume linear relationships. Accordingly, post-treatment smoking abstinence rates and our understanding of smoking dynamics, or more specifically the non-linear relationships between risk factors related to smoking, have improved little over the last few years. We suggested that non-linear methods could better describe such relationships between risk factors while providing valuable insight in the development of smoking behaviour. As such, we compared traditional linear models to the non-linear *cusp catastrophe* model. The aim was to model the discontinuities often observed in addiction data while taking smoker subgroups into account, and to investigate whether such non-linear models would outperform comparable linear models. Additionally, various smoking-related variables were evaluated as we aimed to determine their contribution to the model and their optimal specification. A dataset offering subgroup information about a Dutch-speaking sample of 510 hardcore smokers who had little to no intention to quit and 338 non-hardcore smokers, was re-analysed using the *cusp catastrophe model*. We investigated alternative cusp model specifications and compared them to linear models based on model goodness-of-fit indices. The results showed that the non-linear cusp models were superior descriptions of the observed data than linear models, and an optimal model specification was obtained. Finding such non-linear relationships between smoking behaviour and smoking-related factors supported that smoking behaviour does show both continuous and discontinuous transitions, with a number of smoking-related factors facilitating such transitions. The cusp catastrophe model is thus a useful tool to investigate discontinuities in subgroup smoking behaviour, and can provide valuable information about which factors could influence transitions in such behaviour.

Keywords: cusp catastrophe modeling, smoking behaviour, subgroup analysis, discontinuous transitions, quantitative analyses

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Smoking is the most important cause of preventable death worldwide, claiming about 6 million lives each year (World Health Organization, 2016). Smoking thus continues to be an important public health issue. Furthermore, despite years of research, post-treatment abstinence rates have barely improved over the years (e.g. Shiffman, 1993). One reason for such a lack of theoretical and practical improvements might be the traditionally assumed linear relationships for smoking behaviour and smoking-related factors, while these might in fact be fundamentally non-linear. Research designs and statistical approaches that have almost exclusively focused on static predictors of change only had limited success (Hayes et al., 2007). As such, in recent years more and more evidence is accumulated that conceptualising change as a non-linear process is more successful than seeing change as a mere linear process with static predictors. For example, non-linear methods can help predict transitions in otherwise complex and interwoven systems (Dai, Korolev, & Gore, 2013). In addition, such methods can help in providing early warnings of loss of resilience (Dai, Vorselen, Korolev, & Gore, 2012; Scheffer et al., 2009). Non-linear methods proved successful in predicting phase transitions in, for example, cognitive development (Jansen & van der Maas, 2001), attitudes (van der Maas, Kolstein, & van der Pligt, 2003), and e-commerce competition (Dou & Ghose 2006). Catastrophe models started out as being limited to purely deterministic systems, which made their use in sciences dealing with systems subject to random influences problematic. However, modern catastrophe models obviated these limitations by adding a stochastic white noise to the catastrophe equation while treating the resulting equation as a stochastic differential equation (Grasman, van der Maas, & Wagenmakers, 2009). Thus, catastrophe models are nowadays also available in the biological and social sciences, as they are now able to investigate systems influenced by random processes.

In addiction research, linear relations are still commonly assumed between smoking-related factors and smoking behaviour (Byrne, Mazanov, & Gregson, 2001). However, Clair (1998) showed that such linear models did not perform as well as non-linear models for alcohol consumption in adolescents, and such results were also found for smoking behaviour (Byrne et al., 2001). Hence, the application of non-linear modeling in addiction research remains a promising idea, which might lead to increased insight into the relationships between risk factors related to smoking and smoking itself.

Cusp Catastrophe Models

To improve understanding of smoking risk factors and their relations, we suggested to consider dynamical models as an alternative to linear models. One dynamical model, called the *cusp catastrophe model*, has shown promise in the field of addiction research. Guastello (1984) theorised the feasibility of catastrophe modeling for addiction processes, while Clair (1998) found that catastrophe models indeed predicted alcohol consumption better than alternative linear models. Similar studies reported catastrophe models to facilitate the prediction of alcohol relapses (Hufford, Witkiewitz, Shields, Kodya, & Caruso, 2003; Witkiewitz & Marlatt, 2007; Witkiewitz, van der Maas, Hufford, & Marlatt, 2007). Conceptually an addiction such as smoking often shows one of two stable states, present or absent. However a third uncommon state is also possible, which is intermediary and unstable, wherein the variables that predict smoking behaviour or smoking abstinence can have the same values for both states (Byrne et al., 2001). Cusp catastrophe models can naturally conceptualise these states as locations on a 3-dimensional cusp equation surface (see Figure 1) which accommodates the two stable and one unstable states of smoking addiction. The cusp surface can be seen from a regression perspective, as a response surface wherein the y-axis signals the value of the behavioural smoking variable given the values of two variables that control its behaviour (i.e., the cusp control variables on the α-axis and the β-axis). This surface has the interesting property that the behavioural variable can have two different outcomes instead of one, under certain conditions (Grasman et al., 2009).

[Figure 1 to be placed here]

Catastrophe theory facilitates modeling situations wherein a small continuous change in one variable causes a catastrophic change, or *bifurcation*, in another (e.g., Chen et al., 2014; van der Maas, Kolstein, & van der Pligt, 2003). The cusp model includes one behavioural variable and two control variables. These control variables are important as they can control behavioural phenomena that show sudden transitions, such as smoking addiction, without having to show sudden transitions themselves (Scott, 1985). These two control variables are called the *asymmetry* variable (the α-axis in Figure 1) and the *bifurcation* variable (the β-axis in Figure 1). The asymmetry variable predicts the direction of change in behaviour. The bifurcation variable, on the other hand, has a splitting influence on the behavioural variable. For low values of the bifurcation variable, the asymmetry variable is an ordinary linear predictor of the behavioural variable. However, as the bifurcation variable increases, the behavioural variable diverges and presents two possible outcomes for the same value of the control variables. These two outcomes that can be simultaneously predicted correspond with the two stable states, e.g., smoking numerous cigarettes and smoking only few cigarettes per day. For example, Byrne et al., (2001) showed that smoking attitudes linearly predict smoking behaviour under little or no peer pressure. However, when peer pressure increases smoking behaviour diverges and smoking attitudes predict both increases and decreases. Peer pressure therefore has a bifurcating effect on smoking behaviour, which suggests the presence of complex dynamical relations between smoking-related variables that may be better described with non-linear catastrophe models than with traditional linear models.

Recent research suggested a link between the cusp control variables, asymmetry and bifurcation, and two risk factors often encountered in addiction research, respectively proximal and distal risks (e.g., Hufford et al., 2003; Witkiewitz et al., 2007). *Proximal* risks are factors that immediately precipitate health risk behaviours, such as stress, emotional states, and self-efficacy. *Distal* risks, on the other hand, are factors that have a predisposing influence on health risk behaviours, such as age of onset, and family history. Hufford et al. (2003) performed a Principal Components Analysis (PCA) to reduce the hypothesised relapse risks into two orthogonal components, which where interpreted as representing proximal and distal risk factors. As this interpretation offered an intuitive and elegant link between health risk factors and the cusp control variables, while providing better fit than linear models (Hufford et al., 2003), other scholars followed a similar reasoning (Witkiewitz et al., 2007; Witkiewitz & Marlatt, 2007).

The advent of cusp modeling offered a number of advantages over linear models, such as its' independence from time. Cusp modeling can thus be used to model cross-sectional relationships while being able to detect and quantify potential cusp characteristics including both sudden and continuous states (Chen et al., 2014). Catastrophe models are also parsimonious (Gresov et al., 1993) because they capture complex behaviours with fewer nonlinear equations than the number of linear equations needed to describe the same phenomena (Oliva, Oliver, & MacMillan, 1992).

Smoking and Interventions

Epidemiological studies suggested various relationships between smoking-related risk factors. For example, age of smoking initiation is related to cigarette consumption, nicotine dependence, and smoking cessation (Breslau & Peterson, 1996; Breslau, Fenn, & Peterson, 1993). However, age and age of smoking onset are unchangeable variables and thus less suitable as intervention targets. These variables are the aforementioned *distal* factors, which are long-term risks that are difficult to change. *Proximal* factors are more contingent and changeable individual characteristics (Donovan, 1996), which may thus be easier to change through interventions. For example, the relative malleability of motivational factors has spurred motivational enhancement interventions to become one of the most popular smoking treatments (e.g., Curry, Mermelstein, & Sporer, 2009). Examples of smoking interventions that specifically target motivational components are: motivational interviewing (MI) and cognitive behavioral therapy (CBT). Motivational components also play an important role in theoretical models for smoking, such as the Trans Theoretical Model of change (TTM; Prochaska, DiClemente, & Norcross, 1992).

MI focuses on resolving ambivalence towards negative effects of smoking and increasing *quitting self-efficacy* through a supportive therapeutic style (Miller & Rollnick, 2002). Self-efficacy is the belief of a person that he/she has the potential to successfully accomplish a goal (Bandura, 1977). CBT also targets motivational components and seeks to improve self-efficacy and behavioral control processes by confronting and changing attitudes, thoughts, and affects (Nash et al., 2013; Webb et al., 2010). CBT is an evidence-based technique that emphasises the effects of thoughts and feelings on behaviour (Hollon & Beck, 2004). CBT displayed positive effects on motivational factors such as weight gain concerns, a concern that often motivates women to continue smoking (Perkins et al., 2001). TTM, on the other hand, is a theoretical framework often used for studying smoking, which also includes both motivational factors and self-efficacy. According to TTM, motivation to quit smoking is indicated by perceived pros and cons of quitting (decisional balance), which in turn would predict smoking cessation (Prochaska & DiClemente, 1982; Prochaska & DiClemente, 1983). Progression is hypothesised to be produced by, among others, changes in decisional balance and self-efficacy (Aveyard et al., 2009). In the TTM the pros and cons are seen as change mediators (Velicer et al., 1996).

While the aforementioned studies and intervention strategies elucidate the inner workings of smoking behaviour, their effectiveness is mixed. For example, the capabilities of TTM-based interventions have been questioned based on mixed treatment effectiveness (Aveyard et al., 2003; Aveyard et al., 2009). Additionally, while MI has positive proximal effects, studies on its effectiveness reported mixed results (Colby et al., 2012). One explanation for such mixed results is that motivational components, such as motivation to quit smoking, are not predictive of smoking abstinence (Ussher, Kakar, Hajek, & West, 2016). Similarly, some studies suggest that self-efficacy is also not predictive of smoking abstinence (e.g., Hughes & Naud, 2016).

However, previous studies on cusp catastrophe modeling of smoking behaviour have some noticeable drawbacks. For example, West and Sohal (2006) suggested catastrophe modeling to be appropriate for modeling tobacco addiction processes, however the conclusions based on their study are limited since they did not empirically test the catastrophe models. Another limitation of previous studies is the lack of consensus about the role of the included variables as either proximal, distal, or behavioural factors. For example, substance dependence is seen by some authors as a distal risk factor (Witkiewitz & Marlatt, 2007; Witkiewitz et al., 2007), while others consider it as a behavioural dependent variable (Cupertino et al., 2011).

This study

In the present study, we proposed to investigate the relationships among smoking risk factors with cusp catastrophe modeling. The goal was to elucidate why traditional linear models of smoking are unsuccessful in fully capturing the dynamics of smoking behaviour. In addition, we aimed to elucidate the role of smoking-related variables that are often key factors in smoking interventions, namely, quitting self-efficacy (Bandura, 1977), motivational components (Bommelé et al., 2015), and nicotine dependence (Cupertino et al., 2011; Witkiewitz et al., 2007; Witkiewitz & Marlatt, 2007). We primarily focused on how nicotine dependence is related to actual smoking behaviour, given the aforementioned ongoing debate on whether nicotine dependence should be regarded as either a distal or a behavioural factor.

To investigate the exact relation of nicotine dependence with smoking and smoking-related variables, two differing model specifications were compared based on model goodness-of-fit. The first model included quitting self-efficacy and pros and cons of smoking and quitting as proximal risk factors, age of smoking onset as distal risk factor, and nicotine dependence and average number of cigarettes per day were included as behavioral factors. The second model was identical to the first model, with the exception that nicotine dependence was considered a distal risk factor instead of a behavioral factor. Both cusp models were also compared to their respective linear version.

For our first hypothesis, we predicted the goodness-of-fit indices of the non-linear cusp models to be superior to their linear counterparts. Such a result would illustrate that non-linear modeling should indeed be preferred for the investigation of the relationships among smoking risk factors. For our second hypothesis, we predicted that the first model, wherein nicotine dependence was treated as a behavioural factor, offered better model fit than the second model, wherein nicotine dependence is seen as a distal risk factor. Additionally, we investigated in an exploratory manner whether other combinations of risk factors yielded comparable, or even better fitting, results than the aforementioned cusp and linear models.

While such investigations on a smoker sample might be elucidating, it is possible that subgroups within such a sample might differ widely on smoker characteristics, such as motivation to quit smoking (Bommelé et al., 2015). To account for such different smoker characteristics and to investigate differences between smoker subgroups, a large dataset identifying six smoker subgroups based on number of cigarettes smoked per day and accompanying motivational factors, was used for the analysis. For each smoker subgroup we investigated whether the cusp model obtained better goodness-of-fit indices than comparable linear models, while also investigating the role of nicotine dependence. With this subgroup analysis we aimed to investigate the differences between smoker subgroups in their resilience towards sudden transitions in smoking behaviour, and their patterns of relationships between smoking-related variables.

Method

Participants and Procedure

Bommelé et al. (2015) assessed a Dutch-speaking sample of 510 hardcore- and 338 non-hardcore smokers. While this sample only included Dutch-speaking participants, the participants can be seen as representative of smokers commonly found in developed countries as they share similar socioeconomic conditions. Participants were recruited through an online survey sample (Survey Sampling International) in 2012. As the goal of Bommelé and colleagues (2015) was to obtain a stratified sample, they pursued an equal representation of sex and socioeconomic status (SES). Two SES groups were distinguished based on participants' highest completed level of education. The low SES group had either primary education, lower secondary education (MAVO), or lower to middle level vocational education (LBO, MBO). The high SES group had either higher secondary education (HAVO, VWO) or tertiary education (HBO, University).

Participants were defined as 'hardcore' smokers when they: (1) were aged 35 or older, (2) smoked every day, (3) smoked on average 15 or more cigarettes a day, (4) had not attempted to quit smoking in the previous year, (5) had smoked at least 15 years, and (6) had no intention to quit within 6 months. Conversely, participants were defined as 'non-hardcore' when they: (1) were 35 or older, (2) smoked 'every day' or 'sometimes', (3) had no intention to quit within 6 months, and (4) did not meet all 'hardcore' smoker criteria. Bommelé et al. (2015) identified profiles among hardcore and non-hardcore smokers through two separate series of latent profile analysis (LPA). LPA is a person-oriented technique with which distinct homogeneous subgroups can be identified. Both theoretical and statistical methods, such as goodness-of-fit indices, were employed to determine the most parsimonious number of profiles for their data. Pros and cons of smoking and quitting were used as predictors, wherein participants' scores were used to identify six unique smoker profiles. Variables such as quitting self-efficacy and smoking history were used as covariates. The non-hardcore smoker group included three unique profiles (receptive-, ambivalent-, and disengaged smokers), while the hardcore smoker group also included three profiles (receptive-, ambivalent-, and resistant smokers).

Materials

The dataset of Bommelé and colleagues (2015) contains nine variables of interest: *average number of cigarettes a day, nicotine dependence, quitting self-efficacy, age, age of smoking onset, pros of smoking, cons of smoking, pros of quitting,* and *cons of quitting*. Average number of cigarettes a day was evaluated through a single categorical item asking participants: “On average, how many cigarettes do you smoke per day?” (i.e. 10 or less; 11–20; 21–30; 31 or more). Nicotine dependence was assessed using the Dutch version of the Fagerström Test for Nicotine Dependence (Vink, Willemsen, Beem, & Boomsma, 2005). This test contains six items assessing: number of smoked cigarettes per day, difficulty to abstain from smoking, and time to first cigarette after waking up. The reliability of this measurement instrument showed Cronbach’s α values of .65 for male smokers, .69 for female smokers, .66 for male ex-smokers, and .71 for female ex-smokers. Quitting self-efficacy was evaluated with 16 items on a 7-point Likert scale (α = .95), which measured the perceived ability to abstain from smoking after a hypothetical quitting attempt (Dijkstra, & Vries, 2000b). The four remaining scales assessed the pros and cons of smoking and quitting (Bommelé et al., 2014), on a 7-point Likert scale. For each scale participants indicated their agreement to 16 statements, which assessed various smoking-related topics, such as health, money, and social environment. Internal consistencies of these scales are the following: pros of smoking (α = .81), cons of smoking (α = .85), pros of quitting (α = .89), and cons of quitting (α = .79). Reliability of all scales was found to be acceptable.

Data analysis

Prior to the analysis, one observation was excluded as the participant reported to have started smoking at age zero. No other outliers were identified, thus the final sample size included 847 participants. A common approach to evaluate the applicability of catastrophe models to a measured or observed behaviour is quantitative in nature and involves restructuring the cusp model into probabilistic terms (Witkiewitz & Marlatt, 2007). Our data analysis consisted of different stages: model specification, model fitting, evaluation of goodness-of-fit indices, evaluation of model parameters, and evaluation of the cusp surface plots. These analytical steps were taken for each smoker subgroup. Hence, the research questions were also tested for each smoker subgroup.

To investigate the exact relation of nicotine dependence with smoking and smoking-related variables, we specified two competing models. In Model 1, self-efficacy and pros and cons of smoking and of quitting were considered as proximal risk factors, age of smoking onset was added as a distal risk factor, and nicotine dependence and average number of cigarettes per day were included as behavioral factors. The competing Model 2 was identical except for the inclusion of nicotine dependence as an additional distal risk factor instead of a behavioural variable. To investigate model fit, the cusp models and the respective linear versions were compared to each other based on AIC, BIC, and log-likelihood indices, with lower Information Criteria (IC's) and higher log-likelihood indicating a better fitting model. After identifying the best fitting model, the model parameters were inspected and evaluated on whether significant asymmetry, bifuraction, and dependent variable coefficients were obtained which indicated the presence of a complete catastrophe model. Thereafter, the cusp surface plots were inspected which provided additional information on the proximity of the data to a transition point. The *cusp* package for R was used for the analyses (Grasman et al., 2009).

Results

<u>Descriptive statistics</u>

Means, standard deviations, and ranges of the variables are presented in Table 1. The correlations among all the variables are presented in Table 2. Most of the reported correlations were statistically significant ($p < 0.05$). However, age of smoking onset formed a notable exception as it did not significantly correlate with any of the pros and cons of smoking and quitting. Furthermore, the correlations between pros of quitting and cons of smoking, cons of quitting and pros of smoking, and between number of cigarettes per day and nicotine dependence, were all strong and positive. Probability density functions of all the variables for each subgroup were obtained for general overview purposes and are presented in Figure A in the supplementary materials. These plots showed the distributions of the variables and included means, modes, and medians. Additionally, they indicate both skewness and possible multi-modality as, for example, in the number of cigarettes smoked per day.

[Tables 1 and 2 to be placed here]

<u>Confirmatory analyses</u>

<u>1. Model fitting</u>

The results of the model fitting procedure for Model 1 (nicotine dependence as dependent variable) versus Model 2 (nicotine dependence as bifurcation factor) are shown in Table 3. In all subgroups, Model 2 unequivocally outperformed Model 1, based on higher log-likelihood values and lower AIC and BIC values. This result indicated that nicotine dependence should be conceptualised as a bifurcation variable and not as a dependent variable. In addition, the result showed that the goodness-of-fit indices of the cusp models were superior to those of the linear models, indicating that a cusp catastrophe model best fitted all observed subgroup data. Identical goodness of fit results were obtained for the sample as a whole (see Table A in the supplementary materials), although the current subgroup analysis was seen as more informative as in the whole sample, subgroup differences could balance each other out.

[Table 3 to be placed here]

<u>2. Model parameter estimates</u>

Table 4 shows the coefficients of cusp Model 2 for all subgroups. In all of them, two coefficients always showed up as statistically significant: the bifurcation coefficient for nicotine dependence and the coefficient of the dependent variable number of cigarettes per day. For disengaged non-hardcore smokers, these two coefficients were the only two that were significant, while this group additionally showed lower estimates for number of cigarettes per day when compared to the other groups.

However, for the five remaining subgroups the coefficients for the intercepts of the bifurcation factor and the dependent variable were also found to be significant. Only in the receptive non-hardcore smoker group a significant asymmetry coefficient was found, namely cons of quitting. Although the goodness-of-fit indices in Table 3 indicated that the presence of a catastrophe is very likely, the model coefficients in Table 4 indicated that only for receptive non-hardcore smoker group a complete cusp model was obtained. This is because this group was the only one yielding significant coefficients for asymmetry (α), bifurcation (β), and dependent variables (w), with this model specification.

[Table 4 to be placed here]

3. Cusp surface plots

Figure 2 shows the cusp control surfaces and the plotted data for all the subgroups for Model 2. As can be seen from this figure, for all groups except the disengaged non-hardcore smokers, a portion of the data points lies within the bifurcation region. This demonstrated that most of the subgroups included individuals vulnerable to transitions between few and numerous cigarettes per day, while only the disengaged non-hardcore smokers showed very stable smoking behaviour.

Additionally, the differences in the influence of the proximal risk factors in the subgroups can be observed as the difference in width of the set of data points. The set of data points of the receptive non-hardcore smoker group, for example, is far wider than that of the receptive hardcore smoker group, indicating a larger influence of the proximal risk factors in this subgroup. This is in line with the model coefficients (Table 4), which showed that the receptive non-hardcore smokers were influenced by one significant proximal / asymmetry risk factor (cons of quitting), whilst the hardcore smokers were not influenced by such proximal risk factors (i.e., pros of quitting, quitting self-efficacy).

The disengaged non-hardcore smoker group performed similarly with regard to the distal risk factor nicotine dependence. Interestingly, the set of data points of this group was not located in the bifurcation region. This corresponds to the positive parameter coefficient for nicotine dependence and non-significant bifurcation intercept. For all other subgroups the coefficient for nicotine dependence has a negative value and significant bifurcation intercept.

[Figure 2 to be placed here]

Exploratory analyses

1. Model fitting

Alternative model specifications were investigated to see whether separating the two bifurcation variables, i.e., nicotine dependence and age of smoking onset, would yield more complete cusp models, and helped to clarify their role in the model. The results of the model fitting procedure for Model 3 (only nicotine dependence as bifurcation variable) versus Model 4 (only age of onset as bifurcation variable) are shown in Table 5. Model 3 unequivocally outperformed Model 4 in all subgroups, based on superior goodness-of-fit indices. This result showed that nicotine dependence surpassed age of onset as a bifurcation variable. Such a result seemed indeed conceivable given the stronger correlation between nicotine dependence and cigarettes per day than age of smoking onset and cigarettes per day (see Table 2). In line with the confirmatory model fitting procedure, the cusp models again showed better fit measures than the linear counterpart, indicating that a cusp catastrophe best fitted the observed data.

However, such results questioned whether cusp Model 3 performed better than previous cusp Model 2. The difference between the two models was that Model 2 included both nicotine dependence and age of smoking onset as bifurcation variables, while Model 3 only included nicotine dependence as a bifurcation variable. The results of comparing the goodness-of-fit of these models is shown in Table 6, which shows very minute differences. However, Model 3 should be preferred based on model parsimony.

[Tables 5 and 6 to be placed here]

2. Model parameter estimates

The model parameter estimates of Model 3 are shown in Table 7. Even though Model 3 offered better fit and parsimony than Model 2, this did not decidedly improve the model coefficients. These coefficients largely mimicked those of Model 2 as shown in Table 4. Only the ambivalent non-hardcore smoker group improved in that it provided a complete model wherein significant coefficients for asymmetry- (α), bifurcation- (β), and dependent variables (w) were found.

[Table 7 to be placed here]

3. Cusp surface plots

Figure 3 shows the cusp control surfaces for all subgroups with their respective data points for Model 3. The results shown in this figure mirrored those of Figure 2 regarding Model 2. This was a plausible finding as the exclusion of the age of onset variable did not make large differences in model and coefficient performance. Again, for all groups except the disengaged non-hardcore smokers a number of data points lied within the bifurcation region. These represented individuals vulnerable to transitions between few and numerous cigarettes per day. Similarly, only the disengaged non-hardcore smokers showed stable behaviour.

[Figure 3 to be placed here]

Discussion

The results from this study show that non-linear cusp models outperform comparable linear models when describing the relationships between smoking risk factors. These findings indicate that the linear assumptions underlying much of current research on smoking behaviour are not necessarily the best description for observed smoker behaviour. Indeed, such results do offer support for earlier findings that non-linear models may be better suited for modeling addiction processes than linear models in, for example, alcohol consumption (Clair, 1998) and smoking behaviour (Byrne et al., 2001).

Furthermore, the differentiation along smoker subgroups revealed that in all of the subgroups nicotine dependence should be regarded as a distal risk factor instead of a behavioral factor. This finding supported the notion that, despite the intuitive overlap between nicotine dependence and number of cigarettes per day, nicotine dependence is indeed best considered as a distal risk factor (Witkiewitz et al., 2007; Witkiewitz & Marlatt, 2007). This finding stands in contrast with other scholars who consider it as a behavioural dependent variable (Cupertino et al., 2011). However, nicotine dependence and number of smoked cigarettes are not the same. There are, for example, conceivable situations wherein one goes without the other, e.g., a beginning smoker that does smoke but does not yet experience symptoms of nicotine dependence, or a smoker that quit smoking but is still experiencing symptoms of nicotine dependence. Hence, conceptualising nicotine dependence as a risk factor associated with smoking instead of a behavioural outcome almost identical to smoking does make sense, while it also renders nicotine dependence a possible target for interventions.

Additionally, an exploratory analysis further elucidated the specification of the bifurcation variable, wherein the exclusion of age of smoking onset produced a more parsimonious model. The finding that age of smoking onset did not substantially contribute to the model stands in contrast with earlier studies that suggested that age of smoking initiation was related to cigarette consumption (Breslau, Fenn, & Peterson, 1993). However, when treated as a distal risk factor in the cusp model, age of smoking initiation did not significantly contribute to the probability of sudden transitions between alternate smoking states.

This study illustrates the need to investigate the relationships between smoking risk factors through non-linear modeling. However, only for some smoker subgroups a complete cusp model was found wherein significant coefficients for proximal risk factors (asymmetry; α), distal risk factors (bifurcation; β), and dependent variables (w) were obtained. Such a result suggests that the current model specification can be further improved. In most subgroups a lack of significant proximal risk factors was found. Hence, the decisional balance among perceived pros and cons of smoking and quitting, which affects motivation to quit smoking (Prochaska & DiClemente, 1982; Prochaska & DiClemente, 1983), did not significantly alter how close the subgroups were to a sudden discontinuous change in smoking behaviour. However, the obtained relations between these motivational factors were plausible as related motivational factors did complement each other (e.g., strong positive correlations between pros of quitting and cons of smoking, and cons of quitting and pros of smoking). Improvements could however be found in an alternative specification of the included proximal risk factors. While the current inclusion of both motivational factors and self-efficacy did not produce unequivocal results for each subgroup, other factors such as: stress (e.g., Cui, Rockett, Yang, & Cao, 2012), mood (e.g., Weinstein & Mermelstein, 2013), and/or genetic factors (e.g., Wolock et al., 2013) , might be a substantial addition to the model and allow for a more complete cusp model.

Furthermore, the finding that motivational factors did not significantly contribute to transitions in number of smoked cigarettes per day stands in stark contrast with its prevalence in current smoking interventions and theories. For example, TTM theory hypothesises pros and cons of smoking and quitting to be mediators of change (Velicer et al., 1996) and indicators of progression in smoking behaviour (Aveyard et al., 2009). As such a result was not obtained in the current study, it is possible that the role of these factors differs from the current specification. For example, it could be that these factors should alternatively be regarded as having a bifurcating effect on smoking behaviour. On the other hand, it might be that these results are indicative of the reported mixed treatment effectiveness of TTM-based interventions (Aveyard et al., 2003; Aveyard et al., 2009).

Specific interventions that place an emphasis on the role of motivational factors in smoking behaviour are Motivational Interviewing (MI) and Cognitive Behavioral Therapy (CBT) (Miller & Rollnick, 2002; Nash et al., 2013; Webb et al., 2010). Since in spite of years of research post-treatment abstinence rates have barely improved over the years (e.g. Shiffman, 1993), and intervention effectiveness of MI is mixed (Colby et al., 2012), it is possible that motivational factors play less of a role than previously expected. Our results could indeed not support the role of motivational factors in smoking behavioral change and such results do offer partial support for suggestions that motivational factors are not at all predictive of change in smoking behaviour (Ussher, Kakar, Hajek, & West, 2016). However, our results only offer partial support for such arguments as it only investigated the role of motivational factors as asymmetry factors, and it is still possible that motivational factors would perform well when regarded as bifurcation factors.

Nonetheless, this work did shed some light on interesting differences between smoker subgroups in their resilience towards sudden transitions in smoking behaviour. All but the disengaged non-hardcore smokers showed at least some openness to change, signaled by a number of datapoints lying in the bifurcation region. This finding indicated that these subgroups could be effectively and timely targeted for smoking interventions, as these subgroups are in a moment where they are close to a tipping point between two opposing behavioural states and are thus susceptible to sensible manipulations, such as interventions. The disengaged non-hardcore smoker groups, however, did show very stable behaviour that cannot be easily changed. Such a result is in line with findings that this subgroup is uniquely uninvolved in both smoking and quitting (Bommelé et al., 2015), a result not reproduced in any of the other subgroups. Furthermore, as Bommelé and colleagues (2015) argued, the low tobacco consumption and nicotine dependence of this group makes quitting less urgent and the disadvantages of smoking less visible. This notion is also supported by our finding that this groups displays stable behaviour and can be hard to change. Our finding did, however, add to the Bommelé et al., (2015) study in that it investigated the kind of assumptions underlying smoking research (linear versus non-linear) and that it further elucidated the exact role of nicotine dependence.

One of the tobacco control strategies suggested by Bommelé et al. (2015) for subgroups that are aware of the cons of smoking and pros of quitting focused on decreasing nicotine dependence through, for example, pharmacotherapies and nicotine-replacements. Our results suggest that such a strategy could be effective in influencing the number of cigarettes smoked per day, as most smoker subgroups were susceptible to nicotine dependence, which can influence transitions between smoking states.

The subgroups that did yield significant asymmetry coefficients were the receptive non-hardcore smokers with the cons of quitting variable, and the ambivalent non-hardcore smokers with pros of smoking. Hence, the motivational factors in favor of maintaining smoking seem to largely determine the strength and disparity between their stable states, i.e., smoking numerous cigarettes versus smoking few cigarettes. Such a result indicates that decreasing these variables could decrease the distance between the modes of these stable states, thereby facilitating easier transitions that can be attained through smaller perturbations, such as smoking control interventions. It is therefore also possible that people who do benefit from MI or CBT interventions, belong to the subgroups showing significant asymmetry variables, which could also be a hypothetical reason for the generally mixed or low effectiveness rates of current smoking interventions. However, such an interpretation is speculative for now and would require additional empirical evidence to be fully grounded.

Despite the advantages of using non-linear modeling, there are a number of limitations to the current study. For example, the studied dataset did only assess a small subset of variables that could play a role in influencing smoking behaviour. Examples of such variables that are studied in addiction research but were not included in our current dataset are: craving, stress, family history, family conflict, depression, social cue reactivity, life events, and psychological distress (Donovan, 1996; Hufford, Witkiewitz, Shields, Kodya, & Caruso, 2003). It is thus possible that the inclusion of additional variables would have led to better fitting and more complete cusp models. However, given that our current cusp models did find significant bifurcation variables for all subgroups, such inclusion of alternative variables will not change the main finding that the cusp model offered better fit to the observed data than linear models.

Another limitation relates both to the current dataset and the cusp method as they only offer (solutions for) cross-sectional data. As such, the data did not contain any information of how subgroups and variables would change over time. Conversely, the cusp software we used in this study (Grasman et al., 2009) is yet only suitable for cross-sectional data given the mathematical challenges in obtaining solutions to time-dependent stochastic differential equations. However, it could thus be argued that the employed cusp software and the current dataset match well given that they are both focused on cross-sectional data, while the cusp model does provide an indication of potential for change in the absence of true longitudinal methods.

Lastly, a limitation can be found in the difficulty in finding correct control variables for the cusp model (e.g., Witkiewitz et al., 2007) and in the implicit, theoretical link between proximal and distal risk factors and cusp asymmetry and bifurcation variables. Such an implicit and elegant link between the cusp control variables and the two types of risk factors did perform adequately in an earlier study (Hufford, Witkiewitz, Shields, Kodya, & Caruso, 2003). In this study, the authors performed a PCA yielding two orthogonal components, which were interpreted as proximal and distal risks. This conceptualisation yielded better results than a comparison linear model which included all of the same variables. Thereafter, other scholars also began using this conceptualisation of risk factors (e.g., Witkiewitz et al., 2007; Witkiewitz & Marlatt, 2007). However, it is still possible that these variables could relate in a less convenient and straightforward way. Additionally, this link implies that bifurcation variables are hardly changeable as they are regarded as distal risk factors. However, our bifurcation variable nicotine dependence might be more malleable than a true distal risk, such as age. Such a line of reasoning would also question the theoretical interpretation of cusp control variables, since their link with risk factors may not apply to all modeled phenomena. The implied link between cusp control variables and proximal and distal risk factors might be best regarded as still open to debate, and not as a univocal conceptualisation of cusp control variables. Such debate on this topic and more thorough experimental evidence are warranted to also avoid potential biases or misinterpretations in future work.

Despite these limitations, the current study did show that cusp models fitted better to the data than linear models. Hence, the commonly assumed linear relationship between smoking-related variables and smoking behaviour is hereby questioned. Such a finding could pave the way for catastrophe and other non-linear methods to become interesting alternatives to linear models in addiction research. Furthermore, the current subgroup analysis of smokers did show interesting differences between subgroups which highlights the advantages over analyses only taking the whole sample into account. Lastly, the current study could offer insight in the identification of possible cusp control variables, or conversely, why interventions based on motivational factors offer mixed results. However, before definitive conclusions can be reached, further investigations on the role of motivational factors as possible bifurcation factors are needed.

References

Aveyard, P., Griffin, C., Lawrence, T., & Cheng, K. (2003). A controlled trial of an expert system and self-help manual intervention based on the stages of change versus standard self-help materials in smoking cessation. *Addiction*, *98,* 345-345. http://dx.doi.org/10.1046/j.1360-0443.2003.00302.x

Aveyard, P., Massey, L., Parsons, A., Manaseki, S., & Griffin, C. (2009). The effect of Transtheoretical Model based interventions on smoking cessation. *Social Science & Medicine, 68,* 397-403. http://dx.doi.org/10.1016/j.socscimed.2008.10.036

Bandura, A. (1977). *Social learning theory.* Englewood Cliffs, N.J.: Prentice Hall.

Bommelé, J., Kleinjan, M., Schoenmakers, T., Burk, W., van den Eijnden, R., & van de Mheen, D. (2015). Identifying Subgroups among Hardcore Smokers: a Latent Profile Approach. *PLOS ONE, 10,* e0133570. http://dx.doi.org/10.1371/journal.pone.0133570

Breslau, N., Fenn, N., & Peterson, E. (1993). Early smoking initiation and nicotine dependence in a cohort of young adults. *Drug And Alcohol Dependence, 33*, 129-137. http://dx.doi.org/10.1016/0376-8716(93)90054-t

Breslau, N. & Peterson, E. (1996). Smoking cessation in young adults: age at initiation of cigarette smoking and other suspected influences. *Am J Public Health, 86,* 214-220. http://dx.doi.org/10.2105/ajph.86.2.214

Byrne, D. G., Mazanov, J., and Gregson, R. A. M. (2001). A cusp catastrophe analysis of changes to adolescent smoking behaviour in response to smoking prevention programs. Nonlinear Dynamics, *Psychology, and Life Sciences, 5*, 115-137. http://dx.doi.org/10.1023/A:1026467401041

Carter, B. and Tiffany, S. (1999). Meta-analysis of cue-reactivity in addiction research. *Addiction, 94,* 327-340. http://dx.doi.org/10.1046/j.1360-0443.1999.9433273.x

Chen, D., Lin, F., Chen, X., Tang, W., & Kitzman, H. (2014). Cusp Catastrophe Model.*Nursing Research, 63* 211-220. http://dx.doi.org/10.1097/nnr.0000000000000034

Clair, S. (1998). A cusp catastrophe model for adolescent alcohol use: An empirical test. *Nonlinear Dynamics, Psychology, and Life Sciences, 2,* 217-241. http://dx.doi.org/10.1023/A:1022376002167

Cobb L (1998). *An Introduction to Cusp Surface Analysis.* Technical report, Aetheling Consultants, Louisville, CO, USA.

Colby, S., Nargiso, J., Tevyaw, T., Barnett, N., Metrik, J., & Lewander, W. et al. (2012). Enhanced motivational interviewing versus brief advice for adolescent smoking cessation: Results from a randomized clinical trial. *Addictive Behaviors*, *37,* 817-823. http://dx.doi.org/10.1016/j.addbeh.2012.03.011

Cui, X., Rockett, I., Yang, T., & Cao, R. (2012). Work stress, life stress, and smoking among rural–

urban migrant workers in China. *BMC Public Health*, *12*(1). http://dx.doi.org/10.1186/1471-2458-12-979

Cupertino, A., Berg, C., Gajewski, B., Hui, S., Richter, K., Catley, D., & Ellerbeck, E. (2011). Change in self-efficacy, autonomous and controlled motivation predicting smoking. *Journal Of Health Psychology*, *17*, 640-652. http://dx.doi.org/10.1177/1359105311422457

Curry, S., Mermelstein, R., & Sporer, A. (2009). Therapy for Specific Problems: Youth Tobacco Cessation. *Annual Review Of Psychology*, *60*, 229-255. http://dx.doi.org/10.1146/annurev.psych.60.110707.163659

Dai, L., Korolev, K. and Gore, J. (2013). Slower recovery in space before collapse of connected populations. *Nature*, 496, 355-358. http://dx.doi.org/10.1111/ele.12066

Dai, L., Vorselen, D., Korolev, K. S., & Gore, J. (2012). Generic indicators for loss of resilience before a tipping point leading to population collapse. *Science*, *336*, 1175-1177. doi:10.1126/science.1219805

Dijkstra, A, & De Vries, H. (2000). Clusters of precontemplating smokers defined by the perception of the pros,cons, and self-efficacy. *Addict Behav.*, *25:* 373–385. doi: 10.1016/S0306-4603(99)00073-8 PMID: 10890291

Donovan, D. (1996). Assessment issues and domains in the prediction of relapse.*Addiction*, *91*, 29-36. http://dx.doi.org/10.1080/09652149638782

Dou, W. and Ghose, S. (2006). A dynamic nonlinear model of online retail competition using Cusp Catastrophe Theory. *Journal of Business Research*, 59, 838-848. http://dx.doi.org/10.1016/j.jbusres.2006.02.003

Grasman, R., Maas, H., & Wagenmakers, E. (2009). Fitting the Cusp Catastrophe in R : A cusp Package Primer. *Journal Of Statistical Software*, *32*. http://dx.doi.org/10.18637/jss.v032.i08

Gresov, C., Haveman, H., & Oliva, T. (1993). Organizational Design, Inertia and the Dynamics of Competitive Response. *Organization Science*, *4*, 181-208. http://dx.doi.org/10.1287/orsc.4.2.181

Guastello, S. (1984). Cusp and butterfly catastrophe modeling of two opponent process models: Drug addiction and work performance. *Syst. Res.*, *29*, 258-262. http://dx.doi.org/10.1002/bs.3830290405

Hayes, A. M., Laurenceau, J. P., Feldman, G., Strauss, J. L., & Cardaciotto, L. (2007). Change is not always linear: The study of nonlinear and discontinuous patterns of change in psychotherapy. *Clinical Psychology Review*, *27*, 715-723. doi:10.1016/j.cpr.2007.01.008

Hollon, S. D., & Beck, A. T. (2004). Cognitive and cognitive behavioral therapies. In M. J. Lambert (), *Bergin and Garfield's handbook of psychotherapy and behavior change* (5th ed., pp. 447–492). Hoboken, NJ: Wiley.

Hufford, M., Witkiewitz, K., Shields, A., Kodya, S., & Caruso, J. (2003). Relapse as a nonlinear dynamic system: Application to patients with alcohol use disorders. J*ournal Of Abnormal Psychology, 112*(2), 219-227. http://dx.doi.org/10.1037/0021-843x.112.2.219

Hughes, J. and Naud, S. (2016). Perceived role of motivation and self-efficacy in smoking cessation: A secondary data analysis. *Addictive Behaviors*, 61, pp.58-61. http://dx.doi.org/10.1016/j.addbeh.2016.05.010

Jansen, B. and Van der Maas, H. (2001). Evidence for the Phase Transition from Rule I to Rule II on the Balance Scale Task. *Developmental Review*, 21, 450-494. http://dx.doi.org/10.1006/drev.2001.0530

van der Maas, H., Kolstein, R., & van der Pligt, J. (2003). Sudden Transitions in Attitudes.*Sociological Methods & Research*, *32,* 125-152. http://dx.doi.org/10.1177/0049124103253773

Miller, W. and Rollnick, S. (2002). *Motivational interviewing.* New York: Guilford Press.

Nash, V., Townsend, C., Ponto, J., & Nelson, P. (2011). Cognitive Behavioral Therapy, Self-Efficacy, and Depression in Persons with Chronic Pain. *Pain Management Nursing*, *12*, e6. http://dx.doi.org/10.1016/j.pmn.2010.10.019

Oliva, T., Oliver, R. and MacMillan, I. (1992). A Catastrophe Model for Developing Service Satisfaction Strategies. *Journal of Marketing*, *56*(3), 83. http://dx.doi.org/10.2307/1252298

Perkins, K., Marcus, M., Levine, M., D'Amico, D., Miller, A., Broge, M., Ashcom, J. and Shiffman, S. (2001). Cognitive-behavioral therapy to reduce weight concerns improves smoking cessation outcome in weight-concerned women. *Journal of Consulting and Clinical Psychology, 69,* 604-613. http://dx.doi.org/10.1037/0022-006X.69.4.604

Prochaska J. O., DiClemente C. C. (1982). Transtheoretical therapy: Toward a more integrative model of change. *Psychother Theory, Res Pract., 19:* 276–288. doi: 10.1037/h0088437

Prochaska J. O., DiClemente C. C. (1983). Stages and processes of self-change of smoking: Toward an integrative model of change. *J Consult Clin Psychol., 51:* 390–395. doi: 10.1037/0022-006X.51.3.390 PMID: 6863699

Prochaska, J.O., DiClemente, C.C., Norcross, J. C. (1992). In search of how people change: applications to addictive behaviors. *Am Psychol., 47,* 1102–14.

Scheffer, M., Bascompte, J., Brock, W., Brovkin, V., Carpenter, S., Dakos, V., Held, H., van Nes, E., Rietkerk, M. and Sugihara, G. (2009). Early-warning signals for critical transitions. *Nature*, 461, 53-59. http://dx.doi.org/10.1038/nature08227

Scott, D. W. (1985). Catastrophe theory applications in clinical psychology: A review. *Current Psychological Research and Reviews. Spring*, 69–86. doi: 10.1007/BF02686568

Shiffman, S. (1993). Smoking cessation treatment: Any progress? *Journal of Consulting and Clinical Psychology, 61,* 718–722. http://dx.doi.org/10.1037/0022-006X.61.5.718

Ussher, M., Kakar, G., Hajek, P. and West, R. (2016). Dependence and motivation to stop smoking as predictors of success of a quit attempt among smokers seeking help to quit. *Addictive Behaviors,* 53, pp.175-180. http://dx.doi.org/10.1016/j.addbeh.2015.10.020

Velicer, W., Rossi, J., Prochaska, J. and Diclemente, C. (1996). A criterion measurement model for health behavior change. *Addictive Behaviors, 21*, 555-584. http://dx.doi.org/10.1016/0306-4603(95)00083-6

Vink, J., Willemsen, G., Beem, A. and Boomsma, D. (2005). The Fagerström Test for Nicotine Dependence in a Dutch sample of daily smokers and ex-smokers. *Addictive Behaviors, 30,* 575-579. http://dx.doi.org/10.1016/j.addbeh.2004.05.023

Webb, M., de Ybarra, D., Baker, E., Reis, I. and Carey, M. (2010). Cognitive–behavioral therapy to promote smoking cessation among African American smokers: A randomized clinical trial. *Journal of Consulting and Clinical Psychology, 78,* 24-33. http://dx.doi.org/10.1037/a0017669

Weinstein, S. & Mermelstein, R. (2013). Dynamic Associations of Negative Mood and Smoking Across the Development of Smoking in Adolescence. *Journal Of Clinical Child & Adolescent Psychology, 42*(5), 629-642. http://dx.doi.org/10.1080/15374416.2013.794698

West, R., Sohal, T. (2006). "Catastrophic" pathways to smoking cessation: findings from national survey. *BMJ, 332*(7539), pp.458-460.

Witkiewitz, K. & Marlatt, G. (2007). Modeling the complexity of post-treatment drinking: It's a rocky road to relapse. *Clinical Psychology Review, 27,* 724-738. http://dx.doi.org/10.1016/j.cpr.2007.01.002

Witkiewitz, K., van der Maas, H., Hufford, M. and Marlatt, G. (2007). Nonnormality and divergence in posttreatment alcohol use: Reexamining the Project MATCH data "another way.". *Journal of Abnormal Psychology, 116,* 378-394. http://dx.doi.org/10.1037/0021-843X.116.2.378.

Wolock, S., Yates, A., Petrill, S., Bohland, J., Blair, C., & Li, N. et al. (2013). Gene × smoking interactions on human brain gene expression: finding common mechanisms in adolescents and adults. *J Child Psychol Psychiatr*, *54*(10), 1109-1119. http://dx.doi.org/10.1111/jcpp.12119

World Health Organization,. (2016). *Tobacco*. Retrieved 24 February 2016, from http://www.who.int/mediacentre/factsheets/fs339/en/

Appendix A: Figures and Tables

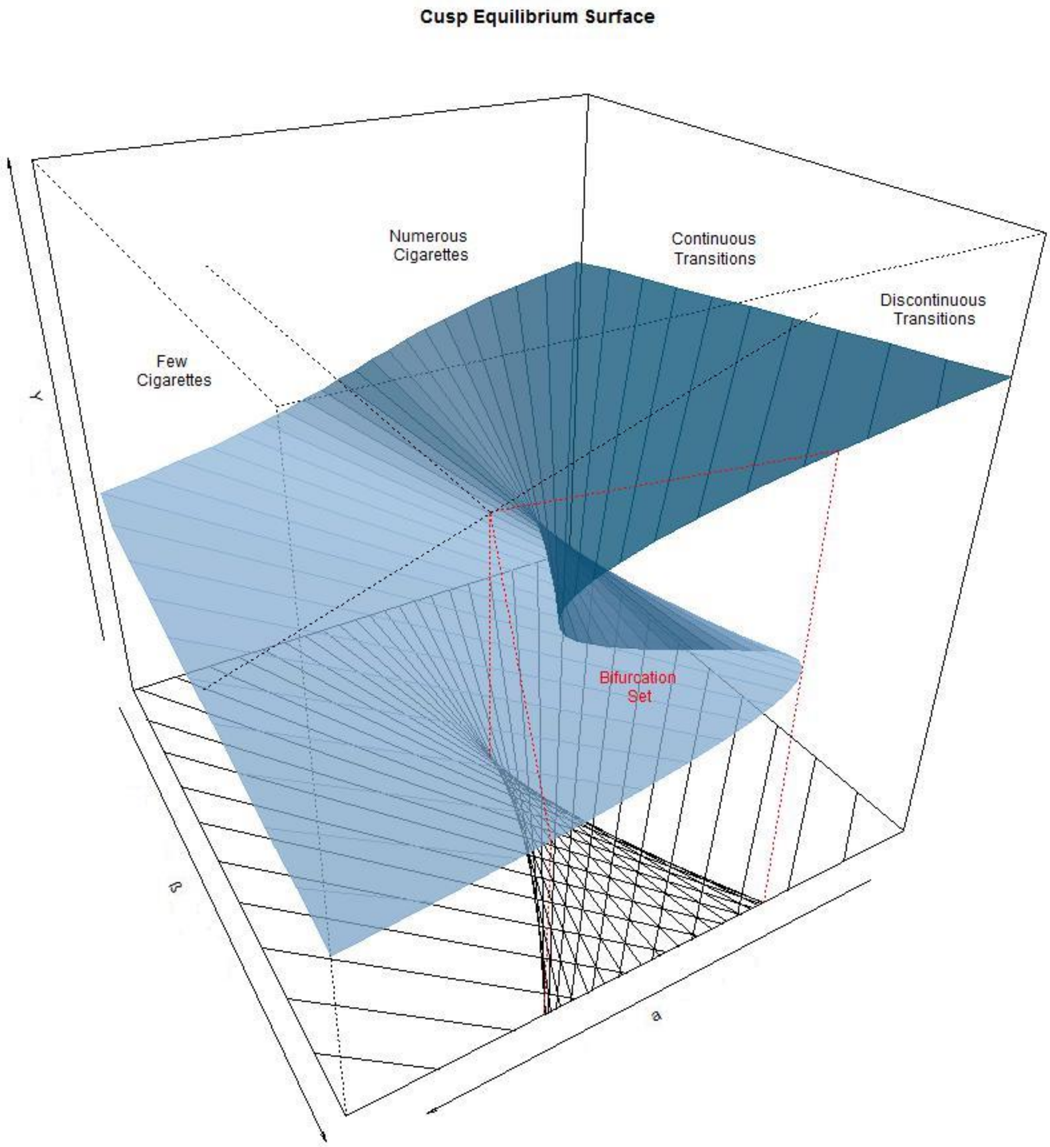

Figure 1: The Solutions of the Cusp Equations can be represented as a 2-Dimensional Surface occupying a 3-Dimensional Space. The Floor of this Image is called the Cusp Control Plane, whereas the Red delineated area represents the Bifurcation Area. For some Values of the Control Variables the Surface predicts Two Possible Values.

Table 1

Descriptive Statistics for all Variables included in the Models. Means, Standard Deviations, and Lower and Upper Bounds for their Values.

	Mean	*SD*	*Min.*	*Max.*
1. Nicotine dependence	4.42	2.29	0.00	10.00
2. Cigarettes per day	17.18	8.87	1.00	90.00
3. Age started smoking	16.70	5.24	6.00	65.00
4. Pros of smoking	3.45	0.70	1.38	5.88
5. Cons of smoking	4.43	0.79	1.69	7.00
6. Pros of quitting	4.54	0.85	1.69	7.00
7. Cons of quitting	3.46	0.69	1.00	6.00
8 Self efficacy	3.98	1.06	1.00	7.00

Note. SD = standard deviation, Min. = lower range boundary, Max. = upper range boundary.

Table 2

	1	2	3	4	5	6	7	8
1. Nicotine dependence								
2. Cigarettes per day	0.69***							
3. Age started smoking	-0.14***	-0.14***						
4. Pros of smoking	0.17***	0.07*	-0.02					
5. Cons of smoking	0.26***	0.19***	-0.07	-0.26***				
6. Pros of quitting	0.15***	0.12***	-0.03	-0.26***	0.76***			
7. Cons of quitting	0.28***	0.12***	-0.06	0.59***	-0.01	-0.07*		
8. Self efficacy	-0.39***	-0.25***	0.08*	-0.19***	-0.16***	-0.07*	-0.29***	

Pearson Correlations for all Variables included in the Models.

Note. $p < 0.05$ (*), $p < 0.01$ (**), $p < 0.001$ (***).

Table 3

Model Fitting Results and Goodness of Fit Indices for Model 1 and Model 2 for both Cusp and Linear Methods. Model 1 represents the Model Specification wherein Nicotine Dependence is taken as a Dependent Variable, whereas Model 2

Subgroup	Model	Method	No.of.parameters	R.squared[a]	Log.likelihood	AIC	BIC
Receptive Hardcore smokers	1	Linear	9	0.20	-454.40	926.80	955.83
Receptive Hardcore smokers	1	Cusp	11	0.03	-246.55	515.11	550.59
Receptive Hardcore smokers	2	Linear	9	0.35	-574.77	1167.54	1196.57
Receptive Hardcore smokers	2	Cusp	11	0.33	**-199.70**	**421.40**	**456.88**
Ambivalent Hardcore smokers	1	Linear	9	0.14	-780.80	1579.60	1612.93
Ambivalent Hardcore smokers	1	Cusp	11	0.20	-370.49	762.97	803.71
Ambivalent Hardcore smokers	2	Linear	9	0.23	-952.42	1922.85	1956.18
Ambivalent Hardcore smokers	2	Cusp	11	0.40	**-314.09**	**650.17**	**690.91**
Resistant Hardcore smokers	1	Linear	9	0.59	-43.90	105.79	116.01
Resistant Hardcore smokers	1	Cusp	11	0.07	-26.84	75.68	88.17
Resistant Hardcore smokers	2	Linear	9	0.56	-65.61	149.21	159.43
Resistant Hardcore smokers	2	Cusp	11	0.77	**-16.29**	**54.57**	**67.06**
Receptive non-Hardcore smokers	1	Linear	9	0.32	-215.88	449.76	473.30
Receptive non-Hardcore smokers	1	Cusp	11	0.62	-95.89	213.77	242.54
Receptive non-Hardcore smokers	2	Linear	9	0.49	-351.39	720.78	744.32
Receptive non-Hardcore smokers	2	Cusp	11	0.77	**-64.90**	**151.80**	**180.56**
Ambivalent non-Hardcore smokers	1	Linear	9	0.20	-484.73	987.45	1016.39
Ambivalent non-Hardcore smokers	1	Cusp	11	0.03	-250.20	522.40	557.76
Ambivalent non-Hardcore smokers	2	Linear	9	0.44	-570.47	1158.94	1187.87
Ambivalent non-Hardcore smokers	2	Cusp	11	0.42	**-175.22**	**372.44**	**407.81**
Disengaged non-Hardcore smokers	1	Linear	9	0.28	-146.21	310.41	328.14
Disengaged non-Hardcore smokers	1	Cusp	11	0.00	-70.66	163.32	184.99
Disengaged non-Hardcore smokers	2	Linear	9	0.55	-132.22	282.43	300.16
Disengaged non-Hardcore smokers	2	Cusp	11	0.56	**-53.95**	**129.91**	**151.58**

represents the Model Specification wherein Nicotine Dependence is taken as a Bifurcation Factor.

Note. Best performing fit values for each smoker subgroup are printed in bold.

[a] Reported R^2 values for the cusp models are Cobb's (1998) pseudo-R^2 values.

Table 4

Overview of Model Coefficients for all Smoker Subgroups for Cusp Model 2. Estimates, Standard Errors, Z-Values, and P-Values, for all Parameters.

<table>
<tr><th></th><th></th><th colspan="6">Asymmetry (α) / Proximal risk factors</th><th colspan="3">Bifurcation (β) / Distal risk factor</th><th colspan="2">Dependent (w) / Behavioural variable</th></tr>
<tr><th>Smoker subgroup</th><th>Model parameters</th><th>α Intercept</th><th>αj Pros of quitting</th><th>αj Cons of smoking</th><th>αj Cons of quitting</th><th>αj Pros of smoking</th><th>αj Self-efficacy</th><th>β Intercept</th><th>βj Nicotine dependence</th><th>βj Age started smoking</th><th>w Intercept</th><th>w Cigarettes per day</th></tr>
<tr><td>Receptive Hardcore Smokers</td><td>Estimate</td><td>-0.11</td><td>0.13</td><td>-0.14</td><td>0.20</td><td>0.25</td><td>0.03</td><td>5.23</td><td>-0.67</td><td>0.01</td><td>-3.80</td><td>0.11</td></tr>
<tr><td></td><td>Standard error</td><td>1.76</td><td>0.23</td><td>0.33</td><td>0.23</td><td>0.24</td><td>0.12</td><td>0.75</td><td>0.08</td><td>0.03</td><td>0.16</td><td>0.01</td></tr>
<tr><td></td><td>z value</td><td>-0.06</td><td>0.56</td><td>-0.44</td><td>0.87</td><td>1.04</td><td>0.29</td><td>7.00</td><td>-8.58</td><td>0.43</td><td>-24.26</td><td>11.78</td></tr>
<tr><td></td><td>Pr(>|z|)</td><td>0.95</td><td>0.57</td><td>0.66</td><td>0.39</td><td>0.30</td><td>0.77</td><td><0.001***</td><td><0.001***</td><td>0.67</td><td><0.001***</td><td><0.001***</td></tr>
<tr><td>Ambivalent Hardcore smokers</td><td>Estimate</td><td>-0.34</td><td>-0.15</td><td>0.09</td><td>0.04</td><td>0.11</td><td>-0.05</td><td>5.60</td><td>-0.45</td><td>0.02</td><td>-3.69</td><td>0.08</td></tr>
<tr><td></td><td>Standard error</td><td>1.48</td><td>0.19</td><td>0.26</td><td>0.23</td><td>0.22</td><td>0.11</td><td>0.54</td><td>0.05</td><td>0.02</td><td>0.08</td><td>0.00</td></tr>
<tr><td></td><td>z value</td><td>-0.23</td><td>-0.77</td><td>0.36</td><td>0.20</td><td>0.49</td><td>-0.47</td><td>10.31</td><td>-8.51</td><td>1.45</td><td>-45.80</td><td>21.49</td></tr>
<tr><td></td><td>Pr(>|z|)</td><td>0.82</td><td>0.44</td><td>0.72</td><td>0.84</td><td>0.62</td><td>0.64</td><td><0.001***</td><td><0.001***</td><td>0.15</td><td><0.001***</td><td><0.001***</td></tr>
<tr><td>Resistant Hardcore smokers</td><td>Estimate</td><td>-0.55</td><td>0.05</td><td>0.26</td><td>0.25</td><td>0.09</td><td>-0.03</td><td>8.82</td><td>-0.96</td><td>-0.02</td><td>-4.58</td><td>0.15</td></tr>
<tr><td></td><td>Standard error</td><td>3.98</td><td>0.52</td><td>0.73</td><td>0.51</td><td>0.79</td><td>0.26</td><td>2.01</td><td>0.25</td><td>0.03</td><td>0.43</td><td>0.02</td></tr>
<tr><td></td><td>z value</td><td>-0.14</td><td>0.10</td><td>0.36</td><td>0.49</td><td>0.11</td><td>-0.10</td><td>4.40</td><td>-3.82</td><td>-0.65</td><td>-10.73</td><td>6.69</td></tr>
<tr><td></td><td>Pr(>|z|)</td><td>0.89</td><td>0.92</td><td>0.72</td><td>0.62</td><td>0.91</td><td>0.92</td><td><0.001***</td><td><0.001***</td><td>0.51</td><td><0.001***</td><td><0.001***</td></tr>
<tr><td>Receptive non-Hardcore Smokers</td><td>Estimate</td><td>2.37</td><td>0.90</td><td>-1.16</td><td>1.79</td><td>-0.67</td><td>-0.06</td><td>7.95</td><td>-0.69</td><td>-0.01</td><td>-3.11</td><td>0.06</td></tr>
<tr><td></td><td>Standard error</td><td>4.10</td><td>0.63</td><td>0.67</td><td>0.61</td><td>0.42</td><td>0.23</td><td>1.02</td><td>0.09</td><td>0.04</td><td>0.13</td><td>0.00</td></tr>
<tr><td></td><td>z value</td><td>0.58</td><td>1.42</td><td>-1.72</td><td>2.92</td><td>-1.59</td><td>-0.26</td><td>7.76</td><td>-7.87</td><td>-0.33</td><td>-23.96</td><td>17.95</td></tr>
<tr><td></td><td>Pr(>|z|)</td><td>0.56</td><td>0.16</td><td>0.09</td><td><0.001***</td><td>0.11</td><td>0.79</td><td><0.001***</td><td><0.001***</td><td>0.74</td><td><0.001***</td><td><0.001***</td></tr>
<tr><td>Ambivalent non-Hardcore smokers</td><td>Estimate</td><td>-0.21</td><td>0.15</td><td>-0.03</td><td>0.22</td><td>0.28</td><td>-0.04</td><td>5.25</td><td>-0.59</td><td>0.02</td><td>-2.89</td><td>0.08</td></tr>
<tr><td></td><td>Standard error</td><td>2.71</td><td>0.41</td><td>0.35</td><td>0.33</td><td>0.30</td><td>0.20</td><td>0.74</td><td>0.06</td><td>0.02</td><td>0.10</td><td>0.01</td></tr>
<tr><td></td><td>z value</td><td>-0.08</td><td>0.37</td><td>-0.07</td><td>0.66</td><td>0.92</td><td>-0.21</td><td>7.14</td><td>-9.66</td><td>0.93</td><td>-29.16</td><td>13.78</td></tr>
<tr><td></td><td>Pr(>|z|)</td><td>0.94</td><td>0.71</td><td>0.94</td><td>0.51</td><td>0.36</td><td>0.84</td><td><0.001***</td><td><0.001***</td><td>0.35</td><td><0.001***</td><td><0.001***</td></tr>
<tr><td>Disengaged non-Hardcore smokers</td><td>Estimate</td><td>11.08</td><td>-0.16</td><td>-1.20</td><td>-0.74</td><td>0.84</td><td>-0.29</td><td>-2.14</td><td>0.90</td><td>-0.10</td><td>0.35</td><td>0.12</td></tr>
<tr><td></td><td>Standard error</td><td>8.26</td><td>0.83</td><td>1.04</td><td>0.64</td><td>0.67</td><td>0.35</td><td>2.53</td><td>0.19</td><td>0.08</td><td>0.79</td><td>0.04</td></tr>
<tr><td></td><td>z value</td><td>1.34</td><td>-0.19</td><td>-1.15</td><td>-1.16</td><td>1.26</td><td>-0.83</td><td>-0.84</td><td>4.69</td><td>-1.24</td><td>0.45</td><td>3.40</td></tr>
<tr><td></td><td>Pr(>|z|)</td><td>0.18</td><td>0.85</td><td>0.25</td><td>0.24</td><td>0.21</td><td>0.41</td><td>0.40</td><td><0.001***</td><td>0.22</td><td>0.66</td><td><0.001***</td></tr>
</table>

Note. Best significant model coefficients for each smoker subgroup are printed in bold.

Note. $p < 0.05$ (*), $p < 0.01$ (**), $p < 0.001$ (***).

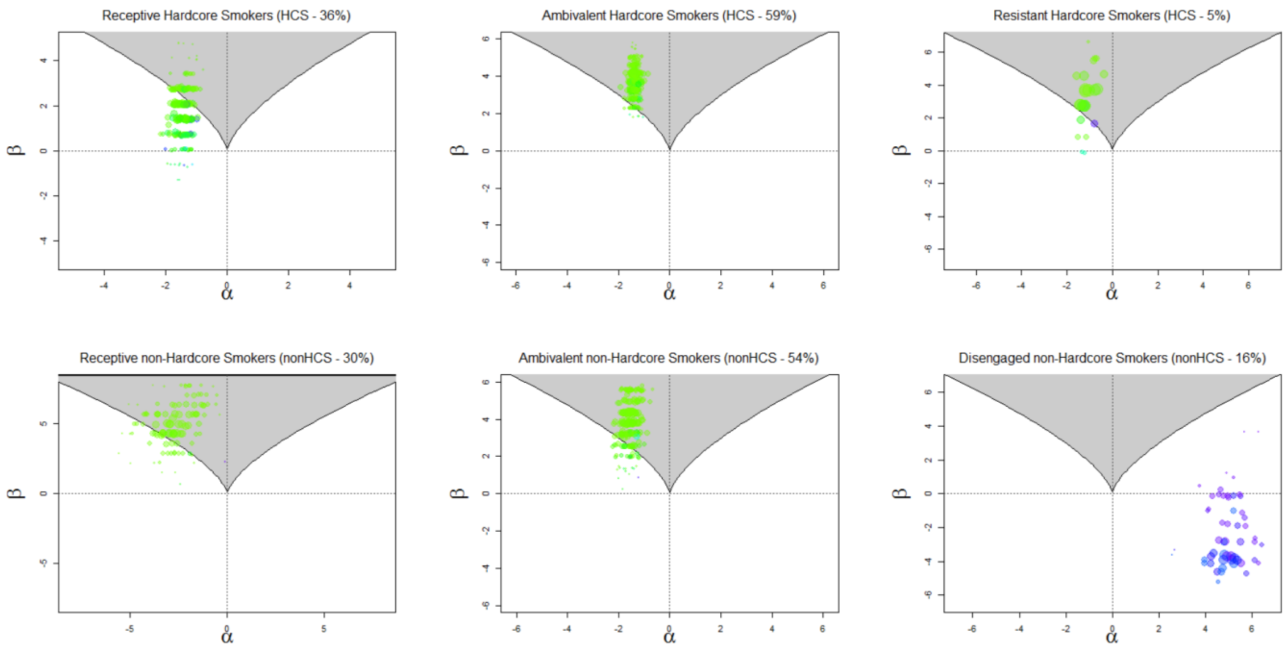

Figure 2: Cusp control surfaces of cusp Model 2 for all smoker subgroups with plotted data points. The grey area represents the bifurcation area.

Table 5

Model Fitting Results and Goodness of Fit Indices for Model 3 and Model 4 for both Cusp and Linear Methods. Model 3 represents the Model Specification wherein only Nicotine Dependence is taken as the Bifurcation Variable, whereas

Subgroup	Model	Method	No.of.parameters	R.squared	Log.likelihood	AIC	BIC
Receptive Hardcore smokers	3	Linear	8	0.34	-574.95	1165.91	1191.71
Receptive Hardcore smokers	3	Cusp	10	0.32	**-199.79**	**419.58**	**451.84**
Receptive Hardcore smokers	4	Linear	8	0.05	-609.34	1234.67	1260.48
Receptive Hardcore smokers	4	Cusp	10	0.05	-235.63	491.26	523.52
Ambivalent Hardcore smokers	3	Linear	8	0.22	-953.55	1923.11	1952.74
Ambivalent Hardcore smokers	3	Cusp	10	0.40	**-315.13**	**650.26**	**687.30**
Ambivalent Hardcore smokers	4	Linear	8	0.04	-984.86	1985.73	2015.36
Ambivalent Hardcore smokers	4	Cusp	10	0.21	-350.34	720.68	757.72
Resistant Hardcore smokers	3	Linear	8	0.53	-66.34	148.67	157.76
Resistant Hardcore smokers	3	Cusp	10	0.77	**-16.50**	**53.00**	**64.36**
Resistant Hardcore smokers	4	Linear	8	0.08	-74.09	164.18	173.26
Resistant Hardcore smokers	4	Cusp	10	0.52	-23.91	67.83	79.18
Receptive non-Hardcore smokers	3	Linear	8	0.48	-352.17	720.34	741.26
Receptive non-Hardcore smokers	3	Cusp	10	0.77	**-64.95**	**149.90**	**176.06**
Receptive non-Hardcore smokers	4	Linear	8	0.09	-380.36	776.72	797.64
Receptive non-Hardcore smokers	4	Cusp	10	0.52	-103.45	226.91	253.06
Ambivalent non-Hardcore smokers	3	Linear	8	0.44	-570.59	1157.18	1182.90
Ambivalent non-Hardcore smokers	3	Cusp	10	0.42	**-175.65**	**371.31**	**403.46**
Ambivalent non-Hardcore smokers	4	Linear	8	0.05	-619.65	1255.30	1281.02
Ambivalent non-Hardcore smokers	4	Cusp	10	0.04	-222.41	464.83	496.98
Disengaged non-Hardcore smokers	3	Linear	8	0.53	-133.28	282.57	298.33
Disengaged non-Hardcore smokers	3	Cusp	10	0.54	**-54.84**	**129.68**	**149.38**
Disengaged non-Hardcore smokers	4	Linear	8	0.25	-145.92	307.84	323.60
Disengaged non-Hardcore smokers	4	Cusp	10	0.28	-66.87	153.74	173.44

Model 4 represents the Model Specification wherein only Age of Smoking Onset is taken as the Bifurcation Variable.

Note. Best performing fit values for each smoker subgroup are printed in bold.

[a] Reported R^2 values for the cusp models are Cobb's (1998) pseudo-R^2 values.

Table 6.

Subgroup	Model	No.of.parameters	R.squared [a]	Log.likelihood	AIC	BIC
Receptive Hardcore smokers	2	11	0.33	**-199.70**	421.40	456.88
Receptive Hardcore smokers	3	10	0.32	-199.79	**419.58**	**451.84**
Ambivalent Hardcore smokers	2	11	0.26	**-314.09**	**650.17**	690.91
Ambivalent Hardcore smokers	3	10	0.40	-315.13	650.26	**687.30**
Resistant Hardcore smokers	2	11	0.77	**-16.29**	54.57	67.06
Resistant Hardcore smokers	3	10	0.77	-16.50	**53.00**	**64.36**
Receptive non-Hardcore smokers	2	11	0.77	**-64.90**	151.80	180.56
Receptive non-Hardcore smokers	3	10	0.77	-64.95	**149.90**	**176.06**
Ambivalent non-Hardcore smokers	2	11	0.42	**-175.22**	372.44	407.81
Ambivalent non-Hardcore smokers	3	10	0.42	-175.65	**371.31**	**403.46**
Disengaged non-Hardcore smokers	2	11	0.56	**-53.95**	129.91	151.58
Disengaged non-Hardcore smokers	3	10	0.54	-54.84	**129.68**	**149.38**

Model Fitting Results and Goodness of Fit Indices for Cusp Model 2 and Cusp Model 3.

Note. Best performing fit values for each smoker subgroup are printed in bold.

[a] Reported R^2 values are Cobb's (1998) pseudo-R^2 values.

Table 7.

Overview of Model Coefficients for all Smoker Subgroups for Cusp Model 3. Estimates, Standard Errors, Z-Values, and P-Values, for all Parameters.

		Asymmetry (α) / Proximal risk factors						Bifurcation (β) / Distal risk factors		Dependent (w) / Behavioural variable	
Smoker subgroup	Model parameters	α Intercept	αj Pros of quitting	αj Cons of smoking	αj Cons of quitting	αj Pros of smoking	αj Self-efficacy	β Intercept	βj Nicotine dependence	w Intercept	w Cigarettes per day
Receptive Hardcore Smokers	Estimate	-0.07	0.13	-0.15	0.21	0.25	0.03	5.44	-0.67	-3.8	0.11
	Standard error	1.85	0.19	0.31	0.23	0.21	0.1	0.12	0.02	0.12	0
	z value	-0.04	0.72	-0.48	0.91	1.2	0.33	47.18	-37.59	-33	24.03
	Pr(>|z|)	0.97	0.47	0.63	0.37	0.23	0.74	**<0.001*** **	**<0.001*** **	**<0.001*** **	**<0.001*** **
Ambivalent Hardcore smokers	Estimate	-0.35	-0.17	0.12	0.03	0.14	-0.05	5.95	-0.46	-3.69	0.08
	Standard error	1.47	0.19	0.25	0.23	0.22	0.11	0.48	0.05	0.08	0.00
	z value	-0.24	-0.91	0.49	0.11	0.65	-0.44	12.30	-8.65	-45.72	21.50
	Pr(>|z|)	0.81	0.36	0.63	0.91	0.52	0.66	**<0.001*** **	**<0.001*** **	**<0.001*** **	**<0.001*** **
Resistant Hardcore smokers	Estimate	-0.38	-0.01	0.29	0.23	0.11	-0.04	8.00	-0.88	-4.60	0.15
	Standard error	3.78	0.49	0.70	0.50	0.79	0.25	1.57	0.22	0.42	0.02
	z value	-0.10	-0.02	0.42	0.46	0.14	-0.14	5.10	-4.02	-10.94	7.10
	Pr(>|z|)	0.92	0.99	0.68	0.65	0.89	0.89	**<0.001*** **	**<0.001*** **	**<0.001*** **	**<0.001*** **
Receptive non-Hardcore Smokers	Estimate	2.35	0.90	-1.15	1.81	-0.66	-0.06	7.74	-0.69	-3.11	0.06
	Standard error	4.11	0.64	0.68	0.61	0.42	0.23	0.81	0.09	0.13	0.00
	z value	0.57	1.42	-1.70	2.95	-1.58	-0.26	9.55	-7.88	-24.30	18.07
	Pr(>|z|)	0.57	0.15	0.09	**<0.001*** **	0.11	0.79	**<0.001*** **	**<0.001*** **	**<0.001*** **	**<0.001*** **
Ambivalent non-Hardcore smokers	Estimate	-0.29	0.16	-0.02	0.21	0.25	-0.05	5.61	-0.59	-2.90	0.08
	Standard error	2.70	0.40	0.35	0.28	0.03	0.20	0.03	0.01	0.04	0.01
	z value	-0.11	0.39	-0.07	0.77	7.88	-0.27	177.90	-56.24	-69.91	15.76
	Pr(>|z|)	0.91	0.70	0.94	0.44	**<0.001*** **	0.78	**<0.001*** **	**<0.001*** **	**<0.001*** **	**<0.001*** **
Disengaged non-Hardcore smokers	Estimate	11.13	-0.12	-1.25	-0.77	0.84	-0.32	-4.01	0.93	0.34	0.12
	Standard error	8.04	0.83	1.04	0.60	0.63	0.35	0.35	0.18	0.93	0.04
	z value	1.38	-0.14	-1.20	-1.28	1.33	-0.91	-11.41	5.09	0.37	3.21
	Pr(>|z|)	0.17	0.89	0.23	0.20	0.18	0.37	**<0.001*** **	**<0.001*** **	0.71	**<0.001*** **

Note. Best significant model coefficients for each smoker subgroup are printed in bold.

Note. $p < 0.05$ (*), $p < 0.01$ (**), $p < 0.001$ (***).

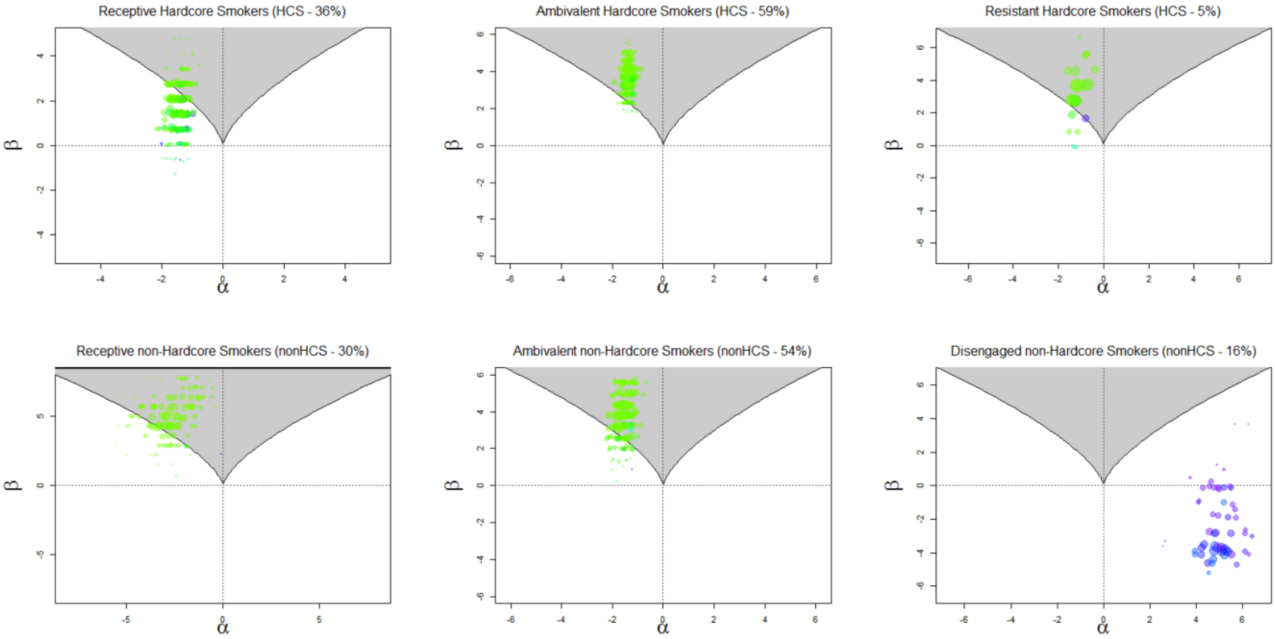

Figure 3: Cusp control surfaces of cusp Model 3 for all smoker subgroups with plotted data points. The grey area represents the bifurcation area.

Appendix B: Supplementary materials

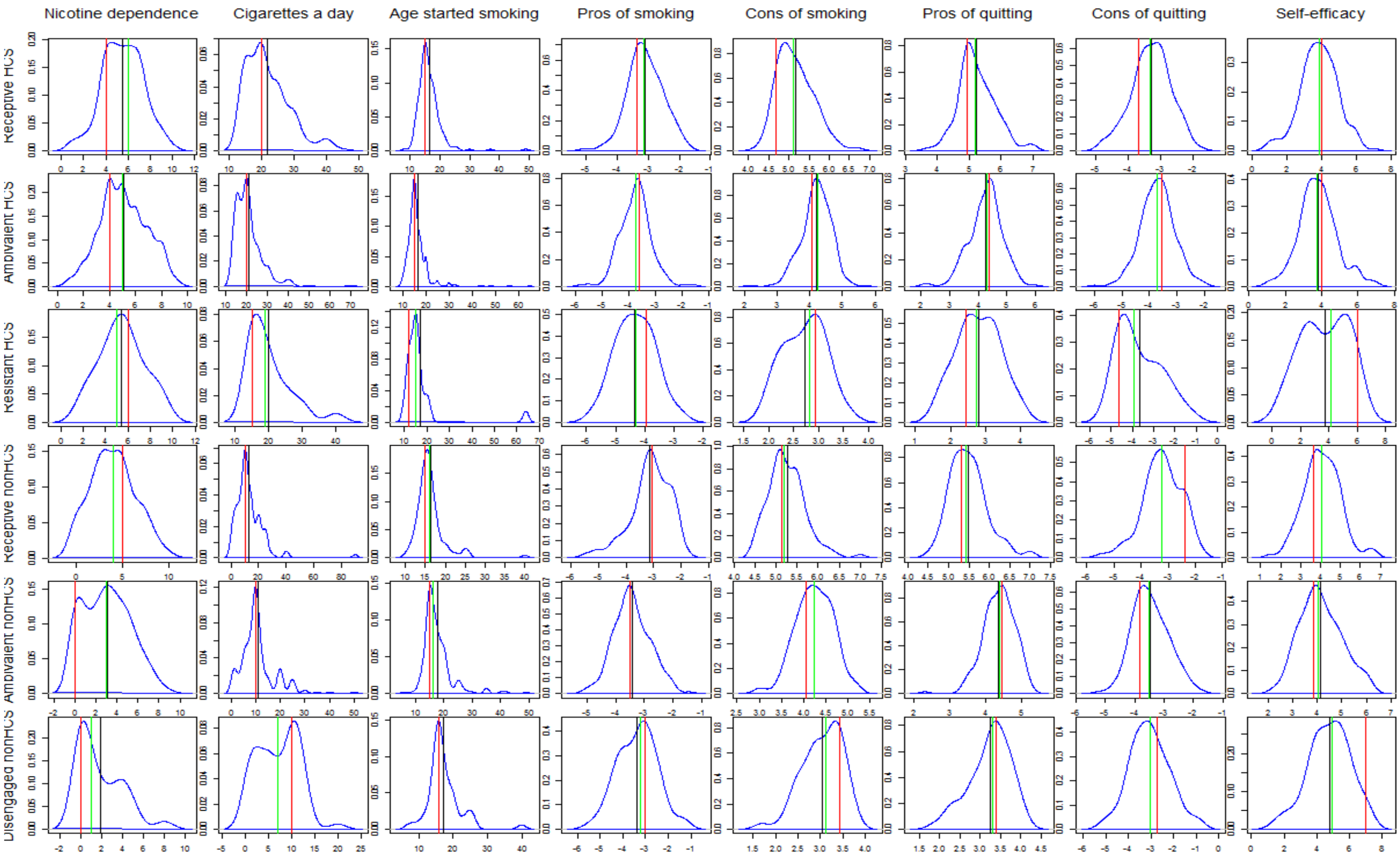

Figure A: Variable distributions for each smoker subgroup. Distributions in blue, black line = variable mean, green line = variable median, red line = variable mode.

Table A.

Model fitting results for linear and cusp versions of model 1 and 2 for the whole sample taken together.

	No..of.parameters	R.squared [a]	Log.likelihood	AIC	BIC
Model 1: Linear	9	0.24	-2591.84	5201.68	5244.34
Model 1: Cusp	11	0.12	-1122.66	2267.32	2319.46
Model 2: Linear	9	0.48	-2769.54	5557.07	5599.74
Model 2: Cusp	11	0.45	-1029.12	2080.23	2132.38

[a] Reported R^2 values for the cusp models are Cobb's (1998) pseudo-R^2 values.