Original Investigation

Acute physiologic effects of secondhand smoke exposure in children

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Abstract

Introduction: We investigated the hypothesis that exhaled carbon monoxide (eCO), heart rate (HR), and blood pressure (BP) would increase acutely in exposed but not in unexposed children.

Methods: In a nonrandomized controlled design, parent–child dyads were assigned to groups based on exposure: child subjects, 7–18 years of age, exposed to smoking daily in the home (exposed) or with no household exposure (unexposed control). HR, BP, and eCO were measured before and after exposure to a parent smoking 1 cigarette (exposed) or a time-lapse equivalent (control). The primary analysis compared mean *acute* changes in physiological measures following intervention or control procedure.

Results: Forty-one dyads were enrolled. At baseline, no differences in child gender, race, ethnicity, HR, BP, lipids, or fasting glucose were noted between exposure groups. Following experimental or control procedures, the median change in eCO was similar between groups (−0.1 ppm exposed vs. 0.0 ppm unexposed, $p = .27$). Acute hemodynamic changes were also similar between exposed and unexposed children, respectively: (HR change 4.2 vs. 2.6 beats per minute, $p = .62$; systolic blood pressure change 0.08 vs. 0.41 mm Hg, $p = .91$; diastolic blood pressure 0.08 vs. 2.27 mm Hg, $p = .37$).

Discussion: This is the first study to report on acute physiologic changes of secondhand smoke exposure in children in a naturalistic setting. A single acute dose of passive smoke exposure in children did not alter the physiologic variables of HR or BP. Further in-home study using continuous monitoring is needed. Demonstration of acute effects may serve as clinical feedback to motivate parents to quit smoking.

Introduction

Nearly 40 million children in the United States are exposed to secondhand cigarette smoke (Department of Health and Human

Services, 2006), and the majority of this exposure takes place in the home. In 2002, 59.6% of nonsmoking children aged 3–11 years had serum cotinine levels greater than 0.05 ng/ml (Pirkle, Bernert, Caudill, Sosnoff, & Pechacek, 2006), which indicates recent exposure to smoke. Chronic exposure to secondhand smoke (SHS) in children is associated with a range of adverse health consequences, including increased rates of sudden infant death syndrome, asthma, pneumonia, and impaired lung growth. The Surgeon General Report of 2006 stated that even brief exposure to SHS can be harmful and, based on a synthesis of evidence, stated conclusively that there are no safe levels of exposure to SHS.

Much of the data pertaining to harms of SHS exposure have been extrapolated from analyses of direct smoking effects. The acute physiologic effects of standardized smoking have been well described in adults (Winniford, 1990). Specifically, cigarette smoking is associated with an acute increase in heart rate (HR) and systolic blood pressure (SBP), and a dose–response relationship exists between nicotine level and the degree of rise in these hemodynamic factors (Spohr et al., 1979). While these effects have been confirmed by other studies (Benowitz, Hansson, & Jacob, 2002; Perkins, Epstein, Jennings, & Stiller, 1986), few studies have looked at acute physiologic changes related to SHS exposure (Corwin, McCoy, Whetzel, Ceballos, & Klein, 2006; Mahmud & Feely, 2004; McMurray, Hicks, & Thompson, 1985; Otsuka et al., 2001; Pimm, Shephard, & Silverman, 1978) and none, to our knowledge, have reported the physiologic effects of SHS exposure in children.

The current study was conducted to investigate the acute physiologic changes related to secondhand exposure in children in a naturalistic setting. By studying exposure in conditions most closely mimicking household exposure, the investigators aimed to generate findings with clinical implications for parental smokers. Specifically, we investigated the hypothesis that exhaled carbon monoxide (eCO), HR, and blood pressure (BP) would increase acutely in exposed but not in unexposed children.

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Methods

Study design

A nonrandomized controlled design was used to examine the physiologic effects of acute exposure to tobacco smoke in a group of children who were chronically exposed to smoking daily in the home as compared with a control group of children with no household smoke exposure. Acute smoke exposure involved the child sitting within 7 feet of the parent while the parent smoked one cigarette. The rationale for choosing exposure to a single cigarette is based on previous study findings of physiologic changes related to this unit of exposure (Kool, Hoeks, Struijker Boudier, Reneman, & Van Bortel, 1993; Mahmud & Feely, 2003; Rubenstein, Jesty, & Bluestein, 2004). The study was designed to measure changes in eCO, HR, and SBP and diastolic blood pressure (DBP). The physiologic variables were measured before and after acute smoke exposure and compared with changes in an unexposed control group who experienced a time-lapse equivalent to the duration of the acute smoke exposure procedure.

Subjects

The current study was conducted as part of a larger ongoing community-based research study on health disparities in heart disease (Aiyer et al., 2007); recruitment strategies focused on communities with disproportionate rates of heart disease/risk factors. Study subjects, recruited at community health fairs and through referral from local pediatric offices in collaboration with the community-based research study, were healthy parent-child dyads who fit the study criteria for one of two study groups, exposed and unexposed. "Parent" subjects had to be at least 18 years old, be the parent or guardian of, and live at home with the child subject. Child participants were between 5 and 17 years of age. The study was approved by the Institutional Review Board of the University of Pittsburgh.

Exposed group. The exposed group consisted of parent-child dyads in which the parent subject reported current daily smoking and a history of smoking at least 100 cigarettes over his/her lifetime, and the child subject was a self-reported nonsmoker. In order to confirm eligibility criteria for exposure group assignment for teenage children, a confidential written screening questionnaire was administered separately to children at least 12 years of age. Parent and child smoking status prior to intervention was biochemically verified during baseline testing of eligible consenting subjects using urine cotinine as a marker for smoke exposure.

Unexposed (control) group. The unexposed group consisted of parent-child dyads in which the parent and child were self-reported nonsmokers. For the parent, nonsmoking was defined as no current smoking or no smoking within the past year. For the child, this was defined as not living in a home with any smokers.

Exclusion criteria. Parents or children with known asthma or diabetes and adults with SBPs above 170 or DBP greater than 110 were excluded. Mothers who were pregnant or suspected they were pregnant were likewise excluded.

Procedures

Baseline measures

Parent participants completed baseline health history and smoke exposure questionnaires; a tobacco use and exposure

survey was separately administered to teen subjects 12 years or older. Exhaled carbon monoxide (Micro Medical Ltd, Chatham, Kent, UK) and urine cotinine (Nicalert [TM], Nymox Corp., Hasbrouck Heights, NJ) values were collected on all child participants for biologic confirmation of smoke exposure. For eCO measurement, subjects were asked to take a deep breath, hold it for 10 s, and exhale forcefully into the mouthpiece of the CO monitor. A research assistant recorded the average of three such exhalations (Jarvis, Belcher, Vesey, & Hutchison, 1986; Jarvis, Tunstall-Pedoe, Feyerabend, Vesey, & Saloojee, 1987). Of the 41 subjects, eCO readings from 3 were unavailable: in 2 cases, established protocol was not followed, and in 1 case, the parent BP was too high to continue with study procedures. For urine cotinine strip measurements, results were interpreted as follows: 0 corresponds with no exposure, 1–2 corresponds with SHS exposure, and values of 3–6 correspond with direct smoking. HR was measured by palpation and BP measured manually with an appropriate-sized cuff.

Testing procedures

Following consent and blood testing, subject dyads were moved to a designated hotel testing room: a smoking room for the exposed group and nonsmoking room for the unexposed group. Hotel rooms were standard size, and room temperatures were maintained at 72 °F. After a standardized time (15 min) to acclimate to the room, Time 1 measures of eCO, HR, and BP were made on both the parent and the child. These measures were repeated 5 min after the smoking exposure in the exposed group or 10 min time lapse for the (control) unexposed group.

Acute smoking exposure procedure

Exposed parents were instructed to smoke one cigarette of their own brand at a measured distance (within 7 feet) from their child; during the smoking/time-lapse period, both exposed and unexposed subjects were instructed to remain in the same positions as during the testing procedures and were allowed to engage in conversation or watch TV.

Statistical methods

Baseline descriptive statistics including means, SD, and frequencies were used to compare the two study groups prior to intervention in terms of demographic, physiologic, and biochemical variables. This included comparisons of parents between the two groups as well as children between the two groups. For continuous variables, Student's *t* tests or the Wilcoxon's rank sum tests were used; for categorical variables, Fisher's exact test of proportions was used.

The primary analysis consisted of comparing mean *acute* changes in physiological measures (HR, SBP, DBP, and CO) following parent intervention (i.e., smoking one cigarette vs. no parental smoking). Analysis of covariance (ANCOVA) was used for each physiological outcome adjusting for child age, gender, and body mass index. In addition, in secondary analyses aimed more at evaluating effects of *chronic* exposure, alternate definitions of parent exposure status were assessed based on questionnaire information and physiological measures at baseline. This included (a) self-reported smoking frequency (never, some days, and every day), (b) hours child was in a room with a smoker (never, <3 hr/week, ≥3 hr/week but <1 hr/day, ≥1 hr/day but <3 hr/day, and >3 hr/day), (c) household rules about smoking (no one permitted, smoking permitted in some rooms/

times, and smoking permitted anywhere), (d) urine cotinine (0 or 1, 2–5, and 6), and (e) eCO (<4.3, 4.3 to <13, and ≥13). For these analyses, ANCOVA was used with a linear test of trend for each outcome across exposure levels.

By study design, we sought a target sample size of 20 children exposed and 20 children not exposed to SHS to compare acute physiological changes. Assuming two-sided Type I error rate of 0.05 and no correction for multiple comparisons, this corresponded to 80% power to detect a large effect size of 0.91 or higher.

Results

Sample description

Forty-one parent–child dyads were enrolled and completed all study measures. First, to verify accurate chronic exposure classification via biochemical analyses, distributions of parent and children urine cotinine levels prior to intervention were examined. “Exposed” parents had a median urine cotinine level of 6.0 ng/ml compared with 1.0 ng/ml in “unexposed” parents ($p < .0001$). Importantly, the distributions between the two groups did not overlap, indicating accurate biochemical classification of parent smokers and nonsmokers. Similarly, levels of urine cotinine were much lower, on average ($p = .003$), in unexposed compared with exposed children, albeit less pronounced than the differences observed in parents. These observations indicate that children of parents who smoked cigarettes were indeed recently exposed to smoke. Prior to intervention, mean eCO levels (ppm) were 23.0 ± 14.0 in exposed parents compared with 4.1 ± 2.8 in unexposed parents ($p < .0001$), again validating accurate classification of smoking status in parents. In contrast to urine cotinine levels in child subjects, mean eCO levels (ppm)

at baseline were similar in exposed (3.2 ± 3.1) and unexposed children (3.6 ± 3.5 , $p = .87$). This suggests similar smoke exposure to both groups of children immediately prior to (e.g., morning of) the study intervention. According to confidential self-report questionnaires completed by teen subjects (those 12–17 years), none of the teen subjects reported smoking in the last 30 days regardless of exposure history. However, two child subjects in the exposed group were found to have elevated baseline urine cotinine levels (values of 3 and 5).

Baseline characteristics of the parent–child dyads are shown in Table 1. Nearly all parents were female and approximately 66% were Black. Among these parents, age, race, ethnicity, lipid levels, HR, and BP were similar by exposure (smoking) status. Although not statistically significant, exposed parents tended to be thinner than unexposed parents, yet had nominally higher fasting glucose levels. Among child participants, gender, race, ethnicity, lipids, HR, BP, and fasting glucose were similar by exposure status. However, unexposed children were, on average, 2 years older than exposed children while significantly heavier.

Acute physiological changes following smoking

After smoking one cigarette, exposed parents experienced a median change of 8.5 ppm in eCO compared with a median change of 0.0 ppm in unexposed parents ($p = .0002$). The change in eCO was highly variable in exposed parents following smoking compared with very little change (as expected) in unexposed parents. In contrast to parents, the median change in eCO was similar between exposed and unexposed children (-0.1 ppm vs. 0.0 ppm, $p = .27$; Figure 1) and did not vary by gender. Nonetheless, the variability in change in eCO was greater in exposed compared with unexposed children (SD of 2.5 vs. 1.1 ppm).

Table 1. Baseline characteristics of parents and children by exposure status

Baseline characteristic	Parents			Children		
	Exposed (N = 20)	Unexposed (N = 21)	p value	Exposed (N = 20)	Unexposed (N = 21)	p value
Age in years (mean, SD)	40.2 (8.3)	41.9 (7.4)	0.49	10.6 (2.8)	12.5 (3.6)	0.06
Female gender (%)	95.0	85.7	0.61	45.0	47.6	1.0
Race (%)			1.0			1.0
Black	70.0	61.9		70.0	66.7	
White	30.0	33.3		25.0	28.6	
Other	0.0	4.8		5.0	4.8	
Hispanic (%)	10.5	9.5	1.0	0.0	4.8	1.0
Body mass index	29.9 (9.4)	34.4 (9.1)	0.13	19.3 (4.0)	23.7 (7.1)	0.02
Total cholesterol, mg/dl (mean, SD)	199.6 (41.8)	192.8 (32.9)	0.57	158.4 (36.3)	164.5 (37.2)	0.62
HDL cholesterol, mg/dl (mean, SD)	52.6 (13.1)	52.0 (16.9)	0.91	46.4 (18.2)	47.6 (14.0)	0.82
LDL cholesterol, mg/dl (mean, SD)	116.7 (36.1)	117.2 (22.9)	0.96	94.8 (31.2)	102.7 (35.2)	0.54
Triglycerides, mg/dl (mean, SD)	114.3 (88.2)	117.2 (15.8)	0.36	75.0 (33.3)	93.4 (63.6)	0.66
Heart rate, beats per minute (mean, SD)	66.8 (8.0)	67.7 (10.4)	0.77	66.3 (8.8)	69.8 (8.4)	0.22
Systolic blood pressure, mmHg (mean, SD)	127.4 (28.4)	123.8 (16.8)	0.62	97.8 (12.2)	103.7 (12.9)	0.14
Diastolic blood pressure, mm Hg (mean, SD)	83.6 (16.3)	82.5 (11.9)	0.80	65.7 (9.2)	72.0 (10.7)	0.05
Fasting glucose, mg/dl (mean, SD)	105.2 (44.5)	95.3 (10.1)	0.79	84.5 (5.7)	87.0 (6.2)	0.25

Note. HDL = high density lipoprotein; LDL = low density lipoprotein.

Figure 1. Distributions of change in exhaled carbon monoxide (ppm) among children based on parent exposure status. Exposed parents smoked one cigarette; unexposed parents did not smoke.

Consistent with similar changes in eCO, acute hemodynamic changes were not significantly different between exposed and unexposed children (Table 2). Specifically, absolute levels of HR, BP, and eCO were relatively similar both before and after intervention and irrespective of exposure status. Spearman rank correlations among all children for acute changes in eCO were as follows: HR ($r_s = .16, p = .36$), SBP ($r_s = .11, p = .50$), and DBP ($r_s = -.09, p = .59$). When assessed by gender, correlation between eCO and SBP following exposure was significantly higher among male compared with female child subjects ($r_s = .56$ male, $-.22$ female, $p = .02$). Collectively, these data indicate weak relationships between acute hemodynamic changes in children following brief exposure to SHS.

Assessment of chronic smoke exposure in parents

According to self-report data, of the parents in the exposed group (smokers), 95% reported daily smoking (an enrollment criteria), 25% smoked at least one pack per day, and 50% reported smoking within 5 min of waking. Only 10% of the

exposed group had explicit household rules prohibiting smoking in the home compared with 83.3% of the unexposed group, and 45% of smoking parents allowed smoking anywhere in their home compared with none of the unexposed.

Table 3 shows adjusted mean physiological values among both exposed and unexposed children based on completed selected measures of chronic exposure to SHS. As seen, measures of HR, BP, and eCO in children were seemingly unrelated to how often parents smoked, the average time in a room with a smoker(s), household rules about smoking, and parent biological measures of urine cotinine and eCO. The only apparent exception was a borderline association between levels of parent eCO and child eCO ($p = .05$).

Discussion

Our study is the first to report on the acute physiologic changes of SHS exposure in children in a naturalistic setting. We used an experimental model of a child's exposure to a parent's smoking

Table 2. Adjusted mean physiological measures among children before and after acute parent smoking intervention^a

Physiological measure	N	Intervention group (N = 20)			Control group (N = 21)			p value (difference)
		Time 1	Time 2	Difference	Time 1	Time 2	Difference	
Heart rate	36	65.4	70.7	4.2	70.7	72.7	2.6	0.62
SBP	40	100.0	100.3	0.08	101.0	101.3	0.41	0.91
DBP	40	66.7	67.7	0.08	70.9	72.4	2.27	0.37
Exhaled carbon monoxide	37	3.9	3.7	-0.12	3.0	3.1	0.16	0.77

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure.

^aAdjusted for child age, gender, and body mass index percentile. The difference scores are also adjusted for Time 1 values. The p value compares the adjusted mean differences between the Intervention and Control groups.

Table 3. Adjusted mean physiological measures among children by parent and home smoking status (chronic exposure)^a

Measure of parent/home smoking exposure	HR (N = 37)	SBP (N = 40)	DBP (N = 40)	Exhaled CO (N = 38)
How often smoke cigarettes				
Never	70.7	100.9	70.7	3.0
Some days	67.6	111.3	84.9	1.6
Every day	65.3	99.6	66.0	4.0
<i>p</i> value (trend)	0.10	0.72	0.16	0.36
Average hours/day child in room w/smoker				
Never	71.2	98.7	67.0	3.9
Less than 3 hr/week	68.4	100.3	72.4	1.7
Less 1 hr/day, ≥ 3 hr/week	69.0	100.6	69.8	2.5
Less 3 hr/day, ≥ 1 hr/day	67.7	103.1	69.3	5.1
More than 3 hr/day	61.2	101.1	66.0	4.3
<i>p</i> value (trend)	0.10	0.58	0.69	0.25
Household rules about smoking				
No one allowed to smoke in home	68.6	98.7	68.9	2.8
Smoking permitted some rooms/times	68.3	103.1	71.8	3.5
Smoking permitted anywhere	66.9	100.2	64.0	4.7
<i>p</i> value (trend)	0.70	0.74	0.27	0.17
Urine cotinine				
0 or 1	70.3	97.3	67.0	2.8
2–5	70.6	100.1	72.0	2.0
6	65.5	102.8	67.5	3.7
<i>p</i> value (trend)	0.32	0.31	0.93	0.43
Exhaled carbon monoxide				
<4.3	73.2	100.4	67.9	2.1
4.3 to <13	65.8	100.3	71.1	3.2
13 or more	67.0	101.9	67.3	4.0
<i>p</i> value (trend)	0.12	0.74	0.88	0.05

Note. HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure.

^aAdjusted for child age, gender, and body mass index percentile.

to investigate the acute effects of passive smoke exposure. Our results indicate that there were no significant changes in eCO, HR, or BP among child subjects after acute exposure to one cigarette smoked by their parent in a controlled environment. Given the body of literature on the adverse effects of acute smoking exposure in adults, these negative findings were unanticipated. Nevertheless, the importance of these findings is particularly notable because of the strengths of our study design. Specifically, the study was carefully conducted to avoid confounding factors that could reduce the effect size of acute exposure. Exposure groupings (exposed to SHS vs. not exposed) were accurately classified according to a combination of history and biochemical verification of parental smoking status. In addition to controlling for exposure effect and the effect of repeating physiologic measures, we controlled for risk factors that could affect HR or BP, including room temperature, acclimation to study environment, and cardiovascular risk variables. The child exposure groups were comparable for these factors, except that unexposed children were, on average, 2 years older than exposed children while significantly heavier. The study was further strengthened by each subject acting as his/her own control. Therefore, in light of the study strengths, these findings raise questions regarding the possibility of a unique response to SHS exposure in children as opposed to that of adults. Based on these findings, we postulate that children may be resistant to

the acute toxic effect of a limited dose of acute SHS exposure or need a higher or more subacute or chronic exposure to affect physiologic variables.

Alternative explanations may account for the lack of physiologic changes measured in response to acute SHS exposure in this study. This study's focus on household smoke exposure may have underestimated each subject's total SHS exposure, thereby accounting for the similar eCO levels between the two groups at baseline and a lack of difference in response to acute exposure. Alternatively, the actual dose of the acute smoke exposure to the subjects may have been less than expected due to factors consistent with a nonlaboratory setting. For example, in order to achieve consistent room temperatures (72 degrees) for each study visit, necessary room ventilation or air-conditioning may have contributed to reducing the actual smoke exposure. Nevertheless, our experimental setting is more consistent with the home environment of children who are exposed to SHS by their parents. Likewise, a higher dose of exposure may be required to effect physiologic changes (Mucha, Mutz, Stephan, & Pauli, 1996); most previous studies of acute exposure involved a more concentrated exposure, involving exposure to the smoke from at least two cigarettes (Hausberg, Mark, Winniford, Brown, & Somers, 1997; McMurray et al., 1985).

While the current study did not find changes in children's eCO following acute exposure to parental smoking, secondary findings related to measures of exposure are further notable. While the sample size is small, and most measures did not differ by gender, the differential correlation of eCO and SBP by gender confirms a previous finding by Mahmud, which suggested an increased susceptibility to the hemodynamic effects of SHS among males (Mahmud & Feely, 2004). Taken together, these findings warrant further study in larger samples. Additionally, we found that urine cotinine levels are higher in children whose parents smoke but that both biomarkers of exposure in children (eCO and cotinine) were highly variable and may suggest varied host susceptibility to SHS effects in children. These results in exposed children support the findings from adult exposure studies and extend our knowledge of the effects of SHS from adults to children. In the absence of predictors of SHS susceptibility, these findings further support the recommendations of the Surgeon General (Department of Health and Human Services, 2006) that there is no safe level of SHS exposure.

Few previous studies exist on effects of acute exposure and all have been conducted on adult populations, primarily in controlled laboratory environments. While many of these studies have found acute changes in physiologic parameters associated with SHS exposure (Heiss et al., 2008; McMurray et al., 1985; Otsuka et al., 2001; Pimm et al., 1978), others have not (Hausberg et al., 1997; Rummel, Crawford, & Bruce, 1975). Importantly, none of these studies investigated the effects of acute SHS exposure in children.

In addition to carefully controlled laboratory studies, where SHS exposure is accomplished by pumping in side-stream smoke from burning cigarettes to a chamber, few studies of acute exposure have been conducted in *naturalistic settings*. In a nonsmoking area abutting smoking-allowed areas (bingo hall), both smokers and nonsmokers exposed to passive smoke had increase in eCO (Olshansky, 1982). Pope et al. (2001) reported increased HR variability among 16 nonsmokers during periods exposed to smoking areas in a commercial airport. Most naturalistic studies have examined physiologic effects of chronic exposure (Corwin et al., 2006; Felber Dietrich et al., 2007). Our study was the first to report the effects of acute passive smoking in children in a naturalistic setting.

Limitations

In addition to the aforementioned issues, the study has several limitations. The choice and timing of outcome measures may have been too narrow. That is, HR variability or continuous BP measurements including a longer interval from the exposure may have been more sensitive indicators of physiologic effect. Exhaled CO may not be a reliable marker for measuring acute exposure of SHS in children (Matt, Bernert, & Hovell, 2008; Matt et al., 2007). Likewise, while limited investigative use of the Micromed eCO monitor demonstrated expected elevations in eCO among adolescents exposed to household SHS (Gourgoulanis, Gogou, Hamos, & Molyvdas, 2002), use of this device in younger children may not be as reliable. The sample size was calculated to detect acute change in continuous variables but was not powered to detect differences variables related to chronic exposure.

Conclusion

A single acute dose of passive smoke exposure in children did not alter the physiologic variables of HR or BP. Given the inconsistent findings to date regarding effects of acute exposure, and the relative paucity of data in children, more study is clearly needed. In-home study with continuous monitoring of exposure and effects could advance our understanding of children's susceptibility to acute and chronic effects. Demonstration of acute effects may ultimately serve as a clinical feedback tool to motivate parents to quit smoking.

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Declaration of Interests

None declared.

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References

- Aiyer, A. N., Kip, K. E., Marroquin, O. C., Mulukutla, S. R., Edmundowicz, D., & Reis, S. E. (2007). Racial differences in coronary artery calcification are not attributed to differences in lipoprotein particle sizes: The Heart Strategies Concentrating on Risk Evaluation (Heart SCORE) Study. *American Heart Journal*, 153, 328–334.
- Benowitz, N. L., Hansson, A., & Jacob, P., III (2002). Cardiovascular effects of nasal and transdermal nicotine and cigarette smoking. *Hypertension*, 39, 1107–1112.
- Corwin, E. J., McCoy, C. S., Whetzel, C. A., Ceballos, R. M., & Klein, L. C. (2006). Risk indicators of metabolic syndrome in young adults: A preliminary investigation on the influence of tobacco smoke exposure and gender. *Heart Lung*, 35, 119–129.
- Department of Health and Human Services. (2006). *The health consequences of involuntary exposure to tobacco smoke: A report of the Surgeon General*. Atlanta, GA: U.S. Department of Health

and Human Services, Centers for Disease Control and Prevention. Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion. Office on Smoking and Health.

Felber Dietrich, D., Schwartz, J., Schindler, C., Gaspoz, J. M., Barthelemy, J. C., Tschopp, J. M., et al. (2007). Effects of passive smoking on heart rate variability, heart rate and blood pressure: An observational study. *International Journal of Epidemiology*, 36, 834–840.

Gourgoulisanis, K. I., Gogou, E., Hamos, V., & Molyvdas, P. A. (2002). Indoor maternal smoking doubles adolescents' exhaled carbon monoxide. *Acta Paediatrica*, 91, 712–713.

Hausberg, M., Mark, A. L., Winniford, M. D., Brown, R. E., & Somers, V. K. (1997). Sympathetic and vascular effects of short-term passive smoke exposure in healthy nonsmokers. *Circulation*, 96, 282–287.

Heiss, C., Amabile, N., Lee, A. C., Real, W. M., Schick, S. F., Lao, D., et al. (2008). Brief secondhand smoke exposure depresses endothelial progenitor cells activity and endothelial function: Sustained vascular injury and blunted nitric oxide production. *Journal of the American College of Cardiology*, 51, 1760–1771.

Jarvis, M. J., Belcher, M., Vesey, C., & Hutchison, D. C. (1986). Low cost carbon monoxide monitors in smoking assessment. *Thorax*, 41, 886–887.

Jarvis, M. J., Tunstall-Pedoe, H., Feyerabend, C., Vesey, C., & Saloojee, Y. (1987). Comparison of tests used to distinguish smokers from nonsmokers. *American Journal of Public Health*, 77, 1435–1438.

Kool, M. J., Hoeks, A. P., Struijker Boudier, H. A., Reneman, R. S., & Van Bortel, L. M. (1993). Short- and long-term effects of smoking on arterial wall properties in habitual smokers. *Journal of the American College of Cardiology*, 22, 1881–1886.

Mahmud, A., & Feely, J. (2003). Effect of smoking on arterial stiffness and pulse pressure amplification. *Hypertension*, 41, 183–187.

Mahmud, A., & Feely, J. (2004). Effects of passive smoking on blood pressure and aortic pressure waveform in healthy young adults—Influence of gender. *British Journal of Clinical Pharmacology*, 57, 37–43.

Matt, G. E., Bernert, J. T., & Hovell, M. F. (2008). Measuring secondhand smoke exposure in children: An ecological measurement approach. *Journal of Pediatric Psychology*, 33, 156–175.

Matt, G. E., Hovell, M. F., Quintana, P. J., Zakarian, J., Liles, S., Meltzer, S. B., et al. (2007). The variability of urinary cotinine

levels in young children: Implications for measuring ETS exposure. *Nicotine & Tobacco Research*, 9, 83–92.

McMurray, R. G., Hicks, L. L., & Thompson, D. L. (1985). The effects of passive inhalation of cigarette smoke on exercise performance. *European Journal of Applied Physiology and Occupational Physiology*, 54, 196–200.

Mucha, R. F., Mutz, G., Stephan, E., & Pauli, P. (1996). Smoking produces a smaller increase in heart rate in the natural smoking environment than in the laboratory. *Drug and Alcohol Dependence*, 42, 209–215.

Olshansky, S. J. (1982). Is smoker/nonsmoker segregation effective in reducing passive inhalation among nonsmokers? *American Journal of Public Health*, 72, 737–739.

Otsuka, R., Watanabe, H., Hirata, K., Tokai, K., Muro, T., Yoshiyama, M., et al. (2001). Acute effects of passive smoking on the coronary circulation in healthy young adults. *Journal of the American Medical Association*, 286, 436–441.

Perkins, K. A., Epstein, L. H., Jennings, J. R., & Stiller, R. (1986). The cardiovascular effects of nicotine during stress. *Psychopharmacology*, 90, 373–378.

Pimm, P. E., Shephard, R. J., & Silverman, F. (1978). Physiological effects of acute passive exposure to cigarette smoke. *Archives of Environmental Health*, 33, 201–213.

Pirkle, J. L., Bernert, J. T., Caudill, S. P., Sosnoff, C. S., & Pechacek, T. F. (2006). Trends in the exposure of nonsmokers in the U.S. population to secondhand smoke: 1988–2002. *Environmental Health Perspectives*, 114, 853–858.

Pope, C. A., III, Eatough, D. J., Gold, D. R., Pang, Y., Nielsen, K. R., Nath, P., et al. (2001). Acute exposure to environmental tobacco smoke and heart rate variability. *Environmental Health Perspectives*, 109, 711–716.

Rubenstein, D., Jesty, J., & Bluestein, D. (2004). Differences between mainstream and sidestream cigarette smoke extracts and nicotine in the activation of platelets under static and flow conditions. *Circulation*, 109, 78–83.

Rummel, R. M., Crawford, M., & Bruce, P. (1975). The physiological effects of inhaling exhaled cigarette smoke in relation to attitude of the nonsmoker. *Journal of School Health*, 45, 524–529.

Spohr, U., Hofmann, K., Steck, W., Harenberg, J., Walter, E., Hengen, N., et al. (1979). Evaluation of smoking-induced effects on sympathetic, hemodynamic and metabolic variables with respect to plasma nicotine and COHb levels. *Atherosclerosis*, 33, 271–283.

Winniford, M. D. (1990). Smoking and cardiovascular function. *Journal of Hypertension Supplement*, 8, S17–S23.