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Measurement of lipoprotein(a) via a novel point of care approach with comparison to established laboratory assays

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Abstract

Objectives: Lipoprotein(a) is an atherogenic particle causative of atherosclerotic cardiovascular disease. Novel treatments have been developed that lower lipoprotein(a) to unprecedented levels with cardiovascular outcomes trials ongoing. Many guidelines recommend testing once in the lifetime of everyone, but testing rates remain low. In this study we compare a lipoprotein(a) point of care testing device to laboratory analysers and assess its performance.

Methods: Lipoprotein(a) concentrations on residual patient samples measured on the Randox and Roche assays were compared to a novel point of care device, iProtin. Furthermore, assessment of iProtin performance characteristics were performed, including intra- and inter-assay coefficient of variation and dilutional studies.

Results: Lipoprotein(a) concentrations measured on the Randox and Roche assays showed strong correlation with iProtin. Regression analysis using Passing-Bablock showed the best fits for iProtin based on 58 serum samples were: 1.15 × Randox + 7.28 nmol/L and 1.02 × Roche + 17.54 nmol/L. The R^2 values for Randox/iProtin and Roche/iProtin were 0.906 and 0.912 respectively. Correlation between Roche and Randox showed Roche=1.15 × Randox − 13.33 nmol/L with an R^2 value of 0.973. Inter-assay coefficient of variation of the iProtin device showed a day-to-day imprecision over 5 days of 15.5 % (low concentration quality control) and 6.2 % (high concentration quality control). Within day imprecision was 13.2 % (lower concentration patient sample) and 14.3 % (higher concentration patient sample).

Conclusions: Point of care testing could be a complimentary option to laboratory testing of lipoprotein(a), especially in remote areas. It may help (re-)stratify cardiovascular risk and help tailor treatment decisions.

Keywords: iProtin; lipoprotein(a); point of care; Roche; Randox

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Introduction

Lipoprotein(a) (Lp(a)) is a highly atherogenic lipoprotein causative of atherosclerotic cardiovascular disease (ASCVD) and calcific aortic stenosis [1, 2]. Australian guidelines classify Lp(a) concentrations>200 nmol/L as high risk for ASCVD and >400 nmol/L as very high risk for ASCVD [3]. As such, accurate measurement of Lp(a) is clinically important as the risk of ASCVD and associated treatment recommendations change with rising Lp(a) concentrations. While most lipid lowering therapies do not have a significant effect on Lp(a) concentrations, novel therapies have been developed that can lower Lp(a) to unprecedented levels and cardiovascular outcome trials are underway investigating if sustained lowering of Lp(a) translates into cardiovascular benefit [4]. This makes accurate laboratory measurements even more important.

Lp(a) can be measured via various methods, including enzyme-linked immunosorbent assays, immunonephelometry, immunoturbidimetry, fluorescent immunoassays and mass spectrometry-based reference methods [5, 6]. However, laboratory measurement of Lp(a) can be challenging due to the unique structure of this molecule. Lp(a) has a structure similar to low-density lipoprotein (LDL) but it has an

additional apolipoprotein(a) attached via disulfide bonds. The apolipoprotein(a) consists of repetitive domains, called kringles (K). Apolipoprotein(a) contains one KV repeat, a plasminogen-like domain and ten KIV repeats, known as KIV_1 to KIV_{10}. KIV_2 can be present in varying repeat sequences. Depending on the number of KIV_2 repeats, the molecular weight of Lp(a) can vary. Furthermore, the number of KIV_2 repeats (and resultant molecular weight of Lp(a)) can differ significantly both within an individual and even more so between individuals [7, 8]. The different size and weight of Lp(a) molecules makes measurement of Lp(a) challenging analytically. It is recommended that Lp(a) should be measured with an assay that is isoform independent (i.e. not influenced by the various KIV_2 repeats), traceable to an internationally accepted calibrator and reported in molar units [9–11]. However, such ideal assays do not exist in routine laboratories. Furthermore, the currently used International Federation of Clinical Chemistry and Laboratory Medicine (IFCC) immunoassay-based reference measurement system for apolipoprotein(a) was developed more than 20 years ago [12]. Promisingly, a mass spectrometry based reference measurement procedure was developed for apolipoprotein(a) measurement more recently [6].

Previous studies comparing Lp(a) measurements on different assays have shown significant variability and current Lp(a) assays are not harmonised [13, 14]. A recent interlaboratory comparison study with eight participating laboratories demonstrated that interassay variability persists, particularly at concentrations used to guide clinical decision making [15].

In addition to analytical difficulties, uptake of Lp(a) testing remains low despite its confirmed causative role in ASCVD and calcific aortic sclerosis [16, 17]. While some guidelines recommend testing of Lp(a) at least once in the lifetime of each individual [2], Australian guidelines currently only recommend testing of Lp(a) in certain high-risk populations [3]. Low testing rates may be due to a lack of awareness amongst healthcare providers. Another reason could be difficulties in access to locally available and cost-effective/government rebatable Lp(a) testing, especially in the Australian context. Australia is a vast country and access to Lp(a) testing in rural and remote areas can be limited. Point of care testing (POCT) can help overcome the barrier of limited test availability in regional and remote areas and has been shown to be successful and cost-effective in areas such as POCT for troponin and several infectious diseases [18–20].

To the best of our knowledge, no POCT systems for Lp(a) are currently available or have been tested for accuracy in Australia. To bridge this gap, we compare a Lp(a) POCT system to two commercially available Lp(a) assays in Australia.

Materials and methods

Serum samples

We collected residual, adult serum samples from patients attending Sullivan Nicolaides Pathology (Brisbane, Australia), in whom Lp(a) measurements had already been requested for clinical reasons and for whom Lp(a) results were available. We selected samples that cover the entire measuring range of the POCT device. All samples were stored at 4 °C and were less than 7 days old. The study was approved by the Ethics Committee at Sullivan Nicolaides Pathology.

Determination of Lp(a) concentration

We measured Lp(a) on two commercially available Lp(a) assays platforms and a POCT device. Lp(a) patient samples were measured on the Randox assay on the Abbott Alinity C platform (Abbott Laboratories, IL, USA), and the Roche Cobas c 502 (Roche Diagnostics, Basel, Switzerland) platform. For Lp(a) POCT we used the iProtin device by tst biomedical electronics Co., Ltd (Taoyuan City, Taiwan).

For measurement on the Roche Cobas c 502, the Roche Tinaquant R® Generation 2 assay was used. This is a particle enhanced immunoturbidimetric assay using a multipoint calibration design with each calibrator being independent [21]. This molar assay is standardized against the IFCC reference material SRM2B for nmol/L and has a measuring range of 7–240 nmol/L. Samples with a higher concentration are diluted via a rerun function using a 1:3 dilution with 0.9 % normal saline [22]. Measurement on the Abbott Alinity C was performed using the Randox Lp(a) assay (Crumlin, United Kingdom). The Randox assay is calibrated in nmol/L and is also traceable to the IFCC reference material SRM2B. Similar to the Roche assay design, it is an immunoturbidimetric assay using a 5-level calibration. The Randox assay is based on the Denka assay, which is considered to be amongst the assays to be least isoform-sensitive and most reliable in terms of commercially available methods [5]. The quoted measuring range is 5–200 nmol/L [23]. As the manufacturer-recommendeddilution with 0.9 % normal saline for values>200 nmol/L resulted in inadequate results, we developed an in-house dilution method using a commercially available Lp(a) quality control (QC) material in a 1:3 dilution [24]. On the day of analysis of patient samples, both assays were calibrated, and both internal and external QC material were run.

The iProtin Lp(a) POCT device by tst biomedical electronics Co., Ltd (Taoyuan City, Taiwan) is a small, handheld device developed for measuring various lipoproteins, including Lp(a)

(Figure 1). Both whole blood and serum can be used. In our study we used 5 µL of serum obtained from a serum separator tube (SST). The calibration reference material for the iProtin Lp(a) product is the Roche Tina-quant® Generation 2 assay traceable to the IFCC SRM 2B reference standard. The calibration curve is generated using a four-parameter logistic model. Calibration parameters are embedded in a QR code on the cartridge packaging and scanning the QR code automatically completes the calibration process. The linearity range of the POC device is 25–200 nmol/L but measurements can reach up to 350 nmol/L [25]. The assay employs shear horizontal surface acoustic wave (SH-SAW) biosensors that sense changes in mechanical vibrations (such as velocity and amplitude changes) to detect binding events on the surface of a chip. Briefly, an electrical signal applied to interdigital transducers creates a SH-SAW with the help of piezoelectric substrates. When a substrate binds to a sensing layer, it adds mass to the surface of the device altering the wave's properties. The change of the wave's characteristics can be measured and is proportional to the concentration of the substrate measured. Lp(a) in an individual's serum is captured by a pre-immobilized monoclonal mouse anti-Lp(a) antibody pre-fixed on the iProtin biosensor. When Lp(a) particles bind to the antibodies on the biosensor, the velocity of the acoustic waves changes which is detected and measured by the reader [26].

Method comparison

Prior to analysis, all specimens were checked for clots and were then re-assayed using the Randox assay (on the Abbott Alinity) on the day of the experiment. This was followed by measurement on the iProtin device on the same day. Analysis of samples on iProtin was performed using the same cartridge kit lot number for all samples. In the absence of a gold standard method, samples were additionally sent to a partner laboratory for measurement on the Roche Cobas c 502 as soon as analysis on both the Randox assay (on Abbott Alinity) and the iProtin device was complete. Passing-Bablok regression analysis was performed and slope, intercept, correlation coefficient and bias were calculated. Method comparisons were plotted for each individual assay pair. We initially selected samples to test the linearity range of the iProtin device (25–200 nmol/L) and subsequently extended this range to include samples with higher concentrations to test the upper measuring limit of 350 nmol/L of the iProtin device.

Imprecision studies

Intra-assay imprecision of the iProtin POCT was assessed by testing a low and high concentration Lp(a) patient serum sample in six replicates in a single day. Inter-assay imprecision was assessed by testing a low and high concentration sample of a human serum-based Lp(a) QC material in daily use in our laboratory: BioRad Liquicheck Immunology QC. The two concentrations were tested in duplicate over five days. Care was taken to use the same bottle of QC material throughout the duration of this study.

Dilution studies

Four patient serum samples with extreme Lp(a) concentrations were selected and measured before and after a 1:3 dilution using a low Lp(a) patient pool (Lp(a)<5 nmol/L) as a diluent on both the Randox assay and the iProtin POCT device. For the Roche assay, the automatic, onboard 1:3 dilution using 0.9 % normal saline was used.

Data analysis

All data analysis was performed using Microsoft Excel, version 14.0, (Microsoft, Redmond, Washington, USA).

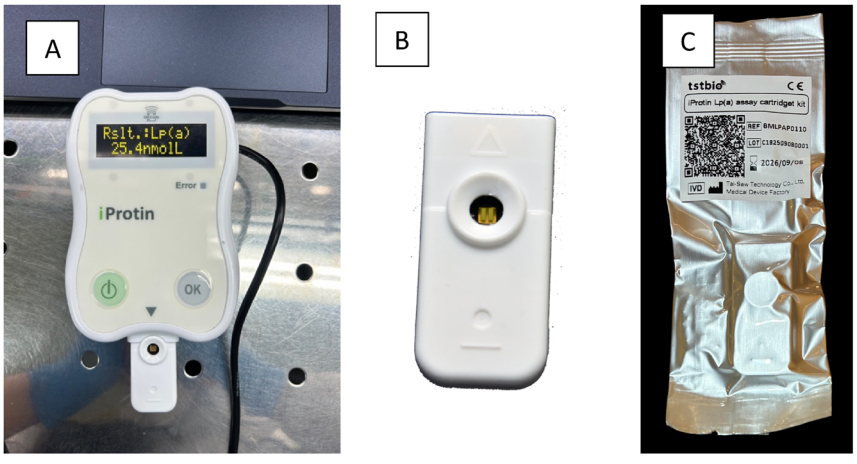

Figure 1: iProtin device in detail. (A) iProtin point of care device displaying lipoprotein(a) concentration in nmol/L. (B) iProtin cartridge. (C) iProtin cartridge package with QR code.

Method comparison was assessed using Passing-Bablok regression analysis, including residual plots and histograms with Analyse-It for Microsoft Excel (version 5.90, Leeds, United Kingdom). Data are presented as mean, standard deviation (SD), and percentage unless otherwise indicated.

Results

Method comparison

A total of 58 samples were analysed on all the three analysers. All assays compared well across the measuring range of the iProtin POCT device. Regression analysis using Passing-Bablock analysis showed the best Passing-Bablok fit for the iProtin POCT device were as follows: 1.15 × Randox + 7.28 nmol/L and 1.02 × Roche Cobas + 17.54 nmol/L. A positive constant and proportional bias was observed for the iProtin assay, more pronounced at lower concentrations. The R^2 values for Randox/iProtin and Roche/iProtin were 0.906 and 0.912 respectively. A total of 65 samples were analysed on both Randox (on Alinity) and Roche with the following regression line and correlation: Roche=1.15 × Randox − 13.33 nmol/L and R^2 value of 0.973. The comparison data is summarized in Figure 2A–F.

Imprecision studies

Table 1 shows the absolute results, means, coefficient of variations (CV) and SD of Lp(a) concentrations obtained from two Lp(a) QC samples measured in duplicate on the iProtin POCT device over five consecutive days. The laboratory target values for the Lp(a) QC were 56 nmol/L and

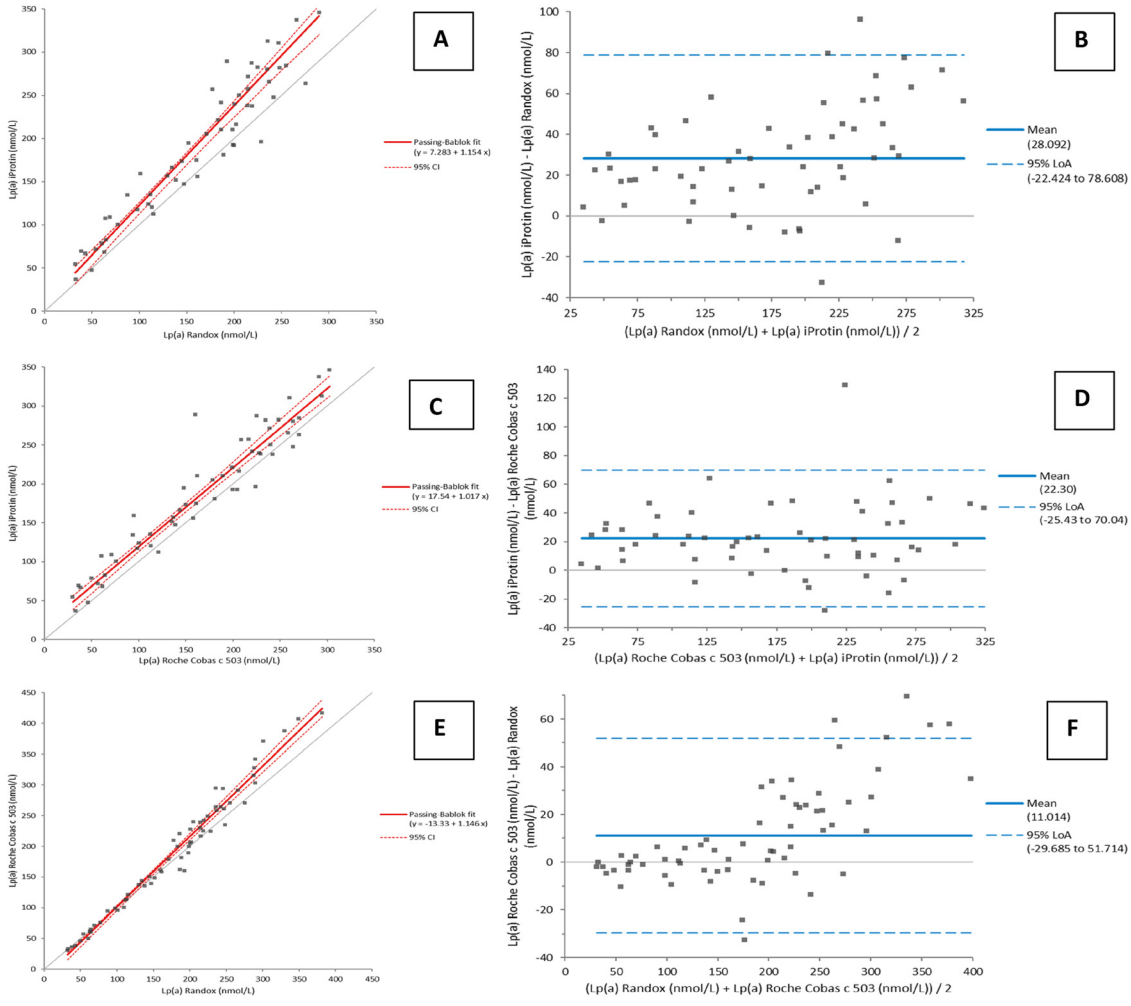

Figure 2: Linearity and Bland Altman difference plots for lipoprotein(a) measurements. (A) Linearity plot Randox (on Alinity) vs. iProtin. (B) Bland-Altman difference plot Randox (on Alinity) vs. iProtin. (C) Linearity plot Roche vs. iProtin. (D) Bland-Altman difference plot Roche vs. iProtin. (E) Linearity plot Randox (on Alinity) vs. Roche. (F) Bland-Altman difference plot Randox (on Alinity) vs. Roche.

Table 1: Inter-assay imprecision using a low and high level QC sample. The mean and SD (in nmol/L) and CV (in %) of duplicate measurements over 5 days are displayed.

Inter-assay imprecision – iProtin (all values in nmol/L)		
Day of analysis	**Low level QC**	**High level QC**
1	61.7	110.9
1	59.2	117.4
2	47.6	119
2	36.5	121.3
3	62.5	136.1
3	57.9	124.4
4	64	123.5
4	60.5	135.2
5	50.6	127.7
5	61.8	123.5
Mean	**56.2**	**123.9**
SD	**8.7**	**7.7**
CV, %	**15.5**	**6.2**

Table 2: Intra-assay imprecision using two different patient samples. The mean and SD (in nmol/L) and CV (in %) of six same day repeated measurements are displayed.

Intra-assay imprecision – iProtin (all values in nmol/L)		
Repeat	**Low level sample**	**High level sample**
1	108.4	180.8
2	115.4	247.2
3	108.5	221.8
4	133.0	200.1
5	144.4	270.2
6	Not available	223.7
Mean	**121.9**	**224**
SD	**16.1**	**32**
CV, %	**13.2**	**14.3**

100 nmol/L as measured on the Randox assay (on Alinity). The day-to-day imprecision was 15.5 % for the lower concentration QC and 6.2 % for the higher concentration QC.

Table 2 shows the intra-assay CV for the iProtin POCT device using a low and high concentration patient sample previously measured on the Randox assay on the Alinity platform (74 and 161 nmol/L) run consecutively for six times on the same day.

Dilutional studies

Three high concentration samples were diluted in a 1:3 manner using a low Lp(a) patient sample (Lp(a)<5 nmol/L as measured on Randox on the Abbott Alinity platform) and subsequently measured on both the Randox (on Alinity) and iProtin assays.

Table 3: Dilutional studies of Lp(a) using three different assays.

High concentration lipoprotein(a) diluted 1:3 on three different assays, nmol/L			
	Sample 1	**Sample 2**	**Sample 3**
Randox (on Alinity)	381.15	451.89	595
Roche	399.7	447.4	613.4
iProtin	365.7	467.4	642.9

The Roche assay uses an automatic, onboard 1:3 dilution with 0.9 % saline. All three assays showed good correlation (Table 3).

Discussion

In this study we compared a novel POCT device for Lp(a) to two established mainstream laboratory assays. Despite an appreciable constant and proportional positive bias of the iProtin assay compared to both Roche and Randox, our results indicated that the POCT device compares well to two other Lp(a) assays within the linearity range of the POCT device (25–200 nmol/L). In particular, at decision points of 75 nmol/L, which is the upper limit of normal of Lp(a) in our laboratory, and 200 nmol/L, which is the threshold for a high-risk result, the three assays compared well. We were not able to assess the 400 nmol/L cut-off, which indicates very high-risk results, as the iProtin POCT device only measures up to 350 nmol/L. The iProtin device was calibrated against the Roche assay (manufacturer internal communication) which was notable in our comparison study that indicated best comparison to the Roche assay. It must be stressed that neither the Roche nor Randox assay are reference methods or a gold standard for measuring Lp(a) – as such the comparison to these two methods is flawed. Recent papers have shown that there is still variability between commercially available Lp(a) assays despite efforts by manufacturers to increase standardisation [11, 13, 15, 27, 28]. There was a previously established ELISA-based reference assay [12], but it is no longer available, and development of a mass spectrometry-based reference measurement procedure has been underway with successful development of an IFCC mass spectrometry-based reference method with values assigned in SI units [6]. Comparison to an Lp(a) reference method would have been ideal, but in the absence of its availability, the use of two commercially available assays calibrated to SRM2B for comparison was the best option available.

The intra- and inter-run CVs were higher in our study compared to the manufacturer's data. The manufacturer's intra-run CV on three whole blood samples tested 20 times in the same assay with an average concentration of 199 nmol/L, 133.5 nmol/L and 43.6 nmol/L yielded CVs of 5.42 , 6.77, and 8.07 % respectively. Inter-group CV tested on 3 whole blood samples tested on 20 consecutive days at concentrations of 158.75, 98.7 and 47 nmol/L resulted in CVs of 6.06 , 6.83, and 9.87 % [25]. We tested inter-assay imprecision over 5 days measuring QC material in duplicate with mean concentrations of 56.2 nmol/L and 123.9 nmol/L. This showed CVs of 15.5 % and 6.2 %. Intra-run CV as tested on two serum samples run six times each with mean concentrations of 121.9 nmol/L and 224 nmol/L showed CVs of 13.2 % and 14.3 % respectively. Direct comparison of our data to the manufacturer's needs to be interpreted with caution as the sample type used in our laboratory differs from the manufacturer's (human-based QC material and serum samples compared to whole blood samples used by the manufacturer). Furthermore, our sample numbers were smaller due to limited POCT cartridge availability.

Comparison of Randox (which is directly based on the Denka Seiken assay design) vs. Roche showed a positive bias on the Roche assay at higher concentrations similar to previously published literature, where it was speculated that this may be the result of the use of different reference standards and calibrators across the measuring range of Lp(a) [13]. From the results of a comparative study using an isoform independent enzyme-linked immunosorbent assay (ELISA) as the reference method, it was claimed that the Denka Seiken assay demonstrated excellent correlation with the ELISA and that it was largely isoform insensitive – in part due to the 5-point calibration curve with each point using multiple different apolipoprotein(a) sizes. However, an overestimation of Lp(a) concentrations in samples with large apolipoprotein(a) isoforms was noted [5]. One study suggested that due to possible undermeasurement of Lp(a) at higher concentrations, manufacturers have increased their calibration for the high-concentration standard to compensate for this. It was speculated that this 'up-calibration' may have been overdone with some assays such as Roche or possibly underdone with the Denka Seiken high level standard resulting in differences at higher concentrations [13].

Screening for Lp(a) has been endorsed by multiple guidelines, including many guidelines that now recommend testing at least once in the life of an individual [2]. Due to the large size of Australia, reaching rural and remote populations for Lp(a) testing is challenging. In addition, it is well described in international studies, that testing rates for Lp(a) remain low worldwide, even in non-rural settings [16, 29]. This is also true for high-risk populations in Australia as demonstrated by a Western Australian study [30]. Measurement of Lp(a) via POCT was easy and relatively fast when tested in our laboratory. It did not require time-intensive laboratory training and (although we did not test this in our study), the sample type can be capillary without the requirement of sample centrifugation. This could be an attractive option for Australia's rural and remote populations where readily available access to biochemistry testing can be difficult. One consideration is that the testing process per sample took approximately 3 min and the device needed to be switched off between consecutive sample measurements, which would need to be considered to optimize workflow in busy clinics. POCT could be used during or before a consultation, particularly in patients with cardiovascular risk factors to help re-stratify ASCVD risk and to help tailor treatment. Patients will still require regular monitoring of their other biochemistry parameters, such as lipid profiles or (if applicable) glycated haemoglobin, renal and hepatic function via a mainstream laboratory. Concentrations above the measuring range of the POCT device would need to be confirmed in a laboratory and the device should be viewed as a screening tool. Introduction of Lp(a) POCT in Australia would require a more rigorous assessment than our current study and formal approval by regulatory authorities such as the Therapeutic Goods Administration. POCT should preferably be overseen by a National Association of Testing Authorities (NATA) accredited laboratory.

This study comes with limitations. While almost all experiments were run using the same cartridge kit lot, the intra-assay CVs were tested using a different cartridge kit lot, which was older but within its expiry date. This may have impacted the results of the intra-assay CV assessment and could be repeated in future assessments. Additionally, the number of patients studied, particularly for the precision assessment, was small and could be expanded. However, the numbers used for our experiment were compliant with the Australian Association of Clinical Biochemists (AACB) recommendation for verification of POCT assays [31]. Furthermore, none of the comparator assays used can be considered gold standard and as such the comparison needs to be interpreted with caution. However, the Denka Seiken assay (from which the Randox assay is derived) was reportedly comparable to an old ELISA-based reference method [5, 23]. Additionally, we did not compare our findings to capillary or whole blood measurements, which would be the preferred measurement sample types in the community. Whole blood samples cannot be run on our laboratory analysers and our study protocol and associated Ethical Approval did not include a simultaneous capillary blood collection. The dilutional studies should be interpreted with caution. A diluted sample with a high concentration (often of a small Lp(a) isoform) may incorrectly meet a calibrator with a large apo(a) isoform size in the

multipoint calibrated assays in the second assay run leading to inaccurate results [32].

The strengths of this study include the use of two different laboratory assays from two different NATA accredited laboratories to assess performance of the POCT device. Furthermore, care was taken to run all experiments via a small group of investigators to minimize pipetting or operational errors. Both laboratories' Lp(a) assays are assessed with daily internal QC and regular external quality assurance programmes. While the overall sample number tested was small, for a POCT comparison, the samples numbers tested exceeded the minimum requirements as set out by local testing guidelines such as the AACB [31]. To the best of our knowledge, it is the first published study comparing a POCT device for Lp(a) to two commercially available, NATA-accredited laboratory Lp(a) assays.

Conclusions

The iProtin POCT device for Lp(a) measurement compares well to two widely available laboratory assays and is easy to use. It may aide in increasing Lp(a) testing in rural and remote areas where access to Lp(a) testing may be limited. It may help with ASCVD risk (re-) classification and subsequent treatment.

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Research ethics: This study was reviewed by the Sullivan Nicolaides Ethics boards and was found to meet the NHMRC criteria for a Quality Assurance and Evaluation activity (HE/2025/SNP/40) on 1 September 2025.
Informed consent: Not applicable.
Author contributions: All authors have accepted responsibility for the entire content of this manuscript and approved its submission. Conceptualization, methodology: C.B. and G.W.; writing – original draft: C.B.; writing – review: K.K, A.A.; data collection and collation: C.B, C.E, A.D., A.L.
Use of Large Language Models, AI and Machine Learning Tools: None declared.
Conflict of interest: The authors received the iProtin device free of charge from tst biomedical electronics Co., Ltd (Taoyuan City, Taiwan) for use in this study. The company was not involved in the study design, data collection, analysis, or interpretation, nor in the decision to submit the manuscript for publication.
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