DIAGNOSING ALCOHOLISM IN HIGH-RISK DRINKING DRIVERS: COMPARING DIFFERENT DIAGNOSTIC PROCEDURES WITH ESTIMATED PREVALENCE OF HAZARDOUS ALCOHOL USE

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Abstract — In several European countries, drivers under influence (DUI), suspected of an alcohol use disorder (AUD, 'alcoholism') are referred for diagnostic examination. The accuracy of diagnostic procedures used in diagnosing AUD in the DUI population is unknown. The aim of this study was to compare three prevalence estimates of AUD based on a structured clinical interview (SCID), a restrictive diagnostic procedure (RDP) and usual clinical diagnostic procedure (CDP), with a prevalence estimate based on sensitivity and specificity data of biological markers of excessive use of alcohol in non-judicial samples. The latter unbiased estimate provides an external yardstick against which the biased patient-based prevalence estimates in this special sample can be evaluated. The unbiased estimate derived from sensitivity and specificity data resulted in a prevalence estimate of excessive use of alcohol between 74 and 82%, which is much higher than the three diagnostic procedures. SCID identified maximally 5% of alcoholics found with the unbiased estimate. RDP identified ≥31% of the unbiased estimate, while CDP identified ≥60% of the unbiased estimate. The high chance of false positive diagnosis, however, makes CDP unacceptable in the legal context of AUD diagnosis in DUI populations.

INTRODUCTION

Alcohol use disorder (AUD, 'alcoholism') increases the risk of involvement in road traffic accidents (Waller and Turkel, 1966; Dunbar *et al*., 1985; Papoz *et al*., 1986; Öström and Erkisson, 1993). The collision rates of alcoholics are twice as much as those of non-alcoholic drivers (Vingilis, 1983). These findings have engendered traffic law regulations in several European countries. The regulations stipulate that drivers under influence (DUI), suspected of being alcoholics, need to submit to a medical examination, in order to refute or sub-stantiate that suspicion (Nickel *et al*., 1995). According to Dutch regulations on driving ability (1994), different groups of DUIs are examined (see the Subjects and methods section). Offenders are informed that they will lose their driving licences in cases of non-cooperation with the examination.

Diagnostic procedures in this context are part of an administrative legal procedure to evaluate whether the subject has the right to have a driving licence. Under Dutch law, it is demanded that the subject has refrained from alcohol misuse for the last 12 months. In cases where alcoholism is diagnosed, the licence is withdrawn.

The legal context causes two problems in identifying alcoholics. The first problem is the understandably low validity of self-reporting of alcohol problems in DUI subjects (Mischke and Venneri, 1987). Secondly, in many instances, a diagnosis of alcoholism has to be defended in legal procedures. Diagnoses, based on clinical judgement and data with an unreliable correlation with alcoholism, are increasingly challenged in court with questions about the chance of a false positive diagnosis. The accuracy of diagnostic procedures used in diagnosing alcoholism in DUIs is unknown and has, to the best of our knowledge, never been investigated before.

Research suggests a considerable prevalence of AUD in DUI populations. In a review of prevalence reports up to 1986, Vingilis (1989) estimated the prevalence to be between 25 and 50%, depending on the sampling of the population and the criteria used for alcoholism. More recent studies, using DSM-III criteria (American Psychiatric Association, 1980) and biochemical tests show the same prevalence range (Table 1). However, there is reason for scepticism about the validity of these prevalence values. The number of cases with elevated biochemical markers cannot be equated with true cases of alcoholism, as was done in most of the reviewed studies, mainly because elevated biochemical markers are not in a strict sense markers of alcoholism, but of hazardous use of alcohol. More importantly, research indicates that the sensitivity of biochemical markers drops dramatically in young alcoholics, and also in drinkers with less severe alcoholism (Nystrom *et al*., 1992; Allen *et al*., 1994; Litten *et al*., 1995; Huseby *et al*., 1997*a*; Salaspuro, 1999). As young drivers represent a relatively high proportion of DUI populations, and severe alcoholics represent only a small minority, the reported prevalence values can be considered as conservative (Vingilis, 1989; Hasin *et al*., 2000).

However, the number of cases with elevated biochemical markers can be used to obtain a better estimate of prevalence, if one takes into account sensitivity and specificity data of biochemical markers of alcoholism in non-judicial samples. With a formula derived from Bayes' theorem, one can calculate the prevalence of hazardous use in a population by incorporating test results with knowledge of the sensitivity and specificity. This population-based method can be used as an external criterion for the accuracy of different diagnostic procedures.

In evaluating diagnostic procedures, one must consider the differences between diagnosing alcoholism in health-care settings and in legal settings. In health care, the main diagnostic aim is to enhance health. Therefore it is important to identify all alcoholic patients. In order to minimize the risk

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Table 1. Summary of investigations on prevalence of alcoholism in drivers under influence (DUI) populations using DSM-III and biochemical tests

Author(s)	No.	Selection	Criterion: alcohol use disorder	Prevalence (%)	Correction for test parameters
Scoles *et al*. (1986)	500	DUI: population prior to trial	DSM III alcoholism classification	27.4	
Miller *et al*. (1986)	461	Convicted DUIs; 25% involved in traffic accident	DSM III alcoholism classification	54	
Iffland and Grassnack (1995)	534	Arrested DUI with BAC >0.8% (>80 mg/dl)	CDT >20 U/l	54.5	No
Dunbar *et al*. (1985)	58	Male and female DUIs >30 years old and involved in accident	GGT >50 U/l	48	No
Dunbar *et al*. (1985)	140	Male and female DUIs >30 years old	GGT >50 U/l	24.3	No
Papoz *et al*. (1986)	3427	Male accident victims presented at emergency ward	GGT >40 U/l	30	No
Papoz *et al*. (1986)	3427	Male accident victims presented at emergency ward of Hospital	Combination of abnormal values of GGT and MCV corresponding to values of 90% of control population that used 80 g pure alcohol daily	27	Yes
Pikkarainen and Pentilla (1989)	176	Apprehension at road block DUIs with BAC >0.5‰ (>50 mg/dl)	GGT >50 U/l (method not described)	25	No
Pikkarainen and Pentilla (1989)	183	Apprehension not at road block, on suspicion for alcohol use, DUIs and BAC >0.5‰ (>50 mg/dl)	GGT >50 U/l (method not described)	29	No
Pikkarainen and Pentilla (1989)	176	Apprehension road block. DUIs with BAC >0.5‰ (>50 mg/dl)	Combination of DUI recidivism and GGT >50 U/l (method not described)	20.5	No
Michiels and La Harpe (1996)	877	Male and female DUIs	GGT >56 U/l in males GGT >42 U/l in females (method not described). Combination of elevated GGT, recidivism DUI and BAC >2.0% (200 mg/dl)	32 16 14	Yes
Ruud *et al*. (1993)	150	Males convicted for DUI	CDT >74 mg/l GGT >50 U/l	35 23	No
Gjerde *et al*. (1986)	269	Male DUIs. 61% <30 years old	GGT >50 U/l in males	21	
Gjerde and Mørland (1987)	50		CDT >74 mg/l	60	No
Jaster and Wegener (1993)	110	Male DUIs	Combination of two abnormal values: GGT >58 mmol/l; MCV >96 fl; CDT index >5%	34	Yes
Lutz and Bausach (1992)	219	Male and female DUIs	GGT >28 U/l in males GGT >18 U/l in women	20.5	No
Morgan and Major (1996)	93	Male DUIs with BAC ≥200 mg/dl, or repeated conviction in last 10 years, or failure to provide specimen for analysis	CDT >20 GGT abnormal (method not described)	28 21.5	No

DSM-III (American Psychiatric Association, 1980); CDT, carbohydrate-deficient transferrin; GGT, gamma-glutamyltransferase; MCV, mean corpuscular volume; BAC, blood-alcohol concentration.

of missed diagnoses, a high sensitivity of diagnostic procedures and tests is important. Usual clinical diagnostic procedures (CDPs) in health care depend on clinical judgement which incorporates all available historical, clinical and laboratory data. In the legal setting of medical examination in a DUI population, the aim is not to enhance health, but to enhance traffic safety. Because diagnosis may be challenged in court, diagnosis is restricted to sure cases. In order to minimize the risk of false positive diagnoses, a high specificity of the diagnostic procedure is important. Therefore more restrictive diagnostic procedures (RDP) are used. Ideally, legal diagnostics must rely on objective, reliable and specific data, such as recent history of alcohol problems, physical signs of alcoholism or specific biochemical tests of hazardous alcohol use. In legal settings, high specificity of diagnostic tests is more important than high sensitivity, because incorrect diagnoses have unacceptable legal consequences.

Understanding the legal dilemma is essential in choosing between the different diagnostic procedures. The dilemma is to find a balance between two opposite aims. On the one side, the requirement is to enhance traffic safety (for the public) — each missed diagnosis endangers traffic safety. On the other side, the requirement is to protect the rights of the individual — each incorrect diagnosis may have unacceptable consequences (for the individual), such as losing employment after being disqualified from driving.

In the present study, three prevalence estimations, obtained with different diagnostic procedures, were compared with each other and with an unbiased prevalence estimate based on sensitivity and specificity data of biological markers of alcoholism in non-judicial samples; i.e. compared to the estimate of hazardous alcohol use which would be obtained by applying population-based findings to the blood test marker results obtained in the examination.

SUBJECTS AND METHODS

Subjects

The study population consisted of 241 consecutive male DUIs who were referred for medical examination between September 1996 and May 1998 after driving under the influence of alcohol. Of these, 29 were excluded because of incomplete clinical or blood test data, leaving a study population of 212.

In accordance with Dutch traffic regulations the following groups were included for referral and examination. (1) DUIs with at least one arrest with a blood-alcohol concentration (BAC) $\geq 2.1\text{‰}$ (≥ 210 mg/dl) or three DUI arrests with any BAC $> 0.5\text{‰}$ (≥ 50 mg/dl) within 5 years, or refusal to co-operate with breath analysis (examination group). This group is referred for examination by the Dutch Traffic Test Organization, Disqualification Division, which covers the costs. Some basic information on the characteristics of drivers processed under these regulations were obtained from the Dutch Traffic Test Organization, which supplied data on all DUIs examined in The Netherlands in 1997. (2) DUIs who apply for re-granting of the driving licence after previous DUI, medical examination and loss of permanent driving licence for 12 months because of diagnosis of alcoholism (re-examination group). In this group almost all individuals are self-referred, applying for re-licensing, and have to pay for the examination.

Standardized clinical data collection

All DUIs were examined and diagnosed by the same physician. The examination was recorded in a standardized clinical report, which was part of a legal procedure on behalf of the Dutch Traffic Test Organization. The clinical report of each subject consisted of extensive history taking, instruments to assess AUD, namely Structured Clinical Interview (SCID) and the CAGE questionnaire, physical examination, biochemical measurements and a conclusive clinical judgement as to whether it was probable the subject had AUD in the last 3 or 12 months. History taking was focused on clinical symptoms of alcoholism and on possible and probable non-alcoholic causes for elevated biochemical markers. The latter included questions about current and past illness, specifically diabetes, liver diseases, blood transfusions and intravenous drug use [because of the possibility of hepatitis C which can also affect carbohydrate-deficient transferrin (CDT)] (Perret *et al.*, 1997), anaemia, and drugs that could affect biochemical markers (anti-epileptics, folate antagonists, anti-AIDS medication, phenothiazines, some diuretics and thyrostatics).

Alcoholism or AUD is defined as either alcohol abuse or alcohol dependence according to DSM-IV (American Psychiatric Association, 1994).

Blood tests

Venous blood samples for determination of haemoglobin (Hb), haematocrit (Ht), red blood cell count (E), mean cell volume (MCV), carbohydrate-deficient transferrin (CDT), gamma-glutamyltransferase (GGT), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) were taken. Serum samples for CDT were frozen within 4 h after collection and stored at $-20°C$ until use. CDT was analysed in duplicate, using a commercial kit, CDTect, from Pharmacia and Upjohn, Uppsala, Sweden. Measurement of serum GGT, ALT and AST was executed within 4 h with Vitros (Ortho Clinical Diagnostics, Rochester, NJ, USA) at $37°C$ and re-coded for the value at $30°C$. Hb, Ht, E, MCV and samples were kept at room temperature and analysed within 4 h with a Technicon H2 analyser (Bayer Diagnostics, Terrytown, WA, USA). The upper reference limits were: CDTect ≥ 20 U/l, GGT ≥ 40 U/l, ALT ≥ 34 U/l, AST ≥ 33 U/l, MCV ≥ 100 fl.

Diagnostic procedures

For reasons of comparability with the population-based method, as the diagnostic window of biochemical markers does not exceed 3 months, only current AUD diagnosis (within the last 3 months) was used in the different diagnostic procedures.

Data from clinical reports of every subject were processed in three diagnostic procedures: SCID, RDP and CDP. The three diagnostic procedures are not independent; SCID is incorporated into the RDP and both SCID and RDP are incorporated into CDP. The diagnostic procedures are described below in detail. Essentially SCID identifies alcoholics who are willing to report alcohol problems, RDP identifies those positive with SCID and with elevated biochemical markers which can be seen as proof of current hazardous drinking, whereas CDP identifies those positive on SCID and RDP and subjects with more 'soft signs' of alcoholism. All resulting diagnoses refer to AUD in the 3 months prior to examination.

Diagnostic procedure 1: SCID. Recent alcohol problems were assessed with the Structured Clinical Interview for DSM-IV Axis I disorders, clinician version, module E (alcohol use disorders) over the last 3 months (SCID-CV; First *et al.*, 1997). The SCID-CV is a semi-structured interview for making the major DSM-IV diagnoses (American Psychiatric Association, 1994). It was designed for use in clinical settings as a way of ensuring standardized assessments. A study, using earlier versions of SCID, reported test–retest kappas for current diagnoses of AUD of 0.75 (Williams *et al.*, 1992).

AUD diagnosis was made if the subject scored positively on one of the SCID criteria of alcohol abuse or scored positively on three criteria of alcohol dependence, in the 3 months prior to interview.

Diagnostic procedure 2: RDP. We devised a RDP for detection of alcoholism, with the aim to maximize reliability and specificity of diagnosis. From the standardized recorded history, only data from the SCID interview, the four CAGE questions (Mayfield *et al.*, 1974) and data from history and medication were used to check for possible non-alcohol causes for raised test results. From physical examination, only liver palpation was used. All biochemical measurements were used. The RDP is described in Fig. 1.

AUD diagnosis was only made if SCID was positive, or if a simultaneous combination of elevated biochemical tests, or simultaneous combination of biochemical tests and clinical signs were present. When there was a possible non-alcoholic cause for positive biochemical and clinical signs, AUD diagnosis was not made. When two or more of the enzymes ALT, AST and GGT were simultaneously elevated, no AUD diagnosis was made. In the presence of indication of liver illness, such as liver enlargement, highly elevated ALT and AST, or highly elevated GGT, no AUD diagnosis was made. We introduced this rule, not only because of the possibility of

Positive SCID in last 3 months prior to examination? → NO → Abnormal or marginal biological markers?

YES

YES

One of:
- CDT≥20 + GGT≥40,
- CDT≥20 + ALT≥34
- CDT≥20 + AST≥33
- CDT≥20 + MCV≥97,
- GGT≥40 + MCV≥100,
- CDT≥20 + recent Cage ≥ 2
- CDT≥20 + hepatomegaly
- MCV≥100 + ALT≥34
- MCV ≥100+ AST≥33

One of:
- CDT≥20 + GGT≥36
- GGT≥40 + CDT≥18
- MCV≥97 + GGT≥40
- CDT≥20 + ALT≥31
- CDT≥20 + AST≥30
- MCV≥100 + CDT≥18
- CDT≥50

One of:
- CDT≥18
- GGT≥36
- ALT≥31
- AST≥30
- MCV≥97

Possible* confounding effect of illness or drug?

Possible* confounding effect of illness or drug?

Probable** confounding effect of illness or drug?

NO

NO   YES   NO   YES   NO   YES

Probable** confounding effect of illness or drug?

NO   YES

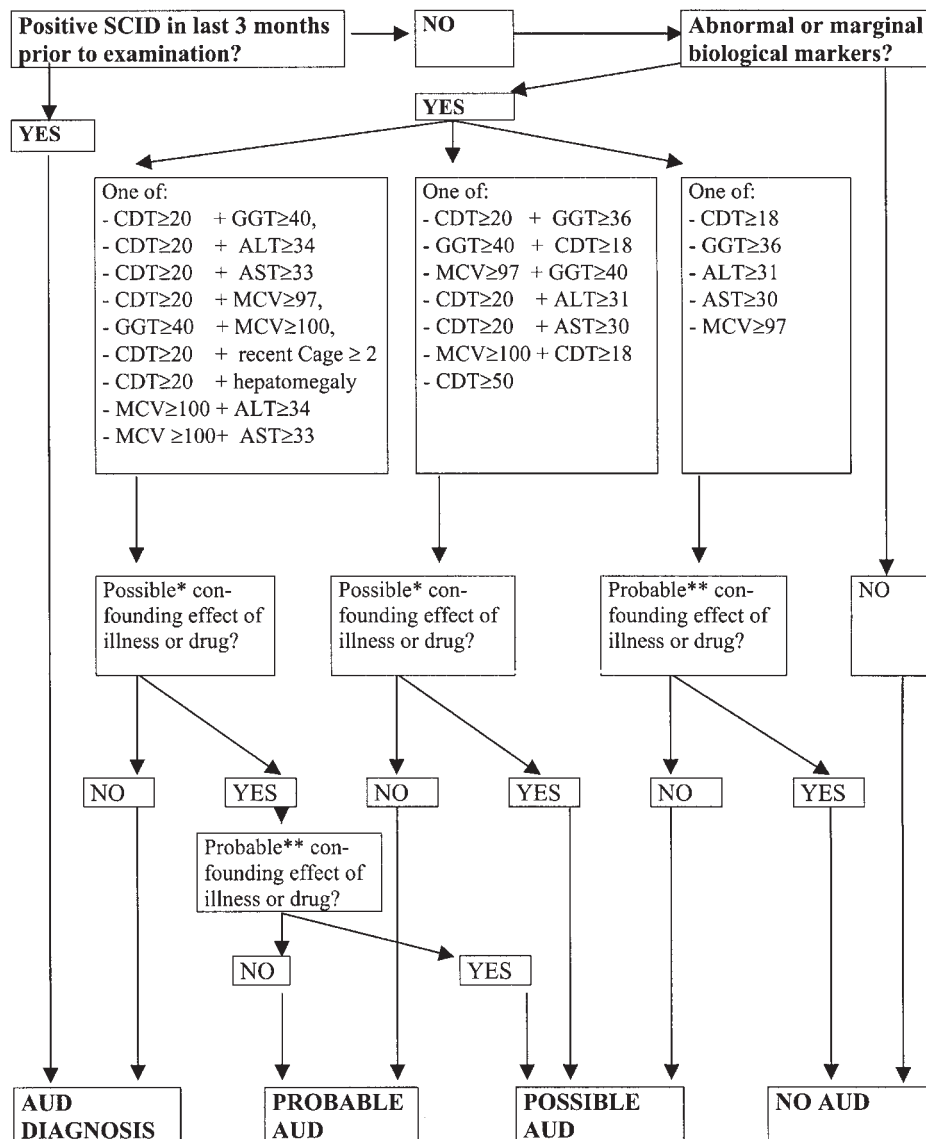AUD DIAGNOSIS   PROBABLE AUD   POSSIBLE AUD   NO AUD

Fig. 1. Restrictive diagnostic procedure: three alternative criteria of increasing strictness for testing excessive use of alcohol.
*Possible confounding effect when the chance that abnormal biochemical or clinical signs are caused by non-alcoholic illness or drug is estimated to be >5%. **Probable confounding effect when the chance that abnormal biochemical or clinical signs are caused by non-alcoholic illness or drug is estimated to be >50%. (These criteria derive from the albeit sparse published data on the rates of elevated marker tests in various non-alcoholic conditions, and the authors' 'intelligent guesswork'.) SCID, structured clinical interview; CDT, carbohydrate-deficient transferrin; GGT, gamma-glutamyltransferase; MCV, mean corpuscular volume; ALT, alanine aminotransferase; AST, aspartate aminotransferase; AUD, alcohol use disorder.

non-alcoholic liver disease, but also because some of the alcoholics who abstain from alcohol for longer than 6 months still have elevated ALT, AST or GGT, probably because of steatosis or cirrhosis. In the case of a moderately elevated GGT, ALT or AST, there is a difficulty whether to interpret a simultaneous MCV elevation as an independent test in diagnosing AUD, as this elevation may be possibly caused by the same liver illness.

In order to diminish the small chance (in this population) of incorrectly diagnosing subjects with non-alcoholic liver disease, without increasing the much greater chance (in this population) of missing diagnosis in non-abstinent subjects

with alcoholic liver disease, we used a higher cut-off value of MCV in the combinations of elevated MCV, with elevated ALT, AST or GGT, as an extra safeguard against incorrect diagnosis.

The algorithm for RDP was made before analysis. Two items ('subject states that he feels no effect of drinking ≥4 AU' and blood pressure ≥160/95) were deleted post-hoc, as these items did not provide any additional differentiating value.

The choice of the different combinations of elevated bio-chemical measurements in RDP was motivated by our aim to achieve a specificity of ~95%. In non-clinical settings, reported specificities are: GGT 80–90%, ALT >80%, AST >90%,

elevated MCV in men >90% and elevated CDT >80% (Sillanaukee *et al.*, 1992; Conigrave *et al.*, 1995; Salaspuro, 1999). As hazardous use of alcohol elevates CDT, MCV and the enzymes GGT, ALT and AST through partly independent biological pathways, these markers can be considered as partly independent tests (Bean and Daniel, 1996). Demanding that a pair of diagnostic tests are simultaneously elevated before making a positive diagnosis of alcoholism maximizes specificity and minimizes false positive labelling of innocent patients, at the price of many missed diagnoses (Sackett *et al.*, 1991). If CDT, GGT and MCV are independent tests, simultaneous elevation of CDT and GGT, or CDT and MCV, or MCV and GGT will result in specificity values between 96 and 99%. This assumption has been partly confirmed in a study that measured specificity of simultaneously elevated CDT and GGT in non-alcoholic controls (van Pelt, 1997).

Diagnostic procedure 3: CDP. In this diagnostic procedure, a diagnosis was reached through clinical judgement after evaluation of all available data, according to usual clinical practice. Besides biochemical measurements, historical data, clinical signs, and instruments to assess alcohol problems were used.

Histories included time and circumstances of arrest. A police report of BAC, data of earlier DUI and reports on earlier medical examinations after DUI were available. No information from the applicants' general practitioner was requested (which is the procedure in the UK DUI system, see Morgan and Major, 1996).

Recent alcohol intake was assessed by means of a structured interview. This included questions about the exact amount of alcohol units (AU) in the week prior to the examination, an estimate of the average AU per week during the last year, and questions about changes in quantity and frequency of drinking in the last year.

Hazardous drinking is defined as the level of persistent alcohol consumption being likely to result in adverse health effects: >280 g ethanol/week (Saunders and Lee, 2000). As 1 AU is defined as a standard drink of ~10 g alcohol, hazardous use signifies an average of more than 28 AU weekly.

Lifetime and current alcohol problems were assessed using the CAGE questions, SCID, and questions about any past treatment for alcohol problems. A subject was considered to have had a lifetime alcohol problem if CAGE score of ≥2 was obtained, or if a subject was ever treated for alcohol problems, or received a SCID diagnosis in the 12 months prior to the interview.

As in RDP, history also included questions about different diseases and drugs, to control for possible confounders in regard to non-alcoholic causes for elevated biochemical markers or liver enlargement.

Physical examination included breath smelling of alcohol during examination (but no alcohol breath test), blood pressure, liver palpation and observation of skin abnormalities indicative of liver dysfunction and neurological dysfunction indicative of polyneuropathy or withdrawal symptoms.

Diagnosis of recent AUD in the CDP procedure was based on clinical reasoning. All data and clinical signs were assessed as either diminishing the chance of recent AUD, increasing the chance of recent AUD, or confirming AUD diagnosis. A positive diagnosis of recent AUD was made if the above-described SCID and RDP procedures resulted in an AUD diagnosis, or if several AUD chance-increasing data were present without the presence of confounding effects of illness or drugs.

Population-based prevalence estimate of hazardous use

Studies have shown that sensitivity and specificity of markers of hazardous alcohol use depend on the distribution of severe and mild cases of alcoholism in the studied cohort. A high ratio of severe/mild cases heightens, while a low ratio lowers, sensitivity. Because we assumed that our population consists of a high-risk population of hazardous users, alcoholics and social drinkers without AUD, we used sensitivity and specificity values found in studies with two high-risk populations (Sillanaukee *et al.*, 1993; Huseby *et al.*, 1997*b*). Sillanaukee *et al.* (1993) compared hazardous drinkers, with some signs of AUD, to social drinkers and found a sensitivity of 57% and a specificity of 79%. Huseby *et al.* (1997*b*) compared alcohol-dependent patients to non-dependent patients from a population of men admitted to a surgical ward and found a sensitivity of 55% and a specificity of 85%. Sensitivity and specificity values refer to the relation of AUD and elevated CDT or GGT.

The estimated prevalence of AUD was computed with the following formula:

$$P = [T - (1 - Sp)]/(S + Sp - 1) \text{ (Poole } et\ al.\text{, 1996)},$$

where: P = prevalence; T = proportion of elevated tests (CDT or GGT) = (true positives + false positives)/all tests; S = sensitivity = number of true positives/(number of true-positives + number of false-negatives); Sp = specificity = number of true negatives/ (number of true negatives + number of false positives). Below: PPV = positive predictive value = number of true positives/ (number of true positives + number of false positives); NPV = negative predictive value = number of true negatives/ (number of true negatives + number of false negatives).

Statistical analysis

Statistics Package for Social Sciences (SPSS for Windows 9.0, 1999) was used for computation of frequencies. Comparison of groups was performed with the *t*-test. Comparisons of multiple groups were conducted with analysis of variance.

RESULTS

Sample characteristics

The sample characteristics of the examination group were not significantly different from all DUIs examined in The Netherlands in 1997, when compared for age, average BAC and mean number of DUI arrests. The mean age of our cohort was 42.1 years and 31% of the DUIs were younger than 35 years (Table 2).

The re-examination group, that consisting of subjects who applied for regranting the driving licence, reported much less alcohol use (5.5 AU/week) than the examination group (10.4 AU/week). In comparison, the average self-reported alcohol intake in the Dutch male population is 21 AU per week (de Zwart, 1998). Only seven subjects (7.5%) from the examination group and four subjects (3.4%) from the re-examination group reported drinking more than 28 AU average per week in the 3 months prior to the interview.

Table 2. Sample characteristics of 212 drivers under influence (DUI) subjects and of all 'first examined DUIs' in 1997 in The Netherlands

	Study sample			First examination in The Netherlands in 1997 ($n = 2045$)	Compared to study: first examination sample (P)
	First examination ($n = 93$)	Re-examination ($n = 119$)	P		
Age (years)	40.17 ± 11.8	44 ± 11.2	0.025*	40.29 ± 10.3	0.92
BAC at arrest (‰)	1.98 ± 0.56	1.90 ± 0.61	0.428	2.12 ± 0.58	0.064
No. of DUI arrests in last 5 years	1.91 ± 1.23	1.41 ± 1.20	0.003*	1.93 ± 1.19	0.459
Months between last DUI and study medical examination	6.2 ± 3.2	47 ± 35	0.000*		
Reported AU/week	10.4 ± 12.9	5.5 ± 8.0	0.002*		

Values are mean ± SD.
*$P < 0.05$, independent sample t-test.
BAC, blood-alcohol concentration; AU, alcohol units.

In all, 107 out of 212 DUIs reported lifetime alcohol problems according to our definition (if the CAGE score is ≥2, or if a subject was ever treated for alcohol problems, or received a SCID diagnosis in the 12 months prior to the interview). As expected, the re-examination group reported more lifetime alcohol problems (61.3%) than the examination group (36.6%). The last percentage is probably the result of under-reporting. According to an epidemiological study performed in 1996, the 1-month, 12-month and lifetime prevalence of AUD in the Dutch male population was respectively 8.5, 13.4 and 28.3% (Bijl *et al.*, 1998).

Prevalence according to different diagnostic procedures

SCID. Applying SCID over the last 3 months as a diagnostic procedure identified seven DUIs with AUD in the examination group and only one in the re-examination group. According to SCID, the estimated prevalence of AUD over the last 3 months in the examination group was 7.5%, but only 0.8% in the re-examination group (Table 3). Applying SCID over the last 12 months identified 21 DUIs with AUD in the examination group (22.6%) and four in the re-examination group (3.4%).

RDP. The RDP resulted in an AUD diagnosis in 32 DUIs from the examination group (prevalence according to RDP 34.4%) and 18 from the re-examination group (prevalence of AUD in the re-examination group 15.1%).

CDP. Using all data, this resulted in an AUD diagnosis in 54 DUIs in the examination group (prevalence according to CDP 58.1%) and 43 DUIs in the re-examination group (prevalence according to CDP 36.1%).

Population-based prevalence computation

The total number of subjects with elevated CDT or GGT was 101 (51 from the examination group and 50 from the re-examination group). The proportion of DUIs with elevated biochemical markers was 101/212. Using the sensitivity and specificity values found by Sillanaukee *et al.* (1993) resulted in an estimated prevalence of AUD for all DUIs in our study of 74%. Using the sensitivity and specificity values found by Huseby *et al.* (1997a) resulted in an estimated prevalence of 82%.

DISCUSSION

The three diagnostic procedures used in the present study are not independent. SCID is incorporated in RDP and both SCID and RDP are incorporated in CDP. Not surprisingly, additional data resulted in higher AUD prevalence values: 3.8% with SCID only, 23.5% with the RDP and 45.8% with clinical judgement. But how can we explain the great difference between prevalence found with diagnostic procedures and the prevalence found with the population-based method?

On the one hand, one has to consider the possibility that the low sensitivity of biochemical markers, used in the population-based method, inflates the estimated prevalence of AUD beyond results of earlier research and beyond face validity. Another possible explanation is that the estimated prevalence found with the population-based method, either 82 or 74%, can be considered as maximal prevalence only. As 'hazardous drinking' encompasses a larger group than the group with AUD, the criterion can be only used as maximal reference level. On the other hand, one has to consider the possibility that the diagnostic tools to detect alcoholism in DUIs result in considerable under-diagnosing. SCID identifies maximally 5% of all AUD found with the unbiased estimate. This performance was not unanticipated; SCID identifies only those alcoholics who are aware of, and willing to be open about, their problems. For obvious reasons, most DUIs will not be open about their alcohol consumption (which was reported as three times lower than the mean in the Dutch population) or about the frequency of experiencing alcohol problems (which was also reported as lower than in the Dutch population).

RDP identified six times as many as the SCID procedure only, and at least 28% and maximally 31% of the unbiased AUD estimate. This is a significant gain, compared to SCID. At the same time, it is evident that the sensitivity of the RDP is low. This result is also according to expectation: RDP will result in under-diagnosis, because physical signs of alcoholism are late symptoms of alcoholic disease, because ~5–20% of alcohol-dependent patients and 40–60% of alcohol abusers show no elevations of biochemical tests (Sillanaukee *et al.*, 1993; Litten *et al.*, 1995; Hillman *et al.*, 1998) and because 31% of our population consisted of subjects younger than

Table 3. Estimate of prevalence of alcohol use disorders (AUD) according to different diagnostic procedures and compared to population-based estimates of rate of excessive use of alcohol

Group and parameter (n = 212)	SCID 3 months		Restrictive diagnostic procedure				Clinical diagnostic procedure		Population-based method ('unbiased estimate')
	+ (n = 8)	− (n = 204)	AUD (n = 50)	Probable AUD (n = 21)	Possible AUD (n = 59)	No AUD (n = 82)	AUD (n = 97)	No AUD (n = 115)	Elevated CDT or GGT, n = 101 (51 examination group; 50 re-examination group)
Total study population (n = 212)									
Prevalence AUD (%)	3.8		23.6				45.8		82[a] or 74[b]
Lifetime alcohol problems (n = 107)	7	100	31 (62.0)	17 (81.0)	25 (42.4)	34** (41.5)	60	47*	
Mean ± SD AU/week	25.6 ± 29.6	7.1* ± 8.7	15.5 ± 15.4	8.6 ± 8.0	6.7 ± 8.2	3.5** ± 6.1	11.9 ± 13	4.1 ± 6.4	7.7 ± 10.7
Examination group (n = 93)	7 (7.5)	86* (92.4)	32 (34.4)	11 (11.3)	19 (20.4)	31 (33.3)	54 (58.1)	39 (41.9)	
Re-examination group (n = 119)	1 (0.8)	118* (99.2)	18 (15.1)	10 (8.4)	40 (33.6)	51** (42.9)	43 (36.1)	76* (63.8)	

Total study population, $n = 212$. Values in parentheses are percentages.
*$P < 0.05$ independent sample t-test; **$P < 0.05$ one-way analysis of variance.
[a]After Huseby (1997b); [b]after Sillanaukee et al. (1983).

35 years. In young subjects, biochemical markers have a low sensitivity for detection of alcoholism. Another reason for under-diagnosis by RDP is that any possible non-alcoholic cause had also automatically led to exclusion of the diagnosis.

CDP identified at least 56% of the unbiased AUD estimate and up to 60% of the examination group. Even if one assumes that the prevalence of AUD found with CDP is rather high, one has to consider that the prevalence values in this study refer to prevalence of AUD found several months after the DUI arrest. It seems reasonable to assume that prevalence of AUD at the time of arrest would be much higher.

The above-mentioned prevalence is dependent on the administrative selection of DUIs for examination, which varies in different countries. The issue here is to provide the clinician, working within a legal situation, with a method to calculate the PPV and NPV for different diagnostic procedures. The diagnostic gain of CDP above RDP has significant legal disadvantages that can be illustrated by the consequences for the PPV of this procedure. If we use this procedure in a population with a 40% prevalence of AUD, under the optimistic assumption that CDP has a specificity of 80% and a sensitivity between 60 and 95%, the PPV of CDP will vary between 66 and 75%. This may be quite acceptable in healthcare settings, but is evidently not acceptable in legal settings. The high chance of false positive diagnosis makes CDP unacceptable in the legal context of AUD diagnosis in DUI populations. Until better markers are available, we advise physicians who participate in diagnosing AUD in DUI populations to use RDP enhanced with secondary data such as circumstances of arrest.

It remains to be researched if RDP (enhanced or not) has a high enough PPV and an acceptable NPV. However, it is too optimistic to hope that such research will be able to replace clinical reasoning completely (Gilg, 1999). As different subgroups of DUIs have different *a priori* prevalences (Table 3), and test parameters of biochemical markers are dependent on age and gender, different norms must be used in diagnostic procedures. Even if precise knowledge of the PPV of different diagnostic procedures in different groups becomes available, one has still to answer a social, as well as the legal, question: how sure one has to be of diagnosis in diagnosing alcoholism in DUI populations?

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