Firefighters' Occupational Exposure to Carbon Monoxide*

J. H. Sammons, Ph.D. (LCDR, MSC USN), and R. L. Coleman, Ph.D.

In an era of unparalleled concern over the occupational environment and for the protection of the health of the worker,[1] it is ironic that firefighters, one of the most essential and heavily stressed occupations, have been virtually overlooked. Consequently, it was considered imperative that the effects of their daily exposures to components of the fire smoke complex, such as carbon monoxide (CO), be evaluated. The scientific literature contained only one reference[2] with any data specifically relating to firefighting as an occupation. Furthermore, there is no data pertaining to morbidity among firefighters although almost without exception this profession is subjected to increased insurance rates. The professional fire literature contained numerous references to the occupational hazards related to smoke inhalation;[3-6] however, there is little proven scientific basis for their conclusions. Since 1969 members of the fire service have had the highest death and injury rates of any occupation.[5] This same publication[5] reported that of the 233 fire service deaths from occupational disease, 222 (95%) were from heart and lung related diseases. Furthermore, of the 463 fire fighters who left the fire service because of occupational diseases, 390 (84%) had heart and lung related diseases.

The present study was designed to determine whether an occupational group exposed repeatedly to the components of the fire smoke complex had a significantly higher residual carboxyhemoglobin (COHb) level than members of a non-exposed control group, and to investigate the possibility of myocardial damage as a result of the occupational exposure. Evidence of myocardial damage was sought by assaying for Total Lactic Dehydrogenase (LDH), Heat Stable Lactic Dehydrogenase (LDH-S) Heat Labile Lactic Dehydrogenase (LDH-L), Hydroxybutyric Dehydrogenase (HBD) and Creatine Phosphokinase (CPK). Based on an extensive review of the literature with emphasis on the effects of CO on the myocardium,[7] it was concluded that CO in low concentrations appears to have a definite deleterious effect on the body and in particular the myocardium.

Methods and Materials

Population Selection. — The test population was a random selection from members of the Oklahoma City Fire

Table 1. — Test Population Pairing Data.

Test Subject	Cigs per Day	Age Start Smoke	Age	Height Inches	Weight Pounds	Years in Fire Svc
1	40+	18	49	69	150	26
2	10-20	20	42	72	170	21
3	0	0	40	68	180	15
4	0	0	37	73	230	10
5	0	0	37	70	200	9
6	0	0	35	70	200	3
7	40+	11	35	72	172	6
8	10-20	16	34	72	225	2
9	10-20	23	34	71	195	10
10	21-39	17	33	69	175	5.5
11	0	00	33	69	187	6
12	10-20	19	33	71	175	3
13	40+	15	33	69	175	2
14	21-39	16	32	68	165	2
15	0	00	30	72	180	6
16	10-20	20	30	70	200	5
17	0	0	29	70	185	6
18	40+	20	29	71	175	7
19	10-20	18	29	73	210	8
20	10-20	14	29	70	175	7
21	1-9	20	28	71	175	5
22	10-20	16	28	71	160	5.5
23	0	0	28	71	205	2
24	21-39	17	27	71	185	2
25	10-20	21	26	70	180	1
26	10-20	16	25	70	160	3
27	40+	18	25	68	148	0.25

From the University of Oklahoma College of Health, Department of Environmental Health, 641 N. E. 15th Street, Oklahoma City, 73104.

Reprint requests to Navy Industrial Environmental Health Center, 3333 Vine St., Cincinnati, Ohio 45220. (Dr. Sammons' present address).

*Condensed from a dissertation submitted to the University of Oklahoma, July 1973.

Department who were paired with a control group of non-firefighters as to age, weight, height, race, smoking habits and family history of cardiovascular and pulmonary disease.

All subjects were treated in exactly the same way and blood specimens were taken at 28-day intervals throughout the 5-month duration of the study.

The pairing of the two populations was accomplished by extensive interviews to assure the most exact pairing possible. All test and control subjects were chosen from a randomly selected pool on the basis of matching as many criteria as possible. This resulted in a close pairing as to age, weight, height, smoking habits, sex, race and family history of cardiac or pulmonary disease. Effort was made to exclude any control subjects who might have been engaged in an occupation or hobby that would have offered significant exposures to CO. Similarly, test subjects were screened closely for second jobs or hobbies that would have offered additional exposure to CO. The descriptive data of the test and control populations is presented in Tables 1 and 2.

Analytical Procedures. — Carboxyhemoglobin (COHb) and reduced hemoglobin saturations were determined by utilizing an Instrumentation Laboratory IL-182 CO-Oximeter. Serum enzyme activities were determined by Sigma Chemical procedures and reagents.

Results and Discussion

The results of laboratory measurements are presented in Table 3 for the test population and Table 4 for the controls. With the exception of COHb all other values fell generally within normal ranges. The fact that there was no significant difference in hemoglobin substantiated the homogeniety of the populations and strengthened the pairing.

When the test and control pairs were singly subjected to the paired t-test, probabilities as shown in Table 5 were obtained. When the 27 test subjects and the 27 controls were considered as a group the probabilities obtained were: Hemoglobin — Non-significant; Carboxyhemoglobin — $p < 0.001$; Total Lactic Dehydrogenase (LDH) — $p < 0.001$; Heat Stable Lactic Dehydrogenase (LDH-S) — $p < 0.001$; Heat Labile Lactic Dehydrogenase (LDH-

Table 2. — Control Population Pairing Data.

Control Subject	Cigs per Day	Age Start Smoke	Age	Height Inches	Weight Pounds
1	40+	14	48	69	148
2	10-20	18	41	71	167
3	0	0	40	68	180
4	0	0	38	72	224
5	0	0	37	70	195
6	0	0	36	70	200
7	40+	14	35	72	174
8	10-20	18	34	72	230
9	10-20	20	34	72	189
10	21-39	18	34	70	176
11	0	0	33	68	185
12	10-20	19	33	70	170
13	40+	16	33	69	170
14	21-39	17	31	68	170
15	0	0	30	71	180
16	10-20	19	30	70	195
17	0	0	29	70	190
18	40+	20	29	70	168
19	10-20	18	29	73	220
20	10-20	16	29	70	176
21	1-9	19	28	71	181
22	10-20	16	28	71	158
23	0	0	27	70	210
24	21-39	13	27	70	190
25	10-20	18	26	70	175
26	10-20	17	25	69	165
27	40+	13	25	69	150

Table 3. — Test Group Data - Five Month Mean Values.

Test Subject	COHb[a]	Hb[b]	Total[c] LDH	Stable[d] LDH	Labile[e] LDH	CPK[f]	HBD[g]
1	11.12	14.6	262	198	28	17	72
2	9.88	15.4	330	137	8	6	82
3	2.76	15.7	284	122	56	7	82
4	4.52	15.5	324	146	49	0	81
5	2.5	17.2	268	114	78	2.0	73
6	4.5	14.6	362	184	68	10	81
7	8.3	16.7	264	126	0	0	74
8	10.7	14.7	562	370	52	39.0	117
9	4.3	14.9	242	156	12	7.0	72
10	13.9	15.8	260	173	0	12	96
11	6.9	15.1	272	182	53	15.0	77
12	11.4	17.2	246	126	60	0	75
13	12.55	14.1	336	138	58	17	87
14	8.4	15.9	213	110	42	0	71
15	7.3	15.5	238	142	58	12	72
16	9.1	16.5	212	120	0	5	84
17	5.9	16.0	226	126	16	5	73
18	13.1	16.1	306	134	0	17	73
19	9.4	16.2	270	134	42	3	96
20	7.2	14.5	268	146	0	0	73
21	12.1	16.3	324	112	22	7	78
22	8.8	16.5	346	160	80	9	78
23	6.1	15.3	240	170	0	0	76
24	13.5	15.4	224	154	46	0	73
25	8.1	16.3	278	173	52	0	78
26	6.4	13.5	318	106	0	0	72
27	12.5	14.4	214	134	30	15	74

[a] Carboxyhemoglobin (grams per 100 ml blood).
[b] Hemoglobin (grams per 100 ml blood).
[c] Total lactic dehydrogenase (Sigma units).
[d] Heat stable lactic dehydrogenase (Sigma units).
[e] Heat labile lactic dehydrogenase (Sigma units).
[f] Creatine phosphokinase (Sigma units).
[g] a-Hydroxybutyric dehydrogenase (Sigma units).

L) — $p < 0.02$ (-); Hydroxybutyric Dehydrogenase (HBD) — $p < 0.02$; and Creatine Phosphokinase (CPK) — $p < 0.01$.

The most significant findings in the COHb study were among the nonsmoking firefighters. The mean COHb value for these nonsmoking test subjects was 5.0 mgm% as compared to 2.3 mgm% for the nonsmoking controls. The latter value for the nonsmoking controls falls within previously reported ranges.[2,8] The 5.0% COHb saturation for the test group nonsmokers compares well with Lawther[2] who found the COHb levels in nonsmoking firemen to range from 0.4 to 8.8% with a mean of 3.2%. The Surgeon General[8] considers COHb levels in smokers to average 4%. It is most significant to note that the current recommended time weighted average (TWA) for an occupational exposure to CO[9] is predicated on an exposure of 42 mg/M^3 CO for an eight hour period to achieve no greater than a 5% COHb saturation. Therefore, it would seem that the nonsmoking fire fighter, by virtue of his occupation, has already achieved the maximum allowable COHb concentration and any additional exposure could be considered detrimental to his health.

In the populations tested, which had no evidence of any specific organ disease or clinical heart disease, members of the fire service had significantly higher LDH, LDH-S, and CPK levels than their paired counterpart. Statistical comparison of the enzymatic batteries of the two groups (firefighters and nonfirefighters) revealed levels of significance with probabilities from $p < 0.02$ to $p < 0.001$ in four of the five components. Those components found to be significantly different are considered to be myocardial related. With few exceptions the individual enzymatic values obtained in this study were within or close to the established ranges of normal; but normal ranges are designed to accommodate individual rather than group variability. When taken as group values the overall differences between the two groups become highly significant. Because of the closeness of the pairing, with the occupation being the major difference, the presumption is strong that for the professional firefighter, their occupation is the main contributing factor to explain the observed enzymatic differences.

Table 4. — Control Group Data - Five Month Mean Values.

Control Subject	COHb [a]	Hb [b]	Total [c] LDH	Stable [d] LDH	Labile [e] LDH	CPK [f]	HBD [g]
1	11.12	15.3	236	154	45	4	78
2	8.62	14.9	173	100	16	1	72
3	2.12	11.9	171	85	22	1	75
4	1.8	15.0	310	124	85	7	72
5	1.0	15.6	166	75	10	0	72
6	3.3	16.2	190	112	74	0	73
7	7.3	15.6	178	76	70	3	74
8	8.5	15.3	163	86	33	5	74
9	3.9	15.0	168	106	55	3	72
10	8.8	16.8	189	96	88	5	81
11	4.0	15.0	184	89	24	0	72
12	7.5	15.9	198	80	10	0	75
13	11.7	15.4	242	46	45	0	90
14	7.3	17.3	202	132	64	0	76
15	0.9	15.2	157	86	60	4	63
16	7.4	15.9	252	78	44	4	65
17	3.2	17.1	202	64	74	5	82
18	10.8	16.1	200	103	58	0	71
19	8.3	15.9	192	102	72	1	61
20	7.4	15.4	164	61	82	0	82
21	7.5	16.6	207	128	62	4	64
22	4.5	16.8	236	118	96	0	71
23	2.0	16.1	216	90	103	0	78
24	7.6	17.1	232	93	89	0	77
25	2.1	15.0	176	87	72	0	71
26	7.5	12.9	257	115	52	4	70
27	5.9	15.0	134	70	44	0	71

[a] Carboxyhemoglobin (grams per 100 ml blood).
[b] Hemoglobin (grams per 100 ml blood).
[c] Total lactic dehydrogenase (Sigma units).
[d] Heat stable lactic dehydrogenase (Sigma units).
[e] Heat Labile lactic dehydrogenase (Sigma units).
[f] Creatine phosphokinase (Sigma units).
[g] a-Hydroxybutyric dehydrogenase (Sigma units).

Table 5. — Probabilities Determined by Pairing T-Test (Five Months of Data).

Test Cont. Pair	COHb [a]	Hb [b]	LDH(T) [c]	LDH(S) [d]	LDH(L) [e]	CPK [f]	HBD [g]
1	NS [h]	-0.01	0.3	0.05	-0.4	0.01	0.02
2	0.05	0.2	0.001	0.3	-0.3	0.2	0.05
3	0.2	0.001	0.001	0.1	0.1	0.05	0.1
4	0.001	0.05	0.4	0.3	-0.001	-0.02	0.05
5	0.001	0.02	0.001	0.001	0.001	0.4	NS
6	0.2	-0.02	0.001	0.01	-0.5	0.001	0.2
7	0.02	0.01	0.03	0.01	-0.2	-0.2	NS
8	0.02	-0.5	0.001	0.001	NS	0.001	0.01
9	0.1	-0.4	0.05	0.02	-0.05	0.2	NS
10	0.001	-0.1	0.01	0.02	-0.001	0.1	0.1
11	0.01	NS	0.01	0.001	0.2	0.001	0.3
12	0.01	0.01	0.02	0.1	0.01	NS	-0.4
13	0.5	-0.1	0.05	0.01	0.3	0.001	NS
14	0.2	-0.02	NS	-0.2	-0.2	NS	-0.2
15	0.001	0.02	0.001	0.001	NS	0.02	0.1
16	0.2	0.2	-0.4	0.1	-0.05	NS	0.01
17	0.01	-0.1	NS	0.01	-0.05	NS	-0.3
18	0.01	NS	0.1	0.2	-0.2	0.001	NS
19	0.02	0.4	0.01	0.001	-0.02	0.2	0.001
20	NS	NS	0.05	0.02	-0.01	NS	-0.1
21	0.01	-0.2	0.1	-0.5	-0.2	0.3	0.5
22	0.01	NS	0.02	0.2	-0.3	0.01	0.2
23	0.05	-0.05	NS	0.01	-0.01	NS	-NS
24	0.01	-0.1	-NS	0.01	-0.05	NS	-NS
25	0.001	0.1	0.001	0.01	-0.5	NS	0.4
26	-0.1	0.3	0.2	-0.5	-0.1	-0.1	NS
27	0.01	-0.3	0.01	0.01	-NS	0.001	0.5

[a] Carboxyhemoglobin (grams per 100 ml blood).
[b] Hemoglobin (grams per 100 ml blood).
[c] Total lactic dehydrogenase.
[d] Heat stable lactic dehydrogenase.
[e] Heat labile lactic dehydrogenase.
[f] Creatine phosphokinase.
[g] a-Hydroxybutyric dehydrogenase.
[h] No significance.
[i] Negative t-value.

Summary

Thirty-six men were selected at random from the Oklahoma City Fire Department and interviewed extensively relative to their occupation and health status. These men were paired as closely as possible with members of local military reserve units.

Every 28 days, blood was drawn from each test and control subject and analyzed for hemoglobin, carboxyhemoglobin, total lactic dehydrogenase, heat stable lactic dehydrogenase, isoenzymes, heat labile lactic dehydrogenase isoenzymes, and creatine phosphokinase and hydroxybutyric dehydrogenase.

In the populations tested, members of the fire service had significantly higher COHb, LDH, LDH-5, HBD and CPK levels than their paired controls.

Regression analyses comparing the various determinations with parameters such as age, length of time in the fire service and smoking habits were generally inconclusive except for COHb which predictably increased with increased smoking. Possible effects of occupational exposures to the fire smoke complex were not demonstrated by the individual determinations, but the results taken as a battery do indicate that such an occupational effect could exist in this study group.

Conclusions

It was concluded from this study that in the population studied: (1) The non-smoking fire fighter has already achieved the maximum allowable COHb saturation under NIOSH guidelines; (2) The test group, as a whole, exceeded the COHb content that would be achieved if they labored at heavy work for 1400 minutes in an atmosphere of 42 mg/M^3 CO; (3) As a group, the test population exhibited changes in enzyme activities that suggest myocardial damage; (4) There was no demonstrable difference in the hemoglobin values of the test and control groups; (5) For this group of fire fighters and their controls, the observed differences could best be attributed to occupation; (6) The significance of the observed differences between the populations demands further studies to better understand and define the role of CO as it relates to the health of the fire fighter.

Continued and expanded studies are already in progress and more are planned.

References

1. Occupational Safety and Health Act of 1970, Public Law 91-596, 91st Congress, S. 2193, December 29, 1970; 84 STAT. 1590.

2. Lawther PJ: Pollution at work in relation to general pollution, *J RSH* 5:250-253, 1971.

3. *Proceedings of a Symposium on Occupational Health and Hazards of the Fire Service.* Sponsored by the John P. Redmond Memorial Fund of the International Association of Fire Fighters at Notre Dame University, South Bend, Indiana, 1971. These proceedings are not generally available.

4. Thomas DM: Health Hazards of Smoke Inhalation, *International Fire Fighter*, 8-10, 1971.

5. Annual Death and Injury Survey, *International Fire Fighter*, 1971.

6. Gordon GS and Rogers RL: Project monoxide, A medical study of an occupational hazard of fire fighters, A project of the John P. Redmond Memorial Fund of the International Association of Fire Fighters, Merkel Press, Washington, D.C. 1969. This report is not generally available.

7. Sammons JH: Firefighters Occupational Exposure to Carbon Monoxide, Dissertation, University of Oklahoma, 1973.

8. *The Health Consequences of Smoking.* A Report to the Surgeon General; 1972 DHEW Pub. No (HSM) 72-7516, 1972.

9. *Occupational Exposure to Carbon Monoxide: Criteria for a Recommended Standard* DHEW Pub. No. (HSM) 73-11000, 1972.

This research was funded by the John P. Redmond Fund of the International Association of Firefighters (I.A.F.F.), and Local 1542, I.A.F.F., Oklahoma City, Oklahoma. The authors are indebted to the leadership of the union and its membership as well as the Fire Command and the officials of the city of Oklahoma City. Special recognition is extended to the 36 firemen and members of the local U.S. Navy and Marine Corps Reserve Units who so generously volunteered their time and blood for the project.

The opinions expressed herein are those of the authors and do not necessarily reflect the view of the Navy Department or the Naval Service at large.