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Shisha smoking: impact on cognitive functions impairments in healthy adults

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Abstract. – OBJECTIVE: Shisha smoking is becoming highly prevalent and is a predominant mode of tobacco use particularly amongst the young generation around the globe. This study aims to investigate whether or not shisha smoking impairs the cognitive functioning in adolescents.

SUBJECTS AND METHODS: We recruited 65 (32 control subjects and 33 shisha smokers) apparently healthy male volunteers. The first group consisted of 33 volunteer male shisha smoker subjects with mean age 24.45±2.93 (mean ± SD) years. The second group consists of 32 subjects with mean age 23.31±2.68 (mean ± SD) years who did not smoke shisha. Age, gender, weight, height, ethnicity, educational and socioeconomic status of a matched design was used to appraise the impact of shisha smoking on cognitive functions. The cognitive function's outcome variables were the response time (Attention Switching Task-AST), (Complex Reaction Time-CRT task) and the percentage of correct answers (Pattern Recognition Memory PRM-task). Cognitive functions were assessed by using Cambridge Neuropsychological Automated Battery (CANTAB).

RESULTS: Shisha smokers showed significant decline in cognitive performances AST-Latency (p=0.03); AST-Congruent (p=0.05); AST-Incongruent (p=0.05); mean CRT (p=0.001); CRT % (p=0.01); PRM (p=0.024) compared to their matched control.

CONCLUSIONS: Cognitive functions including attention, alertness, and memory were significantly impaired in healthy adult shisha smokers compared to non-shisha smokers.

Key Words:
Shisha Smoking, Cognitive functions, Attention task, Memory.

Introduction

Shisha smoking bars are rapidly opened in various parts of the globe and commonly seen around the residential, commercial and educational vicinities. Shisha smoking is a cultural and customary behavior of the people in the various regions of the word including Middle East, North Africa and Southeast Asia. Shisha smoking is well-known by its numerous names in different countries such as "water pipe, hubble bubble, hookah, narghile, ghoza smoking" [1]. Shisha smoking is a communal globally practice[2] especially in the urban population of the high-income posh localities[3], particularly among the young generation of Arabic countries[4]. It has been steadily spread among the adolescents around the globe, gaining more popularity in the Middle East. The prevalence of smoking shisha is ranging from 5-17% in American adolescents and 6-34% Middle Eastern youths[4]. Some studies reported that the shisha smoker's prevalence reached up to 27% of school students and 54% of university students[5,6].

Shisha smoke contains enormous amount of fruit-flavored (apple, plum, mango, mint, coconut, strawberry and cola) fine and ultrafine PM, nicotine, carbon monoxide, polycyclic aromatic hydrocarbons, volatile aldehydes, phenolic compounds and carcinogenic PAH[7-11] and some substantial metals including arsenic and lead. The nicotine contents of shisha tobacco are 2-4% compare to 1-3% for cigarettes and carbon monoxide concentrations 0.34-1.40%[12]. In addition, shisha smokers are wide-open to extra smoke over a lengthier time period as compared to cigarette smoking. A Cigarette smoker typically takes 45-75 puffs in 5-8 minutes and inhales 0.5 to 0.6 liters of smoke whereas a shisha smoking session lasts for 25-80 minutes and shisha smoker takes 45-200 puffs and inhales 0.15-1 liter of smoke in one session of shisha, which is equivalent to smoking about hundred cigarettes[12,13]. Researchers have studied the association between shisha smoking and lung functions[14] but no study has determined the impact of shisha smoking on cognitive functioning. Therefore, this study aimed to investigate

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the impact of shisha smoking on cognitive functions.

Subjects and Methods

Subjects' Selection

This study was conducted in the Department of Physiology, College of Medicine, King Saud University, Riyadh, Saudi Arabia between June to November 2015. Age, gender, weight, height, ethnicity, educational and socioeconomic status matched design were used to investigate the association of shisha smoking on cognitive functions. A detailed clinical history and examination of all the subjects was conducted for the selection of subjects to conclude whether a particular subject was to be included in the study or not. All the participants were inquired with regards to alcohol consumption, cigarette, shisha smoking and other tobacco products. Detail history regarding any drug intake, diseases like seizures, and psychiatric problems, was obtained. The subjects were asked to maintain 6-8 hours of sleep and we excluded those subjects with sleep disturbances history[15]. After the interview, clinical history and examination, the shisha smokers group consisted of 33 volunteers, all male subjects with a mean age of 24.45±2.93 (mean ± SD) years who smoked shisha regularly. These 33 shisha smokers were harmonized with another group called non-shisha smokers (control) group. The control group was selected in a comparable way. 32 healthy male volunteers that matched age, ethnicity and socioeconomic status, with a mean age 23.31±2.68 (mean ± SD) years, were recruited. The control group mainly comprised of university technicians, secretaries, research assistants and receptionists. All subjects were exclusively harmonized for age, ethnicity and socioeconomic and educational status. All subjects provided their informed consent for inclusion before they participated in the study. This study was approved by the Ethics Committee of the College of Medicine (King Saud University, Riyadh, SA).

Exclusion Criteria

Subjects with identified cases of diabetes mellitus, marked obesity, anemia, chronic obstructive lung diseases, malignancy, chronic alcoholics, drug addicts and cigarette smokers (other than shisha) were excluded[14]. Subjects with known cases of anxiety, vision problems, attention deficit, psychiatric disorders, skeletal muscle disorders and those on sedatives, hypnotics and who had disturbed sleep history, were also excluded from the study[16,17].

Cognitive Function

We performed the neuropsychological testing by using "Cambridge Neuropsychological Test Automated Battery (CANTAB)" research suite software, version 6.0.37, Cambridge cognition". CANTAB is a computerized based standard software system to assess the cognitive functions across the life age. It has the ability to detect the declines in the performances of executive functions among the healthy adults and subjects with different disorders. The test process takes 25-30 min to complete the entire procedure. The subjects were made to sit comfortably on a chair and asked to keep pressing the response button with the index finger of the dominant hand.

Attention Switching Task (AST)

AST measured the up-down cognitive control processes involving the prefrontal cortex. The test displayed an arrow cue at the top on the screen for the right or left side of the computer and subject could point on the bases of stimuli in either direction to the right or the left. The direction in which the arrow was pointing (same direction, congruent stimuli) and to the side on which arrow appeared or incongruent stimuli (e.g., the arrow on the right side of the screen pointing to the left).

Choice Reaction Time (CRT)

CRT measured the motor speed and attentiveness. This 2-choice stimulus (left and right) has two possible outcomes measurement of reaction time. Reaction time was measured when the subject pressed the left or right hand button on the press pad on the basis of left and right stimuli on the screen.

Pattern Recognition Memory (PRM)

Visual pattern recognition memory is paradigm designed for visual patterns presented in the center of the screen in a sequence for verbal labels. In this task, subjects were required to select a pattern they had already seen in the form of a unique pattern.

Statistical Analysis

AST test outcome was determined including response latencies and error scores that showed the participant's attention switching ability and the prying of congruent and incongruent task-irrelevant information. CRT test outcome assess the correct and incorrect responses and latency (response speed). PRM task allows the measurement of the number of correct patterns selected.

Table I. Anthropometric data of shisha smokers and control group.

Parameters	Control Group (n=32)	Shisha Group (n=33)	*p*-value
Age	23.31±2.68	24.45±2.93	0.107
Height	169.9 ±8.16	172.27±5.82	0.187
Weight	81.21±24.1	79.27±17.21	0.709

Values are presented in mean ±SEM; NS= Non-Significant.

Table II. Comparison of cognitive functions between shisha smokers compared to their control group.

Parameters	Control Group (n=32)	Shisha Group (n=33)	*p*-value
AST-Switching	256.17 ± 142.8	286.48±124.22	0.365
AST-Latency	544.8± 116.1	604.7±105.8	0.033
AST-Congruent	518.3 ± 108.3	577.8±99.5	0.023
AST-Incongruent	574.8 ± 129.8	635.2±118	0.054
AST-Congruent-cost	62.79± 44.7	63.5±39.2	0.023
AST %	94.7 ± 5.4	93.2±4.8	0.242
CRT	321 ± 84.4	377.6±73.8	0.006
CRT %	97.6 ± 4.18	99.6±.84	0.011
PRM	91.8 ± 7.9	86.74±9.4	0.024

Values are presented in mean ±SEM.

Statistical analysis was executed using SPSS software (version 22.0; SPSS Armonk, NY, USA). Comparison of quantitative data between shisha group and control group was performed using paired t-test. Two-tailed statistics were used and statistical significance was set at $p<0.05$.

Results

Table I demonstrates the anthropometric variables including age, height and weight of shisha smokers and control group. The control group consisted of 32 subjects with mean age 23.31±2.68 years; height 169.9±8.16 cm and weight 81.21±24.1 kg (mean ± SD). The shisha smoker group consisted of 33 volunteer, male subjects with mean age 24.45±2.93 years; height 172.27±5.85 cm and weight 79.27±17.21 kg (mean ± SD). Table II demonstrates the evaluation of the cognitive function test parameters between the subjects who smoke shisha and their matched control subjects. There was a decline in the cognitive function parameters including AST-Switching task in control group at 256.17±142.8 *vs.* shisha smokers 286.48±124.22 (p=0.365); AST-Latency in control group was 544.8±116.1 *vs.* shisha smokers 604.7±105.8 (p=0.033); AST-Congruent in control group was 518.3±108.3 *vs.* shisha smokers 577.8±99.5 (p=0.023); AST-incongruent in control was 574.8±129 *vs.* shisha smokers 635.2±118 (p=0.054); AST-Congruent-cost in control group was 62.79±44.7 *vs.* shisha smokers 63.5±39.2 (p=0.023); AST % in control group was 94.7±5.4 *vs.* shisha smokers 93.2±4.8 (p=0.242); CRT in control group was 321±84.4 *vs.* shisha smokers 377.6±73.8 (p=0.006); CRT % in control group was 97.6±4.18 *vs.* shisha smokers 99.6±0.84 (p=0.011); PRM in control group was 91.8±7.9 *vs.* shisha smokers 86.74±9.4 (p=0.024) (Table II; Figure 1).

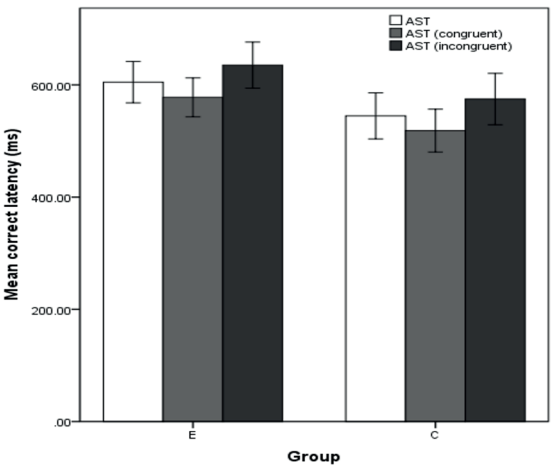

Figure 1. Cognitive functions of shisha smokers and control group.

Discussion

This is the first study added in the existing literature on the effect of shisha smoking on cognitive functions in young adults. We found that shisha smoking impaired the cognitive performance compared to their matched control subjects. The impairment achieved the significant levels for attention, reacting time and memory tasks. Confounders such as sleep, vision, neuro-psychiatric problems, were carefully considered. The shisha and control group did not differ in demographic or clinical characteristics; thus, it is unlikely that these variables influenced the results.

Smoking is allied with neurocognitive functioning impairment, smoking cessation interferences[18] have potential important target for improving functional outcomes. Zhang et al[19] observed the potential effect of smoking and drinking on cognition. They reported that smoking is allied with cognitive deterioration, but not with BDNF (brain-derived neurotrophic factor) levels in a healthy population. However, smoking magnitude was positively allied with BDNF levels.

Doiron et al[20] showed that smoking was more likely to cause cognitive impairment. Their findings are in line with results in healthy adults that have linked smoking to cognitive impairment, brain atrophy and functional changes. Hotta et al[21] determined the relationship between smoking status and numerous domains of cognitive function among middle-aged subjects. Smoking status was linked with a decline in processing speed and the deterioration varies by gender. In some neurocognitive tests, females who had no smoking history showed better performance than past and/or current smokers. Among males, non-smokers had significantly higher marks in the symbol digit substitution test.

Chamberlain et al[22] conducted a study in judgement to non-smokers and nicotine users. They reported significant cognitive impairments on attention sustained, spatial working memory and executive planning functions. Similarly, Sabia et al[23] determined the relationship between smoking and cognitive deterioration in midlife old age subjects. They observed that the middle aged male smokers experienced faster cognitive decline in overall cognition and executive function compared to non-smokers. More recently, Bashir et al[24] found that smokers had significant cognitive impairments in AST-Latency, AST-Congruent, and AST-Incongruent condition. Their findings indicate that attention and alertness were significantly impaired in smokers compared to non-smokers.

Timothy et al[16] summarized the highly acknowledged literature on the neuro-cognitive and neuro-biological implications of cigarette smoking and cognitive disorders. They found that cigarette smoking was associated with impaired learning and memory processing speed, working memory, general intellectual abilities, cognitive flexibility and executive functions. Congruently, in the present study we found that shisha smoking significantly impaired the cognitive performance compared to their control subjects.

Mechanism Contributing Shisha Smoking Induced Cognitive Dysfunctions

Literature revealed that smoking altered the brain structures such as thinner frontal cortical areas, frontal gray matter aberrations[25] decrease insula-based functional connectivity between orbitofrontal cortex, superior frontal gyrus, temporal lobe and insula[26]. Smoking also results in changes in blood counts[27] and decreases the cerebral flow rate in the anterior, middle and posterior cerebral arteries[28]. The most probable mechanism in line with shisha smoking and cognitive impairment is the presence of significant number of possibly cytotoxic compounds in shisha smoke including carbon monoxide, ketones, nitrosamines, aldehydes, dihydroxy benzenes, etc. These compounds may impair the neuronal and cellular membrane function of the cerebral hemisphere[29]. These compounds besides being cytotoxic, also promote oxidative damage to neuronal or glial cell organelles[30]. It has also been reported that, carbon monoxide concentration is significantly higher in smokers[31] and this raise is linked with reduced hemoglobin (Hb) concentrations and oxygen (O2) carrying capacity[32], ultimately reducing the efficiency of the mitochondrial respiratory chain mechanism[33]. Additionally, cigarette smoke contains higher levels of free radical species, which promote oxidative damage to neuronal cellular anatomy and physiology[34]. *In vivo* animal model experimental studies reported that brain tissue exposure to cigarette smoke significantly declines the membrane-bound ATPases, which changes ion homeostasis and enhances Na+ and Ca2+ concentration in the numerous neuronal cells[35], which is allied with neuronal injury or death[36]. Nicotine administration in juvenile rats evokes cell injury and brain function loss with significant effects in the hippocampus[37]. In the present study, we believe that shisha smoking impairs the cognitive functions in a similar fashion.

This is the first study to explore the impact of shisha smoking on cognitive functions. Subjects who were exposed to shisha smoking had significantly impaired cognitive functions. This novel research provides awareness to the public and health administrators about the effects of shisha smoking on cognitive functions.

Study Strengths and Limitation

This study has some strength and certain limitations. To our knowledge this is the first study added in the global literature on shisha smoking and cognitive functions. In this study the subject selection criteria, inclusion and exclusion strategies were highly standardized. We considered the age, height, weight, race, ethnicity, and educational level while using the CANTAB protocol to determine the cognitive functions. The limitation of the present study is the involvement of limited number of shisha smokers because most of the people use both shisha and cigarette and we selected only male shisha smokers. Therefore, this study has a relatively small sample size and because of the cross-sectional design of the study, we could not establish the exact causation.

Conclusions

The young adult shisha smokers show significant impairment in their cognitive functions compared to the matched control group. The results are convincing that young adult smokers should quit shisha smoking. Shisha smoking is allied with demonstrable abnormalities in the brain neuro-cognition. For a better understanding the factors linked with the neurocognitive abnormalities, longitudinal studies with comprehensive neurocognitive assessment combining neuroimaging of brain metabolites, brain function, morphology and genetic vulnerabilities are essential to understand. Electronic and print media should also play a role that shisha smoking should not be a part of cultural identity and societal acceptability and perception that it is less damaging or less addictive than cigarettes. Moreover, the educational and health officials must establish and implement the effective policies and visible health warnings on control shisha smoking.

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Conflict of interest

The authors declare no conflicts of interest.

References

1) Maziak W, Ward KD, Soweid RAA, Eissenberg T. Tobacco smoking using a waterpipe: a re-emerging strain in a global epidemic. Tob Control 2004; 13: 327-333.
2) Kiter G, Ucan ES, Ceylan E. Water-pipe smoking and pulmonary functions. Respirator Med 2000; 9: 891-894.
3) Sohn RM, Shishani BK, Okada RA, Froelicher ES. Approaches to smoking cessation in a cardiovascular population, 1st ed.; Ellen AD, Ed. Springer: New York, NY, USA, 2012; pp. 345-372.
4) Maziak W. The global epidemic of waterpipe smoking. Addict Behav 2011; 36: 1-5.
5) Anjum, Q, Ahmed F, Ashfaq T. Knowledge, attitude and perception of water pipe smoking among adolescents aged 14-19 years. J Pak Med Assoc 2008; 58: 312-317.
6) Jawaid A, Zafar AM, Rehman TU, Nazir MR, Ghafoor ZA, Afzal O, Khan JA. Knowledge, attitudes and practice of university students regarding waterpipe smoking in Pakistan. Int J Tuberc Lung Dis 2008; 12: 1077-1084.
7) Monn C, Kindler P, Meile A, Brandli O. Ultrafine particle emissions from waterpipes. Tob Control 2007; 16: 390-393.
8) Daher N, Saleh R, Jaroudi E, Sheheitli H, Badr T, Sepetdjian E, Al Rashidi M, Saliba N, Shihadeh A. Comparison of carcinogen, carbon monoxide, and ultrafine particle emissions from narghile waterpipe and cigarette smoking: sidestream smoke measurements and assessment of second-hand smoke emission factors. Atmos Environ 2010; 44: 8-14.
9) Sepetdjian E, Shihadeh, A, Saliba NA. Measurement of 16 polycyclic aromatic hydrocarbons in narghile waterpipe tobacco smoke. Food Chem Toxicol 2008; 46: 1582-1590.
10) Sepetdjian E, Abdul Halim R, Salman R, Jaroudi E, Shihadeh A, Saliba NA. Phenolic compounds in particles of mainstream waterpipe smoke. Nicotine Tob Res 2013; 15: 1107-1112.
11) Torrey CM, Moon KA, Williams DA, Green T, Cohen JE, Navas-Acien A, Breysse PN. Waterpipe cafes in Baltimore, Maryland: carbon monoxide, particulate matter and nicotine exposure. J Expo Sci Environ Epidemiol 2015; 25: 405-410.
12) Shafagoj YA, Mohammad FI. Levels of maximum endexpiratory carbon-monoxide and certain cardio vascular parameters following hubble-bubble smoking. Saudi Med J 2002; 23: 953-958.

13) Knishkowy B, Amitai Y. Water-pipe (narghile) smoking: an emerging health risk behavior. Pediatrics 2005; 116: 113-119.

14) Meo SA, AlShehri KA, AlHarbi BB, Barayyan OR, Bawazir AS, Alanazi OA, Al-Zuhair AR. See comment in PubMed Commons belowEffect of shisha (waterpipe) smoking on lung functions and fractional exhaled nitric oxide (FeNO) among Saudi young adult shisha smokers. Int J Environ Res Public Health 2014; 11: 9638-9648.

15) Luister FS, Stollo J, Zee PC, Walsh JK. Sleep: a health imperative. Sleep 2012; 35: 727-734.

16) Timothy C, Durazzo DJ, Meyerhoff S. Nixon J. Chronic cigarette smoking: implications for neurocognition and brain neurobiology. Int J Environ Res Public Health 2010; 7: 3760-3791.

17) Vajravelu HR, Gnanadurai TK, Krishnan P, Ayyavoo S. Impact of quantified smoking status on cognition in young adults. J Clin Diagn Res 2015; 9: 1-3.

18) Glass JM, Adams KM, Nigg JT, Wong MM, Puttler LI, Buu A, Jester JM. Fitzgerald HE, Zucker RA. Smoking is associated with neurocognitive deficits in alcoholism. Drug Alcohol Depend 2006; 82: 119-126.

19) Zhang XY, Tan YL, Chen DC, Tan SP, Yang FD, Zunta-Soares GB, Soares JC, Effects of cigarette smoking and alcohol use on neurocognition and BDNF levels in a Chinese population. Psychopharmacology (Berl) 2016: 233: 435-445.

20) Doiron M, Dupré, N, Langlois M, Provencher P. Smoking history is associated to cognitive impairment in Parkinson's disease. Aging Ment Health 2015; 28: 1-5.

21) Hotta R, Doi T, Shimada H, Makizako H, Yoshida D, Anan Y, Tsutsumimoto K, Uemura K, Park H, Suzuki T. Cigarette smoking and cognitive health in elderly Japanese. Am J Health Behav 2015; 39: 294-300.

22) Chamberlain SR, Odlaug BL, Schreiber LR, Grant JE. Association between tobacco smoking and cognitive functioning in young adults. Am J Addict 2012; Suppl 1: S14-19.

23) Sabia S , Elbaz A, Dugravot A, Head J, Shipley M, Hagger-Johnson G, Kivimaki M, Singh-Manoux A. Impact of smoking on cognitive decline in early old age: the Whitehall II cohort study. Arch Gen Psychiatry 2012; 69: 627-635.

24) Bashir S, Alghamd F, Alhussien A, Alohali M, Alatawi A, Almusned T, Habib SS. Effect of smoking on cognitive functioning in young Saudi adults. Med Sci Monit Basic Res 2017; 23: 31-35.

25) Akkermans SEA, van Rooij D, Rommelse N, Hartman CA, Hoekstra PJ, Franke B, Mennes M, Buitelaar JK. Effect of tobacco smoking on frontal cortical thickness development: a longitudinal study in a mixed cohort of ADHD-affected and -unaffected youth. Eur Neuropsychopharmacol 2017; 27: 1022-1031.

26) Zhou S, Xiao D, Peng P, Wang SK, Liu Z, Qin HY. Effect of smoking on resting-state functional connectivity in smokers: an fMRI study. Respirology 2017; 22: 1118-1124.

27) Tulgar YK, Cakar S, Tulgar S, Dalkilic O, Cakiroglu B, Uyanik BS. The effect of smoking on neutrophil/lymphocyte and platelet/lymphocyte ratio and platelet ındices: a retrospective study. Eur Rev Med Pharmacol Sci 2016; 20: 3112-3118.

28) Song Y, Kim JG, Cho HJ, Kim JK, Suh DC. Evaluation of cerebral blood flow change after cigarette smoking using quantitative MRA. PLoS One 2017; 12: e0184551.

29) Fowles J, Bates M, Noiton D. The chemical constituents in cigarettes and cigarette smoke: priorities for harm reduction. Epidemiology and Toxicology Group; Porirua, New Zealand, 2000; pp. 1-65.

30) Muscat JE, Kleinman W, Colosimo S, Muir A, Lazarus P, Park J, Richie JP. Enhanced protein glutathiolation and oxidative stress in cigarette smokers. Free Radic Biol Med 2004; 36: 464-470.

31) Deveci S, Deveci F, Acik Y, Ozan A. The measurement of exhaled carbon monoxide in healthy smokers and non-smokers. Resp Med 2004; 98: 551-556.

32) Macdonald G, Kondor N, Yousefi V, Green A, Wong F, Aquino-Parsons C. Reduction of carboxyhaemoglobin levels in the venous blood of cigarette smokers following the administration of carbogen. Radiother Oncol 2004; 73: 367-371.

33) Alonso JR, Cardellach F, Casademont J, Miro O. Reversible inhibition of mitochondrial complex IV activity in PBMC following acute smoking. Eur Respir J 2004; 23: 214-218.

34) Moriarty SE, Shah JH, Lynn M, Jiang S, Openo K, Jones DP, Sternberg P. Oxidation of glutathione and cysteine in human plasma associated with smoking. Free Radic Biol Med 2003; 35: 1582-1588.

35) Anbarasi K, Vanim G, Balakrishna K, Devi CS. Effect of bacoside A on membrane-bound ATPases in the brain of rats exposed to cigarette smoke. J Biochem Mol Toxicol 2005; 19: 59-65.

36) Xiao AY, Wei L, Xia S, Rothman S, Yu SP. Ionic mechanism of ouabain-induced concurrent apoptosis and necrosis in individual cultured cortical neurons. J Neurosci 2002; 22: 1350-1362.

37) Abreu-Villaca Y, Seidler FJ, Tate CA, Slotkin TA. Nicotine is a neurotoxin in the adolescent brain: critical periods, patterns of exposure, regional selectivity, and dose thresholds for macromolecular alterations. Brain Res 2003; 979: 114-128.