International Journal of Biomedical and Advance Research
ISSN: 2229-3809 (Online); 2455-0558 (Print)
Journal DOI: https://doi.org/10.7439/ijbar
CODEN: IJBABN **Original Research Article**

Cross-sectional association between cigarette smoking and inflammation, lipid profile and anthropometric parameters

Abinet Teshome[1] and Natesan Gnanasekaran[*2]

[1]*Deprtment of Biomedical Sciences, College of Health Sciences, School of Medicine, Arba Minch University Ethiopia*
[2]*Department of Medical Biochemistry, School of Medicine, College of Health Sciences, Addis Ababa University, Ethiopia*

Abstract

Background: Smoking is a noteworthy hazard factor of cardiovascular vascular diseases (CVD) and, the portrayal of the ailment as a chronic low-grade inflammatory condition is currently to a great extent acknowledged for CVD. The inflammation itself is on the causal pathway connecting cigarette smoking to CVD outside of these inflammatory states.
Objective: To determine the cross-sectional association between cigarette smoking and inflammation, lipid profile and anthropometric parameters
Methods: A community-based, cross-sectional study, 99 individuals were enrolled. Participants divided in to two groups' smokers 50 and non-smokers 49. BMI, Blood pressure, plasma lipid profiles and hs-CRP were measured.
Results: There were no significant in body mass index, systolic blood pressure and diastolic blood pressure between smokers and non-smokers. However, the elevated levels of total cholesterol ($p = 0.001$), LDL-C ($p = 0.025$) triglycerides ($p=0.001$), HDL-C ($p= 0.028$) and hs-CRP ($p=0.001$) were observed in smokers.
Conclusions: In conclusions, we show here that cigarette smoking was a hazard factor for inflammation. This impact might be interceded to some extent by an increase in the hs-CRP level. Further examination utilizing a longitudinal study and a larger populace study is important to affirm the conceivable clinical implication of this present outcome.
Keywords: Blood pressure; vascular complications; hs-CRP; Inflammation; Lipid profile; Smoking.

***Correspondence Info:**
Dr. Natesan Gnanasekaran M.Sc; M. Phil; PhD
Department of Medical Biochemistry,
School of Medicine, College of Health Sciences
Addis Ababa University,
Ethiopia P.O. Box 9086. Ethiopia

***Article History:**
Received: 02/12/2019
Revised: 30/01/2020
Accepted: 30/01/2020
DOI: https://doi.org/10.7439/ijbar.v11i2.5322

QR Code

How to cite: Teshome A and Gnanasekaran N. Cross-sectional association between cigarette smoking and inflammation, lipid profile and anthropometric parameters. *International Journal of Biomedical and Advance Research* 2020; 11(02): e5322. Doi: 10.7439/ijbar.v11i2.5322 Available from: https://ssjournals.com/index.php/ijbar/article/view/5322

1. Introduction

Tobacco utilize is the most common reason for preventable death around the world. It is in charge of one out of ten adult deaths or one in each two smoker's death. In the twentieth century, tobacco-related sicknesses caused the deaths of 100 million individuals and it is anticipated that one billion individuals will die in the 21st century from the tobacco-related ailments. Various unfavorable impacts are related to tobacco utilize, including coronary heart disease, stroke, peripheral arterial disease, abdominal aortic aneurysm, chronic obstructive pulmonary disease and cancer [1]. According to the World Health Organization (WHO) report 2011, in Ethiopia 34% peoples suffer from non-communicable diseases, a cardiovascular disease prevalence of 15%, malignancy and cancer and chronic obstructive pulmonary disease 4% each, and diabetes mellitus 2% [2]. In Ethiopia a moderately low predominance of cigarette smokers, with fewer than 10% of guys, and under 1% of females, being smokers [3]. In one rural town in eastern Ethiopia, 28% of the peoples were smoked daily [4]. Other investigations have demonstrated that the utilization or abuse of addictive substances, for example, cigarettes, liquor, and khat increasingly common in Ethiopia [5]. Moreover, there is proof that smokers have an increased danger of HIV infection and tuberculosis (TB), more regrettable seriousness of infection manifestations, and expanded mortality of HIV and TB [6]. Tobacco smoke

is an intricate blend of more than 5000 to 7000, chemical compounds, including around 70 known cancer-causing agents [7,8]. A few examinations have additionally demonstrated that different components of tobacco smoke likewise have proinflammatory impacts [9].

To be sure, a high burden of inflammation has been appeared to distinguish smokers at high hazard for CVD. Systemic immunologic modifications by smoking were found to correlate with local processes in the vascular wall, portrayed by inflammation and increased articulation of matrix metalloproteinases [10]. Inflammation is known to establish a basic component in atherogenesis [11]. One of the most imperative hazard factors for CVDs, smoking, impacts and enacts the immune system both systemically and locally[12]. A generally acknowledged marker for the event of aggravation is hoisted serum C-receptive protein, which was observed to be expanded in smokers. Interestingly, the systemic proinflammatory impact of smoking isn't limited to dynamic smokers. Likewise peoples exposed to secondhand smoke show increased concentrations of inflammatory markers [13]. In a past report in the Multi-Ethnic Study of Atherosclerosis (MESA) associate, distinguished hsCRP as the sensitive marker of subclinical cardiovascular damage contrasted with markers of thrombosis, subclinical myocardial damage, endothelial damage and vascular function [14,15]. The point of this investigation was, specifically, to scan for the relationship among smoking and inflammatory marker such as hs-CRP in a representative population-based example in Adama (Nazret) Ethiopia.

2. Methods

The examination was led in Adama Hospital Medical College, Adama (Nazret), Ethiopia A community-based, cross-sectional study was done, involving99 people who were randomly divided into two groups: smokers (50 individuals) and non-smokers (49 people). The survey included questions about history, of the people. The number of pack-years of cigarettes smoked was computed from the total number of years spent smoking multiplied by the number of packs (one pack=20 cigarette) smoked every day. Arterial blood pressure was estimated after subjects rested in a sitting position for no less than 5 minutes. Blood pressure was taken utilizing standard mercury sphygmomanometer by an expert medical nurse. Weight and height were estimated utilizing a standard weighing scale with height scale connected. Body Mass Index (BMI) was calculated as the weight in kilogram divided by the square of the height in meters. For biochemical investigation, 10 ml of blood was collected and the plasma was separated. Lipid profile of the plasma was done by an autoanalyzer (Humastar300) based on Johns Hopkins Laboratory Procedure Manual (2003-2004). hs-CRP was dissected by immuneprecipitation with the latex-improved turbidimetric test (Cobas Integra 800 Analyzer, Roche Diagnostics).

2.1 Statistical analysis:

The data was analyzed using SPSS version 20 and the values were analyzed using student t test and confidence interval of 95% were taken, with p-values of less than 0.05 considered to be statistically significant.

2.2 Ethical Consideration

The research proposal was reviewed and ethically approved by Departmental Research Ethics Review Committee (DRERC) of the Department of Medical Biochemistry, School of Medicine, College of Health Sciences, and Addis Ababa University with Ref. No. SOM/ BCHM /012/2006. A letter of invitation was written and distributed for residents of Town of Adama and those willing to partake in the research signed letter of consent. The confidentiality of both the information provided in the questionnaire, anthropometric measurements and laboratory assay results were strictly maintained.

3. Results

The frequency distribution of systolic and diastolic blood pressure in all of the sample population is shown in figure 1 & 2. The results of systolic and diastolic blood pressure showed only numerical differences but not statistically different between the smokers and nonsmokers. Similarly BMI also showed no statistical difference between smokers and non smokers (Table 1).

Serum lipid profile of the smoking group, when compared with nonsmokers, had elevated the levels of total cholesterol, triglyceride, low-density lipoprotein, high-density lipoprotein and hs-CRP was observed (Table 2). The hsCRP demonstrated the greatest percent elevation with smoking and burden among current smokers.

Table 1: BMI, systolic and diastolic blood pressure in smokers and non smokers

Blood Pressure	Smokers	Non smokers	p- value
Systolic	117.3 ± 15.22	112.4 ± 8.7	0.177
Diastolic	77.8 ± 12.2	75.35 ± 8.84	0.216
BMI	20.61±2.43	20.84 ± 2.94	0.657

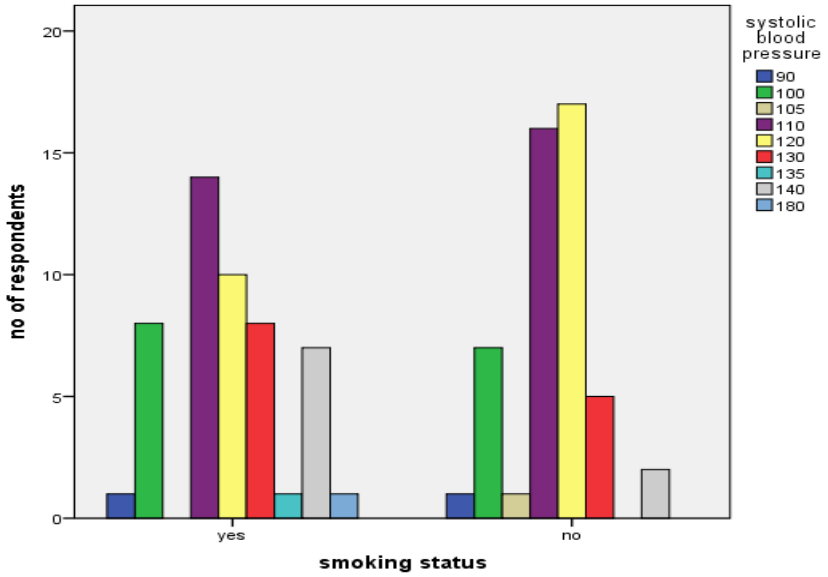

Figure 1: Frequency distribution of systolic blood pressure in respondents

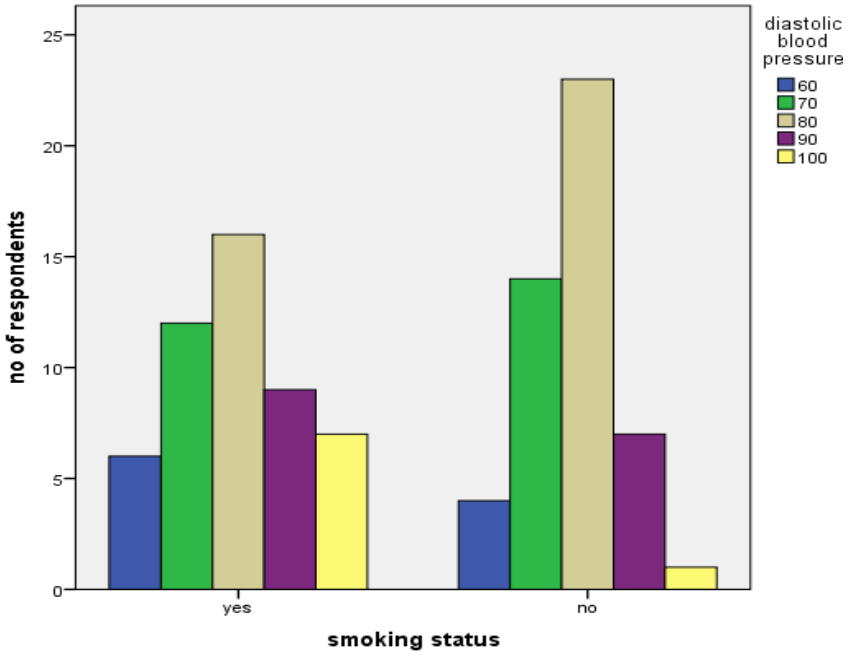

Figure 2: Frequency distribution of diastolic blood pressure in respondents

Table 2: Lipid profiles and hs-CRP in smokers and non-smokers

Blood levels	Smokers	Non smokers	Significance(p-value)
Total Cholesterol(mg/dl)	179.56 ± 28.8	134.05 ± 17.25	0.001
Triglycerides (mg/dl)	254.39 ± 53.21	120.3 ± 22.66	0.001
LDL-C (mg/dl)	89.92 ± 14.70	70.31 ± 9.78	0.025
HDL-C(mg/dl)	47.76 ± 12.09	41.97 ± 10.91	0.028
hs-CRP(mg/l)	4.02 ± 1.04	1.8 ± 0.89	0.001

4. Discussion

There were no difference in the BMI and blood pressure record among smokers. These findings were agreement with Jamal *et al*. findings. They reported that BMI, blood pressure among healthy people, peoples suffer from metabolic syndrome, and smokers had no differences [16]. In contrast from Okubo *et al* finding that longer duration of smoking of cigarettes had a higher danger of hypertension [17]. So not just does smoking or biting tobacco promptly raise the blood pressure, however, the

chemicals in tobacco can harm the coating of the lining of artery walls. This can make arteries to narrow, increasing blood pressure.

The consequence of this work demonstrated elevated amounts of total cholesterol level in smokers. The consequence of this work is in accordance with crafted by Adedeji and Etukudo, where high total cholesterol was recorded in smokers [18]. Interestingly Waheeb and Alharbi in their work, which was on the influence of cigarette smoking on lipid profile [19]. The finding of smoking being related to high TG was predictable with past examinations. One conceivable clarification for this affiliation is that nicotine may increment thoughtful nerve action, which invigorates to the arrival of catecholamines and subsequently prompts lipolysis, with an ensuing increment in plasma convergence of TG [20].

Significantly increased level of LDL was seen among smokers. Khurana *et al* reported that the explanation behind the raised LDL-C because of the down-regulation of LDL receptors and failure of receptor-mediated endocytosis by a metabolite of cigarette [21].

Adedeji and Etukudo, likewise announced an abnormal state of LDL in smokers, proposing that there was an increased LDL-Cholesterol synthesis of smokers who are unsafe to their wellbeing [22].

In our examination level of HDL was significantly increased in cigarette smokers and this finding did not agree with discoveries of Min Yu *et al* who demonstrated that, the levels of HDL to be altogether lower among cigarette smokers [23]. But other researchers reported that smoking did not affect the HDL levels [24].

Our finding might be because of liquor utilization of the smokers as slight and moderate liquor utilization may have a positive impact on HDL levels. Without a doubt, in our examination smokers who drank liquor had higher HDL-C levels [25]. Be that as it may, in clinical work despite the fact that the HDL levels are normal or even high, the huge vascular remaining danger still exists [26-31].

According to the molecule estimate, HDL can be characterized into 10 subtypes (HDL1-HDL10). Among them 1-3 types were large particle types (HDLL were intermediate particle types (HDLM), and 8-10 types were small particle types (HDLS).

Gao *et al* revealed that the basic utilization of HDL level to foresee the danger of Coronary Heart Disease (CHD) was unreasonable. The HDLL in the serum of CHD patients altogether brought down, HDL had development issue. HDLL might be a defensive factor for CHD, whose abatement was firmly identified with the hazard increment of heart sicknesses. The cardiovascular assurance capacity of HDLL might be connected with apoAI content [32].

In the present study demonstrated that the smokers had highest levels of hs-CRP. A few examinations have shown a critical relationship among smoking and elevated hs-CRP levels [15,33]. High sensitivity -CRP has been depicted as an inflammatory biomarker connected to cardiovascular hazard factors and cardiac events. An hs-CRP level of >3 mg/l was autonomously connected with a 60% overabundance danger of episode of the coronary artery disease [34].

Recent literature has recognized that lipids have a significant job in the activation of inflammatory pathways, increasing the generation of inflammatory cytokines, for the most part necrosis factor alpha, interleukin 6 and 1β. Then again, cytokines can promote disturbance of lipid metabolism, in unique cholesterol reverse transport, which is connected to development of atherosclerosis. Inflammation instigates an assortment of alterations in lipid metabolism that may at dampen inflammation or battle infection, however, if chronic could contribute to the increased risk of atherosclerosis [35].

5. Limitations

This cross-sectional study could not establish causal relations, but could generate a hypothesis that can be evaluated by future prospective studies. Therefore, the results are merely reflective of associations observed between smoking and inflammation. The respondents may not have told the exact amount of cigarette smoked and alcohol consumed. We did not assess the respondents dietary and exercise habits which may influence the outcome of the measurements. Longitudinal studies with long-term follow-up among smokers are recommended.

6. Conclusions

In conclusion, we show here that cigarette smoking was a hazard factor for inflammation. This impact might be interceded to some extent by an increase in the hs-CRP level. High sensitive CRP has all the earmarks of being the most sensitive biomarker of inflammation-related with cigarette smoking and could be a helpful biomarker of smoking-related damage for the examination and control of rising tobacco items. Further examination utilizing a longitudinal study and a larger populace study is important to affirm the conceivable clinical implication of this present outcome.

Acknowledgment

The authors thank the study participants who agreed to participate in this study as well as Addis Ababa University and Adama University for their support during the study.

Consent for publication

As a major aspect of the informed consent process, all members in this examination gave the exploration group consent for their information to be recorded, interpreted, anonymised, investigated and utilized in the arrangement of any logical distribution.

Competing interests

The authors declare that they have no competing interests

References

[1]. Mackay J. The global epidemiology of tobacco and related chronic diseases. *Public Health*. 2012; 126:199-201.

[2]. Awoke M, Damen HM, Ahmed A, Tekebash A. Epidemiology of Major Non-communicable Diseases in Ethiopia: A Systematic Review. *J Health Popul Nutr.* 2014; 32:1-13.

[3]. Ng M, Freeman MK, Fleming TD, Robinson M, Dwyer-Lindgren L, Thomson B, *et al*. Smoking Prevalence and Cigarette Consumption in 187 Countries, 1980-2012. *JAMA.* 2014; 311:183-192

[4]. Reda AA, Kotz D and Biadgilign S. Adult tobacco use practice and its correlates in eastern Ethiopia: A cross-sectional study. *Harm Reduct J*. 2013; 10:28

[5]. Tesfaye F, Byass P, Berhane Y, Bonita R, Wall S. Association of Smoking and Khat (*Catha edulis*Forsk) Use With High Blood Pressure Among Adults in Addis Ababa, Ethiopia, 2006. *Prev Chronic Dis*. 2008; 5;1-11

[6]. Van Zyl-Smit RN, Brunet L, Pai M, Yew WW. The convergence of the global smoking, COPD, Tuberculosis, HIV, and respiratory infection epidemics. *Infect Dis Clin North Am*. 2010; 24: 693–703.

[7]. Talhout R, Schulz T, Florek E, Van Benthem J, Wester P, Opperhuizen A. Hazardous compounds in tobacco smoke. *Int. J. Environ. Res. Public Health*. 2011; 8:613-628.

[8]. Rodgman A, Perfetti T. The Chemical Components of Tobacco and Tobacco Smoke. *Boca Raton, FL: CRC Press*. 2009; 1483-1784.

[9]. Lee J, Taneja V, Vassallo R. Cigarette Smoking and Inflammation: Cellular and Molecular Mechanisms. *J Dent Res.* 2012; 91:142-149.

[10]. Kangavari S, Matetzky S, Shah PK, Yano J, Chyu KY, Fishbein MC, *et al*. Smoking increases inflammation and metalloproteinase expression in human carotid atherosclerotic plaques. *J Cardiovasc Pharmacol Ther*. 2004; 9:291–298.

[11]. Ross R. Atherosclerosis an inflammatory disease. *N Engl J Med*. 1999; 340:115–126.

[12]. Ishizaka N, Ishizaka Y, Toda E, Hashimoto H, Nagai R, Yamakado M. Association between white blood cell count and carotid arteriosclerosis in Japanese smokers. *Atherosclerosis*. 2004; 175:95–100.

[13]. Jefferis BJ, Lowe GD, Welsh P, Rumley A, Lawlor DA, Ebrahim S, *et al*. Secondhand smoke (SHS) exposure is associated with circulating markers of inflammation and endothelial function in adult men and women. *Atherosclerosis*. 2010; 208:550–556.

[14]. Al Rifai M, DeFilippis AP, McEvoy JW, Hall ME, Acien AN, Jones MR, *et al*. The relationship between smoking intensity and subclinical cardiovascular injury: The Multi-Ethnic Study of Atherosclerosis (MESA). *Atherosclerosis*. 2017; 258:119-130.

[15]. Wannamethee SG, Lowe GD, Shaper AG, Rumley A, Lennon L, Whincup PH. Associations between cigarette smoking, pipe/cigar smoking, and smoking cessation, and haemostatic and inflammatory markers for cardiovascular disease. *Eur Heart J.* 2005; 26:1765–1773.

[16]. Jamal O, Aneni EC, Shaharyar S, Ali SS, Parris D, McEvoy JW, *et al*. Cigarette smoking worsens systemic inflammation in persons with metabolic syndrome. *Diabetol Metab Syndr*. 2014; 6: 79. doi:10.1186/1758-5996-6-79

[17]. Okubo Y, Miyamoto T, Suwazono Y, Kobayashi E, Nogawa K. An association between smoking habits and blood pressure in normotensive Japanese men; *J Hum Hypertens*. 2002; 16:91-6

[18]. Adedeji OA, Etukudo MH. Lipid Profile of Cigarette Smokers in Calabar Municipality. *Pakist J. of Nutrition.* 2006; 5:237-238

[19]. Waheeb D, Alharbi M. Influence of cigarette smoking on lipid profile in male university students. *Pakist J Pham.* 2011; 28:45-49.

[20]. Chen CC, Li TC, Chang PC, Liu CS, Lin WY, Wu MT, *et al*. Association among cigarette smoking, metabolic syndrome, and its individual components: the metabolic syndrome study in Taiwan. *Metabolism*. 2008; 57:544–8.

[21]. Andersson K, Arner P.Systemic nicotine stimulates human adipose tissue lipolysis through local cholinergic and catecholaminergic receptors; *Int J Obes Relat Metab Disord*. 2001; 25(8):1225–32.

[22]. Khurana M, Sharma D, Khandelwal PD. Lipid profile in smokers and tobacco chewers; a comparative study. J Assoc *Physicians India*. 2000; 28:897-897.

[23]. Min Yu, Chun-Xiao X, Hong-Hong Z, Ru-Ying H, Jie Z, Hao Wang, *et al*. Associations of Cigarette Smoking and Alcohol Consumption with Metabolic

Syndrome in a Male Chinese Population: A Cross-Sectional Study. *J Epidemiol*. 2014; 24; 361-369

[24]. Siekmeier R, Wulfroth P, Wieland H, Grob W and Marz W. Low density susceptibility to *in vitro* oxidation in healthy smokers and non-smokers. *Clin. Chem*. 1996; 42: 524.

[25]. Bleich S, Bleich K. Moderate alcohol consumption and the risk of cardiovascular disease. *Am J Clin Nutr*. 2002; 75: 948.

[26]. Hausenloy DJ, Yellon DM. Targeting residual cardiovascular risk: raising high-density lipoprotein cholesterol levels. *Postgrad Med J*. 2008; 84:590-598.

[27]. Sviridov D, Mukhamedova N, Remaley AT, Chin-Dusting J, Nestel P. Antiatherogenic functionality of high density lipoprotein: how much versus how good. *J Atheroscler Thromb*. 2008; 15:52-62.

[28]. Ridker PM, Genest J, Boekholdt SM, Libby P, Gotto AM, Nordestgaard BG, *et al*. HDL cholesterol and residual risk of first cardiovascular events after treatment with potent statin therapy: an analysis from the JUPITER trial. *Lancet.* 2010; 376:333-339.

[29]. Barter P. HDL-C: role as a risk modifier. *Atheroscler* Suppl. 2011; 12:267-270.

[30]. Tian L, Fu M, Jia L, Xu Y, Long S, Tian H, *et al*. Relationship between apolipoprotein concentrations and HDL subclasses distribution. *Lipids.* 2007; 42:419-426.

[31]. Zhang B, Miura S, Yanagi D, Noda K, Nishikawa H, Matsunaga A, *et al*. Reduction of charge-modified LDL by statin therapy in patients with CHD or CHD risk factors and elevated LDL-C levels: the SPECIAL Study. *Atherosclerosis.* 2008; 201:353-359.

[32]. Gao F, Ren Y,Shen X, Bian Y, Xiao C, Li H. Correlation between the High Density Lipoprotein and its Subtypes in Coronary Heart Disease. *Cell Physiol Biochem*. 2016;38:1906-1914

[33]. Tibuakuu M, Kamimura D, Kianoush S, DeFilippis AP, Al Rifai M, Reynolds LM, *et al*. The association between cigarette smoking and inflammation: The Genetic Epidemiology Network of Arteriopathy (GENOA) study. *PLoS One*. 2017; 12(9):e0184914.

[34]. Buckley DI, Fu R, Freeman M, Rogers K, Helfand M. C-reactive protein as a risk factor for coronary heart disease: a systematic review and meta-analyses for the U.S. Preventive Services Task Force. *Ann Intern Med.* 2009; 151:483-95.

[35]. De Groot LJ, Chrousos G, Dungan K, Feingold KR, Grossman A, Hershman JM *et al*. editors. South Dartmouth (MA): MDText.com, Inc.; 2000.