linda – could you take a look @ this.

– Chris

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6 October 1993

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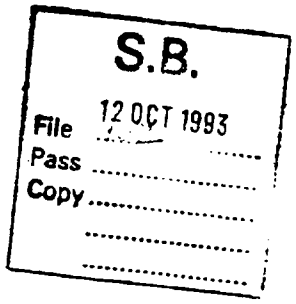

Dear Sharon and Linda

Herewith a copy of our Preliminary Submission to the New Zealand Public Health Commission in reference to their policy paper "Tobacco Products."

Any comments you may have would be very much appreciated.

Yours sincerely

TOBACCO INSTITUTE OF NEW ZEALAND LIMITED

Michael J Thompson
Executive Director

THE TOBACCO INSTITUTE
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PUBLIC HEALTH COMMISSION
TOBACCO PRODUCTS

A PRELIMINARY SUBMISSION

PREPARED BY:
THE TOBACCO INSTITUTE OF
NEW ZEALAND LIMITED

SEPTEMBER 1993

CONTENTS

1.0 INTRODUCTION

The substantially revised version of the Public Health Commission (PHC) document (second draft) entitled 'Tobacco Products' was received by the Institute on 23 August 1993 with a request for a response that was required by 20 September 1993. The necessity to examine properly a semi-technical document of 35 pages and containing 63 references renders this submission a preliminary response.

There is reference material that obviously bears on the issues raised that is not acknowledged in the PHC document that requires to be identified and introduced.

"It is in everyone's interest that the advice is formulated by those who have had the opportunity to review and assimilate the vast amount of relevant information dispassionately ..." (Dr Scott, Chairman of NZ Medical Association, press release of 16 September, commenting on appropriate procedures).

The purpose of this initial submission is to show, by reference to some specific and key examples, that the PHC paper is substantially flawed, and is therefore, in its present form not yet a suitable document for public policy advice to Ministers.

We would not expect the PHC to offer policy advice until it was satisfied as to the validity of that advice. The Institute looks forward to participating in the debate which must surely result from this submission.

2.0 THE INSTITUTE

The Tobacco Institute is the trade association of the tobacco industry in New Zealand. As such it represents the non-competitive interests of Rothmans of Pall Mall (New Zealand) Ltd, W D & H O Wills (New Zealand) Ltd and the New Zealand Tobacco Growers Co-operative Society Limited.

The industry's policy is that the decision as to whether to smoke or not is an adult decision. The industry has responsibly researched young people's smoking behaviour and attitudes to smoking and is on record as discouraging young people from taking up smoking. The industry has supported Government initiatives in this direction.

The public fund benefits from the tobacco industry by some $750 million per annum from high levels of tobacco excise taxation, in addition to Goods and Services tax, personal and company tax associated with tobacco-related economic activity.

The industry, from leaf growing through manufacture to product distribution, contributes not only through the taxation system but also through employment opportunities, wholesale and retail revenue, regional development and general productivity.

For example, the Motueka district, with a total catchment population of approximately 8,500, earns some $4.7 million per annum in sales of tobacco leaf.

The contribution of the tobacco industry to the New Zealand economy is substantial.

3.0 PROCEDURE

This submission represents the Institute's *preliminary* comment on a confidential draft Tobacco Products Policy Paper prepared by the Public Health Commission.

The background to the development of the policy paper is most unsatisfactory.

The Institute first heard of draft one of the PHC paper through a chance media leak. The tobacco interests represented by the Institute had not been approached.

* A copy of the PHC paper was subsequently supplied only following a request from this Institute.

* The Public Health Commission set an impossible deadline for a full response and at that time had no satisfactory procedure for interfacing with the commercial sector.

* The Institute joined with a number of other affected bodies in an approach to the Commission asking them not to impose impossible deadlines and to set up appropriate procedures for policy papers affecting commercial interests. (See Appendix A).

* At the time of the development of this preliminary submission the Public Health Commission has refused to adopt the suggested procedures, has imposed a further unreasonable deadline, and has carried out a major revision to the original Tobacco Products Policy Paper (Draft Two).

The sequence of events shows an unfortunate move away from the consultative processes exemplified by the voluntary code on marketing, which proved such an effective form of regulation of the tobacco industry's marketing activities on a cooperative basis.

4.0 BACKGROUND

The PHC paper proposes a wide range of policy options. The author of the paper is Dr Murray Laugesen. Dr Laugesen was also Scientific Editor of the Toxic Substances Report (TSB) (1989), and the author of the now discredited "Study on the relationship between Government tobacco policies and tobacco consumption trends". Dr Laugesen was also an adviser to one of the 'expert' witnesses representing the advertising ban proponents before a court in Canada (1990).

Dr Harris, the expert witness whom Dr Laugesen was advising, admitted during testimony before the Quebec Superior Court that some of the Laugesen-supplied data were wrong; that some were questionable; that one batch of critical consumption data was incorrect; that perhaps two critical countries were incorrectly classified with respect to their anti-smoking policies; and that this incorrect classification invalidated Dr Laugesen's study.

The Canadian Court had these things to say in its final judgement about the Dr Laugesen supplied data and/or Report:

* "It is a report with an obvious point of view and it's conclusions reflect that point of view.

* "With respect to the TSB Report [authored by Dr Laugesen and C Meads] the Court can only note that it contains serious methodological errors and a lack of scientific rigour which renders it for all intents and purposes devoid of any probative value".

This was the same viewpoint put forward by this Institute when debating the TSB Report in 1989.

It would seem that the essential basis of this PHC paper should be regarded as a set of biased statements based on a demonstrably predetermined viewpoint and as being designed to persuade, rather than to inform; and its findings reflect that point of view.

Dr Laugesen's position, and the PHC paper, exemplify the Lalonde doctrine. Lalonde, a former Canadian Minister of National Health and Welfare, argued that health messages must be vigorously promoted *even if* the scientific evidence was incomplete, ambiguous and divided. He said health messages must be "loud, clear and unequivocal" *even if the evidence did not* support such clarity and definition. There could be no room for confusing viewpoints however uncertain the issues in reality might be. This is a questionable policy wherein "the ends justify the means".

If an advisory body to Government such as the PHC seeks to change lawful consumer behaviour it must have a proper basis for so doing.

As an advisory body the PHC has, in particular, an obligation to assess the scientific evidence and objectively and fairly to present available mathematical calculations.

5.0 SUMMARY : PROCEDURE

* Given the complexity of the PHC document and the tight deadline for response, this Tobacco Institute paper must be regarded as only an initial submission.

* The content of this submission makes it obvious that once it has been considered by the PHC there must be exchanges of information and further consultation.

* The PHC paper in its present form is substantially flawed and incomplete and as such, is not an appropriate document for public policy advice to Government.

* This Institute awaits an authorative response to this submission to ensure that essential exchanges of information take place before the PHC paper proceeds any further.

6.0 HEALTH STATUS

"4,000 Premature Deaths"

The claim is that *"the smoking of cigarettes is a cause of ... over 4,000 premature deaths a year ..."* (p3,4).

The use of the word 'cause' must be a deliberate, and misleading over-simplification. The figure of 4,000 has been so repeatedly used by the anti-smoking proponents that it is now little more than a slogan.

The figure of over 4,000 premature deaths was arrived at by applying cigarette smoking prevalence rates as estimated from Census data in New Zealand, along with relative risks of mortality as estimated in a number of *overseas* countries (the applicability of which to New Zealand is doubtful) to the mortality and morbidity data for New Zealand. The resulting numbers of alleged deaths are *estimates* associated with cigarette smoking. Any estimate derived in this manner hardly deserves the word 'indicative', and certainly not the word 'cause'.

No disease is *uniquely* statistically associated with tobacco use; it is, therefore, invalid to use such a calculation as a reasonable basis for deriving policy options, and the implication is that the figure has been included for its propaganda value. The Institute critiqued the faulty nature of this '*estimate*' in 1989 and has yet to hear from the author of this paper regarding that critique.

Grey et al (1988) forms the basis of the Health Department publication "The Big Kill" wherein deaths are attributed to smoking by geographical areas. If the original *estimates* were reasonable then *actual* cancer deaths would correlate highly; they do not.

Under the heading "Maori", the claim is that "around half of all Maori smoke. Two-thirds of pregnant Maori women smoke. Lung cancer rates in Maori women remain the highest recorded in the world for women" (p8).

The statement naively assumes that the prevalence of lung cancer in Maori is a function of their smoking habit *alone*. It is known that lung cancer rates vary widely in ethnic groups whose rates of smoking are not very different, Hinds 1984. It is probable that these smoking rates are largely associated with lifestyle variables such as lower socio-economic status. The PHC paper states that the lower socio-economic groups have higher than average rates of smoking participation, but the smokers among them tend to smoke fewer cigarettes than higher income smokers (p7).

Lifetime Risks to Smokers (p5)

Under this heading there follows a series of emotive claims.

> *"Exposure to cigarette smoke among adolescents taking up smoking also exposes the lungs to more than forty gene-damaging cancer-causing chemicals".*

At best, this claim is an over-simplification; at worst it is a deliberate attempt to mislead. The author does *not* disclose that the International Agency for Research on Cancer did classify forty three compounds as animal carcinogens, and only two have been *suggested as possible* human carcinogens. All forty three compounds are present in very small amounts in cigarette smoke compared to the doses investigated in animal studies. This is the *second* time the author has made this claim, and the second time the Institute has had to point out the misrepresentation.

> *"Addiction to nicotine ensures prolonged and repeated exposure of the lungs to these chemicals over a lifetime".*

The use of the word 'addiction' is emotive, inapplicable, outdated, and is discussed elsewhere.

The claim relating to smokers dying early is discussed elsewhere.

Environmental Tobacco Smoke

The opinion that smoking is harmful to the health of non-smokers originated mainly from the US Surgeon-General's 1986 Report. The evidence used in support of this opinion is far from conclusive. It was based on three prospective and ten retrospective studies. Nine out of thirteen studies that looked at spousal smoking and lung cancer were **not** statistically significant. The findings of two out of the other four studies were admitted to be highly tenuous by the Surgeon-General.

The PHC alleges that "the exposure of non-smokers to ETS is *causally* related to lung cancer in non-smokers". There is again the deliberate semantic substitution of *cause* for statistical association. The 1986 Reports of the US Surgeon-General *and* the National Academy of Sciences concluded that it has **not** been proved that exposure to ETS resulted in respiratory disease in non-smokers.

Given the above 'concerns' about ETS, and the attempt to set outcome targets for ETS exposure without fully revealing the pros and cons of the technical papers, one can only conclude that the author was pre-occupied with setting an agenda in a less than objective fashion.

EPA

The EPA "considered" thirty studies looking at lung cancer and spousal smoking, twenty four of which showed no significant statistical association. However, the conclusions of the final report are mainly based upon a meta-analysis of eleven US studies. Even when the EPA widened the confidence limits to 90% (a breach of their own standards), only one of these eleven studies showed a significant association. The EPA also ignored two US studies which were published a few months prior to the release of the final report (Brownson et al, and Stockwell et al). The Brownson study was heralded as one of the largest and best designed, and was funded by the National Cancer Institute. It is hard to believe that the EPA did not know about it beforehand. It found a lung cancer relative risk of 1.0 associated with spousal smoking and it has been claimed that if the Brownson study had been included in the EPA's meta-analysis, then they would not have found a significant association between spousal smoking and lung cancer.

Eleven of the thirty studies considered by the EPA also examined workplace ETS exposure, nine of them showed no significant effect; ten of the thirty studies considered childhood ETS exposure, nine of which showed no effect. The EPA did *not* consider these data, ~~which would have~~ been in contradiction with their aim, but instead estimated deaths due to ETS exposure in the workplace and during childhood by extrapolating a relative risk from spousal smoking.

The EPA classification of ETS as a Class A carcinogen rests on two fundamental flaws that invalidate its conclusion. The first was the misuse of the available epidemiological evidence. The statistical manipulations by the EPA constituted speculation without adequate scientific foundation. The second was the assumption that ETS has been shown to be an *undoubted cause* of disease. The American Health Foundation did *not* reach the same conclusion.

The EPA classification of ETS as a Class A carcinogen is now the subject of a Court challenge.

Philip Morris, RJR Nabisco and others, in June 1993, filed suit in the US District Court in North Carolina. They seek a permanent injunction overturning the EPA's findings, on the basis that the agency resorted to "manipulating and cherry-picking" the data to disparage falsely cigarettes. The plaintiffs further contend that the EPA manipulated the results to fit its pre-determined conclusions. The same might be said of the unsubstantiated Figure 1 regarding per capita projections to be found on p8 of the PHC paper.

"Effects of Tobacco Products ..."

Under the heading "Effects of Tobacco Products use on Health Status" (p3), the PHC paper makes a series of allegations that purport to be, *but are not*, supported by the scientific literature. This is an example of selective citation to support a predetermined position.

The first three of these broad allegations are deliberately misleading in that they claim smoking as a *cause*.

A number of epidemiological studies have claimed that smoking is statistically associated with a number of diseases: ie. smokers have a higher rate of certain diseases than non-smokers. However, it is not possible to assert that smoking causes these diseases as the PHC paper has done, since statistical studies *cannot* prove cause and effect.

For most of the diseases mentioned, the studies which suggest the association between the disease and tobacco smoking do not even satisfy the US Surgeon-General's criteria for a *suspicion* of a *cause*: plausibility, consistency, specificity, temporality, strength, comparison with random variability, and finally exclusion of confounding factors.

All six of the broad assertions in this part of the PHC paper have been raised by the author of this paper before, in the Toxic Substances Report of 1989. They were refuted by the Institute then. That author has yet to respond in detail to any of the refutations, but chooses to assert the same claims again for a different audience. It is presumed that no complete refutation is possible.

Again, the conclusion to be drawn is that the assertions are designed to persuade, rather than to inform. This is unacceptable.

The author has cited only three references that support the case and ignored at least ninety references that do not suit the case. The author has had since July 1989 to consider these ninety or so papers. The reiteration of the 1989 claims suggests either an unwillingness to consider viewpoints that do not coincide with his own, or an inability to rebut them.

We examine each of the claims in turn, along with a series of omitted citations.

> *"a cause of increased lifetime risk of, and shortened life due to chronic bronchitis and emphysema, and cancer of the lung ..."*

A number of other findings exist which do not fit comfortably with such claims and which are not mentioned in the PHC paper. For example: Burr and Holiday, 1987; Becklake et al, 1987; Korn et al, 1987; Euler et al, 1987; Gamble et al, 1987;

Garshink et al, 1987; Cohen and Chase, 1978; the Royal College of Physicians, 1983; Reid et al, 1983; and Peach, 1986.

With respect to cancer of the lung, the relationship over time between that disease and smoking is far from clear.

The US Surgeon-General's Report of 1982 has been criticised by Burch, 1983. There are considerable geographic and ethnic differences, Hinds et al, 1984. There are sex differences in the risk factors, Burch, 1978.

The changing rates of lung cancer as between smokers and non-smokers require to be explained: Hanal et at, 1987; Watanabe et al, 1987; Kung et al, 1984; Gao et al, 1987; Gazdar et al, 1988; Anton-Culveer et al, 1988 and Reyes et al, 1987; Todd et al, 1976; Belcher, 1987; Osmond et al, 1982 and 1983; Burch, 1988 and Levi et al, 1987.

Radon, diesel and petrol fumes have been implicated: Axelson, 1984; Archer, 1987; Blumer and Reich, 1980; Garshink et al, 1987; and Gustafsson et al, 1986.

A relationship with occupation has also been noted: Blot, 1984; IARC, 1982; Sterling, 1978; Simonata et al, 1988. Air pollution has also been implicated, Shy, 1984; Churg and Wiggs, 1987; Sasaki et al, 1987; Leung, 1977; and Gao et all, 1987.

Personality and familial relationships have also been associated: Linn and Stein, 1987; Mangan and Golding, 1984; Tokuhata and Liienfield, 1963; Kramer et al, 1987; Jones, 1977; and Ool et al, 1981.

> "... *a cause of shortened life due to premature heart attacks*"

On the assumption that this refers to coronary heart disease, the US Surgeon-General, 1979, noted that smoking was *not* an essential risk factor and the British Medical Journal in an Editorial, 1982, pointed out that dietary changes probably had the most important effect on the incidence of coronary heart disease. Several other commentators have also cast doubts on this assertion regarding tobacco: Shipper and Pocock, 1987; Keys, 1970; Lapidus et al, 1986; and Menotti et al, 1987. Hopkins and Williams, 1981, claimed that 245 different risk factors have been associated with coronary heart disease.

> "... *a cause of strokes; a cause of arterial disease causing reduced blood supply to legs and feet (causing gangrene), a cause of cancer of the lip, tongue, throat, larynx and gullet...*"

With respect to atherosclerotic peripheral vascular disease, the US Surgeon-General, 1979, himself noted inconsistencies in the autopsy literature, and could find no

known mechanism to explain the association. Other pathological and environmental factors have been implicated: Fowkes, 1989.

In the case of cerebrovascular disease, the US Surgeon-General, 1979, noted that the results of the various epidemiological studies on smoking and stroke had *not* been congruent and that no conclusion could be reached with confidence. It was not until 1989 that the US Surgeon-General changed his mind. However, other influential commentators *still* had their doubts; among them, Shinton and Beevers, 1989; and Doll and Peto, 1981. Other suggested risk factors have been raised by Colditz et al, 1988; Olenckno, 1988; Bonita et al, 1986; Zhang et al, 1988; and Wellin et al, 1987.

In the case of cancers of the oral cavity there are differences by sex in the disease that remain to be explained. Other factors have also been associated: Mahbouhi and Sayed, 1982; Graham et al, 1977; Decker and Goldstein, 1981; and Wey et al, 1987.

The situation is equally unclear for the larynx and the gullet. Alcohol abuse has been strongly implicated for the larynx as have other factors: Wynder et al, 1956; Shettigara and Morgan, 1975; Manning et al, 1981; Pedersen, 1973; Olsen et al, 1984; Lynch et al, 1979; Tucker, 1935; Blumiein, 1957; Lin et al, 1979; and Sarma, 1958. There are also unexplained patterns between cancer incidence and smoking rates that have to be explained: Stefani and Carzoglio, 1985; and Osmond et al, 1983.

Similar unexplained differences apply to the gullet as apply to the larynx: the differing rates of incidence by sex, the application of other risk factors, (Brown et al, 1988 and Yu et al, 1988) and temporal trends not matching the incidence of cigarette smoking: Burch, 1984.

> "*a contributing cause of cancer of the bladder pancreas and kidney...*"

The US Surgeon-General, 1982, could *not* find the strong association that the author of the PHC paper asserts in attributing cancers of the bladder and kidney to smoking. Other associations have also been found: Brownson et al, 1988, Miller, 1977; Hoar and Hoover, 1985; Kantor et al, 1988; Slattery et al, 1986; and Najem et al, 1982.

In the case of the pancreas, the epidemiological studies suggest poor methodology or the inability to correct for confounding factors. Other authors have implicated a range of other factors: Morgan and Wormsley, 1977; and Williamson, 1988. The British Medical Journal, 1988, conceded: "We know less about what causes it (pancreatic cancer) than we do about what causes most other common cancers".

> "*...associated with cot death, aortic aneurysm, cancer of the stomach and cancer of the cervix*"

A review of the evidence (Guntheroth and Spiers, 1992) noted that "without exception, all studies demonstrated an increased risk of SIDS associated with the

prone sleeping position". It documented evidence from two cities in New Zealand and one in England of significant reductions in the incidence of SIDS following media campaigns to avoid the prone position for infants. More recently the UK Department of Health has announced that the number of cases of SIDS in England and Wales dropped by over a half following the Government's "Back to Sleep" campaign. This data suggests strongly that the prone sleeping position was the major cause of the disease. In addition, associations of smoking with cot death (SIDS) may easily be artifacts of the association of smoking with lower socio-economic status.

Aortic aneurysm is such an uncommon cause of death that it is hard to see why it should influence public policy. Not surprisingly, there have been correspondingly few studies, so it is not possible accurately to assess the effects, *if any*, of smoking.

With respect to the stomach, diet is believed to be the primary factor of significance, and some industrial processes are also associated: Tomatis, 1988.

The US Surgeon-General, 1982, pointed out that it was by no means certain that smoking is a risk factor for cervical cancer; further, cervical cancer may be associated with high levels of sexual activity: La Vecchia et al, 1986 and the US Surgeon-General, 1982.

> "... *responsible for one in two to one in three smokers dying early from smoking, depending on the length of time a person smokes and the number of cigarettes smoked*".

Using data from the Department of Health's publication on mortality, it is simple to produce average age of death calculations. In fact, the Institute has done so before, (April 1990), and its finding has yet to be answered, and yet the above claim is repeated. The Institute's finding is that *the average age at death from the alleged 'smoking associated diseases' is about five years greater than the average age of death from all other causes, even after removing all motor vehicle accidents and restricting attention to people aged 15-64.*

Simplistic Calculations

Considerable attention has been given in recent years to the calculation of estimates of smoking-attributable mortality. This has been attempted by applying estimates of risk derived from epidemiological studies of survey populations to target populations, many of which differ in location, general environment or time from the corresponding survey populations. The simplistic mathematical model which underlies the conventional methodology has been found to be inadequate as a representation of a complex practical situation. Vital elements are ignored and this model takes no account of the vagaries of the uncontrolled epidemiological data upon which it is based. *Estimates obtained by these methods should be qualified by a*

statement to the effect that they rely upon untested and probably unrealistic assumptions. Smoking attributable mortality as conventionally defined does not establish mortality *caused* by smoking.

From 1964 to 1975, the number of cigarettes smoked per capita in New Zealand was growing; after 1975 it fell reasonably steadily until 1992.

According to the PHC document, death rates due to ischaemic heart disease risk will begin to fall about six years after the change in smoking habits. Conversely, we can expect heart disease death rates to continue to rise for up to six years following the peak in cigarette consumption before beginning to fall. *In fact*, ischaemic heart disease death rates in New Zealand peaked in 1976 and have continued to fall ever since.

While cigarette consumption was rising over the period from 1965 to 1975, the PHC reasoning would have us believe that cancer death rates would therefore be continuing to increase over the period 1975 to 1985, following the ten-year time lag quoted. The facts as presented in "Mortality and Demographic data 1990" show that non-Maori death rates have "changed little since 1974 for non-Maori. Maori cancer death rates ... have declined overall by a fifth for males and a third for females".

The PHC document quotes a number of "significant" reductions in mortality in recent years, but does not give the details of the figures. These figures are not given directly in the Mortality and Demographic Data series, so it is difficult to confirm or refute such claims. However, even the limited analysis above does serve to cast some doubt on the bald assertions made in the PHC paper.

Certainly over *short* periods there will be quite high correlations between the lagged consumption series and mortality rates: *any two series headed in the same general direction will be highly correlated.* Over the last years of the twentieth century, health standards have been generally improving, while social approval of cigarette smoking has steadily eroded, so that consumption and prevalence have both been falling. Consequently, such correlations serve only to reinforce what we already know about the social forces acting in our times.

7.0 "ADDICTION"

The PHC paper mentions the word 'addiction' with some frequency. This, like the use of word 'glamorisation' with respect to the cigarette pack, is an outdated philosophy and shows a lack of appreciation of the current literature. What the PHC paper does not reveal is that the US Surgeon General Report of 1988 regarding 'Addiction' has been superseded by the (new) Surgeon General's Report of 1990 regarding Cessation.

This is simply another example of evidence to support a preconceived viewpoint.

The claim is that *"Ninety percent of New Zealand smokers describe their habit as addictive ..." (p4).*

The distinction between the terms 'addiction', 'habit', 'compulsion' and 'dependence' are blurred. (The PHC paper also blurs 'addiction' and 'habit'). A major problem with the subject of 'addiction; is deciding what the word actually means. There has been a considerable disagreement between scientists and medical bodies in arriving at a satisfactory definition of addiction.

The fact that smokers agree to a statement that smoking is 'addictive', when it is put in front of them only means they *believe* some people have a strong inclination to that particular sort of activity. On this basis, some people in New Zealand would agree that they have an 'addiction' to rugby. This is not a scientific basis for establishing 'addiction'.

Over a period of years there have been a number of suggestions culminating in the publication of the US Surgeon-General's Report entitled "Nicotine Addiction" in 1988, that made the extraordinary claim that cigarette smoking was "as addictive as heroin and cocaine".

That Report notes that nicotine and tobacco did not fit readily into standard definitions of addiction. The response was to broaden the definition of addiction so widely that it could now include smoking or the use of nicotine as well as virtually any other behaviour that people find enjoyable and repeatedly indulge in. There is no substantial scientific evidence that smoking creates a physical dependence on nicotine and no justification for finding that definition as definitive. It should be noted that the World Health Organisation abandoned the use of the term 'addiction' in the late 1950's.

Two years later, the 1990 US Surgeon-General's Report on "Cessation" does not use the word 'addiction' when discussing the psychological consequences of smoking cessation.

In fact there has been a substantial change in the US Surgeon-General's viewpoint. For example: "Short term consequences of smoking cessation include anxiety,

irritability, frustration, anger, difficulty concentrating, increased appetite and urges to smoke. With the possible exception of urges to smoke and increased appetite these effects soon disappear" (p565). There is no suggestion that smoking is "as addictive as heroin and cocaine". This 'official' change of viewpoint is some years old, yet is not reflected by the author of this paper.

Smoking is a personal choice that can be stopped if and when a person decides to do so. The US Surgeon-General's own Public Health figures show that "as at 1987, more than 38 million Americans had quit smoking cigarettes, nearly half of all living adults who had ever smoked" (p13). And further, "... 90% of smokers quit without using cessation programmes, counselling or nicotine gum ..." (p11).

The continued use of the word 'addiction' by an author who persists in quoting the literature selectively can be seen as a deliberate semantic device to subvert the debate from the objective to the emotional.

8.0 SUMMARY : HEALTH STATUS AND "ADDICTION"

Studies based on statistics have been used to support arguments against tobacco. Such studies are about computerised correlations not biological science nor the scientific laboratory.

No autopsy nor any medical laboratory programme has scientifically established cigarette smoking as a cause of lung cancer or heart disease.

* The claim that cigarette smoking is a *cause* of 4,000 deaths a year can at best be an indicative estimate, and certainly **not** proof of causation.

* The author incorrectly cites the International agency for Research on Cancer by not disclosing the real nature of the Agency's studies.

* The author frequently uses the word "causes" when he actually means some form of statistical association.

* In relation to ETS the EPA could only find ETS to be a Class A carcinogen by selective inclusion and exclusion of studies, by departing from the normal confidence limits, and extrapolating improperly from spousal-related information to the workplace situation. That EPA classification is now the subject of a Court challenge.

* Under six broad allegations the author cites only three references to support his preconceived viewpoint and has ignored (for four years) some ninety references that do not coincide with his viewpoint, and that he has not rebutted publically.

* The paper mentioned "addiction" with some frequency and does not take into account the changed stance of the US Surgeon-General between 1988 and 1990, and the number of people who have quit smoking cigarettes: another example of selecting data to fit a preconceived viewpoint.

9.0 TAXATION ISSUES

The Alleged Cost of Smoking

The Australian Estimate

"The cost of tobacco use in Australia in 1988 was estimated at A$6.84 billion ..." (p34). It is difficult to understand why this particular estimate has been brought into the paper, as it has no direct relevance to the New Zealand situation. However, given that it has been introduced into the debate, the following points are relevant.

This approach fails in the following areas:

> "it fails to recognise the wider production and resultant income effects in the economy of a cessation of consumption expenditure in a designated area
>
> "it wrongly ascribes to the community, costs which are essentially private
>
> "it fails to recognise private *benefits* while including private *costs*
>
> "it seeks to provide a simple definition of "economic cost of drug abuse" but effectively creates even further confusion as to what actually is being measured
>
> "it fails to distinguish between value added effects which in national terms represent the well-being of the community, with total expenditure or total output effects which might have little or no impact upon the value-added available to the community".

In summary, the Australian study has a number of serious methodological and data deficiencies which render meaningless its estimates of the cost of what they inappropriately refer to as "drug abuse". (Price Waterhouse, Canberra, 1991).

The PHC paper relies on Australian 'evidence' and does not analyise the New Zealand situation. Such an analysis should be undertaken and is properly the field of expert economists rather than a doctor, particularly one with a subjective viewpoint.

The Canadian Estimates

The PHC paper does *not* cite a Canadian paper (Raynauld, 1992), perhaps because it was not supportive.

The finding of this paper was that smokers do *not* impose a considerable burden, *even if* claims of hospitalisation and medical costs and lost output due to premature death

are accepted. These costs may give rise to transfers, but these in turn are more than compensated for by other transfers which lead to a net flow in favour of non-smokers. The direction of this flow remains unchanged even considering a wide range of medical hypotheses.

The New Zealand Estimates

In respect of the Grey et al cost estimates, the Institute has maintained for some years that the cost estimates are flawed. It is simply not valid to add up the costs of hospitalisation and medical care from the alleged smoking-related diseases and unquestioningly apply an attribution risk figure to come up with a cost "due" to cigarette smoking. The fallacy of such an exercise is that no evaluation of competing risks is made.

The eradication of smoking will **not** dramatically reduce the call on public health funds since there are a large number of risk factors competing for the hospitalisation of the populace, particularly at the older ages at which the so-called smoking-related illnesses become relevant.

The General Problem with such Estimates

As pointed out in the Canadian paper mentioned above, the argument is that the smoker imposes a cost on others, and the issue is who pays for what, and who benefits. For example, it is said that non-smokers pay a good proportion of supplementary health costs so that transfers go from non-smokers to smokers. On the other hand, it must be recognised that smokers pay taxes on tobacco which benefit non-smokers to a large extent. In this case the transfers go in the opposite direction. Smokers also pay general taxes in the same way that non-smokers do. A complete balance sheet of revenues and expenditures accruing to smokers *and* non-smokers is therefore necessary before one can conclude that smokers are a burden on non-smokers, (even if one accepts the allegations in relation to smoking and health).

The published work in New Zealand has been conducted by people with a well established anti-smoking viewpoint: perhaps for this reason it has been essentially superficial in nature in that it does not take into account a complete balance sheet.

Taxation in General

The Institute does not know of any study establishing the lifelong health costs of a smoker as being any different to the lifelong health costs of a non-smoker.

Excise Taxation

The PHC document appears to be in conflict with recently established Government policy for excise taxation.

Excise Taxation is already at unreasonably high levels and is regressive.

As recently as July of this year legislation was passed setting criteria for the indexation of excise taxation on tobacco products. Against this scenario it is incorrect for a body such as the PHC to be attempting to interfere with such recently enacted legislation.

Additionally, the question of excise taxation is not a matter for consideration by the PHC. Excise taxation matters properly rest with Finance and Customs Ministries and the Treasury. The Institute is of the view that any involvement it may have in any debate on excise taxation should be with the Ministers of Finance and Customs and with Treasury and Customs officials.

Transfer Effects on Taxation and Retail Sales

Smokers' expenditure "will shift gradually to other products" (p34). The PHC paper does *not* take into account that this would result in a *lower* revenue, given that nearly 70% of smoker expenditure goes to the Government in the form of excise as well as GST. With the exception of alcoholic beverages, any substitution for other products will not produce excise tax revenue.

10.0 PLAIN PACKAGING

Glamorisation of the Pack

Anti-smoking proponents have maintained for years that there is a cause and effect relationship between juvenile smoking and first advertising and then sponsorship. The Institute has shown for some years through extensive independent research that peer group associations were the more important cause, as has the WHO. This research has been presented to Dr Laugesen on several occasions and he has assiduously ignored it.

A recent Danish study (Jensen and Overgaard) has confirmed these findings by establishing having friends who smoke is a strong predictor of a young person's decision to take up smoking.

The PHC paper recommends "varied warnings, packet redesign and plain packaging" to "deglamorise the product and give more prominence to health warnings ..." (p17).

The primary information to be imparted by the package in any fast moving consumer goods area is the brand name. Brand name recognition (via the use of the trade mark) to distinguish that brand from a multiplicity of competing products (each with their own brand names) is a normal marketing tool to retain, or expand market *share*, not to expand the market.

The trade mark is an essential consumer tool for finding a personally satisfactory product from a host of similar, but personally unsatisfactory products, all within the same product category. *Package stimuli, including the trade mark, are of no interest to people not already within the market for that specific product.*

It has been claimed that "plain packaging has been found to be unattractive and to discourage smoking in adolescents", (Beede et al, 1990). The Beede et al paper was critiqued by the Institute some three years ago. In brief, this paper ignores papers not supportive of its proposition, the methodology is a prime example of tautology and the data was obtained in a biased environment.

The *assumption* is that changes in the packaging will lead to changes in behaviour. **There is no clear evidence that removing the trade mark, or dispensing with the pack design, will affect behaviour.**

The information upon which the Australian Council of Ministers based their recommendation (p18) was questionable, if not frankly specious. In the thirteen studies involved there is *no evidence to suggest that changes to cigarette packaging will alter smoking behaviour*. In the key study it was necessary to bias the survey, by presenting misleading information, to obtain the desired response.

In Australia the situation was such that younger smokers, and those contemplating quitting, already had a better recall of the current health warnings. However, *it has not been shown that public knowledge of warnings is related to smoking behaviour.* There is no Australian evidence that health warnings if read and understood, would affect behaviour.

The PHC proposals would deface and devalue international trade marks. They would interfere with the ability of an industry to market lawful products.

The issues arising from proposals to deface the pack are serious; eg:

* Banning the use of any registered logo, design, pattern or trademark amounts to censorship.

* Freedom of expression is a right recognised by New Zealanders. It is proclaimed in the United Nations Universal Declaration of Human Rights. Any proposed "plain pack" law would fly in the face of New Zealand's commitment to free expression.

* The prohibition of the use of trade marks is at odds with the New Zealand Bill of Rights, an Act ironically made law on the same day as the prohibitive Smoke-Free Environments Act. The New Zealand Bill of Rights purports to protect freedom of expression in the same terms as the Universal Declaration of Human Rights.

* Prohibiting use of the trademarks is contrary to GATT (General Agreement on Tariffs and Trade).

* A trademark is commercial property which represents a substantial financial investment. The removal of the right of a company to display trademarks arbitrarily strips it of these assets or makes them worthless.

* At a time when the Government is actively encouraging a free market economy, a "plain pack" law would represent a major step backwards.

* Under New Zealand law, a trademark to be registered and protected, must be used. If use is prohibited, the protection afforded by registration may not be available.

* A trademark ban would establish dangerous precedents that could affect hundreds of other products and services and the rights of individuals.

* If the prohibition of cigarette company trademarks is proposed, then should not the PHC correspondingly propose prohibiting trademarks for sugar and chocolate (they might tempt diabetics); fat products (bad for people with

cardiovascular problems); all products with preservatives or colourants (bad for hyperactive children)?.

The PHC is wrong to propose the appropriation by Government of intellectual property as such an appropriation would be contrary to international trade conventions to which New Zealand is a signatory.

11.0 CONSUMPTION TARGETS

The History of Smoking Behaviour in New Zealand

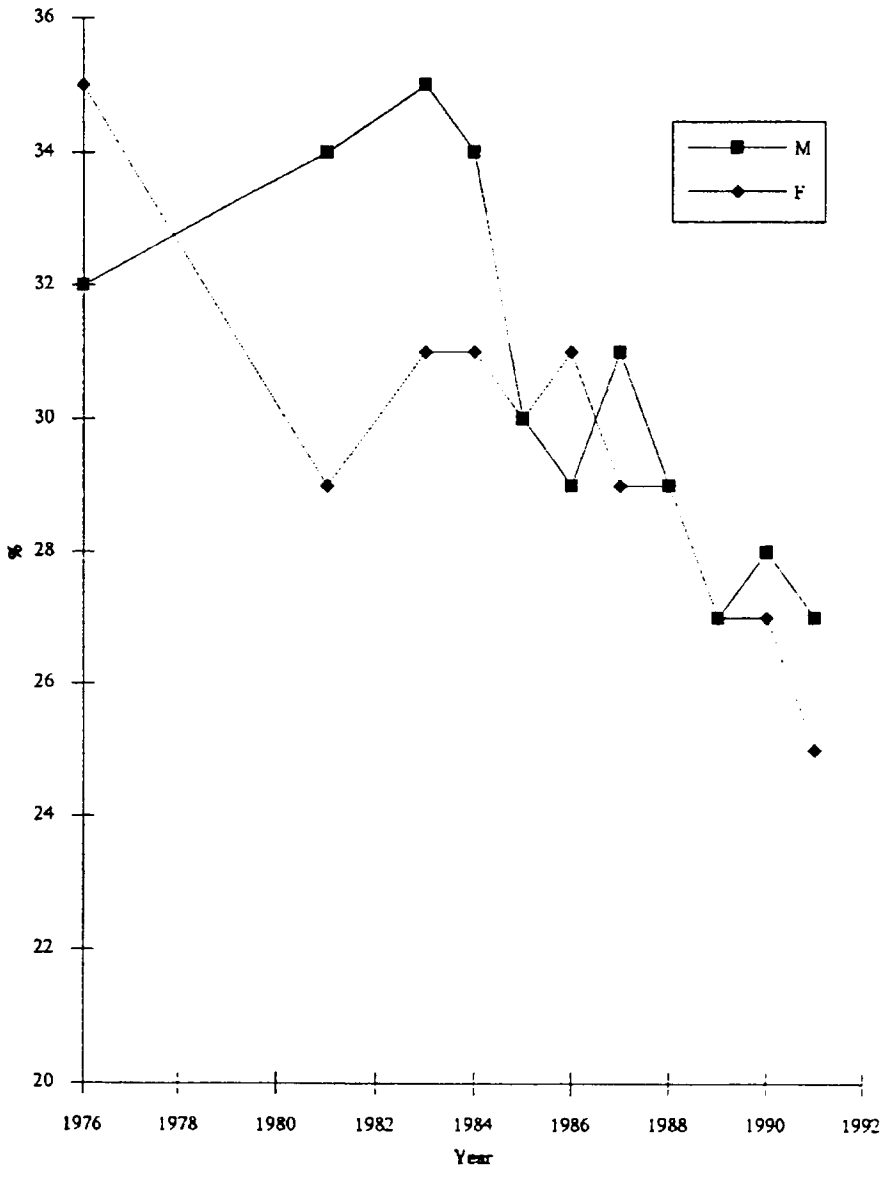
Tobacco products consumption in New Zealand peaked in 1963. The overall level of cigarette and tobacco consumption has fallen sharply since 1975 (Tobacco Statistics, 1991, p13).

Under the heading "Setting a Tobacco Products Consumption Target" (p8), the PHC paper goes on to state: "The target ... is to reduce the consumption of tobacco products from 1,600 cigarette equivalents in 1992 to 1,000 cigarette equivalents in the year 2000. The target requires a decline of steepness intermediate between the two projections shown in Figure 1", (p8).

The source of the data is acknowledged but there is no explanation of the method of modelling the projections. (The paragraph below Figure 1 is obfuscatory).

There follows a chart of the percentage of adult New Zealanders who were smokers, over the period 1976 to 1991, as per Drug Statistics 1992.

Adult smokers as a proportion of the population



The graph is interesting for two reasons: first, because it indicates a declining prevalence in smoking, ongoing since at least 1983, long before the so-called Smoke-free legislation was introduced, and without change in the rate of decline since the introduction of the legislation; secondly, the graph is interesting because the early points on the graph show much greater fluctuations than the later points, and in fact, indicate a female prevalence in 1976 which was 3 percentage points greater than that indicated for males. This does not seem likely, nor does it seem likely that the reversal recorded for the next point was in fact what happened: a male prevalence greater than female prevalence by 5 percentage points - that is a reversal of 8 percentage points.

In respect of this data, Drug Statistics 1992 prepared by Department of Health notes: "Use with caution when comparing the results of surveys using different methodologies".

Tobacco Statistics 1991, jointly attributed to Department of Statistics and Department of Health, was both more revealing and more circumspect: "These data are taken from two different sources. The first of these is taken from the Census of Population and Dwellings (1976 and 1981). The second source is from OTR Spectrum Survey data. There are limitations on the direct comparability between these two sources. Different methodologies have been applied in the collection of these data, for example, in the selection of survey populations and sampling methods; as well as differences in the actual questions on smoking practice. *Therefore, reliable inferences based on the comparability of data from one source to the other cannot be recommended with confidence*". (Emphasis added).

And yet it is data of this nature that is being used to set "Outcome Recommendations" (PHC, p12). The 'actual' adult smoking prevalence is stated as being 27%. Assuming that this was based on sample survey data, based on a survey of N = 1,000, the sampling error is plus or minus 2.7%. The apparently absolute figure of 27% thus becomes somewhere between about 24% and 30%. In the same way the "target for 2,000 becomes somewhere between 17% and 23%. None of this is noted or explained in the "Outcome Recommendations".

For smaller sample sizes, the likely range of error is much greater. If we are interested in estimating prevalence for 15-24 year old females, for example, the sample size is unlikely to exceed 200, in which case the error margin is closer to plus or minus 7%.

Given the sampling error inherent in such survey material the 'actual' and 'target' figures are approximations only. *It may be that the 'target' has already been achieved.*

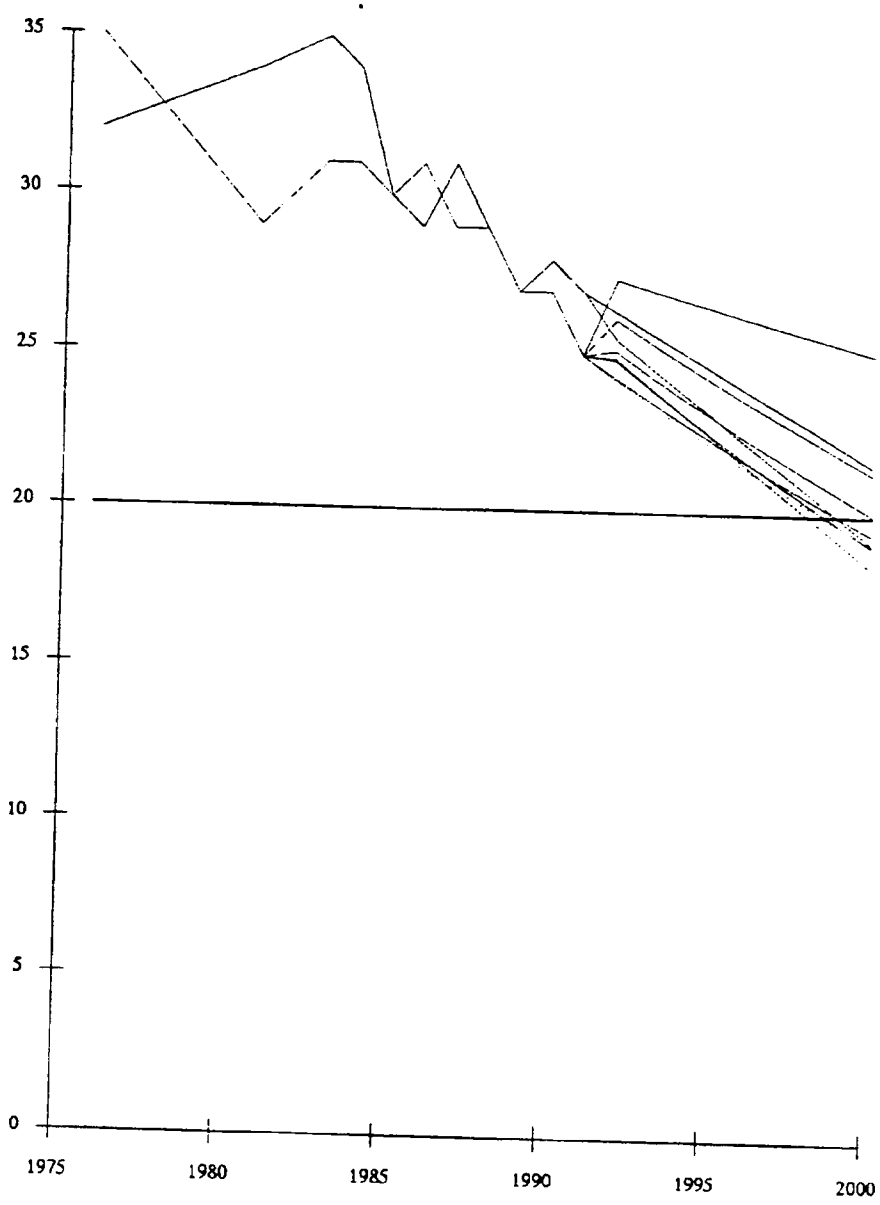
In any case, the 'target' which *appears* to be so precise, is not a suitable target due to its inherent imprecision. Very large sample size surveys would be required to overcome this problem of imprecision.

Given that post-smoking behaviour has undergone *substantial* changes, projecting the slope of the current decline in prevalence is open to several different solutions.

As a preliminary exercise we have adopted nine scenarios. Six of these scenarios suggest that smoking prevalence will meet the target by the year 2000, *without intervention.*

This is demonstrated in the following graph.

Alternative Projections of Prevalence of Adult Smoking



Future Consumption Projections

Extrapolating trends into the future is always difficult, and error-prone; this has to be accepted. However, there are reasonable and unreasonable methods for attempting this procedure. The graph under the heading "Setting a Tobacco Products Consumption Target", Figure 1, (p8), extrapolates two rates of decline into the future, to the year 2000. In neither case is any allowance made for the errors inherent in prediction: we have no idea of how much random variation to expect in these figures.

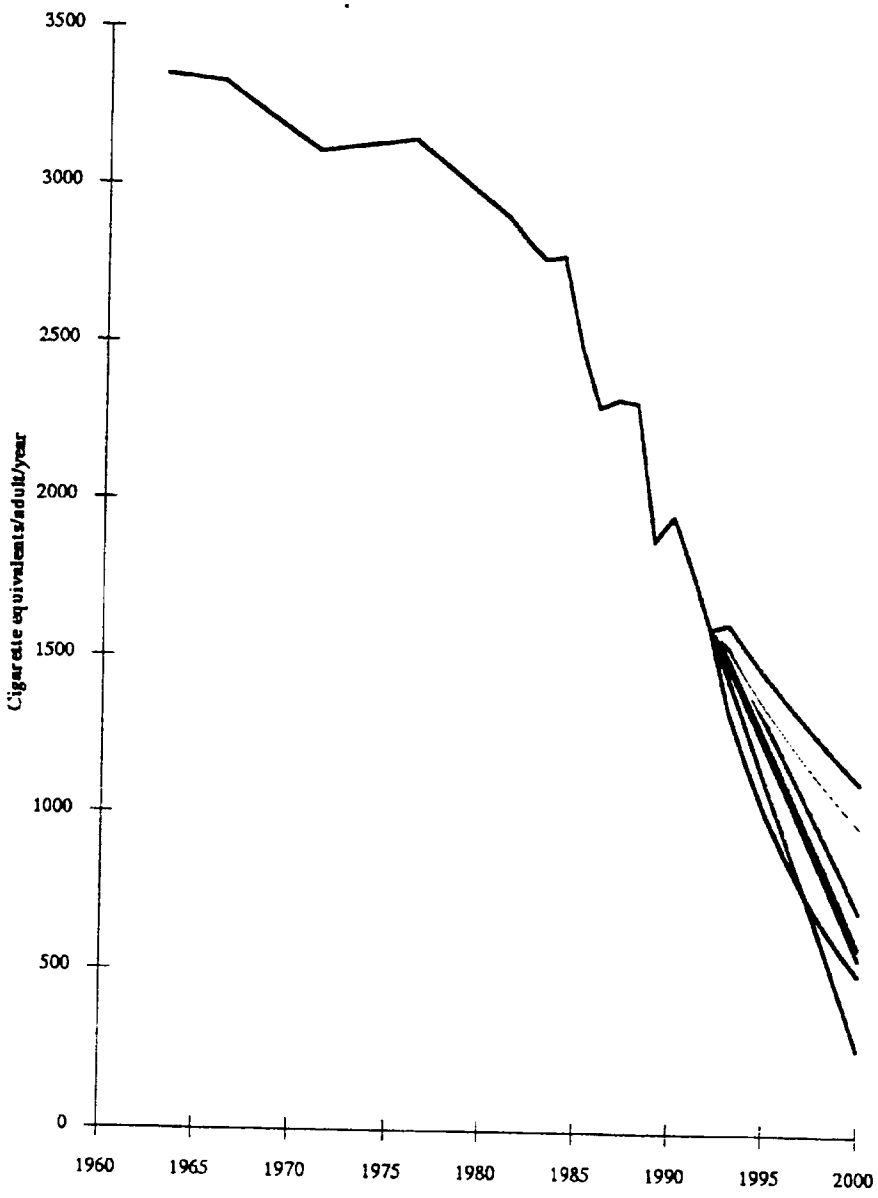
Knowing the author's propensity to error, as witnessed by the Canadian Court, we have had a statistician model some data.

Below is a plot of the per capita consumption figures (aged 15 or more) over the period 1963 - 1992. Clearly, we can construct extrapolations from this curve on a number of bases: use the same average percentage decline as observed over the past two years, past three years, five years, ten years and so on. All of these strategies have their merits, and their drawbacks.

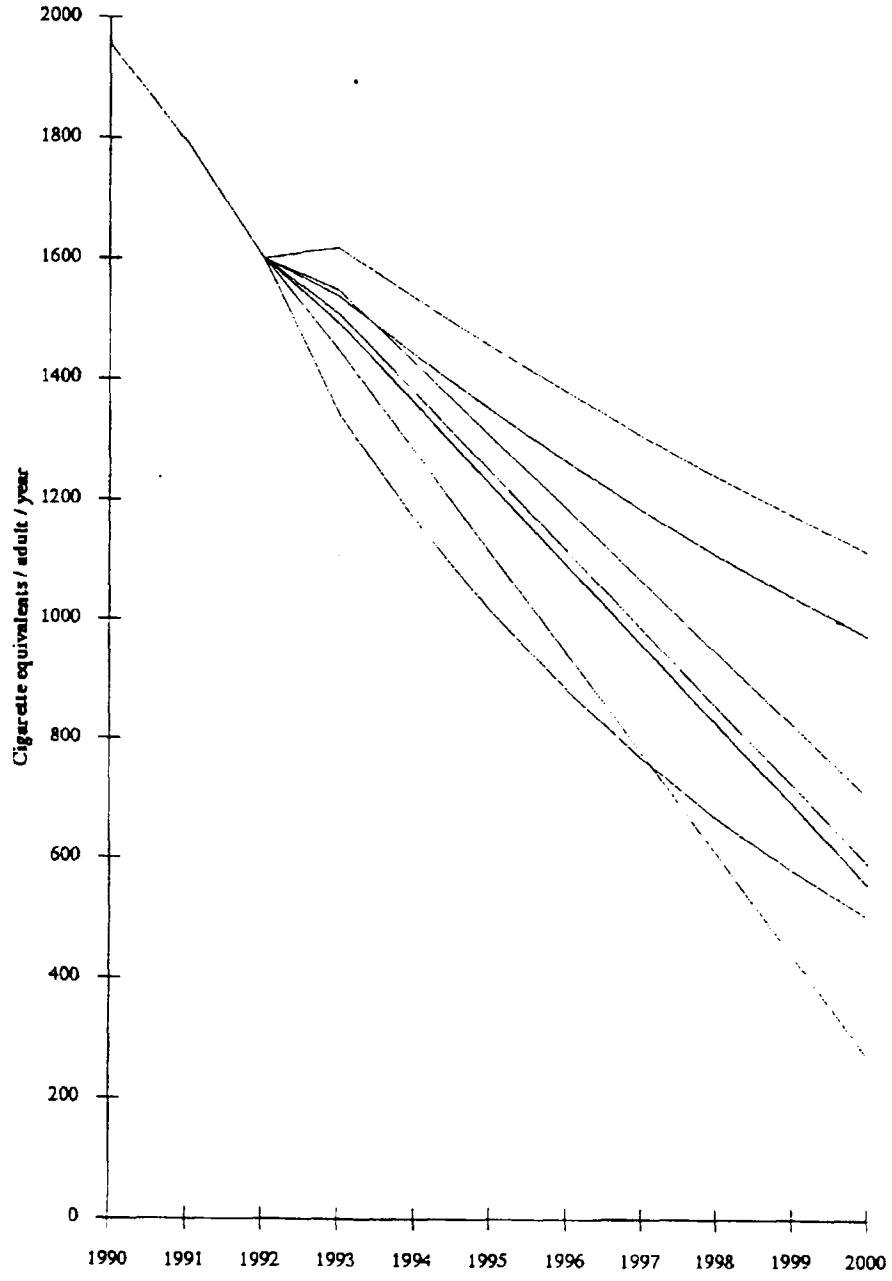
The graph shows projections based on a number of differing but all reasonably justifiable scenarios. The PHC paper states that its projections are based on two possible scenarios, based on data from 1984 - 1990 and 1990 - 1992. The PHC paper does *not* state what model they have used, but there are a number of possibilities, ranging from linear decreases along an established trend line, through a model where there is a linear decrease in the logarithms of the consumption figures, to simplistic models assuming a constant rate of decrease applied to the initial consumption figure. It is also possible, and indeed sensible, to apply these models over a number of differing time periods.

Seven scenarios have been modelled. Six of them show that the 'target' is likely to be reached without any Government intervention. See the following two graphs.

Alternative Projections of per capita Consumption



Alternative Projections of per capita Consumption:
Focus on years 1990 - 2000



Dr Laugesen's modelling techniques, flawed as they have been shown to be, are used to support his preconceived viewpoint. This is yet another example.

Had this modelling been conducted properly, the essential conclusion from the PHC paper would have been to not recommend any Government intervention, thus avoiding wasteful expenditure of scarce taxpayer health funds which could more properly be directed to actual health care.

12.0 SUMMARY : CONSUMPTION TARGETS

* The setting of targets for prevalence from survey data is far less precise than might be thought from the PHC paper. *The targets may have already been achieved.*

* Figure 1 (p8) of the paper is both simplistic and unsubstantiated.

* Re-modelling the same data, to produce seven scenarios, shows that six of those scenarios are likely to be reached by the year 2000 without any Government intervention.

* Had the modelling been conducted with statistical rigour, the essential conclusion would have been not to recommend any Government intervention.

13.0 THE SMOKE-FREE ENVIRONMENTS ACT

The PHC paper proposes further restrictions by way of amendments to the Smoke-free Environments Act 1990. The existing provisions of this Act are such that the Act:-

Interferes in private workplace policies and conditions.

Overrides employee/employer negotiations.

Dictates staff policies for business, small and large.

Forces businesses to introduce and police Government established rules for customers and staff.

Creates situations where just one person in 100 or 1,000 can dictate the behaviour of colleagues.

Compels health bodies to incur increased costs at a time when government is imposing severe budgetary constraints.

Censors New Zealand newspapers and magazines.

Curtails freedom of choice.

Removes sponsorship funds without adequate guarantee of replacement.

Gives preference to overseas sports bodies as against local sports bodies.

Outlaws some trademark usage.

Breaches international trade mark conventions to which New Zealand is a signatory.

Dictates the clothing that citizens may not purchase or wear.

Permits a minister alone to appoint and direct a quango, spending taxpayer funds with less than acceptable accountability.

Requires recipients of government funds to promulgate Government propaganda in return.

Curtails company/shareholder communication.

Expropriates private assets in the form of trademarks.

Compels companies to provide government with commercially sensitive information, with no assurance of confidentiality.

Opts out of long held and clearly workable agreements with industry.

Removes signs from private property.

Creates a hidden agenda of regulatory powers which effectively nullify the ability of manufacturers and retailers to plan their trading future more than a few weeks in advance.

Gives overseas media unacceptable commercial advantages over local media.

Eliminates the right of companies to communicate with customers.

Imposes pressures on sports bodies to act in accordance with Government objectives.

The Tobacco Institute submits that it is reasonable to suggest that the term "enough is enough" appropriately applies.

Such is the restriction of normal freedoms imposed by this Act that if there is to be any revision of the legislation, it should be to return freedoms rather than further remove them.

14.0 ADDITIVES

The PHC paper claims that tobacco products are not required to adhere to any content standards. It further states that levels of additives in each brand are not precisely known and that manufacturers are reluctant to divulge commercially sensitive information.

In fact, since the passage of Smoke-free Environments Act, New Zealand manufacturers and importers have supplied the appropriate regulatory body with lists of additives and a declaration that maximum permitted levels of any additive have not been exceeded.

The additive listings employed in this reporting and declatory procedure are based upon internationally recognised and agreed listings.

15.0 CONCLUSION

The Tobacco Institute of New Zealand welcomes the opportunity to make preliminary comment on the PHC document "Tobacco Products".

We would again emphasise that the PHC should now thoroughly analyse this submission and then involve itself in further extensive consultation with this Institute before any further action on the Tobacco Products paper is considered.

In its present form the draft policy paper is not appropriate public policy advice to Government.

Accordingly, the Institute awaits comment on this submission, and expects to be involved in extensive consultation.

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September 1993