Court File No. T-2030-13

FEDERAL COURT

BETWEEN:

NEIL ALLARD
TANYA BEEMISH
DAVID HEBERT
SHAWN DAVEY

Plaintiffs

and

HER MAJESTY THE QUEEN IN RIGHT OF CANADA

Defendant

AFFIDAVIT OF LEN GARIS

VOLUME II

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Appendix Q: Toonen et al. (2006) Yield of Illicit Indoor Cannibis Cultivation in The Netherlands

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Yield of Illicit Indoor Cannabis Cultivation in The Netherlands

ABSTRACT: To obtain a reliable estimation on the yield of illicit indoor cannabis cultivation in The Netherlands, cannabis plants confiscated by the police were used to determine the yield of dried female flower buds. The developmental stage of flower buds of the seized plants was described on a scale from 1 to 10 where the value of 10 indicates a fully developed flower bud ready for harvesting. Using eight additional characteristics describing the grow room and cultivation parameters, regression analysis with subset selection was carried out to develop two models for the yield of indoor cannabis cultivation. The median Dutch illicit grow room consists of 2.59 cannabis plants, has a plant density of 15 plants/m^2, and 5.10 W of growth lamps per m^2. For the median Dutch grow room, the predicted yield of female flower buds at the harvestable developmental stage (stage 10) was 33.7 g/plant or 505 g/m^2.

KEYWORDS: forensic science, illicit cultivation, flower buds, growth conditions, marijuana

Cannabis is the most commonly used drug in the EU and, depending on the country, is used regularly by 1–10% of all adults (1). The denomination cannabis is commonly used to describe the various products of the cannabis plant (*Cannabis sativa* L.), namely the extracted resin (known as hashish) and the dried female flower buds (known as marijuana, grass, "nederwiet"). The most common mode of administration is smoking in cigarettes (with or without tobacco). Hashish is also eaten, e.g., baked in cookies or cakes (2). The psycho-active effects of cannabis are mainly caused by the cannabinoid Δ9-tetrahydrocannabinol (THC). The most prominent feature of cannabis use is an initial period of euphoria and relaxation, which is followed by a depressant period (3). Use of cannabis affects the execution of complicated mental tasks that require a concerted action of attention, memory, and control of movement (4).

Except for fiber applications, cannabis cultivation is prohibited in most countries. Nonetheless, many EU countries report the growth of cannabis (1). Until the 1980s, cannabis was mainly cultivated outdoors for the production of female flower buds. Cultivation was strongly influenced by weather conditions and day length. The risks for the grower were high, e.g., due to theft or confiscation by the police. In the 1980s, indoor cultivation of cannabis was initiated in The Netherlands in order to evade law enforcement and to become less dependent on environmental conditions. Indoor cultivation became "professionalized" by the growth of nonpollinated female plants (sinsemilla), the use of cuttings taken from high-quality mother plants, and the use of hydro culture systems (5). Indoor cultivation allowed the growth of cannabis the whole year round, with four to six harvests a year. The use of faster and more controlled plant growth under optimal growing conditions in combination with breeding of new high performing varieties resulted in increased yields of flower buds and increased THC levels. For "nederwiet," the average THC level was reported to have increased from 9% in 1999/2000 to 15% in 2001/2002 (6). In 1997, The Forensic Science Service Laboratory in London measured an average THC level in flower buds of 9.4% with extreme levels up to 19% (5).

Little scientific information is available on the yield of female flower buds from cannabis cultivation. In newspaper articles, yields up to 50 g of flower buds per plant have been reported. In 1997, forensic science sources in the U.K. estimated the yield of flower buds at 15–20 g/plant (7). Based on case studies, Hinzen and Poortman-van der Meer (8) estimated the yield for "nederwiet" at 22 g/plant in 1995. This yield estimation is used in Dutch court proceedings to determine the potential financial profits of the illicit cannabis grower.

In order to obtain a scientifically determined estimation of the yield of illicit indoor cannabis cultivation in the Netherlands, cannabis plants seized by the police were used to determine the yield of dried female flower buds. The developmental stage of flower buds of the seized plants was described on a scale from 1 to 10, where the value of 10 indicates a fully developed flower bud ready for harvesting. Using eight additional characteristics to describe the grow room and growing conditions, regression analysis with subset selection (9) was carried out to develop two models for the yield of indoor cannabis cultivation. The model for yield per plant gave a prediction of 33.7 g female flower buds per plant. The second model for yield per m^2 gave a prediction of 505 g flower buds per m^2 for a median Dutch grow room.

Materials and Methods

Samples

Samples of cannabis plants were collected by the police during house searches in buildings or houses that were used for illicit cultivation of cannabis. Grow rooms containing less than 12 plants were excluded from the survey because these rooms cannot supply the minimum sample size of 12 plants. In total, 86 samples of plants in different stages of flower development were collected in 10 different police regions in The Netherlands. The police filled in a form to describe the situation encountered in the grow room. This form requested information on the number of plants, the size of the growing area, the size of the growing area occupied with plants, the type of substrate (soil/potting compost, rockwool,

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TABLE 1—*Morphological characteristics of the female flower buds used to determine their developmental stage.*

Developmental Stage	Morphological Characteristics
1	Onset of flowering
2	Small green female flower
3	Developing green flower
4	Developed green flower
5	Onset of drying
6	Color transition of hairs to red-brown
7	Onset of resin formation
8	Progression of resin formation
9	Almost fully developed flower
10	Fully developed flower, much resin, harvest stage

hydro-culture, or other), the type of heating (no heating, heating, or thermostat-controlled heating), the presence of sticky traps to indicate the presence of insect pests, the type of ventilation (no ventilation, ventilation without aspiration, or ventilation with aspiration to the outside), the type of growth lamps, the wattage of the lamps and the number of lamps in the grow room, the application of additional CO_2, and the presence of fertilizers and additives.

The growth area was sampled randomly according to a defined protocol: cannabis plants were taken along the legs of a virtual X laid over the area occupied with plants. Border plants were excluded from sampling because these have a comparative advantage over other plants and will not reflect the average yield adequately.

Each sample of 12 plants was packed in paper bags and delivered to the laboratory of Plant Research International within 24 h. Upon arrival, the developmental stage of the female flower buds was determined based on morphological characteristics (Table 1). Each sample of twelve plants was randomly separated into two duplicate batches and dried at 35°C for 3 days. From each batch of six plants, the female flower buds were plucked and weighted, resulting in two weight values per sample.

Statistical Analysis

The reliability of the duplicate batches was determined by comparison of the two weight values of each sample. If duplicate values differed by more than three times the standard deviation of the differences of the duplicate values, analytical data were checked for inconsistencies. On the basis of these analyses, three samples were omitted from the analysis. Six additional samples were omitted from the analysis because the accompanying forms lacked essential information.

Linear regression models for yields of female flower buds per plant as well as flower bud yield per m^2 were developed by subset selection (Genstat 7.2 for Windows, VSN International). The value for yield of flower buds per confiscated plant used in the model was calculated by taking the average of the 12 plants in the two batches of six confiscated plants from one sample. The explanatory variables of the model are described in Table 2.

Results

Eighty-six samples of 12 *Cannabis* plants each were collected in 10 different police regions in The Netherlands. The stage of female flower bud development was determined based on the morphological description in Table 1. To predict the yield of female flower buds at the various stages of development, two linear regression models were developed based on 77 of the 86 samples

TABLE 2—*Description of the explanatory variables used for model selection.*

Explanatory Variables	Description
Developmental stage	See Table 1 for description
Plant density	Calculated by dividing the number of plants per grow room by the size of the growing area occupied with plants
Wattage of growth lamps per m^2	Calculated by multiplying the total number of lamps with the wattage of the lamps and dividing this by the size of the growing area occupied with plants
Type of growth lamps	Brand and type of the growth lamps
Type of substrate	Soil/potting compost, rockwool, hydro-culture, or other
Type of heating	No heating, heating, or thermostat-controlled heating
Type of ventilation	No ventilation, ventilation without aspiration, or ventilation with aspiration to the outside
Presence of sticky traps	Not present or present
Presence of additional CO_2	Not present or present
Presence of fertilizer and additives	Not present or present

The size of the growing area was not included in the explanatory variables.

(nine samples were omitted from the analysis due to inconsistencies in the duplicate values or missing data) using the subset selection method described by Furnival and Wilson (9).

The main characteristics of the grow rooms are shown in Fig. 1. In 42 grow rooms, plants were grown in pots with potting soil while in 35 grow rooms hydro culture systems with rockwool were applied. Most grow rooms (23) contained 100–200 plants, while nine grow rooms contained over 1000 plants (Fig. 1*a*). On average, a grow room contained a total of 549 plants, and the median was 259 plants. Thirty grow rooms had a plant density of 9–16 plants/m^2 and in 20 cases the plant density was 17–24 plants/m^2 (Fig. 1*b*). Of the 77 samples analyzed, the average plant density was 18.1 plants/m^2 and the median was 15.3 plants/m^2.

In all grow rooms, horticultural growth lamps of 400 W or 600 W were present. The majority of the lamps were Philips (Master SON-T) lamps. The wattage of the growth lamps was between 500 and 600 W/m^2 in 17 grow rooms and between 300 and 400 W/m^2 in 15 others (Fig. 1*c*). The average wattage was 569 W/m^2, and the median was 510 W/m^2.

Based on these data, the median illicit Dutch grow room consists of 259 plants, with a plant density of 15 plants/m^2 and a wattage of 510 W/m^2. The developmental stage of the confiscated plants varied between developmental stages 2 and 8.5 (Fig. 1*d*).

As input for the models to predict the yield of female flower buds per plant or per m^2, the explanatory variables described in Table 2 were used. The combination of the variables that can predict the yield has been analyzed using subset selection. For both models, yield per plant and yield per m^2, 37% of the variance was accounted for by three explanatory variables: developmental stage, plant density, and wattage per m^2 (Table 3). The model for yield of flower buds per plant with three explanatory variables is described by the following formula:

$$\text{yield of flower buds per plant} = -8.06 + 4.261^{*}[\text{developmental stage}] - 0.482^{**}[\text{plant density}] + 0.01242^{*}[\text{wattage of growth lamps per m}^2]$$

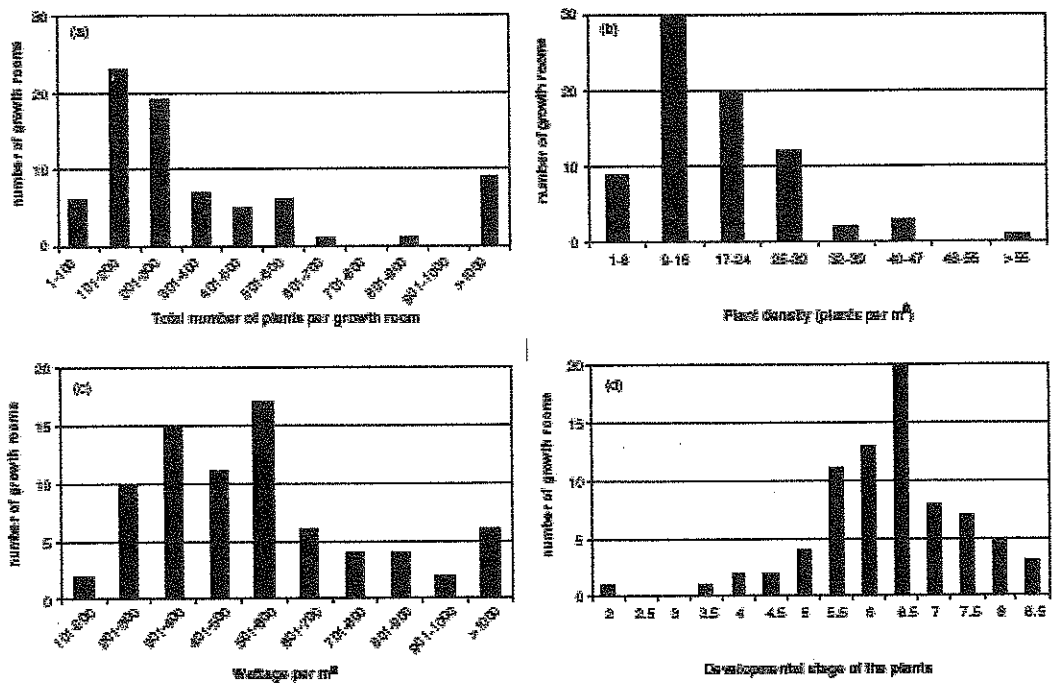

FIG. 1—*Overview of the distribution of the main characteristics of the 77 grow rooms. The distribution of (a) the total number of plants per grow room, (b) the number of plants per m² (plant density), (c) the wattage of growth lamps per m², and (d) and the developmental stage of the plants are shown.*

The model for yield of flower buds per m^2 with three explanatory variables is described by the following formula:

$$\text{yield of flower buds per m}^2 = -386 + 69.8*[\text{developmental stage}] + 6.5*[\text{plant density}] + 0.1838*[\text{wattage of growth lamps per m}^2]$$

Table 4 shows the regression coefficients, standard errors, *t*-values, and *p*-values for both models. On the basis of these models, the yield of female flower buds per plant or per m^2 can be predicted for each developmental stage, given the plant density and wattage of growth lamps per m^2. Table 5 shows the predicted yields for the median Dutch grow room (15 plants/m^2, 510 W/m^2) with the lower bound of the one-sided 95% confidence interval for developmental stages 8–10 where stage 10 represents the fully mature flower buds, ready for harvesting.

Inclusion of the variable ventilation in the models further increased the percentage of variation accounted for to about 41% for both models. Compared with the reference situation with the parameter "ventilation with aspiration to the outside," the parameter "ventilation without aspiration" led to a decrease in yield. The parameter "no ventilation" only applied to one sample and was not significantly different from the reference situation.

Discussion

In order to predict the yield of illicit indoor cannabis cultivation in the Netherlands, data from 77 samples seized by the police were analyzed statistically. The median Dutch illicit grow room consists of 259 plants, has a plant density of 15 plants/m^2, and 510 W of growth lamps per m^2. Based on regression analysis with subset selection, models were developed to predict the yield of female flower buds per plant and per m^2. For both yield of flower buds per plant and yield of flower buds per m^2, the model with three

TABLE 3—*Model selection for the response variables yield of female flower buds per plant and yield of flower buds per m².*

	Explanatory Variable(s)	Adjusted R^2	
		Yield per Plant	Yield per m^2
1	Developmental stage	21.93	17.89
2	Developmental stage, plant density	28.25	32.52
3	Developmental stage, plant density, wattage per m^2	36.56	37.15
4	Developmental stage, plant density, wattage per m^2, ventilation	40.74	41.54
5a	Developmental stage, plant density, wattage per m^2, ventilation, fertilizer	43.69	—
5b	Developmental stage, plant density, wattage per m^2, ventilation, presence of sticky traps	—	41.54

The best subset with 1–5 explanatory variable(s) based on adjusted percentages of variance accounted for (adjusted R^2) is indicated.

TABLE 4—*Regression coefficients with standard error (SE), t-value (t(73)), and p-value (p) for the variables of the models that predict the yield of female flower buds per plant and the yield of flower buds per m².*

	Yield per Plant				Yield per m²			
Variables	Coefficient	SE	t(73)	p	Coefficient	SE	t(73)	p
Constant	−8.06	5.99	−1.35	0.183	−286	184	−1.55	0.001
Developmental stage	4.26	0.857	4.97	<0.001	69.8	16.3	4.27	<0.001
Plant density	−0.492	0.117	−4.13	<0.001	6.5	2.22	2.95	0.004
Wattage per m²	0.01342	0.00380	3.27	0.002	0.1836	0.0724	2.54	0.013

TABLE 5—*Prediction of the yield of female flower buds per plant and per m² for the median Dutch grow room (median value of 15 plants/m² and 510 W/m²) for developmental stages 8–10 with a one-sided (p) 95% confidence interval.*

	Yield per Plant			Yield per m²		
Developmental Stage	Predicted Yield of Flower Buds (g)	SE	Lower Bound of One-Sided 95% Confidence Interval (g)	Predicted Yield of Flower Buds (g)	SE	Lower Bound of One-Sided 95% Confidence Interval (g)
8	25.1	1.76	22.2	365	33.6	309
9	29.4	2.51	25.2	435	47.7	355
10	33.7	3.31	28.1	505	63.1	399

explanatory variables (developmental stage, plant density, and wattage per m²) accounted for 37% of the variance. For both models, the percentage of variance accounted for was increased to about 41% by adding the variable ventilation. However, this variable was not included in the model because the parameter "ventilation with aspiration to the outside" was only significantly different from the parameter "ventilation without aspiration," while it was not significantly different from the parameter "no ventilation." The type of growth lamps used in the different grow rooms was very similar and, therefore, did not influence the percentage of variance accounted for. Also, the type of substrate used did not influence the yield significantly.

It could be possible to further improve the models by incorporating other explanatory variables. Variables like the genotype of the plant, the quality of the starting material (cuttings or seeds), and the presence of diseases may have a significant effect on flower bud yield. Also, other, more difficult to define variables, such as the skill of the grower, may influence the yield of flower buds. Including these factors in the analysis could increase the percentage of variation accounted for and further improve the predictive value of these explanatory models.

There is little scientific information about illicit cannabis cultivation in The Netherlands. No central registration of dismantled grow rooms is carried out. The only data available are from a number of case studies by Bovenkerk and Hogewind (10).

The study described in this paper shows a large variation in the size of grow rooms (from 12 to 7800 plants per grow room). The relatively small number of large grow rooms strongly influenced the average size as shown by the average of 549 plants per grow room compared with a median of 259 plants per grow room. In 2001, a total of 2012 grow rooms were dismantled and 884,609 "nederwiet" plants were confiscated by Dutch police (1). This corresponds to an average of 440 plants per grow room. A case study in Utrecht (The Netherlands) (10) showed a distribution of the size of the grow rooms that is comparable to the distribution shown in Fig. 1a, with an average of about 280 plants per grow room. It has to be noted that all data available are based on data from police confiscations and that the actual average number of plants per grow room might differ from the above values. This has to do with the fact that police searches are probably not random. Searches are initiated based on internal police strategies or carried out after reports e.g., by neighbors. Professionally equipped grow rooms with high-quality air filtering or large grow rooms in the country-side might be detected less frequently.

The yield of female flower buds at a given developmental stage is described by the models as a function of the plant density and the wattage of growth lamps per m². In 1999, Forensic Services in the U.K. estimated that the buds of a female plant can produce 10–15 g of marketable cannabis (7). In The Netherlands, Huizer and Poortier-van der Meer (8) estimated the yield for "nederwiet" at 22 g/plant. In popular cannabis cultivation literature, average yields of 366–610 g/m² are described (11). For the median Dutch grow room with 15 plants/m² and 510 W of growth lamps per m², the models developed here estimate the yield at the harvestable developmental stage 10, at 33.7 g/plant or 505 g/m².

Implementation of these numbers in case law will be the responsibility of the public prosecutor. For The Netherlands, the Dutch Criminal Assets Deprivation Bureau advises to comply to the lower limit of the one-sided 95% confidence interval. In that case, the minimal yield for a median Dutch grow room is 28.1 g/plant or 399 g/m² (12).

Acknowledgments

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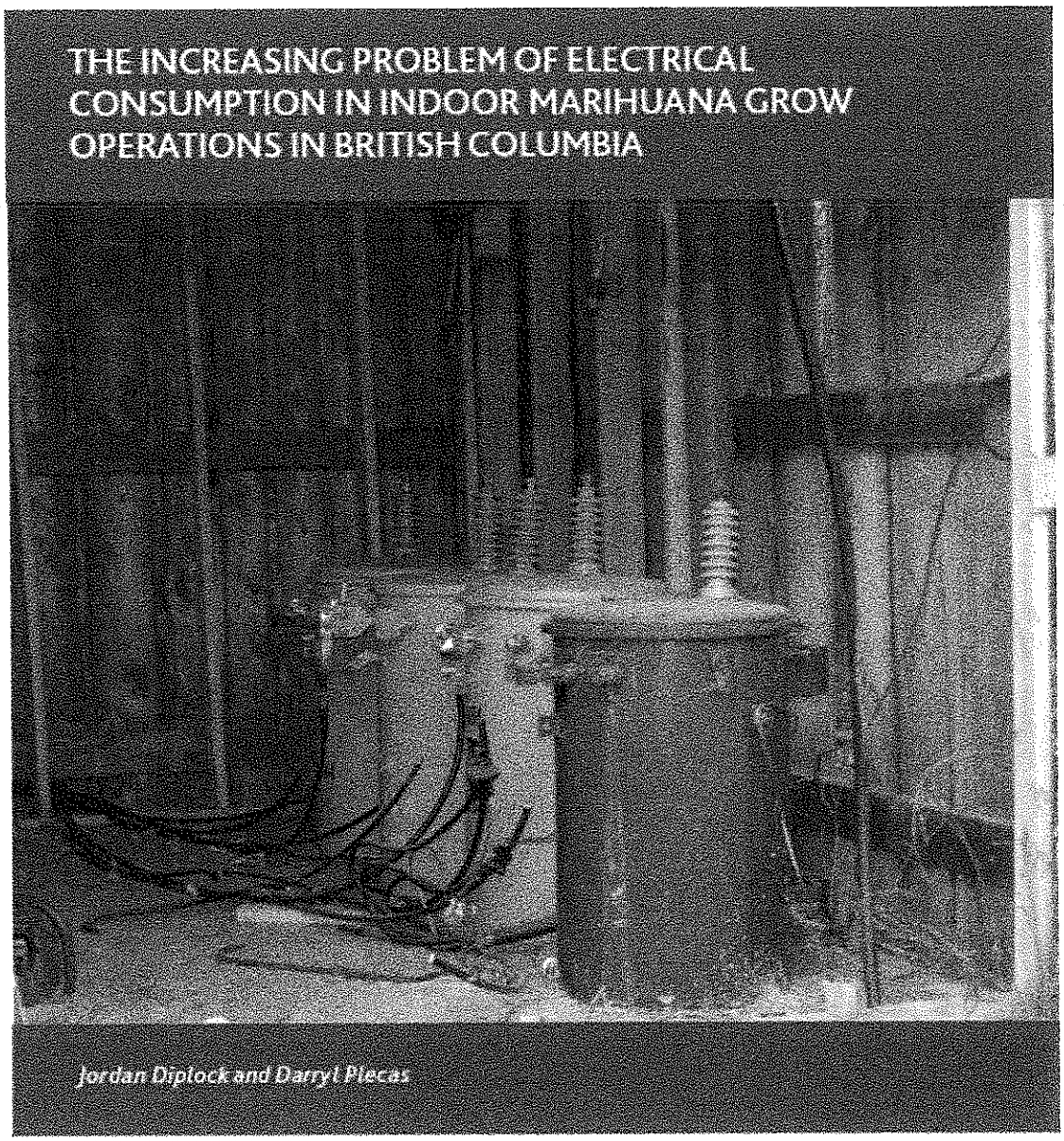
Appendix R: Diplock & Plecas (2011) The Increasing Problem of Electrical Consumption in Indoor Marihuana Grow Operations in British Columbia



UNIVERSITY OF THE FRASER VALLEY

CENTRE FOR PUBLIC SAFETY & CRIMINAL JUSTICE RESEARCH

Introduction

The production of marihuana is a criminal activity that is very profitable for offenders and harmful to communities (Plecas, Diplock, & Garis, 2009). Growers in British Columbia commonly set up their operations indoors, in homes, and other buildings to avoid detection and to cultivate plants year round. Moreover, indoor buildings allow growers the opportunity to set up large and increasingly sophisticated operations that provide greater control over the growing process than can be generally maintained outdoors. These indoor operations are set up with the intention of making commercial profit, referred to as 'commercially viable growing operations' typically use large amounts of electricity to power high-wattage bulbs used for growing, along with other equipment. Along with the enormous consumption of electricity from the thousands of marihuana growing operations in British Columbia comes a myriad of serious problems that affect all British Columbians.

Indoor marihuana growing operations present a serious threat to public safety in the communities in which they operate.[1] For example, electrical hazards pose a very real threat to both occupants of the house and their neighbours. The changes made to houses and other buildings to supply power to marihuana growing operations require special training, certification, and inspection to ensure proper function and safety. However, in the pursuit of high profits, growers are more concerned with avoiding detection than preventing electrical hazards. Therefore, indoor marihuana growing operations, and the risks associated with any improper electrical work done to support them, are not subjected to the regulation and maintenance of safety standards that are in place to protect the public from serious risks.

Indoor growing operations consume much more electricity than normal residential homes, as they run multiple large wattage lights and other equipment (Garis and Plecas, 2007). This increased need for electric power means that the typical grow operation exhibits electrical hazards that can increase the risk of fire and other harms (Garis, 2008). The many electrical hazards combine to make indoor marihuana growing operations at least five times more likely to catch fire than normal residential homes (Plecas et al., 2009). The operations commonly lack electrical protection for fuses and circuit breakers, have improperly installed electrical systems, and show a failure to enclose electrical bypasses. Those within the grow site are at risk of shock and electrocution, as there is commonly water present (Garis, 2008). Not only are these hazards problematic for the growers and others inside the operations, but they also put unsuspecting neighbours, first responders, and utility workers at a great risk.

Recent trends suggest that these risks will get worse. Data from founded marihuana growing operations 'busted' by police in British Columbia in 2003 (Plecas, Malm, & Kinney, 2005) indicated that the average size of an indoor grow operation was 15.5 lights. At that time, growing operations had been increasing in size since 1997 (Plecas et al., 2005). The most current analysis of growing operations in at least several jurisdictions in the province indicated, where the use of electricity could be confirmed, that the average founded growing operation between 2006 and 2010 used approximately 27.5 lights (see Chaisson and Plecas, 2011a; Chaisson and Plecas, 2011b). The substantial increase in the number of lights since before 2006 is consistent with the finding that the average size of growing operations has more than doubled since the release of the Plecas et al. (2005) report (Chaisson and Plecas, 2011a; Chaisson and Plecas, 2011b). It is apparent that the trend towards using more electricity to produce larger crops continues. Growing operations are also more likely to use other

[1] For a detailed discussion of the numerous harms associated to marihuana growing operations see Plecas et al. (2009).

specialized equipment, such as dehumidifiers, machines to increase levels of CO_2, and cooling units to reduce heat (Garis and Plecas, 2007). This equipment increases the energy requirements of the average grow operation. Furthermore, it is clear from the most current analysis of growing operations that a larger proportion of growers are stealing power (Chaisson and Plecas, 2011a; Chaisson and Plecas, 2011b). In fact, the proportion of growers stealing power appears to be approximately 52%, which is more than double the proportion reported by Plecas et al. (2005) based on information from 1997 to 2003.[2] This is not surprising given the increasing size of growing operations and the risks of detection that accompanies the increased energy consumption.

The purpose of this report is to provide further insight into the increasing problems associated with the electrical consumption of indoor marihuana growing operations in British Columbia. The problems are not just related to the well-documented dangers of electrical hazards within growing operations, but the increasing economic and societal threats. The analysis begins by using recent data from the number of founded marihuana growing operation police files from British Columbia to estimate the total number of operations currently operating across the province. This estimate will be based on existing estimation methods and information related to the proportion of indoor marihuana growing operations that steal electricity. Using the estimated number of growing operations in British Columbia, a discussion of the total electricity consumption of illegal marihuana growing operations will be provided, in addition to an analysis of the economic and societal problems caused. This report concludes by examining the need for action beyond current efforts, which may come in the form of new smart metering to curb the theft of electricity and the over-consumption of this limited resource for illicit purposes.

The Number of Indoor Marihuana Growing Operations in British Columbia

In a previous article (Plecas et al., 2009), the authors examined several methods for estimating the total number of marihuana growing operations in British Columbia. These estimates were based on data on the number of founded grow operations that came to the attention of police in 2003. Without current data, the final estimate was intentionally conservative, concluding that at least 10,000 growing operations were producing marihuana. This number was less than, but not substantially different, from estimates that arose from the adaptation of methods originally described by Easton (2004) and Bouchard (2007). With newly acquired recent police data, it is possible to provide a more accurate and up to date approximation of the number of growing operations in the province.

Information from police data indicated that there were 2,348 founded cases of marihuana production in British Columbia in 2010 (RCMP, 2011). Of these cases, approximately 90% were indoor operations; a total of 2,113 founded indoor grows. Without a range of detailed data on the offenders associated to these founded grows, using Bouchard's (2007) capture-recapture model was not possible. However, since the estimate produced

[2] This figure is nearly identical to the estimate provided to the authors from BC Hydro, which indicated that at least 51% of growing operations that came to the attention of their field inspectors were stealing electricity. It is also nearly identical to the estimate provided to the authors by individuals who have operated illegal grow operations and who have a broad knowledge of the industry. These individuals reported that generally "half" of all operators today steal electricity.

from Bouchard's model was very similar to that of Easton's (2004) model[3], Easton's economic model alone will be used to provide one of the alternative estimates of the number of marihuana growing operations in British Columbia. Based on an analysis of the costs and potential profit of operating a marihuana growing operation, Plecas et al. (2009) concluded that the value to cost ratio (1.5) used by Easton (2004) was consistent with their findings of an average of 1.41. Assuming that the risks, the costs of operating a growing operation, and the value of the product have not changed significantly since the analysis by Plecas et al. (2009), Easton's formula can also be used to estimate the number of active grow operations in the province in 2010. Changing only the number of founded indoor growing operations, Easton's method produces an estimated total of 13,2064 active grow operations in British Columbia in 2010. Notably, this figure is also very close to the 13,500 estimate provided to the authors from BC Hydro, who came to this figure by extrapolating from Easton's (2004) calculations of the number of growing operations in 2000.

The Extent and Value of Consumption

The estimated 13,206 active growing operations present a considerable threat to the sustainability of hydro electricity in British Columbia. A typical growing cycle involves at least 18 hours of light each day for the first month, followed by two months of 12 hours per day. As a typical growing light is a 1000 W bulb, a grow operation uses, on average, 14kWh per day for each light over the course of a crop. Using the approximation that a crop takes 90 days to cultivate, and four crops can be produced in a year, the annual consumption of electricity per light is approximately 5,040 kWh. Further, using the findings of Chaisson and Plecas (2011a) and Chaisson and Plecas (2011 b), growers who diverted electricity for their operations used approximately 36 lights. This figure closely reflected the figures provided to the authors by BC Hydro, whose data indicated that, on average, 36.5 lights were used per growing operation that stole electricity. Accordingly, the average growing operation using diverted electricity stole 181,440 kWh per year. Given this, the 52% of growing operation that stole electricity represented 6,867 operations with an overall theft of nearly 1,246 GWh per year across the province.

As of April, 2010, BC Hydro charged $0.0627 per kWh for consumption up to the first 1,350 kWh used over a two month period, with the rate increasing to $0.0878 per kWh for the balance consumed during the period (BC Hydro, 2011). This residential "stepped rate" is the likely rate that would be charged to operators of marihuana growing operations within the company's service territory. Using only the lower rate ($0.0627/kWh), the total value of electricity theft would be $78.1 million per year. Of course, given that the vast majority of the electricity consumed per growing operation would be charged at the higher stepped rate of $0.0878 per kWh, the total annual value of the theft is likely closer to $109.4 million.

What must also be taken into account is the amount of electricity consumed by operators of marihuana growing operations not stealing electricity. This would include another 6,339 cases per year. Again, using the findings of Chaisson and Plecas (2011a) and Chaisson and Plecas (2011 b), each of these operations, on average, would use 21.8 lights or 109,872 kWh of electricity per year. The annual consumption then, which is, in effect, wasted consumption, on account that it is put toward an illegal enterprise, is nearly 696.5 GWh. At

[3] In Plecas et al. (2009), the use of Bouchard's model yielded an estimate of 11,500 total growing operations, while Easton's model produced an estimate of 12,500.

[4] Easton (2004) estimated the number of marihuana growing operation using the formula $T = B[1+PQ/C)]/[(PQ/C)-(1+R^*)]$, where T is the total number of growing operations, PQ/C is a ratio of value to cost = 1.5, R^*=.10 is the assumed return to legal activities, and B is number of founded marihuana growing operations discovered by police during the year.

$0.0627/kWh, this equates to another $43.7 million worth of electricity per year. Priced out at the higher rate, the cost would actually be $61.2 million. That said, BC Hydro would not peg the cost this high, as its investigators have estimated that the average growing operation not involving theft uses just 10 lights. BC Hydro's estimate would be particularly accurate in those locations that currently employ electrical and fire safety inspection (EFSI) initiatives, as growing operations with 10 or more lights would consume more electricity than the 93kWh per day threshold for over-consumption, and would come to the attention of EFSI inspection teams, rather than BC Hydro's own inspectors. According to the BC Hydro estimates, at the higher rate ($0.0878), we should expect their estimate to be substantially lower at $28.1 million.

The Economic and Societal Problems

There are numerous economic problems associated with this level of energy consumption going toward illegal ventures. Perhaps the most obvious is the threat to British Columbia's electricity suppliers, primarily BC Hydro, as the nearly $109.4 million dollars of lost revenue presents a real challenge to supplying British Columbians with sustainable, low-cost energy. Those revenue losses will be ultimately borne by legitimate electricity customers in British Columbia, who will face higher rates for their electricity consumption. This should be especially concerning for legitimate customers because the actual revenue lost by BC Hydro translates into much higher costs for British Columbians.

The current supply of electricity that can be offered relatively cheaply by BC Hydro as a result of their existing Heritage Resources is not enough to meet the growing demands of the province (BC Hydro, 2011). As such, the company must contract to independent power producers (IPPs) to meet the demand. According to BC Hydro's Clean Power Call (2010), this additional source comes at a much higher cost of $0.124 per kWh. Given that the production of marihuana is illegal, the power consumed by this industry illegitimately increases the province's demand for electricity, requiring the purchase of the more expensive electricity from IPPs. Therefore, if all theft of electricity from growing operations were eliminated, the savings to all other electricity consumers would be nearly $154.5 million. Furthermore, although electricity providers do not lose revenue from 'paid' growing operations, legitimate electricity consumers are still affected. These customers must pay increased rates because these operations still require a great deal of electrical power, which increases the overall demand for electricity above what would normally be needed, causing the rates to account for the higher priced energy provided by IPPs. Therefore, the total economic cost to legitimate electricity consumers in British Columbia of indoor marihuana growing operations is even higher than $154.5 million.

There are societal costs of this electricity consumption as well. Putting the very real problems of organized crime and substance abuse aside, the illicit marihuana production industry is a constant drain on British Columbians. Adding to the problem is the fact that the increased consumption caused by marihuana growing operation requires electricity providers to spend more money and more natural resources to develop new sources of power. BC Hydro (2011) reported that it was investing $6 billion to improve its capacity meet growing demand and provide electricity to its consumers. Building the infrastructure to supply electricity has an environmental impact, as well as an economic one.

It is particularly troubling that the illicit marihuana production industry profits so greatly, stealing valuable resources from legitimate users and negatively impacting communities and the environment, without contributing any money in taxation to even begin to offset the high societal costs. Using the Marihuana Indoor Production Calculator (Plecas, Diplock, Garis, Carlisle, Neal, & Landry, 2010) with the new figures from the current article, and assuming that the rate of domestic cannabis use in British Columbia has decreased, as it has across Canada (Health Canada, 2010), the total annual revenue generated by the domestic and export

wholesale distribution of marihuana is in the range of $3.6 billion to $4.5 billion.[5] The calculation indicates that only 9% to 12% of the marihuana produced in the province is consumed by British Columbians. Overall, this is an enormous amount of money generated tax free by criminals at the expense of British Columbians.

The Need For Action

This report has demonstrated that the indoor marihuana production industry is extremely costly for British Columbians, as it increases the economic and societal costs of electricity for the legitimate electricity consumers in the province. While growers who steal electricity are particularly costly for British Columbians, all grow operations negatively affect the costs of providing power for the province. At a time when British Columbians are encouraged to conserve electricity to ensure that this valuable resource can continue to be sustained in the future, a small, criminal segment of the population is profiting from a highly disproportionate level of consumption, leaving the law-abiding population to bear the costs. For the most part, the estimates presented in this article are conservative suggesting that the true costs are much higher. Also, as this report has focused specifically on the issues of electricity consumption, these figures do not come close to reflecting the total costs, which would also include, for example, law enforcement and health care spending.

While this report speaks to the issue of electricity theft in British Columbia, since all indoor grow operations require power, the matter of electrical theft as it relates to marihuana growing operations is one that should be given serious attention by other jurisdictions as well. There have been some successful initiatives targeting marihuana growing in British Columbia, specifically the EFSI initiatives, which uses a public safety approach to curbing marihuana production by focusing on over-consumption of electricity and the inherent hazards those levels of usage create in residential environments. However, the unintended consequences of these initiatives may have been to increase the likelihood that growers will divert electricity, not only reducing their production costs, but also decreasing their chances of being discovered as a result of over-consumption. Furthermore, EFSI initiatives are not viable in all parts of the province, which is potentially leading to the displacement of illegal marihuana production to those parts of the province without EFSI. It is imperative that policy makers, law enforcement, and electric utilities continue to develop innovative responses to this problem in order to reduce the economic and societal burden of this illegal behaviour. Given the British Columbia experience, which shows that growers are increasingly likely to steal power, and given that power costs should be expected to steadily increase significantly most everywhere in the near future, without serious attention, it would be safe to assume that the cost to the public (as high as it is now) will become increasingly expensive in the future.

[5] The Marihuana Indoor Production Calculator estimates the size of the marihuana industry by incorporating estimates of the population of the jurisdiction, the percent of the population who have used the drug in the past year, the average number of lights used in growing operations, and the number of growing operations in the jurisdiction. The tool assumes that each light produces one pound of marihuana for each crop, and that four crops can be produced per year. Using the average number of lights and total number of operations, one can calculate the total amount of marihuana produced in the jurisdiction. The calculator uses the price of $2000/lb for domestic sales and $3000/lb for export sales. Based on the population, the proportion of users, average weight of marihuana cigarettes, and average number of marihuana cigarettes smoked per person per year, the calculator determines the size of the domestic market and assumes that the remaining product is exported. See Plecas et al. (2010) for a detailed description of the tool.

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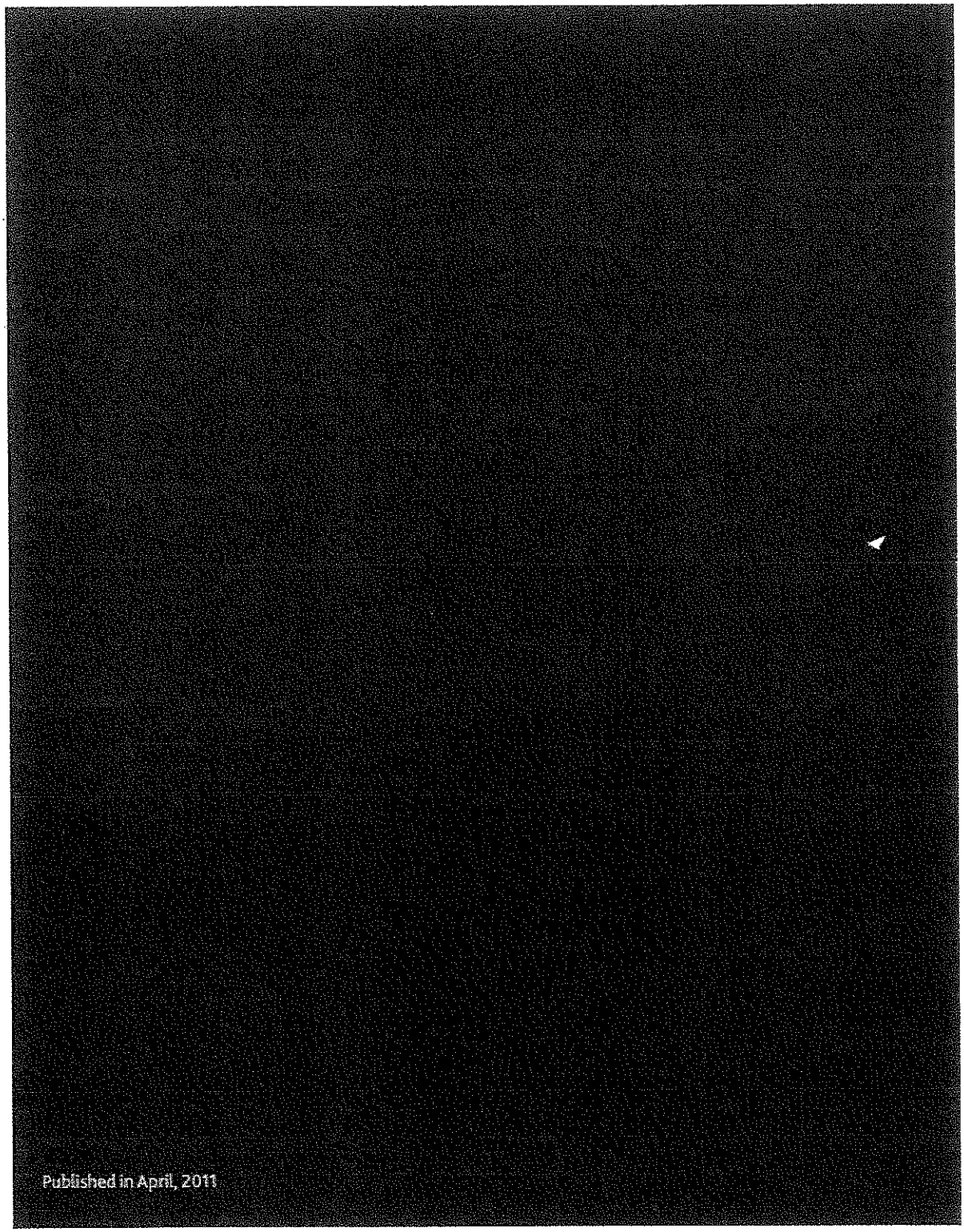
Published in April, 2011

Appendix S: Curriculum Vitae: Darren Waddell

August 7, 2014

Darrell Waddell

Owner & Operator:

MD Waddell Holdings Ltd.
Electrical and Power Line Contracting

26660 60th Ave
Aldergrove, British Columbia. V4W 1V7
Business Phone: 604-856-7954
Cellular Contact: 778-808-8234

Personal Information

- B.C. Electrical Contracting License # 201587
- B.C. Electrical F.S.R. # 18784
- Electrical T.Q. # 7717-EW-94
- Power Line Technician # 00071-10

Work History and Experience

- Employment in Family Electrical Business since 1978.
- Residential wiring.
- High Voltage Installations
- Commercial wiring
- Industrial wiring

I received my Electrical Accreditation and Unrestricted A Contractor license in April 1994.

While working for Family Business Waddell Electric Ltd. Our company was approached in January 1998 by BC Hydro Power Authority for a pilot program to investigate Theft of Electricity from this Utility. This program was initiated to help the bolster crew compliment to attend various locations throughout Greater Vancouver and The Fraser Valley Region of British Columbia as the problem of Theft and Financial Loss to BC Hydro became an Epidemic. Primarily all responses to these Call-outs had to do with Suspected Theft of Electricity associated with MARIJUANA Grow Operations. The program was expanded to a larger amount of response area and a long term Labor Contract was negotiated.
As time went on I was also indentured to detect and locate Energy Diversions (Thefts of Electricity) and eventually hired to full time Contracting to BC Hydro Security Department. I worked across British Columbia as a Primary Electrical Investigator for the detection of these thefts.

From January 1998 to January 2012 I have investigated and dismantled the Electrical Apparatus of over 2500 MARIJUANA Grow Operations.

Detected and Located over 1000 Thefts of Electricity.
I have testified in Federal and Supreme Court on over 300 Criminal cases as an Expert in the area of Electrical Investigating and Theft of Electricity in conjunction with MARIJUANA Grow Operation Electrical Apparatus.
I have given Expert Opinion on Electrical Apparatus and Consumption Records for Police Agencies across Western Canada for the assistance of a Warrant to search relating to Suspected Illegal MARIJUANA Grow Operations.
I have also given Expert Opinion related to the Electrical Apparatus, Theft of Electricity and Damages as a result of a MARIJUANA Grow Operation in regards to Civil Court Cases.

-Darrell Waddell

Appendix T: Electrical Safety Risk Scale

Review of Electrical Hazards at Marijuana Grow Operation Sites

Provided by: Darrell Waddell, owner and operator of MD Waddell Holdings Ltd., Electrical and Power Line Contracting. See CV in Appendix S.

In summer 2014, Adjunct Professor/Fire Chief Len Garis commissioned MD Waddell Holdings Ltd. as part of a study led by the University of the Fraser Valley. The instructions were to:

- review photographs from all Electrical and Fire Safety Initiative case files for residential marijuana grow operations, and
- develop an electrical safety risk scale to be used to assess the electrical hazards seen in the photographs.

Based on the EFSI photographs, the following grading system was developed:

EXTREME: Electrical apparatus is in a state of condition that can/or will cause:

- Electrocution to persons and personnel.
- Fire. Affecting the immediate structure as well as neighbouring buildings.
- Catastrophic failure/and or fault to residential, utility and neighbouring electrical apparatus.

Typically these are a result of Electrical Diversion (Appendix U). In these instances, all electrical apparatus must be immediately disconnected from the utility. Due to the severe danger of this type of illegal application, barricading and non-entry may be employed until at such time as a utility disconnect can be performed.

Repairs must be made by qualified personnel and electrical inspection required before apparatus can be re-energized.

HIGH: Electrical apparatus is in a state of condition that can/or will cause:

- Electrocution to persons and personnel.
- Fire. Affecting the immediate structure as well as neighbouring buildings.
- Catastrophic failure/and or fault to residential, utility and neighbouring electrical apparatus.

Electrical apparatus has been left or is in a condition where there is no guarding or cover over exposed bus bars, circuit panels, junction boxes and/or multiple bare wires. (Appendix U). The electrical apparatus must be disconnected at source (typically utility meter base).

Repairs must be made by qualified personnel and electrical inspection required before apparatus can be re-energized.

MODERATE: Electrical apparatus is in a state of condition that can/or will cause:

- Electrocution to persons and personnel.

Here, electrical apparatus equipment is or has been installed to non-standard specifications. (Appendix U)

Repairs must be made by qualified personnel and electrical inspection required before apparatus can be re-energized.

LOW: Electrical apparatus is in a safe condition or data is inconclusive.

Appendix U: CSA Electrical Code Violations

CSA Electrical Code Violations

Professional assessment provided by Darrell Waddell, owner and operator of MD Waddell Holdings Ltd., Electrical and Power Line Contracting. See CV in Appendix R.

Electrical Diversion:

Typically any Electrical Apparatus attached ahead of the Utility Meter, Electronic Devices to alter the Meter Recording Equipment, Shunts/Jumpers to Bypass main flow of Electricity from Utility Meter Recording Instrument Bus Bars and Internal Tampering of Utility Meter.

CSA Code Rules
2-032
(1) No person shall damage any electrical installation or component thereof.

Exposed Electrical Equipment and Wiring:

CSA Code Rules

2-200 Electrical equipment shall be installed and guarded so that adequate provision is made for the safety of persons and property and for the protection of the electrical equipment from mechanical or other injury to which it is liable to be exposed.

2-202
(1) Bare live parts shall be guarded against accidental contact by means of approved cabinets or of other forms of approved enclosures except where bare live parts are
(a) Located in a suitable room, vault or similar area that is accessible **only** to Qualified Persons; or
(b) as permitted elsewhere by this Code.

(2) Where electrical equipment has mounted on it, within 900 mm of bare live parts, non-electrical components that require servicing by unqualified persons, suitable barriers or covers shall be provided for the bare live parts.

(3) Entrances to rooms and other guarded locations containing exposed bare live parts shall bee marked with conspicuous signs **forbidding** entry to unqualified persons.

Non-Standard Equipment and Wiring:

CSA Code Rules

2-024 Electrical equipment used in electrical installations within the jurisdiction of the inspection department **shall be approved and shall be of a kind or type and rating approved for the specific purpose for which it is to be employed.**

2-300
(1) All operating electrical equipment shall be kept in safe and proper working condition.

Appendix V: Kurup et al. (1983) Allergenic Fungi and Actinomycetes in

Mycopathologia 82, 61–64 (1983).

Allergenic fungi and actinomycetes in smoking materials and their health implications

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Abstract

Street marijuana, commercial cigarettes and pipe tobaccos were studied for the presence of fungi and actinomycetes associated with hypersensitivity pneumonitis. *Aspergillus* species and thermophilic actinomycetes were isolated from the smoking materials. In addition, *Aspergillus fumigatus* spores were isolated from marijuana smoke, indicating the potential hazard involved in developing serious disease. Precipitin antibodies against fungi, particularly *Aspergillus*, showed a higher prevalence in marijuana smokers, whereas only very few cigarette smokers and nonsmokers demonstrated antibodies to fungi. Cigarette smokers and nonsmokers showed more or less similar prevelance of antibodies against thermophilic actinomycetes.

Introduction

A number of fungi and actinomycetes have been shown to cause hypersensitivity pneumonitis (HP) in man (3, 14). These organisms have been isolated from various working and living environments of patients (8). Inhalation of numerous spores of these organisms will sensitize the individuals and, on prolonged inhalation of heavily contaminated air, may result in HP. Hypersensitivity pneumonitis is an immunologic interstitial lung disease which may be incapacitating if not diagnosed early and controlled by avoidance of insulting antigens. The commonly recognized diseases include farmer's lung, bagassosis, mushroom worker's lung and ventilation pneumonitis are caused by inhalation of thermophilic actinomycetes and various fungi or other organic dusts.

Smoking marijuana contaminated with *Aspergillus fumigatus* has been recently attributed to several cases of aspergillosis (2, 5, 11). This led us to evaluate smoking materials such as cigarettes, pipe tobacco and marijuana for the presence of thermophilic actinomycetes and fungi, and the possible role of these organisms in the development of immunologic lung diseases.

Materials and methods

Smoking materials. Twenty-four samples of street marijuana, 12 different brands of cigarettes (both with and without filters) and 3 samples of pipe tobacco were studied. Approximately 100 mg of the sample was suspended in 10 ml of sterile distilled water. After vigorous mixing the suspension was allowed to stand for 10 minutes. About 1 ml of the supernatant was serially diluted and cultured in appropriate media.

Culture

For the isolation of fungi, 1 ml of the suspension was plated in Sabouraud's glucose agar (SGA) and

SGA with streptomycin (40 μg/ml) and penicillin (40 units/ml) to suppress bacterial growth. One set each of the inoculated plates were incubated at 37 °C and at 25 °C. Cultures were examined regularly for up to 2 weeks and growth appearing on the plates were identified by standard procedures.

Cultures for the isolation of thermophilic actinomycetes were made in tryptic soy agar (TSA) and in TSA with novobiocin (25 μg/ml). The inoculated plates were incubated at 55 °C in plastic bags to prevent evaporation. Plates were examined daily for 10 days. Growth appearing on the plates were subcultured and identified according to procedures previously described (6).

Fungal inhalation

Marijuana cigarettes obtained from patients and commercial brands of cigarettes were attached to an Andersen air sampler (1). The cigarettes were then lit and the smoke drawn through the sampler containing SGA or TSA plates. The plates were incubated and studied as described above. Spores present in equal volume of room air were also collected on the sampler impact plates and studied for comparison.

Antigen preparations

The detailed methods of antigen extraction from *Aspergillus* and thermophilic actinomycetes were reported elsewhere (7, 9, 10). Fungi were grown in a synthetic broth for 2–3 weeks at 37 °C in stationary cultures or 5 days in shaker cultures. Following incubation the culture filtrate was separated, dialyzed and freeze-dried. These antigens were used to detect antibodies in the sera of subjects included in the study.

Thermophilic actinomycetes were grown in synthetic medium or by the double dialysis method using dialyzate of tryptic soy broth (TSB) (9). *Micropolyspora faeni* was grown in a synthetic medium incubated at 50 °C in shaker incubators for 5 days (7). Culture filtrate was separated and processed as in the case of fungal antigens. *Thermoactinomyces candidus* and *T. vulgaris* were grown in stationary cultures incubated at 55 °C for 2 weeks in dialyzate medium. The culture filtrate was processed as described above.

Agar gel double diffusion

Antibodies present in the sera against various fungi and actinomycetes were tested by agar gel double diffusion (DD) method (9, 10). Antigens were used at a concentration of 5–10 mg/ml. After filling the wells with antigen and antibody the gels were incubated in a humid chamber for 48 hours. It was then washed to remove the unreacted antigen and antibody and stained by Coomassie blue. Precipitin arcs appearing on the slides were studied in comparison with appropriate positive and negative controls run along with the test sera.

Results

Culture results

The various fungi and actinomycetes isolated from smoking materials are shown in Table 1. *Aspergillus* species were the predominent group in the marijuana samples. *Thermoactinomyces candidus*, *T. vulgaris* and *M. faeni* were also isolated. Only one sample was completely negative for fungi or actinomycetes, but it was heavily contaminated with bacteria. Quantitative cultures yielded 10^5 to 10^7 colonies of fungi per gram of marijuana sample. When lit and unlit cigarettes were studied by the Andersen sampler, they yielded *Aspergillus fumigatus* and *Mucor*.

When studied quantitatively, tobacco cigarettes yielded 1.5×10^4 to 1×10^5 colonies of thermophilic

Table 1. Fungi and actinomycetes isolated from smoking materials.

Organism	Marijuana	Cigarettes	Pipe tobacco
	(24)	(12)	(3)
Aspergillus flavus	12	–	–
A. fumigatus	7	–	–
A. niger	6	–	–
Mucor sp.	10	–	–
Penicillium	2	–	–
Other fungi	4	–	–
Micropolyspora faeni	1	–	–
Thermoactinomyces candidus	7	10	3
T. vulgaris	3	2	–
Number of negative samples	1	1	–

No. in parentheses = No. of samples analyzed.

Table 2. Precipitins against fungi and actinomycetes in the sera.

Fungi or actinomycetes	Marijuana smokers	Cigarette smokers	Normal controls*
	(24)	(19)	(14)
Aspergillus fumigatus	9	3	2
A. niger	2	–	–
A. flavus	1	3	–
Micropolyspora faeni	7	–	–
Thermoactinomyces candidus	13	6	5
T. vulgaris	9	9	6

* No smoking during the past 5 years.
No. in parentheses = No. of patients studied.

actinomycetes per gram. No fungi were isolated from the tobacco products. None of the cigarette samples studied yielded any organism when sampled through the Andersen sampler. *Thermoactinomyces candidus* was the predominent organism isolated from cigarettes and pipe tobacco. Only one cigarette was negative for any growth.

Serology

Of the 24 marijuana smokers studied, 9 showed antibodies to *A. fumigatus* (Table 2). Other fungal antigens reacted only with a few sera. However, cigarette smokers showed only low incidence of precipitins against *A. fumigatus*. All three groups, marijuana and cigarette smokers and control, demonstrated the presence of antibodies against the thermophilic actinomycetes, *T. candidus* and *T. vulgaris*, while *M. faeni* antibody was seen only in marijuana smokers.

Of the 24 marijuana smokers 18 were asymptomatic, while the remaining complained of varying degrees of respiratory symptoms. One of the smokers with proven systemic aspergillosis developed pulmonary granulomas related to a defect in his polymorphonuclear leukocyte oxidation enzyme system. The remaining 5 marijuana smokers experienced coughing and wheezing after exposure to marijuana. None of the cigarette smokers had any respiratory complaints.

Discussion

Previous studies demonstrated the presence of mesophilic and thermophilic fungi in tobacco products (12, 13, 15). However, in the present study, we could not demonstrate any fungi from pipe tobacco or cigarettes. This may be due to the differences involved in tobacco processing. Our study isolated thermophilic actinomycetes from cigarettes, pipe tobacco and marijuana samples. In addition, the results presented indicated that fungal spores present in the marijuana samples can be inhaled during smoking. The three species of thermophilic actinomycetes and the *Aspergillus* (Table 1) are capable of causing hypersensitivity lung diseases in man.

The distribution of antibodies against fungi was higher in the group of marijuana smokers than in the other groups, clearly indicating that the fungi present in marijuana samples led to the sensitization of smokers. On the other hand, there was no difference between the three groups with regard to the presence of antibodies against various thermophilic actinomycetes. *Thermoactinomyces candidus* and *T. vulgaris* are widely distributed in nature and may be the reason for the higher incidence of antibodies in all three groups. However, because of the universal presence of these organisms in cigarettes, cigarette smokers may inhale more thermophilic actinomycetes spores than normals.

Marijuana samples contained fungi which can cause asthma and invasive lung diseases. The respiratory complaints of marijuana smokers are most likely due to an allergic reaction elicited by the inhalation of *Aspergillus* spores. Inviduals with underlying pulmonary or systemic diseases may inhale *Aspergillus* spores during smoking and may develop pulmonary or generalized aspergillosis. Both marijuana and cigarette smoking impair the function of pulmonary alveolar macrophages (4). As a result the inhaled spores may not be cleared by phagocytosis and result in colonization by the organisms and eventual invasion of the lung and other organs. Additional studies on the immunological response of smokers to the prevalent fungal and actinomycetes antigens are essential to determine the impact of smoking, if any, on the development of HP and other diseases.

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Appendix W: Verweij (2000) Fungal Contamination of Tobacco and Marijuana

RESEARCH LETTER

Fungal Contamination of Tobacco and Marijuana

To the Editor: Invasive aspergillosis remains a significant cause of morbidity and mortality in immunocompromised patients, including transplant recipients and those treated for hematological malignancy. Exposure to airborne *Aspergillus* spores is considered a major risk factor for acquiring infection. The guideline of the US Centers for Disease Control and Prevention recommends that potential sources of fungal spores should be eliminated to reduce the exposure of patients at high risk.[1] However, the risk of invasive aspergillosis associated with tobacco or marijuana smoking is unclear. We investigated whether *Aspergillus* spores are present in tobacco of commercially available cigarettes and marijuana (marijuana is sold semilegally in the Netherlands), and whether burning contaminated tobacco causes release of spores.

Methods. Tobacco from 98 cigarettes from 14 different commercial brands and 7 samples of marijuana were cultured for molds. The tobacco or marijuana was placed in 25 mL of distilled water and 1 mL of distilled water with 50 µL of Tween 20 and shaken for 5 hours. The supernatant was recentrifuged, and the residue was plated on Sabouraud dextrose agar. The plates were incubated at 29°C for 3 weeks. Sabouraud broth was added to the original tobacco and incubated at 29°C for 2 weeks.

A vacuum-driven water pipe was used to investigate the level of fungal contamination of tobacco smoke. Tobacco was contaminated with 10^6 CFU/mL *Aspergillus fumigatus* spores and then rolled into cigarettes. Each cigarette was attached to a mouthpiece, lighted, and the smoke was pulled through a 0.22-µm micropore filter for approximately 5 minutes. The smoke of 40 contaminated cigarettes was cultured, including 20 cigarettes with a filter attached and 20 without the filters.

Results. All cigarette brands tested had some degree of fungal contamination, although not every cigarette was found to have a positive culture (TABLE). Between 1 and 5 different fungal species were cultured from each cigarette, but *A fumigatus* was the most frequently isolated organism. Other opportunistic molds recovered from tobacco included *Fusarium*, *Acremonium*, *Rhizopus*, and *Scedosporium* species. Marijuana was also heavily contaminated with molds, with *Penicillium* species predominating.

Table. Culture Results of Tobacco Obtained From Cigarettes and Marijuana*

Result	Cigarettes (n = 98)	Marijuana (n = 7)
Positive culture for molds	68 (64)	7 (100)
CFU/g	200-300	10^4-10^7
Aspergillus fumigatus	36 (37)	2 (28)
Aspergillus flavus	1 (1)	1 (14)
Aspergillus terreus	3 (3)	0
Aspergillus glaucus complex	17 (17)	0
Penicillium species	3 (3)	6 (86)

*Data are presented as No. (%) unless otherwise indicated.

The smoke from the 20 cigarettes with a filter as well as that from 20 cigarettes without was negative for growth of molds.

Comment. We found tobacco and marijuana are heavily contaminated with fungal spores. The finding of molds in tobacco is not surprising because many opportunistic molds are also common plant pathogens. Because a cigarette contains between 500 and 900 mg of tobacco, as many as 270 viable fungal spores may be present in a single cigarette. The absence of fungal spores in tobacco smoke could be due to exposure to heating, nicotine, or other factors.

Previous studies have found that exposure to marijuana or cigarette tobacco is correlated with exposure to *Aspergillus*.[3,4] Although our results indicate that smoking appears to present a limited risk of inhaling fungal spores, the leaves themselves are a source of fungal spores. Given the common policy of reducing the exposure of patients at high risk to fungal spores, we believe that tobacco and marijuana should be eliminated from these patients' environments.

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Appendix X: McLaren et al. (2008) Cannabis Potency and Contamination: A Review of the Literature

Addiction

REVIEW

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Cannabis potency and contamination: a review of the literature

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ABSTRACT

Aims Increased potency and contamination of cannabis have been linked in the public domain to adverse mental health outcomes. This paper reviews the available international evidence on patterns of cannabis potency and contamination and potential associated harms, and discusses their implications for prevention and harm reduction measures. **Methods** A systematic literature search on cannabis potency and contamination was conducted. **Results** Cannabis samples tested in the United States, the Netherlands, United Kingdom and Italy have shown increases in potency over the last 10 years. Some countries have not shown significant increases in potency, while other countries have not monitored potency over time. While there are some grounds to be concerned about potential contaminants in cannabis, there has been no systematic monitoring. **Conclusion** Increased potency has been observed in some countries, but there is enormous variation between samples, meaning that cannabis users may be exposed to greater variation in a single year than over years or decades. Claims made in the public domain about a 20- or 30-fold increase in cannabis potency and about the adverse mental health effects of cannabis contamination are not supported currently by the evidence. Systematic scientific testing of cannabis is needed to monitor current and ongoing trends in cannabis potency, and to determine whether cannabis is contaminated. Additionally, more research is needed to determine whether increased potency and contamination translates to harm for users, who need to be provided with accurate and credible information to prevent and reduce harms associated with cannabis use.

Keywords Cannabis, contamination, marijuana, potency.

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INTRODUCTION

Recently, there has been a resurgence of interest in cannabis. This has been evident in political and media focus on links between cannabis and mental health, and claims that cannabis, particularly the variety known as 'skunk', is much more potent than thought previously. The occurrence of mental health problems among cannabis users and evidence of increases in treatment-seeking for cannabis-related problems [1,2] have been linked to purported increases in cannabis potency [2] and contamination of cannabis [3]. The alleged increase in potency, claimed to be 20–30-fold (e.g. [4–6]), has been used to justify calls for tougher laws [7].

The 2006 World Drug Report [2] stated that increased potency means that '... today, the characteristics of cannabis are no longer that different from those of other plant-based drugs such as cocaine and heroin' ([2], p. 2). Recently, an Australian Government Minister expressed concerns that: 'there is evidence that hydroponically grown cannabis can contain higher levels of toxins and therefore can greatly exacerbate the onset of conditions like schizophrenia ...' [3].

These issues are not new, with claims about escalating cannabis potency made as far back as 1975 [8]; yet we know little about cannabis markets that can help support or reject recent claims. Provision of quality information about harms associated with highly potent or contaminated cannabis has important public health implications. For example, evidence shows that ecstasy users would avoid using ecstasy that is known to contain substances other than 3,4-methylenedioxymethamphetamine

(MDMA) if they were provided with that information [9]. Given the popularity of cannabis it is important we have current, accurate information on the available product, to assist users in making informed decisions about their use, and contribute to evidence-based policy development and media debate about the probable harms associated with cannabis use.

This paper reviews the available international evidence on patterns of cannabis potency and contamination and potential associated harms, and discusses their implications for prevention and harm reduction measures.

METHOD

Scientific databases (e.g. Medline, EMBASE, PsychInfo, Drug, Pubmed, CINCH, Scifinder Scholar, TOXLINE and Commonwealth Agricultural Bureau Abstracts and Biological Abstracts) were searched for papers on cannabis potency and contamination using the terms 'cannabis', 'potency', 'contamination' and related search terms (free text and expanded subject headings). Additional references were obtained from bibliographies and researchers in the field.

'Grey literature' [10] was used to supplement the limited published scientific literature. Information was also gathered from cannabis and drug policy internet sites and illicit or 'folk' literature on cannabis. This enabled the inclusion of up-to-date information to enhance our picture of the current situation [11].

CANNABIS POTENCY

What determines cannabis potency?

The psychoactive drug cannabis comes from the plant belonging to the family *Cannabaceae*, the genus *Cannabis* and the species *Cannabis sativa* and its variants, although there is some debate about species differentiation [12,13]. Most commonly, the flowering tops ('buds') or leaves are dried to prepare 'marijuana', or the resin secreted from the plant is compressed to prepare 'hash'. Less commonly, 'hash oil' is prepared by extracting the psychoactive component of the plant in oil [14]. This section discusses predominantly marijuana, as most potency research assesses this form of cannabis.

The plant contains almost 500 compounds [15], including 70 cannabinoids, which provide the psychoactive effect [16,17]. The cannabinoid with the strongest psychoactive effect is delta-9-tetrahydrocannabinol (THC). While the THC content is used commonly as a measure of potency, the psychoactive effect may also depend on levels of other cannabinoids, which may interact with each other to have either additive or antagonistic effects [18–20]. For example, cannabidiol (CBD) acts as an antagonist for some of the effects of THC [18] and may have anxiolytic and antipsychotic effects [19]—thus, CBD may offset some of the psychoactive effects of THC, thereby affecting the potency of cannabis [20].

A major factor in determining potency is plant variety. For example, 'hemp', grown primarily for use as a fibre, contains very low THC levels and higher CBD levels compared with cannabis that is grown for its psychoactive effects [21], and variations in cannabinoid content occur depending on the plant's geographical origin [22]. Cross-breeding and genetic modification have produced hybrid subspecies with high levels of THC [23,24]. These hybrids are often produced in the Netherlands, and the seeds are available widely over the internet.

The THC content also varies according to the following factors: the part of the plant that is used, with the buds containing the most THC, followed by leaves, stems and seeds; the way it is prepared for administration, with hash oil containing the most THC, followed by hash and marijuana; storage, as THC degrades over time, particularly when cannabis is not stored in an airtight container; and cultivation techniques, such as growing female plants in isolation so they are seedless ('sinsemilla') [14,17,23,25,26]. Hydroponic or other methods of growing cannabis indoors under artificial conditions is thought to produce higher concentrations of THC than cannabis that is grown naturally, particularly in colder climates such as northern Europe [23,27]; however, this assertion is debated, and Australian research assessing this question has not been released by the funding body. The effect of indoor cultivation on potency is discussed in further detail below.

Trends in cannabis potency

Nine studies have analysed the potency of marijuana or hash over time in nine countries (Table 1). In the United States, the THC concentration of confiscated marijuana rose from 2.0% in 1980 to 4.5% in 1997 [28], and reached 8.5% by 2006 [29]. The potency of New Zealand marijuana seized between 1976 and 1996 did not show an increase [27]. A recent European Monitoring Centre for Drugs and Drug Addiction (EMCDDA) report concluded that the potency of cannabis used in Europe has not increased significantly over time, with the exception of the Netherlands, where most of the marijuana is now produced domestically indoors [21].

Recent Dutch data indicate that the THC content of marijuana sold in 'coffee shops'—businesses that are permitted to sell small amounts of cannabis to the public—more than doubled between 2000 and 2004, but has since dropped off [30,31]. A similar pattern was observed for Dutch hash [30,31]. The marijuana samples analysed

Table 1 Summary of studies assessing the potency of marijuana.

Study	Country (date r period)	Sample (no)	Type of cannabis (% of all samples)	Test	Main findings		Comments
					Average cannabinoid level	Range—%THC	
ElSohly et al. [28]	USA (1980–97)	Seizures (35 213)	Marijuana (91%) Sinsemilla (4%) Hashish (6%)	THC, CBD, CBC, CBN content	THC Avg. all samples: 2.0% (1980)–4.8% (1997) CBD <1 for marijuana and sinsemilla; 1.0% (1980)–2.3% (1997) for dichrowed CBC and CBN <1 for all samples	All years: Marijuana: 0–29.86% Sinsemilla: 0.1–33.12% Dichrowed: 0.01–2.40%	Unclear what proportion of marijuana is made up of buds, versus less potent stems, leaves and seeds, and whether this has changed over time Unknown whether the samples grown indoors or outdoors
ONDCP [29]	USA (1983–2006)	Seizures (59 369)	Not reported	THC content	~4.0% (1983)–8.9% (2006)	Not reported	Not stated whether the seizures included cannabis resin as well as marijuana, sinsemilla and dichrowed Unknown whether the samples were grown indoors or outdoors
Braken and Sutherland [27]	New Zealand (1976–96)	Seizures (1066)	Cannabis leaf (57.5%) Flowering tops (42.5%)	THC content	Leaf: 1.6% (1976–82): 1.0% (1994–96) Flowering tops: 3.8% (1976–82)–3.4 (1994–96)	Leaf: 1976–82: 0.3–4.2% 1983–89: 0.3–3.9% 1994–96: 0.2–3.8% Flowering tops: 1976–82: 1.3–9.7% 1907–89: 0.7–9.2% 1994–96: 1.0–8.8%	Leaf and flowering tops were segregated for analysis and may have been from same seizures Almost all samples grown outdoors
EMCDDA [21]	Austria (1997–2003)	Seizures (2268)	Herbal cannabis (100%)	THC content	~2% (1997)–~2% (2003)	Not reported	Unknown whether samples grown indoors or outdoors
	Czech Republic (1997–2003)	Not reported	Herbal cannabis (100%)	THC content	~2% (1997)–~6% (2003)	Not reported	Unknown whether samples grown indoors or outdoors
	Germany (1997–2003)	Seizures (17 403)	Herbal cannabis (100%)	THC content	~5% (1997)–~8% (2003)	Not reported	Unknown whether samples grown indoors or outdoors

	Netherlands (1999/2000–2001/12/2002)	Coffee shops (N = 3)	Imported herbal cannabis (2.8%) Sinsemilla (72%)	THC content	Imported herbal cannabis: ≈6% (1999/2000)–≈9% (2001–02) Sinsemilla: ≈8% (1999/2000)–≈13% (2001–02)	Not reported	The majority of sinsemilla grown indoors
	Portugal (1997–2003)	Seizures (n = 69)	Herbal cannabis (100%)	THC content	≈1% (1997)–≈1% (2003)	Not reported	Only large seizures (over 10 kg) analysed. Very small numbers in later years (e.g. 2003 average based on only 4 seizures)
Nestok et al. [31]	Netherlands (2000/2001–2006/2007)	Coffee shops (N = 2)	Imported herbal cannabis (26%) Nederwiet (sinsemilla) (74%)	THC content; median CBD and CBN content; CBN/THC ratio	THC Imported herbal cannabis: 5.0% (2000/2001)–9.0% (2003/2004)–6.0% (2006/2007) Nederwiet: 11.5% (2000/2001)–20.4% (2003/2004)–16.0% (2006–07) CBD and CBN <1% for all herbal cannabis samples CBN/THC ratio (×100) Imported herbal cannabis: 9.5 Nederwiet: 0.6	2006/2007: Imported herbal cannabis: 0.6–44.6% Nederwiet: 3.8–23.7%	Samples of nederwiet were chosen based on the most popular varieties sold in coffee shops at the time The most popular variety did not differ in %THC from the varieties that were supposed to be the 'strongest' Most nederwiet grown indoors
Baker et al. [32,33]	UK (1975–81)	Seizures (315)	Herbal cannabis (100%)	THC content	3.4% (1975)–4.9% (1981)	1975: 0.2–3.7.0% 1976: 0.4–3.7.0% 1978: 0.4–8.6% 1979: 1.2–11.1% 1980: 0.7–12.0% 1981: 1.8–12.0%	All seizures were imported Unknown whether samples grown indoors or outdoors
Baum et al. [34]	UK (1998–2004)	No reported	'Cannabis leaves'	THC content	7.9% (1998)–12.7% (2004)	Not reported	Not clear what constitutes 'cannabis leaves' Unknown where the samples grown indoors or outdoors

Table 1 Cont.

Study	Country (time period)	Sampling (n)	Type of cannabis (% of all samples)	Test	Main findings		Comments
					Average cannabinoid level	Range—%THC	
Decorte et al. [35]	Italy (1997–2004)	Seizures (94.7)	Loose marijuana (5%) Kilobricks (55%) Buds (26%) Home produced (13%)	THC content	2.5% (1997)–15.8% (2004)	Not reported	Most of the rise in potency occurred when samples of flowering tops replaced kilobricks ('made of stems, seeds, leaves and buds) as the type of cannabis most often seized Unknown whether samples grown indoors or outdoors

THC = delta-9-tetrahydrocannabinol; CBD = cannabidiol; CBN = cannabinol; chromo = chromatogram. 'Marijuana' refers to a mixture of leaves, stems, seeds and buds; 'sinsemilla' refers to the flowering tops/buds of the plant that have not been pollinated; 'kilobricks' refers to the fibrous type cannabis grown in the wild; 'herbal cannabis' refers to leaves and flowering tops, but not stalks, roots or seeds; 'resin' refers to cannabis that is grown domestically and is to the Netherlands is mainly of sinsemilla; 'cannabis leaves' is not defined by the study; 'loose cannabis' refers to samples without defined form; 'kilobricks' refer to samples of pressed cannabis made up of leaves, buds, stems and seeds; 'buds' refer to the flowering tops of the plant. CNDDP: Office of National Drug Control Policy; EMCDDA: European Monitoring Centre for Drugs and Drug Addiction.

were sinsemilla, and thus unlike samples from other countries, which are usually sourced from police seizures, and contain a mixture of sinsemilla and leaf, buds, stems and seeds [31].

In the United Kingdom, the average potency of marijuana varied between 3% and 5% from 1975 to 1989 [21,32,33] and then rose from 8% in 1998 to 13% in 2004 [34]. The potency of hash in the United Kingdom fluctuated during this period, with no discernable trend. A recent Italian study found that the average potency of marijuana seizures increased from 2.5% in 1997 to 15% in 2004 [35]. Most of this increase occurred between 2000 and 2004. During this time there was an increase in the proportion of the seizures that were buds. It is likely that this shift to samples comprising the more potent part of the plant accounts for the potency increase.

Methodological issues

Several methodological issues create difficulties in drawing conclusions about cannabis potency trends. The sample sizes of cannabis products analysed are often small [35] and may not be representative of the cannabis available to users [20]. It has been argued that the potency of marijuana analysed in the United States in the 1970s was underestimated because the samples were not stored properly [36]. However, these explanations cannot account for the rise in US marijuana potency over the last decade. There is wide variation in the potency of different parts of the plant, and it is not always clear which parts have been analysed [29]. The accuracy and precision of potency analyses varies from study to study [21]. Sample selection can also affect the results of analyses: in the Netherlands, coffee shop owners were asked for the most popular samples at the time [31,37], so it remains possible that increases in the potency of domestically produced Dutch marijuana actually reflect changes in consumer preference rather than a potency increase.

Indoor versus outdoor growing: impact on potency

The popularity of growing cannabis indoors has been proposed as one of the main reasons for an increase in potency. The ability to control the environment indoors means that plants can reach their full 'potential', which includes reaching the maximum possible level of THC for the variety being grown. Is there any evidence for a change in cannabis cultivation techniques over time?

Most of the cannabis consumed in Australia is produced domestically. For the last 10–15 years, the proportion of cannabis detected by law enforcement that is grown indoors versus outdoors has increased [38,39]. This move from outdoor to indoor crops has been observed in North American [40] and European [41,42] countries.

Indoor cultivation involves controlling factors such as light, humidity and temperature [43]. The popularity of this method is due probably to the increased yield of plants grown indoors and to the fact that indoor plants can be cultivated year-round and may, under ideal circumstances, produce up to six crops per year as opposed to one (from outdoor methods of cultivation). It also ensures uniform quality due to the practice of cloning from a variety of cannabis with high THC content, and cannot be detected by law enforcement via aerial surveillance [43].

The increase in market share of indoor-grown cannabis may have led to a more consistent product in terms of potency and could, in part, explain the potency increases that have been reported in some countries, such as the Netherlands, United Kingdom and United States, although this is difficult to assess given that it is often unknown whether samples analysed have been grown indoors or outdoors.

Cannabis potency and health effects

Australian and international drug treatment and hospital data suggest that demand for treatment for cannabis-related problems is rising [1]. Cannabis has the potential to have adverse physical, psychological and social outcomes [44–46]. It has been claimed that more potent cannabis increases the risk of cannabis-related harms [17,47]. However, given the antipsychotic and anti-anxiety effects of CBD, it may be that the percentage of CBD is as important in contributing to such a risk as the percentage of THC [48]. This issue requires further research, as most studies assess only THC levels [49].

An alternative possibility is that cannabis users will titrate the amount of cannabis smoked depending on potency [50]. If users did titrate in this way, it is possible that the adverse respiratory effects of smoking would be reduced with more potent cannabis, as users would be inhaling less smoke overall. Such titration behaviour has been found for those who smoke tobacco [51,52].

Some studies have found evidence of titration behaviour (e.g. longer interval between 'puffs', holding smoke in lungs for shorter period of time) when smoking more potent cannabis [53–55]. However, some of these studies found that despite these behaviours, the amount of THC administered was still higher for more potent cannabis, suggesting that effective titration did not occur [55], and other studies failed to find differences in smoking behaviour for different cannabis potencies [54,56–59].

These older studies are hampered by small sample sizes ($n = 6$ to $n = 15$) and the low potency (0.2–2.1% THC) of the cannabis used. Research with larger sample sizes and higher potency cannabis seems to suggest that certain types of cannabis users may adjust the amount they smoke, provided that they are given enough time to feel the effects of more potent cannabis. Users who are seeking the most intense high possible may be exposed to greater harms with more potent cannabis, given that they would be unlikely to adjust how much they smoke based on the potency [60].

It has been suggested that cannabis smoking behaviour is related more to learned habit rather than potency [57]. In contrast, tobacco smokers seem to be able to change the amount they smoke immediately depending on the level of nicotine in the cigarette [51,52]. Levels of nicotine may be experienced more readily by tobacco smokers than are THC levels by cannabis smokers [57].

CANNABIS CONTAMINATION

Recent Australian surveys have indicated that contamination is a concern for the general population and users of cannabis. One in four Australian adults (28%) believed that hydroponic cannabis poses a greater health risk than naturally grown cannabis due to greater potency and contamination [61]. Medicinal cannabis users avoided hydroponic cannabis because of its perceived contamination and adverse side effects [62]. Contamination of cannabis is of particular concern for medicinal cannabis users, given that the health of these users is already compromised.

However, there is a contrasting perception that cannabis is a 'natural' and therefore less harmful product than manufactured drugs such as amphetamines and heroin, and safer to smoke than licit cigarettes, which contain 'chemicals' [63].

There are three major avenues for cannabis contamination. Is there evidence to support concerns regarding contamination of cannabis?

Cultivation and storage: naturally occurring contaminants

McPartland [64] reviewed a number of studies which have found marijuana to be contaminated with fungi and bacteria. In one study, fungi was found in 13 of the 14 samples, and evidence of exposure to *Aspergillus* fungi was found in the majority of marijuana smokers (13 of 23), but only one of the 10 control participants [65]. Another study found fungal and bacterial contamination in all 24 samples, with *Aspergillus* contamination the most common [66]. Nearly half (nine of 24) of the marijuana smokers assessed had antibodies to *A. fumigatus*, and six of the patients reported respiratory complaints. A more recent study found that all seven marijuana samples were contaminated with mould, with the *Penicillium* species being the most common [67]. A Dutch study found that cannabis sold in coffee shops contained fungi and bacteria at levels unsafe for ingestion [68].

Moulds such as *A. flavus* produce mycotoxins, which can be carcinogenic [69]. *Aspergillus* can cause aspergillosis (a fatal lung disease), and studies have found an association between this disease and cannabis smoking among patients with compromised immune systems [70–73]. There is no research on whether contaminated cannabis leads to disease in otherwise healthy individuals. It has been suggested that sterilizing cannabis by heating it to 150°C for 5 minutes will kill these potentially harmful fungi spores [64].

Heavy metals present in soil may also contaminate cannabis, which has the potential to harm the user without harming the plant [22,74]. This contamination is usually restricted to specific areas with heavy metal content in soil, and thus may not represent a widespread problem [64].

Cultivation and storage: growth enhancers and pest control

Chemicals used to destroy pests are associated with risks to the individual using the pesticides as well as the consumer of the end product. Thus, there are strict government controls on pesticides that can be used commercially and domestically [75]. Because cannabis is an illegal drug, there are no equivalent guidelines or controls for cannabis cultivation, and it is not known whether certain pesticides are safe to use on a product that is smoked, even if the substance is safe for use on products that are to be ingested orally.

There is scant research on this issue. A Dutch study found traces of pesticides in cannabis, but in such small amounts that it was unlikely to cause harm to users [37]. Indoor-grown cannabis is often perceived to be more contaminated than cannabis grown naturally [61,62] because of the supposed addition of substances that maximize yield, without the observation of withholding periods or 'flushing out' the plant [62]. The extent to which this actually occurs cannot be determined from current literature; research has not been conducted to investigate this.

Retail: substances added for marketing purposes

Substances may also be used to 'bulk up' the weight of the marijuana or to make it appear more potent. Recently, there were reports of tiny glass beads added to marijuana in order to add bulk and to mimic the crystalline appearance of the resin glands, which contain large amounts of THC. This marijuana ('grit weed') appeared across the United Kingdom [76–78], prompting the Department of Health to issue a public health alert of potential harms associated with smoking the contaminated marijuana including sore mouth, mouth ulcers, chesty persistent coughs and tightness in the chest [79]. The Department estimated that approximately 5–10% of marijuana seized from January to March 2007 was contaminated with glass beads [80]. There have also been reports of marijuana containing other substances such as phencyclidine and tobacco [64], but no systematic research has addressed this.

DISCUSSION AND IMPLICATIONS

There is evidence for a doubling of potency in the United States. The Netherlands recorded a doubling from 2000 to 2004, but the potency has since dropped again. Increases have been reported in the United Kingdom and Italy, although the increase in Italy is due probably to changes in the part of the plant that was sampled. No significant increase has been reported in New Zealand or in European countries other than the United Kingdom and the Netherlands.

There is enormous variation in potency, within a given year, from sample to sample. For example, in 1979 samples analysed in the United Kingdom ranged from 0.2% THC to 17% THC [32]. Thus, cannabis users may be exposed to greater variation of cannabis potency in a single year (due to this natural variation in cannabis products) than over years or decades [21].

Given the potential for other cannabinoids to offset the effects of THC, it is important to analyse percentages of other cannabinoids, particularly cannabidiol, in addition to THC [20]. The studies that did report on the percentage of cannabidiol found very low levels compared to THC [28,31], suggesting that the anxiolytic and antipsychotic effects of cannabidiol would be unlikely to offset effects of THC.

The increase of indoor-grown cannabis is often claimed to be the main factor behind potency increase [81,82], but few studies report on the cultivation technique used. High-potency cannabis existed before the advent of indoor methods of cultivation; samples of cannabis with THC content of 17% were reported in the 1970s [32]. While growing cannabis indoors probably does not in itself cause plants to be more potent, it provides controllable conditions enabling plants to be grown to their full potential so it may indirectly contribute to potency increases seen in some countries.

There is a perception that cannabis—particularly cannabis grown indoors—is contaminated with pesticides and other substances added during cultivation. There is evidence for naturally occurring contaminants such as fungi, which have the potential to cause lung disease among immunocompromised individuals, and possibly respiratory problems in otherwise healthy individuals. Given that cannabis is a commercial crop, it is likely that pesticides and other substances are added to maximize yield and quality of the cannabis plant. Research is

needed to determine how these products are used—are pesticides 'flushed out' of the plants appropriately and, if not, what harms are associated with smoking a product with traces of pesticides still present?

Concerns about increasing cannabis potency are based largely on beliefs that more potent cannabis causes greater harm. However, it may be that cannabis users adjust how much they use depending on potency. The evidence supporting this hypothesis is mixed. Early laboratory-based studies in general do not show evidence of titration, but these studies are small and generally compare cannabis with little variation in potency. More recent studies have reported that certain types of users may adjust the amount of cannabis smoked depending on potency.

This leaves open the question of whether more potent cannabis has contributed to increased treatment seeking for cannabis-related problems over recent years. As suggested by Hall & Swift [24], it is possible that this may be due to an increase in the use of the more potent parts of the plant, rather than an increase in the potency of the plant itself. Another reason for increases in treatment seeking could be the introduction of cannabis diversion programmes, some of which involve mandatory treatment for those who have committed a cannabis-related offence. We could also be seeing the impact of the cohort of people who began using cannabis at an early age in the 1990s, who are now presenting to treatment with problems related to their early initiation and duration of cannabis use [83].

Realistic and accurate information about cannabis potency and contamination and the associated harms are important components of any public health strategy to prevent and reduce cannabis use and related problems, and can contribute to evaluations of the impact of drug strategies. It is unclear whether informing cannabis users of contamination issues would lead to changes in behaviour, although for other drugs there is some evidence that information about quality would affect users' drug-taking behaviour [9].

CONCLUSION

Overall, evidence for cannabis potency and contamination is fragmented and fraught with methodological problems. However, it is clear that claims of a 20- or 30-fold increase in cannabis potency and the adverse mental health effects of cannabis contamination are not supported by the evidence. Systematic scientific testing of cannabis available today is needed urgently to monitor current and ongoing trends in cannabis potency, and to determine whether cannabis is contaminated. Additionally, more research is needed to determine whether increased potency and contamination translates to harm for users, who need to be provided with accurate and credible information to prevent and reduce harms associated with cannabis use.

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13 Allergic Bronchopulmonary Aspergillosis

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CONTENTS

KEY POINTS

- Bronchial reactivity is present in patients with Allergic bronchopulmonary aspergillosis (ABPA).
- ABPA represents an IgE mediated hypersensitivity to fungal antigens.
- Control of the inflammatory response is central to the therapy of ABPA.
- Uncontrolled ABPA leads to progressive airway destruction and respiratory decline.
- Consider the diagnosis of ABPA in difficult to control cases of asthma.
- Antifungal therapy represents a possible steroid-sparing therapy.

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- ABPA is not an invasive fungal infection but may mimic invasive diseases.
- Consider fungi beyond aspergillus as the cause of symptoms, i.e., ABPM.
- Severe asthma with fungal sensitization (SAFS) mimics ABPA.
- Chest imaging helps determine diagnosis and prognosis of disease.

INTRODUCTION

Allergic bronchopulmonary aspergillosis (ABPA) describes a syndrome in which patients with asthma harbor the saprophytic growth of *Aspergillus* species within their airways. An intense allergic inflammation results in response to fungal antigens leading to clinical disease. Recurring clinical exacerbations can lead to bronchiectasis, pulmonary fibrosis and even death. ABPA is present in 1–2% of all asthmatics and up to 15% of patients with cystic fibrosis (CF) *(1)*. While the initial manifestations of disease may be subtle, more severe disease may be dramatic and, at times, life-threatening. ABPA may go unrecognized, since the early clinical course may be as indolent as a moderate to severe persistent asthma. As with rhinosinusitis, gastroesophageal reflux disease (GERD) and COPD, ABPA should be included whenever considering an asthma diagnosis. Assiduous therapy in ABPA can decrease the frequency of exacerbations and may slow the progressive lung damage, leading to pulmonary fibrosis and death.

ABPA DEFINED

ABPA represents a specific hypersensitivity to *Aspergillus* species within the lung manifest immunologically by elevated immunoglobulins specific to this fungus' antigens. An initial inoculum of fungal spores is thought to enter and seed the respiratory airways. Subsequently the fungus grows septated hyphe in a saprophytic manner within the susceptible airways. Susceptibility may arise from a genetic predisposition or abnormal mucociliary clearance (e.g., areas of scarring, bronchiectasis) *(2, 3)*. Since the fungi are fully contained within the airways – without invasion or penetration to the submucosa – they are not considered invasive *(4)*. However, even without invasive growth, an intense and perhaps overly exuberant, allergic exudate and TH-2 dominant inflammatory response are stimulated. The characteristic ABPA inflammation contains large numbers of eosinophils, as well as neutrophils, Curschmann's spirals, desquamated epithelial cells and mucus *(4)*. The often associated parenchymal infiltrates are similar in character to those of eosinophilic pneumonia and are thought to arise in areas distal to mucus obstructed airways. It is the unchecked acute and chronic inflammation which leads to progressive airway damage, airway hyper-responsiveness and bronchiectasis. As bronchiectasis becomes more severe, the density and number of inflammatory cells increases *(5)*. The inflammation persists and fibrotic changes can result.

The inflammatory and interleukin profile of inflammation in ABPA appears to have an asthma-like TH-2 dominant pattern *(6)* with relatively low levels of interleukin-10 (IL-10) *(7)*. *Aspergillus* as a Type II allergen it thought to produce proteases which impair tolerance – T-cell activity *(8)*. An increased sensitivity of the peripheral blood monocytes cells to IL-4 is also observed in ABPA *(9)*.

Since other non*aspergillus* species may cause similar clinical manifestations, an alternative more encompassing name, allergic bronchopulmonary mycosis (ABPM),

Table 1
Implicated Fungi of Allergic Bronchopulmonary Mycosis (APBM)

Fungi	*Reference*
Aspergillus sp	
Candida sp	*(62)*
Fusarium	*(63, 64)*
Pseudoallescheria boydii	*(65)*
Scedosporium apiospermum	*(66)*
Curvularia sp	*(67–69)*
Blastomyces dermatitidis	*(70)*

may better describe the syndrome. ABPA is a more widely recognized term than ABPM. Nonetheless, other fungi should be considered as potentially causative. Clinicians should look for other fungi in the appropriate clinical scenarios, particularly when there is no evidence for *Aspergillus* (*see* Table 1).

Beyond this allergic lung disease, *Aspergillus* and other fungi may cause other pulmonary diseases including: pulmonary aspergilloma, chronic necrotizing aspergillosis, invasive pulmonary aspergillosis and severe asthma with fungal sensitization (SAFS). The invasive fungal infections (IFI) due to *Aspergillus* should not be confused with ABPA since the clinical significance and therapies are different *(10)*. Selected cases of true infection may be difficult to differentiate from ABPA. Uncommonly cases of ABPA may progress to include features of, or progress into invasive disease *(11, 12)*. When asthmatic patients do not meet criteria for ABPM but demonstrate "fungal sensitization" to fungal antigens, treatment ought to be considered *(13, 14)*. Nonetheless, the clinical setting and radiographic findings are often adequate to distinguish these invasive infections from ABPA and SAFS.

OTHER FUNGAL LUNG DISEASES

Pulmonary aspergilloma, or more generically, pulmonary mycetoma, is an anatomically opportunistic fungal infection. A mycetoma typically forms within a cavity previously created by granulomatous lung disease (e.g., sarcoidosis, tuberculosis) or other cavitary lung diseases (e.g., pulmonary abcess). Within the remaining cavity, inflammatory cells, fungi and cellular debris combine to form a sphere or fungus ball, i.e., mycetoma. A posterior–anterior chest radiograph or CT of the chest may incidentally reveal such a mass as the first indication of disease. A suggestive and nearly pathognomonic crescent shaped radiolucency or even a mobile sphere within the cavity may be detected *(15)*. Most patients do not develop overt clinical disease, but should significant hemoptysis (>50–200 ml per episode) or growth occur, surgical therapy may be beneficial.

Chronic necrotizing aspergillosis or so-called "semi-invasive" aspergillosis describes a progressive lung infection with parenchymal destruction and in most cases locally limited. Radiographically, necrotizing aspergillosis may have similar characteristics as ABPA and when they are not clinically distinct may require bronchoscopy and biopsy

and/or search for serum markers of IFI *(10, 16–18)*. Treatment with systemic antifungal therapy has greatly reduced the need for surgical resection *(19)*.

Invasive fungal disease with *Aspergillus* is difficult to recognize early. Fortunately, this severe disease is usually only found in patients with serious immune compromise (e.g., persistent neutropenia, leukemia, or organ-transplant related immune suppression). This distinct clinical setting is most often adequate to distinguish this often disseminating disease from ABPA. Given the high mortality among at-risk individuals, any evidence of fungus should prompt a presumptive diagnosis of invasive disease and initiation of empiric therapy – more definitive diagnosis requires biopsy. The radiographic changes of invasive disease can be focal or diffuse, but typically do not have the prominent airway findings of ABPA *(15)*.

MAKING THE DIAGNOSIS

At its essence a diagnosis of active ABPA requires three elements be present: (1) bronchial reactivity (asthma), (2) noninvasive fungi and (3) an active allergic response to the fungus. While some authors also call for bronchiectasis to be present, we prefer not to require this for diagnosis.

Since the first case series descriptions of the syndrome, various diagnostic criteria have been proposed. The variations in diagnostic criteria seem to reflect clinician preferences and the serologic testing available to them. In his initial description of ABPA in 1952, Hinson identified patients who had fungus in their airways, recurrent fevers, chest radiograph changes and blood eosinophilia *(4)*. In the 1970s Safirstein (1973) and Rosenberg (1977) proposed similar criteria, which called for major (required) and minor (supportive) diagnostic information (Table 2). They added serologic testing data beyond

Table 2
Selected Antifungal Therapy Reports in ABPA

Drug (dosing)	*Description*	*Outcome (reference)*
Ketaconazole (400 mg/day)	12 months	Decreased IgE and symptoms *(71)*
Itraconazole (400 mg/day)	RDBPC, $n=29$, 16 weeks	Decreased IgE, fewer exacerbations *(42)*
Itraconazole (400 mg/day)	RDBPC, $n=55$, 16 weeks	Decrease of corticosteroid dose, improved X-rays and PFTs *(41)*
Itraconazole	Open, $n=14$, 2 years	Decrease of corticosteroid dose, total IgE *(49)*
Itraconazole (≥ 200 mg/day)	Observational, $n=14$	Decreased steroid use, decreased eosinophilia, decreased exacerbations *(49)*
Itraconazole (300 mg/day)	Retro cohort $n=33$ > 6 months	Decreased steroid use, decreased IgE, improved ((FEV??) *(32)*
Voriconazole (wt based dosing)	Retro case review $n=13$ (less than?????)	Decreased of total IgE, increased FEV1/FVC *(51)*

Table 3
Diagnostic Schema for ABPA

Safirstein et al. *(20)*
Major and minor criteria in 50 patients with ABPA
Major
Recurrent pulmonary densities in CXR
Eosinophilia in sputum and blood
Asthma
Allergy to aspergillus fumigatus (Type I or Type 3)
Minor
Recovery of Asp Fumigatus from sputum
Asp fumigatus serum precipitins
History of recurrent pneumonia
History of plugs in expectorated sputum
All patients fulfilled major criteria and 66–88% of patients fulfilled one or more minor criteria
Rosenberg et al. *(72)*
Major and minor criteria
Major
History of pulmonary infiltrates
Peripheral blood eosinophilia
Asthma
Immediate skin reactivity to *Asp.* Precipitins to *Asp.* Antigens
Central bronchiectasis
Elevated serum IgE
Minor
Recovery of *Asp.* from sputum
History of expectoration of brown plugs or flecks
Arthus reaction to *Asp.* antigen (Type 3)

peripheral blood eosinophilia (high serum IgE, immediate skin reaction to *Aspergillus* antigen, precipitating antibodies) and called for central bronchiectasis to be present. They differ subtly on the inclusion of serum precipitins to *Aspergillus* antigens and use of IgE levels (>1,000 ng/mm^3) *(20, 21)* (Table 3).

The diagnosis of ABPA requires bronchoreactvity, intense inflammation and associated fungus. Standard criteria are used to demonstrate asthma as outlined by regional societies. To establish the presence of fungus, we obtain a culture of a high quality expectorated sputum. Rarely, a bronchoscopy will be necessary to obtain culture material or to perform airway assessments. We use total serum IgE levels – in a similar manner as recommended for CF patients – as a screening test in at-risk patients *(22)*. When this general marker of allergic inflammation exceeds 1,000 ng/mm^3 we perform further specific testing.

Enzyme-linked immunosorbent assay (ELISA) or immunoblot is used to identify specific IgE and IgG to Aspergillus or other fungal species. When present this humoral sensitization to fungal antigens fosters the immunologic mechanisms responsible for the chronic airway inflammation of ABPA. These assays are useful as alternatives to or in conjunction with skin prick testing to *Aspergillus* specific antigens or serum precipitans

levels *(23, 24)*. No single testing method of specific immunity has shown ideal statistical sensitivity or specificity *(25)*.

A diagnosis of ABPA or flare of existing disease is made when there is evidence of a specific allergic response to fungus and total IgE levels are greater than 1,000 ng/ml or twice a patient's baseline. A recent study suggests that thymus activation-regulated chemokine (TARC) had a superior diagnostic accuracy to other serologic markers for the diagnosis of ABPA in CF patients *(26)*.

Bronchiectasis, found in the preponderance (>80%) of cases of ABPA, is best detected by noncontrast high-resolution computed tomography (CT) and may not be apparent on posterio-anterior chest radiographs. Bronchiectasis should be considered a late finding in ABPA. Waiting for bronchiectasis to become apparent may significantly delay the recognition of early ABPA and thwart efforts at preventing the permanent airway damage.

CLINICAL MANIFESTATIONS

Individuals with asthma or CF are at risk for ABPA. Nearly 2% of patients with asthma have ABPA. In asthma patients who require oral steroids the incidence triples to some 6% *(27)*. Initially, these patients may have few distinguishing findings other than difficult-to-control or persistent asthma. With progression of disease, patients may report intense bouts of coughing, production of sputum with grit or small bits of hard matter. These represent mucus plugs or small casts of the airways and often contain fungal elements when examined by microscopy. ABPA may be present in 2–15% of patients with CF *(1)*.

Unusual presentations and manifestations of ABPA include eosinophilic pleural effusions, hemoptysis and the development of super infections *(28)*. Hemoptysis in ABPA from bronchiectasis is often minimal and rarely massive.

RADIOLOGY

Chest radiographs of ABPA patients may be normal or show signs of bronchiectasis, mucus plugging or focal infiltrates in an alveolar filling pattern *(20, 21)* (*see* Fig. 1). Central airway bronchiectasis – third to fifth generation bronchi (thickened, dilated or distorted airways) – is characteristic of ABPA. Chest radiographic patterns include circular shadows, parallel nontapering lines (ectatic airways), dense cylindrical shadows (mucus plugs), finger-in-glove (mucus plugs) or signet ring patterns (increased bronchus:artery ratio) *(15, 29)*. The findings of bronchiectasis are most clearly identified by high resolution/thin cut CT scan. Distal to an obstructed airway, an alveolar pattern or infiltrate may develop. These diffuse or "fleeting infiltrates" of ABPA may be wedge-shaped in a pattern representing the affected segment or subsegment obstructed by mucus plugging or intense eosinophilic inflammation. While chest CT is more sensitive and specific then chest radiographs for revealing the findings that support a diagnosis of APBA it is not routinely required. Serial imaging by chest radiographs or CT is important in assessing response to therapy and monitoring for progression of disease or complications *(30)*. This is of particular importance because radiographs may reveal focal infiltrates, mucus plugs or bronchiectasis even in the asymptomatic patient *(20)*.

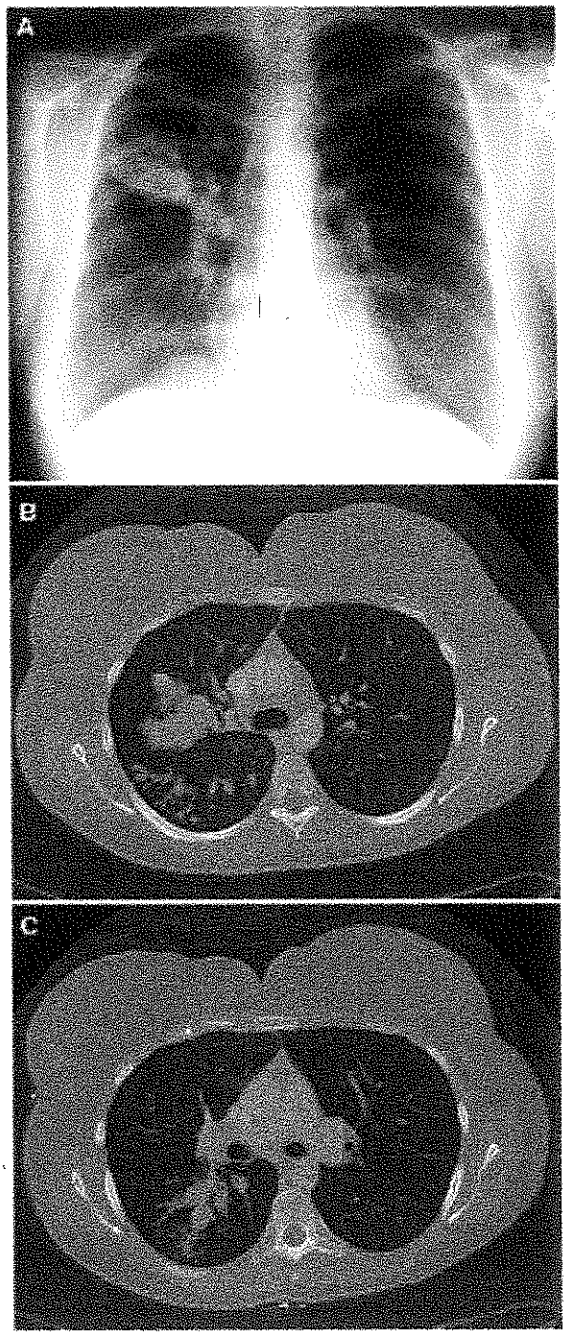

Fig. 1. PA and lateral chest radiographs of a woman with ABPA: dense airway filling and parenchymal infiltrate are present. The companion CT images show finger-in glove finding of mucus filled and grossly dilated airways, and findings of bronchiectasis (dilated, thickened and irregular bronchi).

SEROLOGY

Serologic measures such as total immunoglobulin type E (IgE), and specific IgE/IgG (ELISA, immunoblot and/or immunoprecipitins) are helpful in the diagnosis and management of ABPA. Such immunologic testing in conjunction with antigen specific skin prick testing can detect if a patient has a significant allergic response to specific fungal antigens. Total serum IgE levels are used by most clinicians both for screening and to monitor response to therapy *(13, 22, 23)*. Levels greater than >1,000 ng/ml in patients with CF or others are considered consistent with a diagnosis of ABPA or poorly controlled disease. IgE levels often correlate well with clinical status and identify response to therapy *(31)*. Serial measurements of IgE thus may guide therapeutic efforts *(32)*. However, elevated serum IgE levels are not specific to ABPA and may also be elevated in other fungal infections, asthma without ABPA, SAFS, parasitic infections, and allergic rhinosinusitis. Total and specific IgE levels can be elevated in the absence of ABPA when bronchiectasis is present – some 11% of patients with CF and 17% of those with idiopathic bronchiectasis may have elevated levels with or without the other diagnostic criteria for ABPA *(33, 34)*. Notably, measurements of fungal specific or total IgG or IgA levels do not correlate with disease activity.

CLASSIFICATIONS OF ABPA

At least two different classification schemes have been proposed for assessing the severity of ABPA. The first, a staging system, reflects the various manifestations of ABPA but may not necessarily reflect the progression of disease. This places patients into five stages: I–V. The stages reflect a combination of disease response to steroids, severity of disease, time course and radiographic changes *(35)* (*see* Table 4).

The second classification system orders patients into groups based on radiographic abnormalities. This method, classifying patients based on varying degrees of structural lung damage (no changes, central bronchiectasis, central bronchiectasis and more), appears to order patients by severity of disease (mild, moderate severe) *(36)* (*see* Table 4).

A third classification system, using solely a radiographic scoring system has also been devised which roughly correlates with clinical disease *(37)*. This is supported by other observers who found patients with a greater mucus plugging on CT were less responsive to therapy.

APBA LOOK-ALIKES

A diagnosis of SAFS may represent a continuum along the spectrum of ABPA but is usually reserved for asthma patients with demonstrated reactivity to fungal antigens (20–30% of asthmatics) but do not meet all serologic diagnostic criteria for ABPA *(13, 14)*.

Allergic fungal sinusitis may be confused with ABPA, particularly since many asthma patients have concurrent sinus disease. Allergic fungal sinusitis is similar to ABPA but with the sinus cavities as the site of saprophytic infection and may be coincident with ABPA. (In one study some 13% of patients with ABPA had findings of sinusitis on CT *(29)*). Serologic measures (specific IgE/IgG ELISA, total IgE, specific

Table 4
Classifications of ABPA

Stage *(35)*	
I	Acute – meets diagnostic criteria and is responsive to steroid therapy
II	Remission – free of significant symptoms or asthma after steroid therapy
III	Exacerbation – characterized by periods of worsened symptoms, radiographs or increased serum IgE
IV	Corticosteroid-dependent asthma – patients unable to discontinue steroid therapy
V	Fibrotic disease – significant structural changes (radiographic) and irreversible airflow obstruction with steroid dependent asthma
Severity	
Mild – ABPA-serologic	
	Aspergillus skin test (+), elevated serum IgE and eosinophilia
Moderate – ABPA Central Bronchiectasis (CB)	
	Serologic diagnosis
	Central bronchiectasis on chest CT
Severe – ABPA-CB-Other radiologic features (ORF)	
	Serologic diagnosis
	Central bronchiectasis
	Other radiologic features, including: pulmonary fibrosis, blebs, bullae, pneumothorax, pleural effusion or collapse *(36)*

immunoprecipitins) will not distinguish ABPA from allergic sinusitis. Radiographic imaging (sinus CT) and suggestive findings on history and physical examination may identify this ABPA look alike.

Individuals with CF, an autosomal recessive genetic disease, have a relatively high incidence of ABPA (2–15%) as compared to the general population *(1, 3, 38)*. Whether this reflects a genetic predisposition or an increased risk due to the associated bronchiectasis remains uncertain. Rarely, the onset of ABPA may actually represent the first sign of CF. Based on retrospective analyses of patients with ABPA, some investigators suggest that CF genes are more common in individuals with ABPA *(39)*. They suggest that individuals who are heterozygotic for a gene which causes CF are predisposed to develop ABPA. However, there are no prospective population studies to confirm this interpretation.

Other eosinophilic pneumonias should be kept in mind when considering ABPA. These loosely describe a collection of pulmonary pathologies associated with elevated serum eosinophil levels such as the pulmonary infiltrates with eosinophilia (PIE) syndromes. Of these diseases, Churg–Strauss and infectious eosinophilic pneumonias bear some resemblances to ABPA. Both may be associated with wheezing, eosinophilic pulmonary infiltrates and blood eosinophilia.

The allergic granulomatous, angiitis and periarteritis nodosa of Churg and Strauss often has systemic findings due to vasculitis. The eosinophilia is much greater than typically seen in ABPA. Definitive diagnosis relies on pathologic examination of a biopsy *(40)*. Infectious eosinophilic pneumonias, which may result from infection with an endemic parasite (e.g., Paragonamiasis, Strongyloides) or fungi (i.e., *coccidioides immitis*),

characteristically present with very high levels of eosinophilia, exposure to the infecting agent and prominent systemic infectious manifestations *(40)*.

THERAPY

The related goals of therapy, preservation of respiratory function and control of symptoms guide therapeutic decisions. Total serum (i.e., not *Aspergillus*-specific) immunoglobulin, type E (IgE), levels provide measures of allergic disease and response to therapy *(13, 22, 23, 32)*. Additionally, pulmonary function testing, patient symptoms, other markers of inflammation and radiographs are used singly and in combination to adjust therapy.

The unchecked inflammation and the implication that this inflammation is ultimately destructive to the airways supports the primary therapy with corticosteroids aimed at control of inflammation. There is also an emerging body of literature illustrating the importance of antifungal therapy for ABPA *(14, 16, 32, 34, 41–44)*. Since asymptomatic patients may have progressive disease *(20)*, we also follow total IgE levels, chest radiographs and respiratory function to monitor disease activity. In stable patients, quarterly or semi-annual assessments with improvement prompt a decrease in steroid dosing. Patients with worsened findings trigger increased or resumption of steroid therapy and consideration of antifungal medication. In nonresponsive or recalcitrant cases, we pursue a reevaluation for IFI (using imaging, biopsy, serum or BAL galactomannin *(16)*, and/or serum 1→3-Beta-D-Glucan *(17, 18)*), super infection or a secondary diagnosis.

Despite control of pulmonary function, symptoms and radiographic abnormalities, some patients will have persistently elevated IgE levels. Thus, a 50% decrease in total IgE has been proposed as an alternate measure of significant response to therapy.

CORTICOSTEROIDS

Oral corticosteroids are the mainstay of therapy for ABPA. The use of corticosteroids in ABPA has moderate case-based data and empiric clinical evidence as support. In a 1973 review of 50 patients with ABPA, Safirstein found steroid therapy (average daily dose: 10.5 mg) decreased the frequency of exacerbations *(20)*. Common practice initiates therapy with a daily dose of 0.5 mg/kg. In acute cases, steroids are administered for 2–8 weeks followed by a gradual reduction. The reduction of steroids is guided by symptoms, serology, pulmonary function testing and radiographs. The sequential tapering down of steroids may be to lower daily doses or more commonly to every other day dosing.

In an effort to decrease the adverse effects of systemic steroids, inhaled steroids are often proposed as possible adjuncts to disease control. While many clinicians may prescribe inhaled steroids, they are routinely employed primarily as therapy for the coincident asthma. A few case series suggest some efficacy in control of symptoms using inhaled corticosteroids *(45)*. More formal prospective trials have only shown better asthma control without significant benefit in other ABPA disease manifestations *(46)*.

Allergen avoidance is a long-standing therapeutic recommendation for allergic diseases. Unfortunately this is not easily accomplished in the case of ABPA and has yet to prove clinical benefit. *Aspergillus* is a nearly ubiquitous organism, as such, environmental control is unlikely. Additionally, the patient themselves harbors the antigen source

within their own airways. Since the offending organism/allergen has taken up residence in the patient, antifungal medications have been used in an attempt to decrease the allergen load and saprophytic organisms.

ANTIFUNGAL MEDICATIONS

For several decades antifungal medications have been considered and used in patients with ABPA in attempts to decrease the antigen burden and need for steroid therapy. Initial trials of inhaled antifungals such as natamycin and more recent efforts failed to show clinical efficacy *(47, 48)*. Inhaled, instilled and systemic delivery of amphotericin has been used without formal assessments. However, the azoles, a newer group of antifungal medications, may have some utility in ABPA and have been the subject of a number of studies in ABPA (*see* Table 2).

Ketaconazole, the first studied, showed some clinical improvements but the associated risk of liver toxicity and adrenal suppression has prevented it from gaining much use in ABPA. Itraconazole with moderately good oral preparations, better safety profile and excellent activity against aspergillus species has shown an ability to decrease the average steroid dose (14 mg per day) needed for ABPA *(41, 49)*. Most reports are of case–controlled studies, but recent blinded, prospective trials have shown modest benefits with the use of itraconazole *(42)*. The mechanism of these ascribed benefits may be through inhibition of fungal growth, interference with steroid metabolism, anti-inflammatory effects or some other yet to be recognized process. Voriconazole, another azole with a good oral preparation and excellent efficacy against aspergillus, may also prove useful but lacks rigorous clinical testing *(43, 50, 51)*. One set of two cases reports clinical response in patients treated with posaconazole after azole resistant isolates were identified *(73)*.

For patients who respond rapidly and remain stable after moderate courses of steroids, the added expense, monitoring and additional risks of oral azole therapy may not be justifiable. On the other hand, for patients in whom the adverse effects of corticosteroids are severe and have disease severe enough to need prolonged daily therapy, the corticosteroid sparing effects of adjunctive azole therapy appear worthwhile.

OMALIZUMAB

Omalizumab, a humanized monoclonal antibody of IgE, inhibits IgE binding to receptors on effector cells. Periodic injections of omalizumab improve clinical control in patients with allergic asthma (and moderately elevated serum IgE levels) *(52)*. Off-label use in patients with ABPA has shown clinical improvement and decreases corticosteroid requirements for disease control in small studies *(53–57)*. There is currently a randomized, double-blind placebo controlled study of the use of Omalizumab in CF patients with ABPA. (http://www.clinicaltrials.gov/)

OTHER THERAPEUTIC CONSIDERATIONS

Other considerations in the management of ABPA relate primarily to the complications of bronchiectasis and medical therapies.

The associated bronchiectatic airways of ABPA thwart the normal mucociliary clearance of the lung. Devices and maneuvers to improve airway clearance have become a standard in the management of bronchiectasis *(58)*. As an adjunct to chemotherapy, sputum clearance may be of benefit to patients with ABPA particularly if they have already developed significant bronchiectasis.

The progression from a noninvasive hypersensitive state to a disseminated infection, albeit rare in the immunocompetent host, remains of concern in patients with ABPA. Consideration of a disseminated infection presenting with characteristics of ABPA should be considered even in an immune competent patient. Chronic corticosteroid therapy should be considered as a risk for development of subsequent invasive disease *(59)*. Since corticosteroids are the primary therapy of ABPA, any progression to invasive or disseminated disease must always be kept in mind. Radiographic imaging can usually distinguish an invasive pattern from aspergilloma type or allergic related disease patterns in addition to the BAL and serum markers listed above *(16–18)*.

The damaged bronchiectatic airways of the ABPA patient bring increased risk for other airway infections as well. Bacterial super infection should be considered in the ABPA patient who worsens or has increased symptoms despite appropriate therapy. Probable pathogens are numerous, but are likely to be those most commonly implicated in nonspecific bronchiectasis: *H. influenza*, *Staphylococcus aureus*, *Pseudomonas aeruginosa* and mycobacterial species *(1, 60, 61)*. Intermittent surveillance with sputum cultures may identify the probable pathogens and the chronically infected, at risk patient.

An increased risk of hemoptysis accompanies most diseases with chronic lung infections or bronchiectasis. ABPA is no exception. Small amounts of bloody sputum may be present with cases of more severe ABPA disease. Should severe hemopytsis develop, an aspergilloma or bronchial artery source should be considered. Chest imaging can usually identify an aspergilloma which may require definitive treatment of bleeding with surgical resection *(12)*.

SUMMARY

Allergic bronchopulmonary aspergillosis is a potentially severe and destructive lung disease which may accompany asthma and CF. A diagnosis of ABPA should be suspected in patients with asthma who have difficult to control symptoms, require systemic steroids for control, have sputum production or have abnormal chest radiographs. The diagnosis of ABPA is made when a specific and intense allergic response to *Aspergillus* species is present in a patient with asthma without evidence of invasive fungal disease. Serum IgE levels (>1,000 ng/mm^3) are sensitive for diagnosis and assist in management. Therapy aims to preserve lung function, maintain quality of life, and reduce exacerbation rate through the control of the inflammatory response with prednisone and decreasing antigen exposure. Antifungal therapy with azoles may decrease the need for systemic steroid therapy and improve lung function. Omalizumab has demonstrated some potential to be a steroid-sparing therapy.

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Appendix Z: Pacific EHS : Review of Photos From Marijuana Grow Operations in Surrey, BC—Assessment for Fungal Growth and Chemicals

Pacific EHS
A Total Safety Company

September 16, 2014 Pacific Reference: 15064-SM L01

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Reference: **Review of Photos from Marijuana Grow Operations in Surrey, BC – Assessment for Fungal Growth and Chemicals**

Introduction

In response to your request, Pacific EHS has reviewed photos from nearly 1,800 properties in Surrey, BC, that were known to have sustained marijuana grow operations (MGOs). The MGOs assessed were mainly unlicensed; however several hundred were also reported to be licensed. It was not always clear to Pacific whether or not the properties sustained licensed or un-licensed MGOs.

Pacific's specific role in the project was to determine the presence and extent of fungal (mould) growth and to determine if chemical containers related to the MGO were present, such as pesticides or fertilizers.

Pacific's Experience

Pacific EHS is an experienced multi-disciplinary Environmental Health and Safety (EH&S) firm that has been providing Environmental Site Investigations and Remediation Services, Hazardous Materials Assessment, Industrial Hygiene and Safety Services to our clients across British Columbia (BC) for over 20 years. Growing from a single-owner proprietorship into an industry leading specialty environmental consulting firm and analytical laboratory, Pacific EHS employs over 40 talented individuals in four offices across British Columbia and has become one of the largest and most trusted companies of its kind in BC, offering multi-disciplinary services to a broad spectrum of public and private sector clients.

Pacific has been involved in the assessment of hundreds of MGOs throughout the province of British Columbia. Shannon McIntosh, who led this project, has been conducting assessments of former MGOs since her start with Pacific in 2007. Mrs. McIntosh is a Canadian Registered Safety Professional (CRSP) and a Certified Industrial Hygienist (CIH). Her experience with MGOs includes assessment and sampling plan development, site assessments and sampling, data interpretation, report writing, report review, and project management. Please see Appendix C for a copy of Mrs. McIntosh's CV.

Although many different hazards may be present on properties used to cultivate marijuana, Pacific's expertise as it relates to this project is in Industrial Hygiene, particularly the presence of fungal growth and chemical contamination.

Indoor Marijuana Grow Operations

Background and Hazards

An indoor MGO is a building or other structure which has been physically altered to produce marijuana. MGOs can be located in any type of building including rural, urban or industrial buildings. The growing of marijuana typically uses a significant amount of electricity, water and chemicals (fertilizers and pesticides/fungicides), all of which can alter the structure causing significant damage.

Alterations to a building for a MGO may include tapping into the hydro lines before the electrical meter to steal electricity, installing high-wattage lights, removing structural walls, disturbing plumbing lines, building irrigation systems and re-directing exhaust ducts to increase the amount of carbon dioxide in the growing area. Pesticides and insecticides are used to address fungal diseases and insect infestations that the crop may develop. Large amounts of fertilizers are used to increase crop yields and promote flowering at the correct time. Health and safety concerns may include, but are not limited to:

- **Fungi:** The high humidity and temperatures necessary for a grow room lead to the formation of damaging mold. The presence of mould on building materials may poses a serious health risks to residents or workers entering the property (see additional information below).
- **Pesticides:** In BC, the most commonly used pesticides in a MGO are organochlorines, organphosphates and a number of pyrethroid and synthetic pyrethroid pesticides.
- **Fertilizers:** The fertilizers used in MGOs are usually similar to normal household fertilizers used for other indoor plants; however, the volumes used are much greater.
- **Carbon Monoxide and Carbon Dioxide:** Incomplete combusting of fossil fuels or changes in the exhaust ducts in a MGO can result in elevated levels of carbon monoxide and carbon dioxide.

Remediation

Once a MGO is discovered, it is important that the building be properly remediated to ensure that anyone entering that property in the future, such as a new homeowner is not exposed to anything hazardous that was associated with the illegal operation. After a MGO has been identified, the gas, water and electricity supply are often disconnected and the occupancy permit on the property may ~~be~~ have been removed. Home owners or property managers must ensure that the property is properly remediated to eliminate any contamination associated with former MGO. Please see below for additional information on possible contaminants of concern.

In B.C., several municipalities have by-laws which hold the property owner in part culpable for MGOs discovered in their properties. Generally, the bylaws require proper remediation of the property including an environmental assessment conducted by a person or company registered with the Canadian Registration Board of Occupational Hygiene (CRBOH) or the American Board of Industrial Hygiene (ABIH), to ensure all residual mould, pesticides, fertilizers or other hazards have been removed. Most bylaws also require other inspections, such as structural, electrical and plumbing inspections. In some cases, anything that was not done to code or without a permit must also be rectified.

Fertilizers

The fertilizers used in MGOs are usually similar to normal household fertilizers used for other indoor plants; however, the volumes used are much greater. The main nutrients in these fertilizers are nitrogen, phosphorous and potassium. Most fertilizers also contain some trace minerals such as calcium, copper, zinc and iron. Fertilizers can be organic or synthetic, come in powder or liquid forms and release in immediate or slow modes.

Fertilizers may cause surface contamination and improve fungal growth by providing nutrients in areas where they are applied and spilled. The use of manure may also allow other pathogens such as bacteria, to grow and may get into plumbing systems. The use of fertilizers is not likely to result in significant health hazards to the growers or to emergency personnel; however, exposure to elevated levels of fertilizers can still present a health risk, such as disturbances of the kidneys, lungs and liver and can potentially cause cancer due to the presence of nitrite and heavy metals in fertilizers.

Pesticides

The use of pesticides in MGOs is highly variable based on geographic location, the presence of certain pests and the availability of pesticides. In BC, the most commonly used pesticides in MGOs are organochlorines, organophosphates and a number of pyrethroid and synthetic pyrethroid pesticides. These pesticides may cause illness in exposed individuals if the exposure is high enough or if the pesticides are misused. Pesticide exposure can cause a variety of adverse health effects ranging from simple irritation of the skin and eyes to more severe effects including the nervous system impairment, reproductive problems, and cancer. Pesticide contamination can be hard to mitigate and can persist in the environment for an extended period of time.

Most information and studies available on pesticide fate applies to soil and water in outdoor environments; however, this will differ from indoor applications such as residential buildings. Pesticide behavior on building material surface such as concrete has seldom been studied. There are many factors that affect pesticide fate, including the application method, amount, concentration, timing, frequency and placement. With time, pesticides either break down, are redistributed within the application site, and/or move off site. Pesticide breakdown, or degradation, most often occurs from reactions with oxygen or water, though sunlight is also a significant contributor. Indoors, pesticides tend to break down slower than outdoors, primarily due to the lack of direct sunlight indoors. The ultraviolet light necessary for pesticide breakdown is filtered out by glass (windows) and vapour loss may also be less due to the lack of heat from the sun. Microbes are also a contributing factor to pesticide degradation. The lack of rain and wind indoors also slows the rate of pesticide breakdown. Degradation processes can take anywhere from hours to days to years, depending on the environmental conditions and chemical characteristics specific to the pesticide.

In terms of breakdown, half-life is used to measure the persistence of a pesticide. The values given are for soil, and would likely vary greatly for indoor surfaces. Pesticides are typically categorized, based on their half-life, as non-persistent (half-life of less than 30 days), moderately persistent (half-life of 30-100 days), or persistent (half-life greater than 100 days). Research suggests that depending on environmental conditions, pesticide degradation half-lives may extend as long as years.

Another value used for pesticides is the sorption coefficient, which refers to the tendency of a pesticide to bind to soil. Sorption can slow movement and increase persistence due to protection from degradation. The higher the sorption coefficient, the greater the sorption potential.

Pesticide movement rating is used to determine the potential for the pesticide to move towards water.

Finally, water solubility is used to describe how readily a pesticide will dissolve in water. The higher the value, the more soluble the compound. The higher the solubility, the easier it is to remove the pesticide from a surface.

To put context to the above, Pacific conducted research in 2006 related to commonly found pesticide residues in MGOs. Of those most commonly detected, almost all have low, very low or extremely low movement ratings, half-lives in soil ranging from 1-45 days, low water solubility (most range from 0.002 to 0.8) and high sorption coefficients (most above 1,000). As an example, Permethrin, a synthetic pyrethroid commonly found in MGOs, has an extremely low movement rating, a moderately persistent (30 day) half-life in soil, a low solubility (0.006) and a comparatively high sorption coefficient (100,000).

In summation, pesticide residues indoors, protected from moisture, solar UV radiation, excessive heat, wind and other elements may last for years, though there does not appear to be sufficient research as to half-lives of pesticides indoors such as for MGOs.

Carbon Dioxide and Carbon Monoxide

The growth of marijuana can be enhanced by the presence of high levels of carbon dioxide. Carbon dioxide is usually obtained by compressed gas cylinders or fossil fuel combustion in a MGO. In some MGO sites, the growers change the exhaust for furnaces and other combustion appliances to go into the grow areas so that higher carbon dioxide can be obtained. Incomplete combusting of fossil fuels or changes in the exhaust ducts can also result in elevated levels of carbon monoxide.

The ideal levels of carbon dioxide in a MGO are between 700 ppm to 1,500 ppm. Although the levels are higher than the levels commonly found outdoors (360 ppm ~ 1,000 ppm), it should not pose a significant health hazard.

Carbon monoxide is a colorless, odourless and tasteless gas which is highly toxic to humans in high quantities. Concentrations as high as 667 ppm can result in seizure, coma, and death. Exposure to lower concentrations of carbon monoxide may cause symptoms such as headache, nausea, vomiting, dizziness, fatigue and a feeling of weakness.

Fungi

Fungi are plant-like but lack chlorophyll. Each fungal "colony" is a mass of interwoven mycelium, made up of millions of tiny branching filaments, known as hyphae. The group includes many familiar types such as the mushrooms, toadstools, puffballs, bracket fungi, morels, truffles and yeasts. Those most commonly found growing in indoor environments are often called moulds (i.e.: *Cladosporium*, *Penicillium* and *Aspergillus*).

Fungi grow very quickly almost anywhere, including inside buildings. One of the reasons fungi are so successful is their ability to produce and disperse huge numbers of microscopic spores, which can be transported vast distances. By their sheer numbers, fungi can quickly take advantage of any new food supplies that become available, as all they need to colonize a material is water and a source of carbon, which is present in many building materials.

The air we breathe can contain tens of thousands of spores per cubic meter, while soil holds vast numbers. Many of the spores produced by fungi remain viable for years, therefore, there will always be fungal spores present in the air that we breathe, both outdoors and indoors and it is almost impossible to completely exclude fungi from any environment (including the cheese we seal in plastic and put in the refrigerator).

There are several ways in which fungi can affect the health of building occupants. The primary route of exposure to fungi is the inhalation of the fungal spores, hyphal fragments and portions of other fungal structures. This exposure may result in allergic reactions, increased asthma, upper respiratory tract irritation and even fungal infections in some people. The exact mechanism that results in the health effects is still being researched, but there are likely many contributing factors. These include the mycotoxins produced by the fungi, antigens on the surface of the fungi as well as the presence of B-glucans in the cell walls.

The health effects experienced by people vary significantly. Some people are unaffected by high levels yet others are affected by low levels of fungal spores. As the exposure duration and concentration of fungal spores increases, so do symptoms. The most common symptom is allergies, particularly allergic rhinitis. Allergy-related problems become particularly apparent, when the concentration of airborne spores is relatively high and the majority consists of only 1 or 2 species.

The following are some of the fungal genera which grow indoors and are implicated in causing respiratory problems:

- *Alternaria*
- *Aspergillus*
- *Chaetomium*
- *Cladosporium*
- *Epicoccum*
- *Fusarium*
- *Mucor*
- *Penicillium*
- *Phoma*
- *Pithomyces*
- *Stachybotrys*
- *Trichoderma*

Some fungi can be quite pathogenic (cause systemic illness in people), including *Histoplasma*, *Cryptococcus*, *Sporothrix*, *Blastomyces* and *Candida*. At least three species of *Aspergillus* (*A. fumigatus, A. niger* and *A. flavus*) can be included in this group, however, most others (there are between 100 and 200 species of *Aspergillus*) are relatively benign. People with compromised immune systems are at the greatest risk for fungal infections.

Repeated inhalation and sensitization to a wide variety of organic material, including fungi, can cause hypersensitivity pneumonitis (HP), a lung disease, in a small percentage of exposed people. Additional health effects caused by fungi may include aggravation of pre-existing asthma, sinusitis, histoplasmosis and rhinitis.

Other substances produced by fungi, besides spores, can also cause health problems. These include mycotoxins (substances produced by fungi which may interfere with the growth of other fungi or bacteria) and Volatile Organic Compounds (VOC's – responsible for the musty odor characteristic of fungi). Note however, that health effects associated with mycotoxins are typically associated with only very high exposures that are likely only to occur during the consumption of fungal contaminated food or during high risk activities, such as fungal remediation.

Although there are no standards in Canada for acceptable levels of fungal spores in air, there are several guidelines and standards that exist worldwide. These standards have been summarized below. Note that only those standards that apply to non-viable spore trap sampling reported in fungal structures per cubic meter have been summarized.

Organization	Published Standard (spores/m^3)	Description or additional information
Texas Department of Health Guidelines	≤ 22	This value refers only to *Stachybotrys chartarum* spores used to indicate in an area has been adequately remediated
	≤ 2,000	Total spores – The area has been adequate remediated, provided 1/3 of the spores are *Cladosporium* spores, 1/3 are *Aspergillus/Penicillium*-like spores and 1/3 are others spores
American Academy of Allergy, Asthma and Immunology/National Allergy Bureau (Outdoor Environments)	> 1 – 6,499	Only individuals extremely sensitive will experience symptoms.
	6,500 – 12,999	Many individuals with sensitivities will experience symptoms
	13,000 – 49,999	Most individuals with any sensitivity will experience symptoms
	> 50,000	Almost all individuals with any sensitivity at all will experience symptoms. Extremely sensitive people could have severe symptoms.
mcg Occupational Health & Safety Consulting	< 5,000	Normal Background for Residential Buildings[2]
	< 2,500	Normal Background - filtered HVAC systems[2]
	>10,000	Probable Contamination

1. Symptoms – allergy suffers who are allergic to pollens or molds may experience symptoms of hay fever or asthma
2. types and relative proportions of fungal spores similar to outdoors

Methodology

Pacific was provided electronic photos of nearly 1,800 properties in Surrey, BC, that were known to have sustained MGOs. The photos were grouped by property address. Where there were duplicate files for one property, all files and photos for that one address were analyzed and used for the assessment. Note that Pacific did not conduct any site visits or sampling as part of this project.

Pacific, with help from the University of the Fraser Valley, developed an excel spreadsheet with the parameters to be assessed. The addresses were entered in sequential order by street address. The parameters to be assessed included the following:

- Whether or not fungal growth was visible;
- The extent of fungal growth, if present;
- Whether or not chemical containers (presumably related to the MGO) were present;
- If present, what the chemical containers contained
- If present, whether or not the chemical containers had labels;
- A hazard rating based on the presence of fungal growth and chemicals;
- Photos representing what was found; and,
- A comments summary for the property with additional information on how the assessment was determined.

Following the development of the spreadsheet, Pacific assessed the photos address by address over a period of approximately two months (July and August, 2014).

Assessment of Photos

Fungi

The first component (column one of data table) analyzed related to fungi (mould) and whether or not it was present. The three categories of determination were was mould present – yes (Y), no (N), or suspected (S). Note that determination on the presence of mould is preferably conducted with a visual inspection and confirmatory samples; however we were limited to photos. Therefore when we say that yes, mould was present, this is in our professional opinion only, based on education and experience. If it was determined that mould was suspected, this was typically because either the photos were not clear enough, close enough, or the staining resembled fungal growth but may instead be only water staining, oxidation of materials, or other unknown non-fungal staining.

In terms of the extent of mould growth, we determined the fungal growth to either be minor (1), major (2) or not present (3) (column two of data table). Minor fungal growth was assessed to be less than one square meter of visible growth or staining in total for the photos available for a particular address. When assessed as minor, the fungal growth was typically only visible in one small area of the building. Major fungal growth was assessed to be greater than one square meter of visible growth or staining in total for the photos available for a particular address. Typically if fungal growth was visible in more than one area of the building, the site was assessed as having major fungal growth. For sites assessed as having suspected fungal growth, we still made the assessment on extent (minor vs. major) of suspected growth. Please refer to Photos 1-3 in Appendix A for examples of fungal growth and how they were assessed.

Note that the extent of fungal growth in terms of area covered had an element of subjectivity, as area determination was an estimate only based on photos as opposed to on-site measurements.

MGO-Related Chemicals

The first step in assessing the potential hazards of chemicals was to determine if chemical containers were present that we presumed were used in the MGO (column three of data table). The three categories of determination were; were chemical containers present – yes (Y), no (N), or suspected (S). When sites were assessed as having suspected chemical containers present, this determination was typically made for sites that had large plastic drums/containers that in our experience are used to mix/dilute chemicals for MGOs, or had other containers that resembled those used in MGOs but photos were not enough to confirm. Note that there are some instances where fertilizers/pesticides may be present in non-MGO properties (e.g. residential gardeners); however they are typically found in much smaller quantities that found in MGOs.

Following the determination of chemical containers were present, the next determination was on the contents of the containers, or container identifier (column four of data table). If no chemical containers were identified in column three, column four was labelled as not applicable (n/a). If chemical containers were present of suspected, the containers were identified as; Pesticide/Fungicide/Herbicide (P), fertilizer (F), other (O), or unknown (U). This determination was based on the presence of labels. If labels were not visible or clearly identifiable, the rating of "unknown" was used. Chemicals included in the "other" category included such products such as pH balancers.

Once a determination was made on the suspected contents of the containers (container identifier), it was next determined if labels were present on the containers (column five of data table). If no chemical containers were identified in column three, column five was labelled as not applicable

(n/a). If chemical containers were present or suspected, the presence of labels were assessed as: yes, (Y), no (N), or unknown (U). A determination of unknown would be made if the container(s) in the photo were positioned so that labels may be hidden or covered by other contents/materials/etc.. Note that for some addresses, there were multiple containers present, and therefore the same site may have been assessed as Y, N, and/or U.

Please see Photos 4-6 in Appendix A for examples of assessments related to chemical containers.

Hazard Rating

Following the determination on the presence of mould and chemicals, the site was given a hazard rating (column six of data table). The rating scale was 1-4, with 4 being the greatest hazard. Again, there was an element of subjectivity with this category. Some addresses had more photos than others and therefore provided more information, and some sites were in different stages (e.g. of growing or remediation) that others. Typically, an address would be assessed as having a hazard rating of 1 if there was no mould or chemical containers present, there were sufficient photos and no major residual signs of the site being used as a MGO e.g. all contents gone, finishing materials appeared clean. A site would generally be assessed with a rating of 2 if minor mould and/or chemical containers were present (or major mould was present in non-occupied areas such as attic spaces or crawl spaces), if there was no mould or chemical containers present but the site was dirty, had contents still present related to the MGO, was still an active grow site (e.g. plants present), of if there were a limited amount of photos. Sites were typically assessed with a rating of 3 if there was major mould present, if a fire had been sustained causing notable damage, and/or if chemical residue was suspected on building/finishing materials (e.g. in bathrooms in tubs/showers, sinks, toilets). A site would be rated as a 4 if it was deemed too unsafe to enter e.g. from a fire, and/or other catastrophic damages.

Photos and Comments

Column seven of the data table included photos to support the assessment for the address/property. Not all addresses were linked with a photo. Column eight of the data table included comments on what was found at the address/property or additional information not otherwise included for in the other columns.

Note that for some sites, either there were no photos from inside the building(s) on the property, or it was determined that there were not enough photos to make an assessment. As an example, there were many addresses where the only photos provided were of the electricity meter on the outside of the house.

Conclusions

Based on photos provided to Pacific, we assessed nearly 1,800 addresses/properties in the city of Surrey, mainly for the presence of fungal growth and chemical containers. We have extensive experience in the assessment of former MGOs throughout the province of British Columbia. For each of the addresses/properties, we assessed whether or not fungal growth was visible, and if so, the extent, whether or not chemical containers were visible, and if so, what the chemicals may be and if labels were present. Based on factors such as the presence/absence/extent of mould and chemicals, we provided a subjective hazard rating for each address/property. We provided example photos to confirm or provide examples for our assessments, as well as a written comments section for each address/property summarizing or providing additional information.

Limitations

This report has been prepared in accordance with established Industrial Hygiene and Mycological practices. It is intended for the exclusive use of the University of the Fraser Valley. The use of this document for any other purposes is at the sole risk of the user.

Statement of Qualifications

Pacific EHS has been providing consulting services in the environmental and industrial hygiene fields, since 1990. Our personnel include the following:

- Industrial Hygienists (CIH, ROH)
- Registered Professional Biologist (RPBio)
- Canadian Registered Safety Professional (CRSP)
- Occupational Health and Safety Technicians.

Our company also carries Comprehensive General Liability and Environmental Errors & Omissions Liability Insurance.

Yours truly,

Pacific EHS

SIGNATURE ON FILE COPY

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