

## Letters to the Editor

### Interpretation of Blood and Urine Cannabinoid Concentrations\*

Sir:

Marijuana is very widely used and millions of tests for the metabolites of this drug in urine specimens are being performed. The availability of rather simple and rapid screening methods for marijuana has increased the popularity for testing urine specimens for the drug to about the same as that for testing for alcohol in blood or breath. Most of the programs that test urine specimens for drugs include tests for cannabinoids but not for alcohol. Alcohol, which is more widely used both occasionally and chronically, is more impairing to health, performance, and safety, is addicting with life-threatening withdrawal, and can kill both acutely when an overdose is taken or through degenerative diseases produced by chronic abuse. Marijuana is a much more benign drug.

The objective of this report is to explore the possibilities of interpreting the meaning of concentrations of cannabinoids in various specimens. Alcohol is a very poor model for the interpretation of cannabinoid concentrations as it is for most other drugs. Many studies have produced vast amounts of information on alcohol. Although there is controversy, the opinions of experts of the degree of impairment, symptoms, amount of alcohol consumed, and of alcohol concentrations at some prior time based on alcohol concentrations have been accepted in courts. Experts have calculated the relationship between blood, plasma, breath, urine, saliva, and other specimens. Most people are not aware that this cannot be done at the present time for cannabinoid concentrations. The scientific data are not available. Research on performance and other effects has been done following the use of marijuana of known or unknown strength without establishing the concentration of cannabinoids in the subject. When cannabinoid concentrations have been reported it has not been possible to correlate them to performance changes. This lack of knowledge has not stopped people from drawing conclusions of performance decrements based on cannabinoid concentrations without documentation or adequate scientific foundation.

#### *Urine*

Because of the millions of urine specimens being analyzed for cannabinoids many attempts have been made to interpret the meaning of the findings and also what an immunoassay cutoff value of cross-reacting cannabinoids means. Of the more than 80 metabolites of tetrahydrocannabinol (THC), about 30 have been found in urine [1]. Practically no THC, the major psychoactive cannabinoid, is present in urine. Most testing methods are standardized against 11-nor-tetrahydrocannabinol-9-carboxylic acid (THC-COOH) which represents about 25% of the cannabinoids found in urine. This compound is found both free and bound. The other acid metabolites are the diacid (8%), an hydroxy acid (5%), and six other acids from 1 to 3% in abundance [1]. The result of an immunoassay of urine for cannabinoids is a measure of the total amount of binding produced by the cross-reactivities of the various metabolites compared to the amount of binding produced by a known concentration of THC-COOH.

The urinary cross-reacting cannabinoid excretion patterns are extremely variable for subjects smoking the same or different strengths of marijuana cigarettes [2]. The same is true

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for chronic smokers whose urines no longer test positive to a 100- $\mu\text{g}/\text{L}$  cutoff enzyme multiple immunoassay technique (EMIT<sup>®</sup>) st. Cannabinoid assay in 0 to 45 days or in 3 to 77 days to a 20  $\mu\text{g}/\text{L}$  Emit d.a.u. cannabinoid assay [3]. Some urines which tested negative after a few days tested positive later with no evidence of intervening drug use. The use of more sensitive assays extends the number of days the specimens test positive. Quantitation of THC-COOH in urine specimens using a gas chromatography-mass spectrometric (GC/MS) method revealed the extremely varied excretion patterns obtained from the urines of 10 subjects who each smoked a 2.7% THC cigarette. A urine concentration of 2700  $\mu\text{g}/\text{L}$  of THC-COOH dropped to about 150  $\mu\text{g}/\text{L}$  23 days after the subject stopped smoking marijuana [4]. The subject did not appear to be impaired during this period.

There is general agreement in the scientific community that urine concentrations of cannabinoids cannot be correlated with performance, health, or safety [5]. The identification of cannabinoids in urine can indicate that the person used or had been exposed to marijuana. The concentration found should be high enough to guarantee positive identification.

### *Hair*

The detection of THC in hair has been reported [6]. An example of the distribution of "marijuana" along the length of hair is reported as about 0.2  $\mu\text{g}/\text{g}$  in the first 2 cm from the root and about 1.3 to 1.8  $\mu\text{g}/\text{g}$  from 2 to 18 cm of hair. The first 2 cm equals about two months growth of hair. Attempts to correlate "joints per week" with concentrations of "marijuana" which ranged from 0 to 5  $\mu\text{g}/\text{g}$  of hair were unsuccessful. There was not enough information in this report to evaluate the usefulness of hair as a specimen. It appears that recent use of the drug will not be detected and that confirmation would be difficult or impossible.

### *Saliva and Breath*

THC has been detected in saliva for several hours after marijuana was smoked [7] but not after intravenous injection of radiolabeled THC [8]. The saliva traps the products present in the smoke. The detection of THC in trapped breath samples [8] was most likely due to the direct contact of the smoke on the lung. The finding of THC in saliva or breath indicates marijuana smoking sometime before obtaining the specimen, but its presence cannot be correlated with time of smoking, with blood concentrations, or with impairment. Confirmation of such tests is very difficult or impossible.

### *Blood and Plasma*

THC and THC-COOH concentrations will be about twice as high in plasma than in blood because these drugs are bound to plasma proteins [9]. THC concentrations peak about 7 min after smoking starts, and decline while smoking continues. In Table 1 some reported cannabinoid concentrations versus times are summarized.

Smoking marijuana cigarettes containing 1.3 and 2.5% THC produced peak plasma concentrations of 94 and 155  $\mu\text{g}/\text{L}$ , respectively [12]. About 20 min after starting to smoke, THC concentrations were about half those of the peak; about 1 h  $1/6$ ; and at 2 h less than  $1/10$  the peak concentration. THC-COOH concentrations rose slowly and peaked in about 25 to 30 min at 31 and 45  $\mu\text{g}/\text{L}$ , respectively, for the two cigarettes. THC and THC-COOH concentrations will crossover at about 20 to 30 min after smoking begins. One hour after starting to smoke, plasma contained the following proportions of cannabinoids: 17% THC, 40% THC-COOH, 25% "polar acids," and 9% of different hydroxy metabolites [13]. One of these, the 11-hydroxy THC, is psychoactive and is found in much greater concentrations after oral ingestion of marijuana than after smoking.

TABLE 1—Serum concentrations after smoking marijuana cigarettes.

No. of Subjects	Concentration of THC <sup>b</sup>	Minutes					Hours					Ref
		0	5	10	20	30	1	2	4 to 8	24		
6 <sup>f</sup>	1.8%	100 <sup>c</sup>	40	25	16	13	8.3	3	0.3	0	<sup>d</sup>	
6 <sup>f</sup>	1.8%	107 <sup>c</sup>	63	34	17	12	7.6	4.5	1.1	0.9	<sup>d</sup>	
10	10 mg		85	78	58	42	10	1.1			10	
6	1.0%	0.2 (3.8)	90	46	17	12	6.5	3.8 to 2nd cig.			11	
			71	43	16	11	8	3.8	4.4	3.4	11	
6	1.3%		92	82	59	29	14	7	3.1		12	
6	2.0%		90	104	55	28	17	8	3.7		12	
6	2.5%		134	133	91	42	20	9.6	3.1		12	
					THC-COOH, µg/L							
6 <sup>f</sup>	1.8%	0	15	18	21	22	21	20	14	5	<sup>d</sup>	
6 <sup>f</sup>	1.8%	46	64	67	77	72	76	67	48		<sup>d</sup>	
10	10 mg		7	11	18	26	24	15	4		10	
6	1.0%	3.3 (16)	6	19	24	22	18	16 to 2nd cig.			11	
			18	28	34	28	25	24	24		11	
6	1.3%	4	16	31	31	23	18	11			12	
6	2.0%	4	19	43	37	26	21	13			12	
6	2.5%	4	22	45	45	34	25	14			12	

<sup>a</sup>F = frequent user, I = infrequent user.<sup>b</sup>Conc. = concentration in the cigarette.<sup>c</sup>Time after smoking stops for this study only.<sup>d</sup>M. A. Peat, personal communication, Jan. 1987.

A finding of 20  $\mu\text{g/L}$  of THC in plasma (10  $\mu\text{g/L}$  in blood) probably indicates that marijuana was smoked within the hour and with 10  $\mu\text{g/L}$  in plasma within 2 h. THC concentrations greater than 50  $\mu\text{g/L}$  could indicate smoking within 20 min. Concentrations of THC-COOH of 10  $\mu\text{g/L}$  in plasma are attained in less than 10 min after beginning smoking and can remain over 10  $\mu\text{g/L}$  for 6 h. They will exceed those of THC in 20 to 30 min. Although no relationship has been reported between a subject's rating of "high" and possible effects on driving, it is interesting to relate the high to THC and THC-COOH concentrations. The intensity of "high" increases rapidly for the first 10 min of smoking, peaks in the next 20 min, and is reported to last for up to 4 h after smoking started, and it is unlikely that a range of plasma THC concentrations could be reliably equated with impaired performance [14]. This differs from the way THC concentrations peak and rapidly decline but more closely resembles the curve of THC-COOH concentrations versus time. It is possible that elevated concentrations of THC and THC-COOH remain in chronic heavy smokers for many hours. A group of smokers was reported to have serum concentrations of 2 to 8  $\mu\text{g/L}$  of THC and 27 to 93  $\mu\text{g/L}$  of THC-COOH despite the fact that they were instructed to refrain from marijuana usage for at least 24 h before testing [15].

A plasma concentration of THC of 1  $\mu\text{g/L}$  was found seven days after a heavy user (once daily) discontinued smoking marijuana [16]. Recent smoking of marijuana, that is, within 6 h was indicated by THC concentrations in plasma in excess of 2 to 3  $\mu\text{g/L}$  and 8B,11-dihydroxytetrahydrocannabinol (dihydroxy-THC) concentrations in urine in excess of 15 to 20  $\mu\text{g/L}$  [10]. This was based on acute exposure in ten subjects to about 10 mg of THC. Determinations of dihydroxy-THC are not available from commercial laboratories at the present time. Urines containing 638 and 847  $\mu\text{g/L}$  of THC-COOH were obtained from two operators killed in motor vehicle crashes when no THC, THC-COOH, or hydroxy-THC (THC-OH) was found in their bloods [17].

#### *Concentrations versus Effects*

The effects of smoking 2% THC cigarettes by seven professional and three private pilots have been evaluated using a flight simulator [18]. Individual performances varied considerably from pilot to pilot and from variable to variable. The results indicated that smoking marijuana caused significant deterioration in simulated instrument flying ability for at least 30 min in experienced pilots. The effect probably lasted 2 h and disappeared in 4 h. A preliminary report stated that there were performance decrements on a flight simulator by ten private pilots 24 h after each smoked an entire marijuana cigarette which contained 19 mg of THC [19], but urines and bloods were not tested for cannabinoids, alcohol, or for any other drugs, and the study lacked adequate controls. Another preliminary report dealt with a similar study of ten private pilots after they had drunk enough alcohol for their blood concentrations to reach 100 mg/dL (0.10%). Significant decrements in performance were still evident 14 h later when their bloods should have contained no alcohol [20]. It might be concluded from the two preliminary reports that a pilot whose blood contained no alcohol and no THC but did contain THC-COOH could have had a performance decrement because the blood alcohol concentration could have been 100 mg/dL about 14 h earlier or marijuana might have been smoked 24 h before the time of the blood test or both.

Four studies, one simulator and three on-road studies, looked at extended effects of marijuana and alcohol. Two of the four studies reported that alcohol affects driving for extended time periods, 3 and 4 h [21]. None of the studies revealed any effects of marijuana after the initial test shortly after smoking. In another study, subjects smoked up to 200 mg/kg (14 mg of THC) and drank enough alcohol to produce blood concentrations of 0.08% [22]. Although the plasma concentration of THC was 5  $\mu\text{g/L}$  and of THC-COOH was 30  $\mu\text{g/L}$  the morning after, the only effect was that the alcohol group slowed their speed. No effect was

measurable in the laboratory 2 to 4 h after the use of strong marijuana cigarettes (4% THC), and marijuana had little effect on well trained tasks, but it had a greater effect on the learning of new tasks.<sup>1</sup> Findings [23] suggest that marijuana smoking (2.9% THC) can produce residual (hangover) effects the day after (9 h). The precise nature and extent of those effects as well as their practical implications remain to be determined. THC plasma concentrations the morning after were 3  $\mu\text{g/L}$  for the placebo and 5  $\mu\text{g/L}$  for the 2.9% THC cigarette smokers.

A recent review on marijuana concluded:

Essentially, there are no controversies about the experimental data which examine the issue of whether marijuana impairs psychomotor performance. There remain difficulties in determining from the magnitude and nature of psychomotor performance impairment, the quantitative predictions about the increased probability of accidents in situations such as driving, flying, or industrial work.

No cannabinoid concentration was reported in the review [24].

Concentrations were given in a report which examined the ability of eight male drivers to perform a series of driving tasks following the smoking of marijuana cigarettes or the drinking of alcohol or both [25]. The concentrations of alcohol in bloods were between 80 and 100 mg/dL and those of THC in serum averaged 17  $\mu\text{g/L}$  (range 6 to 31) when each drug was used alone.

The results from the driving portion of this experiment suggest that alcohol and marijuana, when taken in quantities normally associated with social use, adversely affect performance on driving tasks that are typical of every day driving situations. That is, no exaggerated courses or maneuvers had to be employed to demonstrate effects. Moreover, the data obtained from the driving tasks suggest that the effects of alcohol and marijuana are additive when the drugs are taken together. Having said this, however, it is necessary to qualify the term 'adverse.' At no time during this experiment was the driving performance of our eight subject drivers so poor that we were forced to cancel the run. Drivers did not weave down the road under the influence of drugs or brake and accelerate in an erratic manner. The differences in behavior between the drug and placebo conditions that we were able to isolate were so subtle that they would hardly be noticed by observation. Only when the outputs from a number of sensitive transducers are combined and analyzed with the aid of multivariate techniques do the differences between drug conditions become evident. So, the results should not be interpreted as an absolute indictment against either drug.

Attempts to correlate passing or failing of coordination tests with plasma concentrations of THC of 59 subjects who had smoked marijuana cigarettes until a satisfactory level of "high" was obtained revealed that if concentrations measured at 5 min were ignored, failures were almost inevitably associated with plasma concentrations above 25 to 30  $\mu\text{g/L}$  [26]. "Overall, 94% of subjects failed to pass the test 90 min after smoking and 60% after 150 min, despite the fact that by then plasma concentrations were rather low." The plasmas of 28 of 57 had no THC at 150 min.

A "Guide for Presumptive Indication of Intoxication or Being Under the Influence of Alcohol and Drugs" [27] presents concentrations for some drugs in blood and urine that presumably reflect impairment. A blood concentration of THC of 5  $\mu\text{g/L}$  and a urine concentration of THC-COOH of 100  $\mu\text{g/L}$  are presented without scientific foundation.

Very heavy chronic marijuana smokers (24 to 74 mg of THC daily for at least ten years) in Costa Rica earn their living driving trucks, buses, and taxis [28]. The heaviest users had the highest incomes, the least unemployment, and the most stable job histories of the entire user group. It was not reported that their driving was adversely affected. Blood and urine cannabinoid concentrations were not reported.

<sup>1</sup>R. Jones, personal communication, Newport Beach, CA, Jan. 1987.

*Incidence in Drivers*

There have been a few studies of auto crashes where the concentrations of drugs found in bloods of dead operators were reported. These are summarized in Table 2.

Alcohol was found in 57% of the blood specimens obtained from 401 dead drivers in Ontario. Evidence of marijuana, THC, was detected in the bloods of 15 (3.7%) of the victims. Of the 14 bloods, 8 had alcohol concentrations greater than 0.15% [29].

A study in which the bloods of 600 operators killed in single-vehicle crashes in North Carolina were analyzed for drugs revealed that alcohol was found in 79% of the specimens. THC was found in 7.8% and alcohol was found in the majority of these bloods. It was concluded that there was probably only 1 driver who could have been significantly impaired by marijuana use alone [30].

Analyses of the bloods of 440 young male California drivers, 15 to 34 years old, revealed that alcohol was present in 70% of the drivers. Evidence of marijuana was found in 37% of the drivers. Only cannabinoids were found in 4.3% of the drivers. The authors concluded that alcohol was related to crash responsibility but that marijuana was not and that none of the differences were statistically significant. They also concluded that drugs other than alcohol, marijuana, and cocaine are unlikely to be major problems in fatal crashes [31].

Analyses of the bloods of 1169 fatally injured drivers in Ontario for alcohol and THC revealed that alcohol was present in 57% of the drivers. Concentrations of THC of 0.2 to 37  $\mu\text{g}/\text{L}$  were found in the bloods of 127 (10.9%) drivers. Only THC was found in 20 (1.7%) of the drivers' bloods [32].

Some conclusions can be drawn from these 4 studies of fatally injured drivers. Alcohol was found in 1677 (64%) of the 2610 drivers. Cannabinoids were found in the bloods of 351 (13.4%), but cannabinoids alone were found in 72 (2.8%) of the drivers. It is obvious that the majority of drivers in these studies as they have been in other studies had enough alcohol to be impaired when another drug was present. There is no evidence that there is a significant number of drivers who might have been impaired by marijuana alone.

An attempt was made to establish the contribution to crash responsibility of drugs [17]. THC was found in the bloods of 8 (20%) of 40 drivers considered responsible for crashes compared to the finding of THC in the bloods of 38.5% of those not considered responsible for crashes.

The blood and urine specimens of 317 drivers of tractor-trailer trucks who were randomly selected, were analyzed for alcohol and marijuana as well as some other drugs [33]. Only 2 drivers had traces of alcohol in their bloods (0.01 and 0.02%). THC and THC-COOH were found in 9 bloods, and only THC-COOH was found in 25 more bloods. Four drivers had THC concentrations of 5  $\mu\text{g}/\text{L}$  or more (12  $\mu\text{g}/\text{L}$  in blood and 5.5, 7, and 11 in sera). The THC-COOH concentrations were from 45 to 148  $\mu\text{g}/\text{L}$ . Such concentrations could indicate recent marijuana use.

TABLE 2—*Incidence of drugs in dead motor vehicle operator's blood.*

Ref	29	30	31	32	Total
Place	Ontario	N.C.	Cal.	Ontario	
Years	78 to 79	78 to 81	82 to 83	82 to 84	
No. of cases	401	600	440	1169	2610
Alcohol+	229	476	308	667	1680
THC	15	47	162	127	351
THC+ Alc+	13	41	132	107	293
THC+ only	1	4	19	20	44
THC <5 $\mu\text{g}/\text{L}$	13	19	139	107	278
THC >4 $\mu\text{g}/\text{L}$	2	28	23	20	73

### Discussion and Conclusions

All of the studies have some biases and shortcomings. The major problem is the lack of control studies of the frequency of occurrences and the concentrations of cannabinoids in the general driving population. Such studies have been reported for alcohol, but have not been reported for marijuana because of the infinitely more complex problem of obtaining blood specimens from the general driving population and the difficulties and expense of the analyses.

Some recent remarks may be appropriate [21],

In conclusion, marijuana does appear to impair driving behavior. However, this impairment is mediated in that subjects under marijuana treatment appear to perceive that they are indeed impaired. Where they can compensate, they do, for example, by not overtaking, by slowing down and by focusing their attention when they know a response will be required. Unfortunately, such compensation is not possible where events are unexpected or where continuous attention is required. Effects on driving behavior are present shortly after smoking but do not continue for extended periods.

Sufficient information is not available on marijuana for an expert to offer a scientifically defensible opinion that driving impairment is correlatable with cannabinoid concentrations.

There is little in the scientific literature which would alter an opinion offered a decade ago [34]: "At the present time there is no evidence that marijuana is a significant public safety problem or is about to become one. The effects of marijuana reported in these studies are such that it is highly unlikely that a person driving erratically and recklessly would do so because of the influence of the drug."

What forensic scientific testimony can be given about the performance of a person whose blood or plasma contained 1, 5, 10, 25, or 100  $\mu\text{g/L}$  of THC and/or THC-COOH?

One conclusion of a 1980 report to congress might still be appropriate [35]. "The relationship of specific blood drug levels to driving impairment has not been established for drugs other than alcohol, except in case of extreme doses that may be expected to produce gross impairment. Thus even though a blood-drug level may be determined it is often not clear what it means in terms of impairment."

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### **SOFT Position Statement on HHS Guidelines for Forensic Toxicology Laboratory Analyses**

Sir:

Below you will find a Position Statement on HHS Guidelines for Forensic Toxicology Laboratory Analyses. This Position Statement has been voted on and accepted by the voting members of the Society of Forensic Toxicologists, Inc. (SOFT), and now represents the official policy of the organization at this time.

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## **SOFT Position Statement on HHS Guidelines for Forensic Toxicology Laboratory Analyses**

It is the policy of the Society of Forensic Toxicologists, Inc. (SOFT) to neither endorse nor reject proposed government regulations or guidelines. However, the membership of SOFT recognizes and agrees in principle with the goals and objectives of the Scientific and Technical Guidelines for Federal Drug Testing Programs as outlined in the Federal Register dated 14 August 1987. We strongly endorse and support the implementation and application of appropriate quality assurance programs and quality control measures in all laboratories involved in analytical toxicology testing. At the same time, the membership is concerned as to what impact these guidelines may have on other forensic toxicology activities specifically, the activities of postmortem forensic toxicology laboratories.

Forensic toxicology is defined as the analysis of body fluids and tissues for drugs and/or poisons and the interpretation, in a judicial context, of the information generated by such analyses. These analyses frequently are the search for unknown analytes followed by their identification, confirmation, and quantitation.

The subject Scientific and Technical Guidelines are proposed specifically for laboratories engaged in high-volume testing of a single biological specimen (urine) for a limited number of analytes or drug groups (five). In the federal urine drug testing program, the scientific director or the certifying scientist certifies only the test result. They make no *interpretation*

of the test result. This is the role of the medical review officer (MRO), who is not a member of the forensic laboratory staff.

In contrast, in postmortem forensic toxicology, one deals with multiple types of biological specimens (blood, bile, urine, vitreous and tissues such as brain, kidney, liver, and so forth) that vary extensively in their state of postmortem change. Furthermore, in death investigations, the forensic toxicologist must evaluate virtually hundreds of drugs, their metabolites, and a near limitless spectrum of other poisons. In each individual case, the analytical protocols selected are dictated by the type, amount, and physical condition of the specimens available and information available as to the circumstances of the death and the findings at autopsy.

For these reasons, it is the opinion of the membership of the Society of Forensic Toxicologists that, with respect to postmortem forensic toxicology laboratories, the proposed Scientific and Technical Guidelines for Federal (Urine) Drug Testing Programs as outlined in the Federal Register dated 14 Aug. 1987 are analytically impractical, inappropriate, and realistically impossible to apply.

### **Discussion of "An Attempt at Determining Probabilities in Human Scalp Hair Comparison"**

Dear Sir:

It has been 13 years since Gaudette and Keeping's [1] article on probabilities and human scalp hair comparison was published. Since then Gaudette has followed up with research on human pubic hair and other articles to explain/defend his probabilities [2-4]. Other authors have since produced articles to comment on Gaudette's work and forensic hair comparison in general [5-9]. This was certainly one of Gaudette's goals—to stimulate the forensic science community into engaging in dialogue and research into forensic hair comparison. Sadly, though, only dialogue has followed. No one person or organization, including the Royal Canadian Mounted Police, has published any independent research using qualified forensic hair examiners to support or refute Gaudette's works.

Many good comments and observations have come from the analysis of Gaudette's work. It appears as though a shift is developing away from the concern of random positive hair comparisons towards a demand for the significance of specific actual forensic hair comparisons. Aitken and Robertson [7] following the line of Stoney [10] make the point that forensic science comparisons are NOT random events. Objects from crime scenes, once collected, are fixed occurrences—not random. Suspects brought in for questioning are NOT randomly chosen from the general population.

If the suspect has shed hair at the scene of the crime and it is collected by the field-workers, the probability that a qualified forensic hair examiner will correctly match those hairs to the suspect's representative known sample is very high indeed. The time has come for the rehashing of Gaudette's work to stop. It is time to begin forensically oriented research, simulating casework, using qualified forensic hair examiners, both within Canada and internationally. Let us hope that the same enthusiasm used in dissecting 13-year-old research will be exhibited in pursuing this new and vital research.

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### Hazards in the Forensic Science Laboratory: Legal Implications\*

Sir:

In discussing the hazards in the forensic science laboratory, legal considerations must not be overlooked. The simple fact is that whatever is designated as a hazard has correlary resultant factors upon which legal reflections are certain. The hazard, its effect and its cause, most assuredly, will be placed before the jaundiced eye of our American judicial system, there to be examined, scanned, and hopefully remedied.

As more than three decades of experience in forensic science applications are considered, it appears that the law must be cognizant of the types of laboratory hazards present. These hazards may include those that are physical, which are exemplified by noises, temperature variations, traumatic incidents, light intensity, or radiation, for example. Or, they may include those hazards that are biological in essence such as microorganisms and infectuous diseases, parasitic viruses, or blood tissue contaminations, for example, Acquired Immuno-deficiency Syndrome (AIDS), and others found in body cavities. Of course, these hazards may also be of a chemical nature and include drug related hazards. In each instance, there ought to be medical attention promptly available in the event of hazardous exposure.

At one time, not so long ago, benzodeine was utilized for bloodstain processing. Its carcinogenic properties led to cancer of the bladder. Because of improper ventilation, or loose hygienic practices, laboratory personnel frequently have been exposed to hazardous pollutants. These pollutants include exposure to lead while testing weapons and ammunition, exposure to high noise levels while firing guns and testing ammunition, aerosol droplets and dust particles contaminants exposing laboratory personnel to such pollutants as ether and peroxide which may explode upon impact, as well as exposure to unlabeled compounds thought to be innocuous when, in fact, they may not be so at all. Epidemiologically, there are many various, invasive, viral infections that may affect mankind and cause dire consequences. AIDS is the fear of today. Tomorrow there will be something else. Receiving samples of

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blood, particularly if there is present outside blood, contaminants on the specimen received or the vial passed, in fact, may expose the recipient to dire consequences should invasion of the recipient's bloodstream occur.

Trace evidence, in which instance short and long wave radiation or ultraviolet rays are used, may prove hazardous to the eyes. Viral diseases, accidental puncture wounds, and improper collection of blood and tissue samples, for example, may well expose the pathologist, the toxicologist, and other laboratory personnel to hazardous health problems. It would appear, therefore, that all blood and tissue samples, whether labeled properly or not, should be treated as though infection is possible in the handling of it and during the analyzation processes.

Laser technology, when used to lift fingerprints from surfaces from which they could not be lifted before its discovery and implementation, must be viewed from its potential cause of eye and skin damage if not appropriately used.

Other areas of legal interest, when reflecting upon hazards in the forensic science laboratory, may well include consideration of product liability and professional malpractice. For example, a design engineer should consider the laboratory hazard when designing a laboratory facility. Proper ventilation is an extremely important factor, as is the disposal of waste products, the elimination of noise factors, and reduction of exposure to light and radiation, to name but a few considerations. To understand the judicial systems' role in these important matters, it must be remembered that lawyers and judges are constantly mindful of the need to prove "a" competent producing cause when attempting to place liability in each instance of an alleged tort, that is, an unintentional wrong.

The legal burden that must be shown to prove a competent producing cause of the harm resulting from exposure to a hazard within the forensic science laboratory may frequently present a very difficult problem for the lawyer bringing a lawsuit on behalf of an injured or ill client. How does the lawyer prove that a deleterious toxic effect resulted from a hazard existing within the forensic science laboratory? For example, consider testicular atrophy. Was it an alcohol related problem vis-a-vis chemical exposure to a toxic substance that caused the manifestation of the problem? The most common method of proof is evidenced by the frequency, the intensity, and the duration of the exposure to the hazard on each occasion, and then, to compare the results occasioned thereby. Also, consideration must be given to the individual's congenital susceptibility, his or her repeated or chronic exposure or chronic reaction, and the noted side effects of such exposure.

In recognizing the hazards present within the forensic science laboratory, a responsibility to document incidents and safety checks becomes essential. The questions, of course, always present are: (1) How do you create a wall of safety? (2) How do you raise a defense against exposure? (3) How do you develop a safety system?

In 1986 the Environmental Protection Agency (EPA) proposed the regulating of care and safety precautions to reduce laboratory hazards. The regulatory impact of the federal register may assist in: (1) identifying hazards and hazardous substances, (2) requiring proper ventilation, (3) informing laboratory workers of chemical or other hazardous materials within the work environment and when effectuated, and (4) an itemization of individual standards for adopting operating procedures with mandatory precautionary methods and procedures. At least this first attempt at federal regulation in the work place, as it affects the forensic science laboratory, or those within the work environment, will bring some uniformity and consideration of, as well as afford protection from, health hazards. The prior territorial and provincial protectionism, without true review of environmental problems, may become a thing of the past.

It must be recognized that in past years very little consideration has been given to the problems associated with health hazards within the forensic science laboratory. This may have been due in part, at least, to the lack of reported incidents. This, in turn, may have been due to a low incident ratio or to hidden correlations. For example, nurses working

within areas where they have been exposed to anesthesia have been known to have a higher incident ratio of spontaneous abortion. On the other hand, reported incidents may be few because in the professions, and among those persons of higher learning, there is tendency to "get on with life" rather than reporting every adverse incident or taking a sickleave, or time off, as a result of an ill spell. Then, too, the low number of lawsuits brought, or placed into litigation, involving health hazards within laboratories, may well be a reason for so little progress being made in developing health safety measures to protect persons being exposed to hazards within the forensic science laboratory.

What then should the law require regarding the health hazards we recognize as being present? The most obvious answer would appear to be the requirement for routine periodic testing of laboratory personnel, including a complete physical examination. In this regard, it should be noted that hearing, smelling, seeing, touching, and tasting proficiencies are essential from both a neurological and physiological viewpoint. In addition, a required blood sample should be provided and analyzed. First, it could determine the blood count of both red and white blood cells. This would detect any bone marrow deficiencies. Second, liver function and the identification of any toxic effect of certain drugs could be determined. Thirdly, a biochemical profile should be obtained whereby renal functions through urine analysis and creatinines, could be determined.

Certainly, written procedures, posted hazard lists, methods to deal with known hazards, periodic employee meetings during which hazards are explained and discussed, together with periodic blood tests and health evaluations would be an appropriate means to ward off a goodly number of properly conceived legal problems.

Failure to develop proper procedures within the forensic science laboratories may well expose persons, including corporations, to both criminal and civil liability and, in certain instances, to punitive awards for damages. For example, the chemical company that continuously exposes its employees to hazardous waste compounds may encourage a result by which both personal and corporate, criminal and civil liability, will be determined and imposed.

Finally, for those who might desire to hide behind an ill-conceived notion that the existing one-, two-, or three-year statute of limitations has given immunity to legal exposure when questions of liability are considered, note that the majority of states, today, hold that the applicable statutes of limitations do not begin to run until the negligence of the proposed defendant was known, or should have been known, by the party bringing the action for legal compensation and fair and equitable relief. Paraphrasing Yogi Berra, of New York Yankee fame, "a legal problem is never over until it's over." Resolution by preventive measures is the answer.

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