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Advancing forensic SNP typing: Insights from an interlaboratory study of the FORCE panel☆

Andreas Tillmar[a,b,*], Kimberly Sturk-Andreaggi[c,d], Adam Staadig[a,b], Christina Amory[e], David Ballard[f], Erik F. Bergseth[g], Claus Børsting[h], Selena Cisana[i], Michael Coble[j], Lucinda Davenport[f], María de la Puente[k], Katherine Butler Gettings[l], Erin M. Gorden[m], Kelly Grisedale[n], Jennifer Daniels-Higginbotham[c,d], Rachel Houston[o], Sheree Hughes[o], Reet Järving[p], Kevin M. Kiesler[l], Jonathan L. King[j], Maria Victoria Lareu[k], Odile Loreille[q], Dennis McNevin[n,r], Melissa K. Muenzler[j], Walther Parson[e,s], Vania Pereira[h], Christopher Phillips[f,k], Jorge Ruiz-Ramírez[k], Maarja Sadam[p], Jennifer Snedeker[o], Carolyn R. Steffen[l], Monika Stoljarova-Bibb[p], Peter M. Vallone[l], Jodie Ward[n,r], Jessica Watson[n,r], Catarina Xavier[e], Daniel Kling[a,g,t], Charla Marshall[c,s,u]

[a] Department of Forensic Genetics and Forensic Toxicology, National Board of Forensic Medicine, Sweden
[b] Department of Biomedical and Clinical Sciences, Faculty of Medicine and Health Sciences, Linköping University, Sweden
[c] Armed Forces Medical Examiner System's Armed Forces DNA Identification Laboratory (AFMES-AFDIL), Dover Air Force Base, USA
[d] SNA International, LLC, Alexandria, USA
[e] Institute of Legal Medicine, Medical University of Innsbruck, Innsbruck, Austria
[f] King's Forensics, King's College London, United Kingdom
[g] Department of Forensic Sciences, Oslo University Hospital, Norway
[h] Department of Forensic Medicine, University of Copenhagen, Denmark
[i] Centro Regionale Antidoping, Turin, Italy
[j] Center for Human Identification University of North Texas Health Fort Worth, USA
[k] Institute of Forensic Sciences, University of Santiago de Compostela, Spain
[l] Applied Genetics Group, National Institute of Standards and Technology, USA
[m] Signature Science, LLC, USA
[n] National DNA Program for Unidentified and Missing Persons, Australian Federal Police, Australia
[o] Department of Forensic Science, Sam Houston State University, USA
[p] Estonian Forensic Science Institute, Estonia
[q] DNA Support Unit, Federal Bureau of Investigation Laboratory, USA
[r] Centre for Forensic Science, University of Technology Sydney, Australia
[s] Forensic Science Program, The Pennsylvania State University, USA
[t] Biostatistics (BIAS), Norwegian University of Life Sciences, Norway
[u] DNAID Services, LLC, USA

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ABSTRACT

This study evaluated the ability to produce FORensic Capture Enrichment (FORCE) genotypes using amplicon-based and capture-based enrichment assays. The FORCE panel is a standardized set of single nucleotide polymorphism (SNP) markers developed for forensic applications. Twelve DNA samples were prepared and distributed to the laboratories for testing: five control DNA samples, a dilution series ranging from 10 ng to 0.03 ng, two degraded DNA samples with 200 bp and 150 bp average fragment lengths, and one inhibited sample spiked with humic acid. Fifteen laboratories from three different continents participated in this study, choosing from one of four manufacturer-developed enrichment assays to complete the experiments, setting their own parameters for

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* Correspondence to: Department of Forensic Genetics and Forensic Toxicology, National Board of Forensic Medicine, Artillerigatan 12, Linköping SE58758, Sweden.
E-mail address: andreas.tillmar@liu.se (A. Tillmar).

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sequencing and other user-defined steps to accommodate their own preferences and expertise. A total of eighteen methods were evaluated, as three laboratories performed two methods. The results showed that all four assays were successful in producing full FORCE SNP genotypes from high quality samples. However, significant differences between and within assays and methods were observed. Read count variability and enrichment type led to significant differences in call rate. Robust SNP recovery was observed across all assays at 0.3 ng DNA input, with an amplicon-based assay producing high SNP call rates at 0.03 ng DNA input. Capture and single primer extension assays produced consistently high SNP call rates from degraded samples with 150–200 bp fragments. Future research to optimize laboratory parameters may reduce the variation in SNP data, so that labs may equitably adopt SNP methods to make use of these powerful forensic markers.

1. Introduction

Single nucleotide polymorphisms (SNPs) have multiple advantages for forensic identity, kinship, and missing persons analysis [1]. Today, SNP genotyping is largely performed using high throughput sequencing technologies and microarray (SNP chip) techniques [2]. SNPs can be assayed using PCR, primer extension capture, and hybridization capture, along with whole genome sequencing (WGS), which are methods well-suited for forensic samples that may have limited DNA template and/or degraded DNA [3]. Unlike short tandem repeat (STR) markers that are 100–500 base pairs (bp) in length with high mutation rates, SNPs are single base markers with low mutation rates [4]. Therefore, more SNPs are needed to achieve sufficient discriminatory power for use in human identification and kinship [5]. The Human Genome Project and other similar efforts have collectively identified millions of SNPs in global human populations [6,7]. Based on these reference SNPs, scientists within the forensic community have developed several PCR-based SNP panels for human identification purposes. Early examples of these SNP panels include SNPforID [8] and the Kidd panel [9]. With the advent of massively parallel sequencing (MPS), larger forensic SNP panels have been developed such as a 140-SNP Qiagen panel (QIAGEN, Hilden, Germany) [10], Precision ID Identity Panel (Thermo Fisher Scientific, Waltham, MA, USA) [11], ForenSeq DNA Signature Prep Kit (QIAGEN) [12], MPSplex (QIAGEN) [13], and ForenSeq Kintelligence Kit (QIAGEN) [14]. Sequencing of the forensic SNP panels is performed using sequencing-by-synthesis chemistries on platforms such as Illumina and Ion Torrent, which are among the most widely used to date, although other sequencing chemistries and platforms are also capable of analyzing such SNP panels. These sequencing methods differ with respect to their chemistries and thus require different library preparation methods. The bioinformatic data analysis must furthermore be tailored to the methodological approach (e. g., [15]), leading SNP panel manufacturers to create their own software for data analysis. This has led to a diversification of the SNP genotyping workflows being validated for forensic use.

In order to accommodate the diversity in MPS techniques employed by worldwide forensic laboratories, the FORensic Capture Enrichment (FORCE) panel was designed conceptually as a collection of SNPs that could be targeted using multiple MPS methods [16]. The FORCE panel is a set of ≈5500 SNPs for identity, kinship, ancestry, phenotype, and Y-haplogroup prediction, with no known clinical relevance at the time of selection. The initial FORCE panel SNP selection was based on a myBaits hybridization capture assay design (Arbor Biosciences, Ann Arbor, MI, USA), with the intent of balancing the number of SNPs needed for human identification with the cost per probe. Since the initial FORCE publication, several papers have demonstrated the panel's adaptability to alternate enrichment and sequencing methods. These include hybridization capture for Ion Torrent sequencing [17] and QIAseq (QIAGEN), a single-primer extension–based technology, for Illumina sequencing [18,19].

In this report, the practical ability to assay the FORCE panel using a laboratory-chosen MPS method was tested in a global interlaboratory study. In addition to the original myBaits hybridization capture using Illumina sequencing [16], the following MPS methods were evaluated: myBaits hybridization capture with Ion Torrent sequencing [17], QIAseq with Illumina sequencing [19], AmpliSeq (Thermo Fisher Scientific) with Ion Torrent sequencing, and xGen (Integrated DNA Technologies, Newark, NJ, USA) with Illumina sequencing. A FORCE panel consortium was created in 2022, comprised of fifteen participating laboratories representing ten countries from three different continents. This project was not grant funded, relying on individual laboratories to secure the reagents, equipment, and personnel time necessary to produce SNP data from a controlled set of DNA samples. The laboratories were informed of the multiple options for FORCE target enrichment, then permitted to choose whichever option would work best in their respective laboratory environments. This allowed laboratories to leverage their existing equipment, in-house expertise and preferred MPS methods for SNP data production. A set of twelve known samples was prepared and aliquots were sent to each of the test sites for SNP analysis using their chosen FORCE method. The MPS data files were collected and analyzed by one laboratory. SNP recovery and concordance were evaluated for each methodology, noting any interlaboratory variation observed. This study provides the first comprehensive cross-platform assessment of the FORCE panel, demonstrating its adaptability across diverse library preparation methods and sequencing platforms, and establishing benchmarks that pave the way for informed selection and implementation, rather than ranking, of SNP typing methods in forensic laboratories.

2. Materials and methods

2.1. Samples

This project used six commercially available DNA samples obtained from Promega (2800 M), Coriell (NA12877 and NA12878), and NIST (RM 8391 (NIST Genome in a Bottle (GIAB) sample HG-002), SRM 2391d Component A, and SRM 2391d Component B). To assess the sensitivity of each assay, a dilution series was created from 2800 M to allow for DNA inputs of 10 ng, 1 ng, 0.3 ng, 0.1 ng, and 0.03 ng. To evaluate inhibition, a separate aliquot of 2800 M was spiked with humic acid. Soil solution was prepared by mixing soil with nuclease-free water (20% w/w) and shake-incubated for one hour [20]. Subsequently, 5 µL of the soil solution was added to 1 ng of genomic DNA. While the exact humic acid concentration in the inhibited sample was not measured, the amount of soil solution added was intended to mimic levels of inhibition that could reasonably occur in a casework sample affected by similar soil-derived inhibitors. DNA degradation was examined by artificially shearing NIST RM 8391 to two separate DNA size ranges: ≈200 bp (NIST deg1) and ≈150 bp (NIST deg2) by treatment in a Covaris S2 Focused Ultrasonicator (Covaris, Woburn MA, USA). NIST RM 8391 was treated for four minutes (NIST deg1) or 15 min (NIST deg2) using the following instrument settings: Duty cycle: 10%; Intensity: 10; Cycles/burst: 100; Temperature: 6.5°C; Mode: Frequency Sweeping. Shearing was performed in a MicroTUBE AFA vessel (Covaris) in a total volume of 130 µL. With the exception of the dilution series, DNA samples were provided at high DNA concentration to allow each laboratory to utilize as much targeted DNA template as would be used in routine practice. A summary of the DNA samples provided to the participating laboratories

for the interlaboratory study can be found in Table 1. Laboratories were also encouraged to include samples representative of their typical casework in addition to the study-provided samples; however, these samples were not incorporated in these analyses.

2.2. FORCE assays

For the purposes of this study, the term "assay" will refer to the FORCE enrichment approach (AmpliSeq, myBaits, QIAseq, and xGen); whereas the term "method" will refer to the complete workflow employed by each laboratory for their particular assay. Altogether, the four different FORCE panel assays were tested using 18 different methods across the 15 laboratories, as some laboratories tested more than one method (Table 2). For simplicity, the 18 methods are described by their FORCE assay, recognizing that other variables are subsumed within the method as well (such as DNA input, library preparation and sequencing conditions). Overall, the study included five AmpliSeq, eight myBaits, four QIAseq, and one xGen methods utilizing one of the following four MPS instruments: Ion GeneStudio S5 (Thermo Fisher Scientific), MiSeq FGx (QIAGEN), NextSeq 550 (Illumina, San Diego, CA, USA), or NextSeq 2000 (Illumina). Supplementary File S1 summarizes the standard procedures for each assay, including the reagents and instruments used and their respective manufacturers, and the laboratory-chosen parameters of each method are presented in Table 2. The details of the AmpliSeq and xGen designs are presented in Supplementary File S2, as these details have not previously been published.

2.3. Bioinformatics, genotype calling and data analysis

Bioinformatic data analysis was performed at one laboratory (National Board of Forensic Medicine, NBFM) for consistency. Sequence data files were transferred to the NBFM for bioinformatic analysis using the CLC Genomics Workbench v21 (QIAGEN). Due to the differing chemistries, different tools and settings were used for data analysis as appropriate for the specific method. For example, PCR duplicates were removed from myBaits data, whereas PCR duplicates were merged into super reads using unique molecular indices (UMIs) for the QIAseq analysis. The AmpliSeq and xGen chemistries do not allow for PCR duplicate removal; therefore, PCR duplicates were retained in these datasets. An overview of the bioinformatic workflows is provided in Supplementary File S1. Genotype calling was performed using a custom R script [21]. The following thresholds were applied for genotype calling: minimum read depth of 10; allele read frequency (computed as the number of reads for the most common allele divided by the sum of all reads for the specific SNP) of 0.5–0.8 for heterozygous loci and 0.95–1.0 for homozygous loci, other allele read frequencies resulted in no call; minimum quality score of 20; and minimum forward/reverse read ratio of 0.2.

The analysis focused on SNP call concordance among laboratories instead of concordance with published genotypes, as previous studies have shown errors in control DNA genotypes generated in large scale genome sequencing projects [16,22]. The majority genotype was considered to be the concordant genotype across chemistries and assays. The overall performance analysis focused on autosomal SNPs, due to sex chromosomal differences in the control DNA samples tested. In total, 4283 autosomal SNPs were included. These were SNPs where the majority of the laboratories generated genotype data. Separate concordance analyses for X and Y SNPs (241 and 817, respectively) are included as supplementary material.

Univariate and multivariate analyses were performed to detect significant differences in call rate. Overall differences were assessed using a Kruskal–Wallis test, and pairwise differences were evaluated with Dunn tests.

3. Results and discussion

3.1. Overall QC metrics

Overall, each of the four assays successfully produced FORCE genotypes from one or more samples, although considerable variability in SNP call rate was observed across assays and between methods within a particular assay. The analysis described in Sections 3.2–3.4 primarily focuses on genotype call rates and genotype concordance. However, clear variations were observed in some overall quality control (QC) metrics shown in Fig. 1(i.e., mapped reads (%), target specificity (%), insert size (bp) and proportion unique reads (%)), both between assays and among methods using the same assay.

3.1.1. Proportion of mapped reads

Across assays, clear differences in mapping performance were observed when considering all samples included in this study (Fig. 1A). AmpliSeq methods generally showed the highest median proportion of reads mapping (96%), but at the same time also had relatively high within-method variation including some distinct outliers with AmpliSeq-A and AmpliSeq-C. The myBaits methods displayed slightly lower proportion of mapped reads (median 93%) with generally narrower within-method distributions, indicating less sample-to-sample variation compared to AmpliSeq and xGen methods. QIAseq methods showed marked heterogeneity, with most methods achieving high mapping rates comparable to AmpliSeq (medians >95%), while one method (QIAseq-A) demonstrated slightly lower mapping proportion (median 87%). Finally, xGen showed the largest overall variability (ranging from 31% to 95%) when all study samples were considered. Reasons for the presence of un-mapped reads may be high proportions of adapter-dimers, having too unspecific or short reads, or other artifacts being created during the library preparation.

3.1.2. Target specificity

Target specificity (reads mapping proximal to the SNP positions) showed much stronger assay-dependent effects than overall mapping proportions (Fig. 1B). The AmpliSeq methods displayed consistently very high on-target specificity across most laboratories (median 99.7%

Table 1
DNA samples tested with each FORCE panel assay as part of the interlaboratory study.

Sample	Description	DNA Concentration (pg/μL)	Average Fragment Size (bp)	Volume Provided (μL)
A	NA12877	5000	Intact	50
B	NA12878	5000	Intact	50
C	2800M_10ng	1667	Intact	12
D	2800M_1ng	167	Intact	12
E	2800M_0.3 ng	50	Intact	12
F	2800M_0.1 ng	17	Intact	12
G	2800M_0.03 ng	5	Intact	12
H	NIST deg1	2400	≈ 200	10
I	NIST deg2	1000	≈ 150	10
J	NIST 2391d-A	1600	Intact	15
K	NIST 2391d-B	1600	Intact	15
L	2800M_humic acid	1000	Intact	25

Table 2
Overview of the 18 FORCE methods tested for the interlaboratory study, designated by the target enrichment approach.

Assay (Library Preparation)	DNA Input[a] (ng)	Sequencing System (Kit)	Cycles (Illumina) or Flows (Ion)	Samples Per Run[b]
AmpliSeq-A	5	Ion GeneStudio S5 (Ion 530 Chip Kit)	650 flows	8–17
AmpliSeq-B	5–10	Ion GeneStudio S5 (Ion 530 Chip Kit)	650 flows	4–5
AmpliSeq-C	5	Ion GeneStudio S5 (Ion 530 Chip Kit)	500 flows	6
AmpliSeq-D	5	MiSeq FGx (MiSeq FGx Reagent Kit)	301 + 301 cycles	6
AmpliSeq-E	5–10	Ion GeneStudio S5 (Ion 540 Chip Kit)	500 flows	18
myBaits-A (KAPA Hyper)	10	MiSeq FGx (MiSeq Reagent Kit v3)	301 + 301 cycles	5–8
myBaits-B (KAPA Hyper)	10	MiSeq FGx (MiSeq Reagent Kit v2 and v3)	151 + 151 cycles (v2) or 76 + 76 cycles (v3)	3–5
myBaits-C (KAPA Hyper)	10–77	MiSeq FGx (MiSeq FGx Reagent Kit)	151 + 151 cycles or 301 + 301 cycles	3–7
myBaits-D (Ion Plus Fragment)	1	Ion GeneStudio S5 (Ion 540 Chip Kit)	500 flows	8–9
myBaits-E (NEBNext Ultra II)	6–30	MiSeq FGx (MiSeq FGx Reagent Micro Kit)	351 + 151 cycles	12
myBaits-F (KAPA Hyper)	10–26	NextSeq 2000 (NextSeq 1000/2000 P2 Reagents v3)	101 + 101 cycles	22
myBaits-G (NEBNext Ultra II)	5–10	MiSeq FGx (MiSeq FGx Reagent Kit)	301 + 301 cycles	16
myBaits-H (NEBNext Ultra II)	5–10	NextSeq 550 (NextSeq 500/550 High Output Kit v2.5)	151 + 151 cycles	16
QIAseq-A	10	MiSeq FGx (MiSeq FGx Reagent Kit)	151 + 151 cycles	3–6
QIAseq-B	6–24	MiSeq FGx (MiSeq FGx Reagent Kit)	151 + 151 cycles	3–5
QIAseq-C	10–40	MiSeq FGx (MiSeq Reagent Kit v2)	151 + 151 cycles	5–7
QIAseq-D	9–84	MiSeq FGx (MiSeq FGx Reagent Kit)	151 + 151 cycles	8–9
xGen-A	10–257	NextSeq 550 (NextSeq 500/550 High Output Kit v2.5)	151 + 151 cycles	25

[a] DNA input excludes the dilution series.
[b] Samples and positive controls only; no negative controls included in this number.

across all methods), with some displaying greater spread and occasional drops for individual methods and samples. In contrast, the myBaits assay exhibited uniformly low specificity across all methods (median 21%), but with clear differences between methods. The lower specificity for probe-based hybridization capture assays is well known [23]. The myBaits-D method included two consecutive capture steps (Supplementary File S1), resulting in higher specificity (median 55%) compared to the other myBaits methods (median 18%). QIAseq methods showed overall high median specificity (90%) close to AmpliSeq but with broader within-method dispersion, reflecting appreciable method-to-method variability. xGen displayed intermediate specificity with substantial spread across samples (49–97%).

3.1.3. Insert size

Insert size distributions (Fig. 1C), which could only be assessed for paired data (i.e., methods that used Illumina sequencing), revealed pronounced differences both between assays and methods. The myBaits methods used different library preparation kits with both enzymatic and mechanical fragmentation with variable conditions, which resulted in wide insert size ranges (medians across methods from 149 bp to 269 bp) and substantial within-assay variability. By contrast, methods employing the QIAseq assay with its standardized enzymatic fragmentation step yielded more consistent median insert sizes and narrower interquartile ranges (181 bp, and 168 bp to 197 bp, respectively) except for QIAseq-D.

3.1.4. Proportion of unique reads

The proportion of unique reads for myBaits and QIAseq methods (Fig. 1D) showed substantial variability, reflecting differences in sequencing depth and sample multiplexing between the methods (deduplication was not performed for the FORCE PCR-based methods, AmpliSeq and xGen, and therefore the proportion of unique reads is not applicable). The myBaits methods spanned a wide range, with some methods (i.e., myBaits-C, -E, and -G) achieving very high proportions of unique reads (medians >93%), while others (notably myBaits-A and -D) showed consistently low values due to high duplication rates (resulting in medians of < 25% unique reads). All in all, large method-to-method spreads were observed for the myBaits assay, consistent with differences in library preparation parameters and reads per sample driven by multiplexing strategies (Table 2 and Supplementary File S1). QIAseq methods generally exhibited lower proportions of unique reads (median 33% across methods) and narrower ranges, suggesting higher duplication overall but with clear between-method variation. Overall, duplication metrics appeared to be strongly influenced by sequencing depth relative to library complexity, which is attributable to DNA input, in addition to assay-specific characteristics.

These assay and method differences further underscore the impact of both assay design and method-specific parameters. Additional QC metrics (e.g., total number of reads, mean read length, mean coverage) are presented in Supplementary File S3.

3.2. Control DNA samples

3.2.1. Inter-assay variability

When considering the five unaltered control DNA samples (>1 ng DNA input, not degraded or inhibited: samples A, B, C, J, and K), each

assay resulted in a median of more than 4000 (maximum 4283) called autosomal SNPs (Fig. 2). Three out of four assays produced variable numbers of autosomal SNPs, with AmpliSeq ranging from 155 to 4223, myBaits ranging from 903 to 4283, and QIAseq ranging from 79 (a single outlier; otherwise from 4146) to 4277. These ranges are depicted in the probability density distributions of Fig. 2, which may be attributed to both inter- and intra-laboratory differences (Supplementary File S3 through S6). The xGen assay produced nearly 100% autosomal SNP call rates across all five samples (ranging from 4156 to 4206 SNPs) and a compressed probability density distribution, likely due to being represented by only one method performed at a single laboratory (xGen-A).

The proportion of discordant (any allelic difference) SNP genotypes was consistently low across all assays as shown in Fig. 3 (< 0.006 excluding two outliers). The average discordant proportions were 0.0021 for AmpliSeq, 0.0006 for myBaits, 0.0001 for QIAseq, and 0.0043 for xGen, indicating high concordance across assays. Most samples clustered near zero discordance; however, the two outliers, one AmpliSeq (0.019) and one myBaits (0.018), were clearly isolated deviations rather than a systematic trend. Overall, these results demonstrate robust SNP concordance across all tested assays and methods.

Assay type (i.e., AmpliSeq, myBaits, QIAseq, or xGen) had a significant impact on the call rate for the five good quality samples ($p \leq 0.0001$). A more in-depth analysis showed that AmpliSeq differed significantly from both myBaits ($p \leq 0.0001$) and QIAseq ($p \leq 0.0001$), whereas no significant differences were found between the other assay pairs.

3.2.2. *Intra-assay variability*

The AmpliSeq assay exhibited the most intra-assay variability in call rate, with one method (AmpliSeq-C) producing mostly failed samples due to poor quality Ion Torrent sequencing reagents. This can be seen in Fig. 4 with the SNP recoveries shown by method for a high-quality sample (sample B). Root cause analysis by the manufacturer identified five lots impacted by suboptimal purification of a minor component. This was confirmed when the AmpliSeq-C amplicons were also prepared

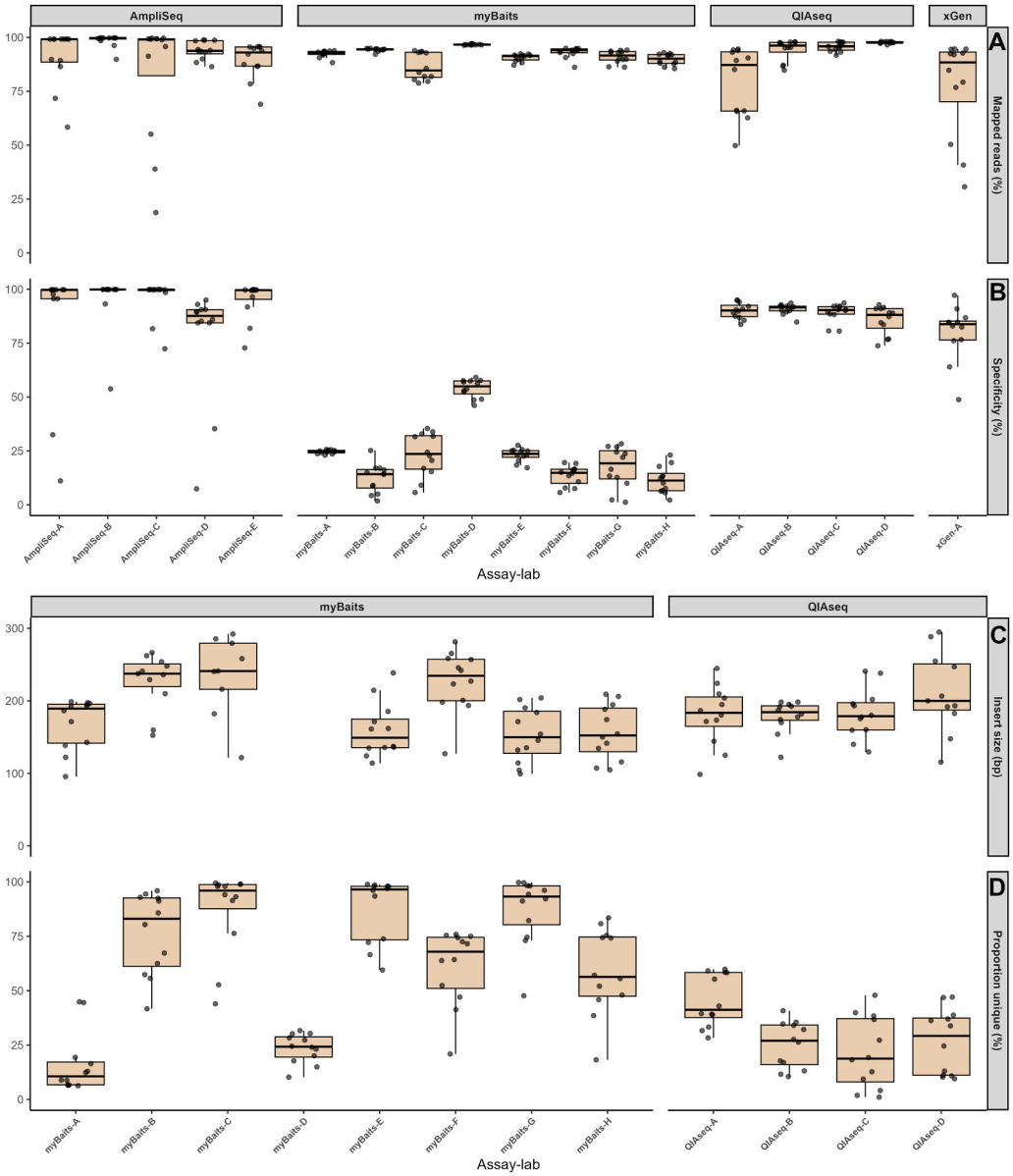

Fig. 1. Box and jitter plots showing the observations for proportion of mapped reads (A), specificity (B), average insert size (C), and proportion of unique reads (D) for the samples included in the study (the degraded samples were excluded from the "Insert size" subplot). The boxplots show the median (central line), interquartile range, and whiskers (1.5 × interquartile range).

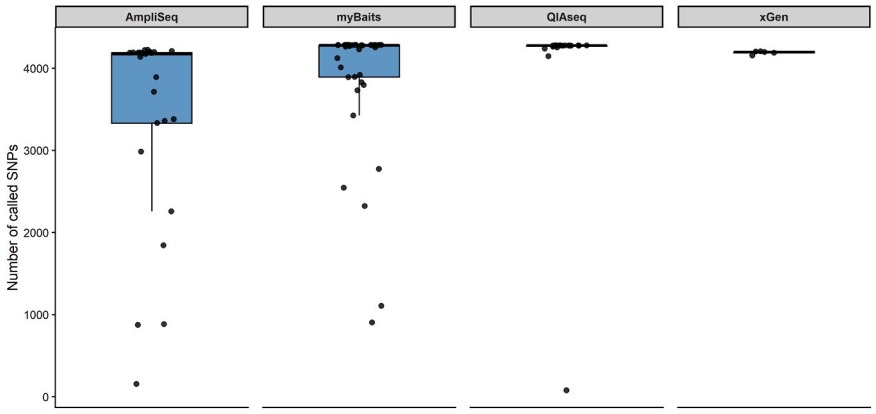

Fig. 2. Box and jitter plots showing the number of called autosomal SNPs for the five control DNA samples (>1 ng DNA input, not degraded or inhibited: samples A, B, C, J, and K). The boxplots show the median (central line), interquartile range (box), and whiskers (1.5 × interquartile range). Individual data points are overlaid as jittered dots to illustrate the distribution. Sample sizes are AmpliSeq = 25 (5 methods); myBaits = 40 (8 methods); QIAseq = 20 (4 methods); xGen = 5 (1 method).

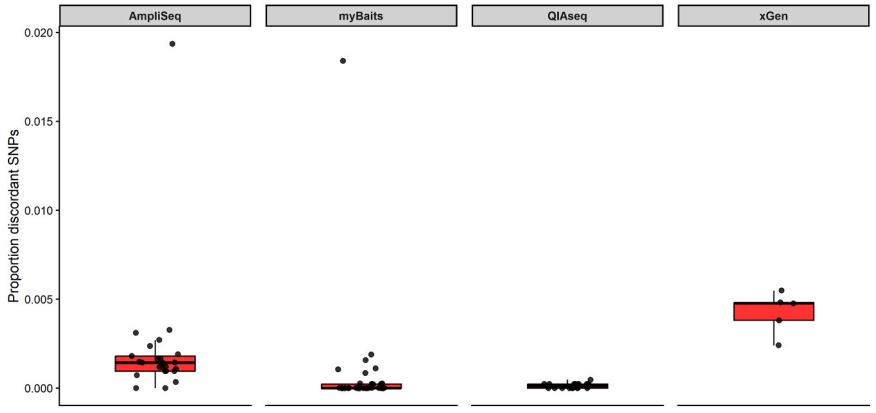

Fig. 3. Box and jitter plots showing the proportion of discordant autosomal SNPs, per sample, for the five control DNA samples (>1 ng DNA input, not degraded or inhibited: samples A, B, C, J, and K). The boxplots show the median (central line), interquartile range (box), and whiskers (1.5 × interquartile range). Individual data points are overlaid as jittered dots to illustrate the distribution. Sample sizes are AmpliSeq = 25; myBaits = 40; QIAseq = 20; xGen = 5. Detailed information of discordant SNP genotypes is available in Supplementary File S4.

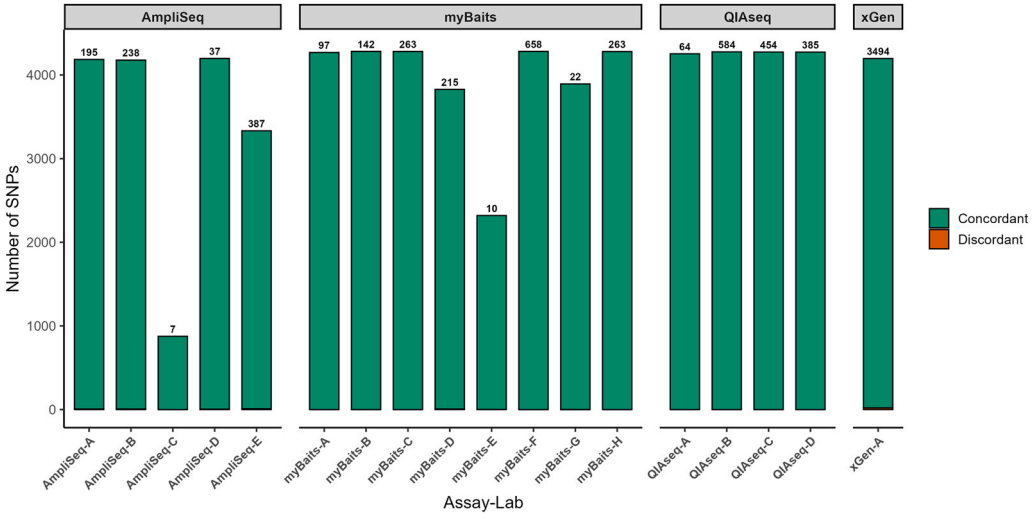

Fig. 4. Column graphs showing the number of called autosomal SNPs by FORCE method for sample B, as an example of high-quality sample results. The green shading indicates concordant SNPs, while the orange shading indicates very low numbers of discordant SNPs. Detailed counts of concordant and discordant SNP genotypes, together with the rs identifications of discordant SNPs, are available in the Supplementary File S4. Average coverage is indicated on top of each bar (see Supplementary File S3 for all samples).

as Illumina libraries and successfully sequenced on a MiSeq instrument (AmpliSeq-D), substantially increasing the call rates for all samples. Another method (AmpliSeq-E) produced significantly lower call rates when compared to methods AmpliSeq-A ($p < 0.01$) and AmpliSeq-B ($p < 0.001$). Unlike the other AmpliSeq methods, AmpliSeq-E used a 540 chip and although it offers more reads, the maximum read length of approximately 200 bp (500 flows) is below the mean amplicon size of the FORCE panel targets (≈300 bp) and therefore limits SNP recovery. Thus, differences in AmpliSeq call rate may be related to aspects of the method employed by the testing laboratory, such as choice of sequencing chip and/or run parameters (e.g., number of flows).

Within-assay differences in call rate were also significant for myBaits and QIAseq. Three myBaits methods generally resulted in lower call rates than the other methods: myBaits-D that used Ion Torrent sequencing, myBaits-E that used a MiSeq FGx Reagent Micro Kit (QIAGEN), and myBaits-G that used a MiSeq FGx Reagent Kit (QIAGEN) with

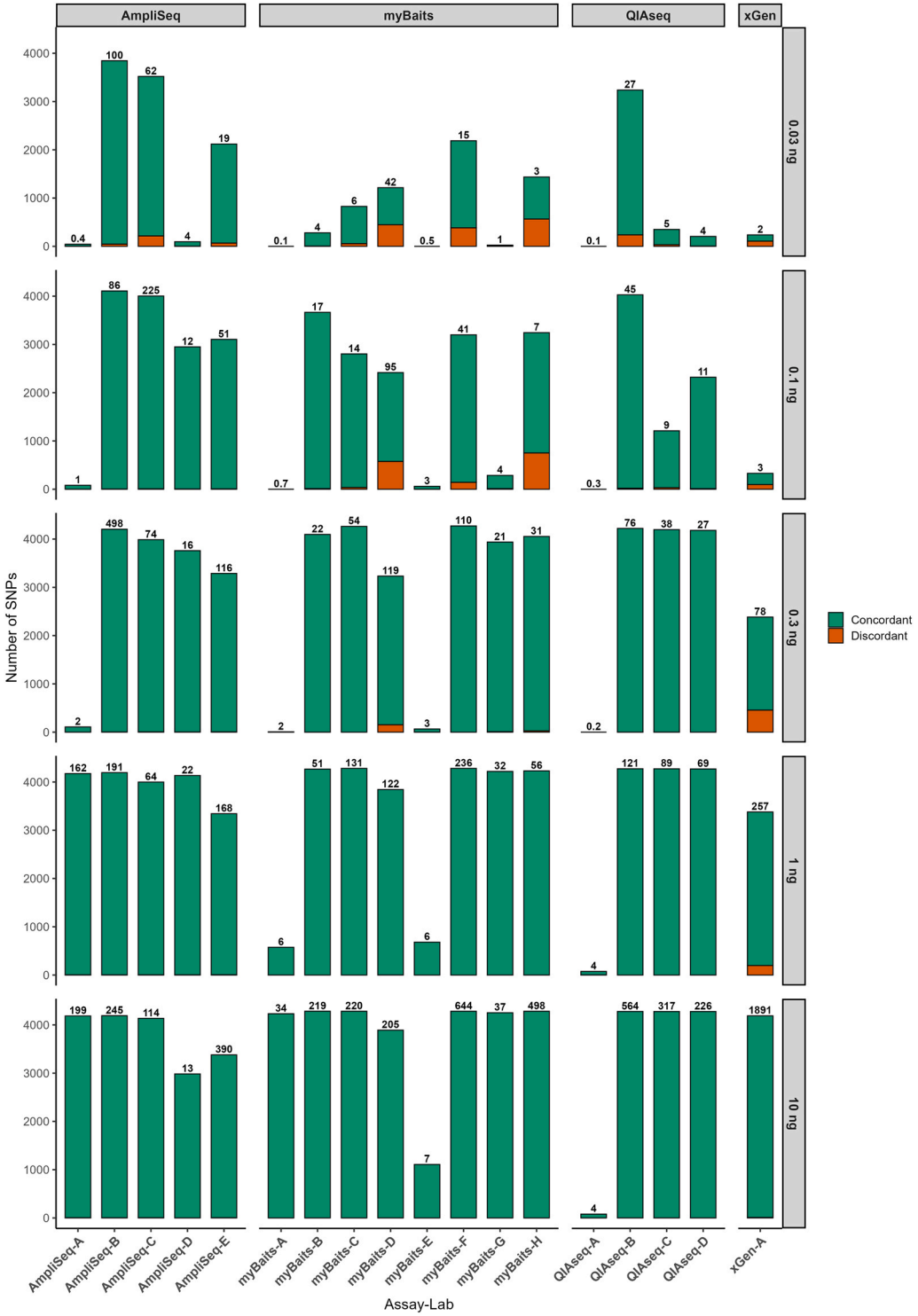

Fig. 5. Column graphs showing the number of called autosomal SNPs for the 2800 M dilution series (sample C – G) by method. The green shading indicates concordant SNPs, while the red shading indicates discordant SNPs. Detailed counts of concordant and discordant SNP genotypes, together with the rs identifications of discordant SNPs, are available in the Supplementary File S4. Average coverage is indicated on top of each bar (see Supplementary File S3 for all samples).

16 samples per run. The two latter sequencing approaches with less output and/or increased multiplexing (typically 3–8 samples per run for the other methods using a MiSeq FGx Reagent Kit) would have resulted in fewer reads per sample, contributing to reduced call rates. Despite relatively high coverage (e.g., 177 × average coverage for sample B), myBaits-D had lower call rates compared to other myBaits methods with similar coverage depths. It was observed that high proportions (5–10% for good quality samples) of sufficiently covered SNPs (i.e., read depth of at least 10) were imbalanced and thus resulted in no calls for the myBaits-D samples. This may be due to stochastic variation exacerbated by an additional post-capture PCR following the second capture reaction of the myBaits-D method. The setup of the bioinformatic analysis workflow could have also been a contributing factor, as PCR duplicate removal was likely suboptimal for the Ion-specific errors [24] in the myBaits-D data. Due to lower read outputs of the sequencing approaches, the myBaits-E and myBaits-G methods resulted in reduced coverage compared to the other myBaits methods, which could explain the reduced call rate. Overall, the lower call rates of these three methods were significant in some of the pairwise comparisons, with myBaits methods having higher call rates. Of note, myBaits-F, which used the NextSeq 2000, produced significantly higher call rates than myBaits-A and myBaits-G, which were both sequenced on the MiSeq FGx platform. Of the four QIAseq methods, QIAseq-A produced a significantly lower call rate than the other three methods ($p < 0.01$) (Supplementary File S4). High proportions of adapter dimers are a likely explanation for the reduced call rate of QIAseq-A. Elevated adapter dimer proportions have been observed previously [25]. However, the remaining three QIAseq methods produced high and comparable call rates and were not significantly different, indicating that adapter dimer formation may not be a consistent issue across QIAseq assays. Overall, the xGen produced consistently high call rates between the five control DNA samples.

3.3. Dilution series

The results of the 2800 M dilution series are shown in Fig. 5. As expected, decreasing the DNA input led to a reduction in called SNPs and an increase in discordant SNPs across all methods regardless of FORCE assay.

All of the methods except two (QIAseq-A and myBaits-E) were successful in producing complete, concordant profiles at 10 ng input (sample C). The QIAseq-A method resulted in only 79 called SNPs, and the myBaits-E method resulted in only 1108 called SNPs, echoing the findings discussed above for the five control DNA samples.

The DNA input of 1 ng (sample D) generated over 4000 concordant SNPs in the majority of methods. The exceptions were, again, QIAseq-A and myBaits-E, as well as myBaits-A and xGen-A. The myBaits-A method produced only 575 SNPs, yet the remaining myBaits methods were successful at this input quantity. The xGen method produced 3182 concordant SNPs but also 197 discordant SNPs.

At 0.3 ng input (sample E), three of the four QIAseq methods and five of the eight myBaits methods yielded ≈ 4000 concordant SNPs, whereas the myBaits-D method showed a reduced call rate with 3079 concordant SNPs and 154 discordant SNPs. Four of the five AmpliSeq methods produced > 3200 SNPs, although AmpliSeq-A generated only 108 called SNPs (all of which were concordant). The xGen-A method yielded 1927 concordant SNPs, with 457 discordant SNPs.

Four of the five AmpliSeq methods yielded ≈ 3000 or more concordant SNPs and minimal discordances at 0.1 ng input (sample F). The myBaits methods produced a wide range of called SNPs (0–3665). Of the called SNPs, the myBaits methods generated between 0 and 752 discordant SNPs. The xGen-A method had a very low number of called SNPs ($n = 330$) with high ($n = 97$) discordant SNPs.

At the lowest DNA input of 0.03 ng (sample G), AmpliSeq had the most concordant SNPs, with two of the five AmpliSeq methods yielding over 3300 concordant SNPs. Of the QIAseq methods, QIAseq-B produced 3000 concordant SNPs and 238 discordant SNPs, yet the remaining QIAseq methods generated < 351 called SNPs. QIAseq-A did not produce any called SNPs, although this was not unexpected given the lower performance of QIAseq-A overall. The myBaits methods generated < 2000 concordant SNPs and up to ≈ 500 discordant SNPs, although with high inter-lab variability. The xGen method generated only ≈ 250 called SNPs, approximately half of which were discordant.

Of note, myBaits-F displayed a different pattern of discordance for the two lowest input samples (sample F with 0.1 ng input and sample G with 0.03 ng input). While other myBaits methods showed a comparable proportion of discordant SNP calls (Fig. 5), the myBaits-F discordances were mostly due to allele drop-in, in contrast to the allele drop-out observed at these lower input levels (Supplementary File S4 through S6). The drop-in alleles observed in these myBaits-F samples were, after investigation, consistent with a high-quality, highly concentrated sample that was prepared in the same library set and sequenced in the same NextSeq 2000 run as these samples.

In summary, the dilution series demonstrates that there is variability in the performance of each assay depending on the method used, with a general trend of decreasing call rates and increasing discordances as DNA input is reduced. At the lowest DNA inputs of 0.1 and 0.03 ng, the amplicon-based AmpliSeq assay had the highest call rate compared to the other assays. This may be related to the bioinformatic handling of amplicon versus non-amplicon data, as the AmpliSeq analysis did not remove PCR duplicates. However, the same minimum coverage threshold was used for all assays regardless of PCR duplicate removal (10 ×).

3.4. Degraded and inhibited samples

Fig. 6 shows the results from the testing of two degraded samples (sample H and sample I) and one inhibited sample (sample L that was spiked with humic acid). For sample H, with a mean fragment size of 200 bp, all four QIAseq methods and five of the eight myBaits methods produced > 4200 concordant SNPs. This includes QIAseq-A, which performed just as well as the other three QIAseq methods in the degraded and inhibited sample experiments. Three myBaits methods (myBaits-A, -D, and -E) generated slightly fewer (2833–3757) concordant SNPs, and 99 discordant SNPs were observed in the myBaits-D data. The AmpliSeq methods produced low call rates for sample H, with only one full profile generated (from AmpliSeq-B with 4190 concordant SNPs). AmpliSeq-E produced a partial profile with 2158 concordant SNPs, and the remaining AmpliSeq methods generated < 400 SNPs from sample H. This is likely due to AmpliSeq's mean amplicon size of ≈ 300 bp, preventing PCR amplification of the fragmented DNA. The xGen method generated 3713 concordant SNPs from sample H. For sample I, with a mean fragment size of 150 bp, QIAseq again produced > 4100 concordant SNPs across all four methods. As with sample H, five of the eight myBaits methods generated nearly full profiles for sample I with > 4200 concordant SNPs, whereas the three remaining myBaits methods (myBaits-A, -D, and -E) generated partial profiles with slightly fewer concordant SNPs (3400–3800). AmpliSeq-B and AmpliSeq-E methods produced partial profiles (1000–2500 SNPs), although ≈ 20–30% of them were discordant. The remaining AmpliSeq methods (AmpliSeq-A, -C, and -D) yielded < 75 SNPs from sample I. The xGen assay produced almost 3000 concordant SNPs and 231 discordant SNPs from this degraded sample.

Sample L spiked with humic acid resulted in > 4100 concordant SNPs from nearly all methods. The exceptions were AmpliSeq-C with 1516 concordant SNPs, AmpliSeq-E with 3370 concordant SNPs, and xGen with 3744 concordant SNPs. The three myBaits methods that produced relatively lower SNP calls in other experiments (myBaits-A, -D, and -E) yielded 3500–4000 concordant SNPs.

Altogether, the data demonstrate that all four FORCE assays were able to overcome humic acid inhibition, but DNA degradation resulted in variable call rates. Assay type had a significant impact on the call rate for the two degraded samples H and I ($p = 0.0001$). Pairwise

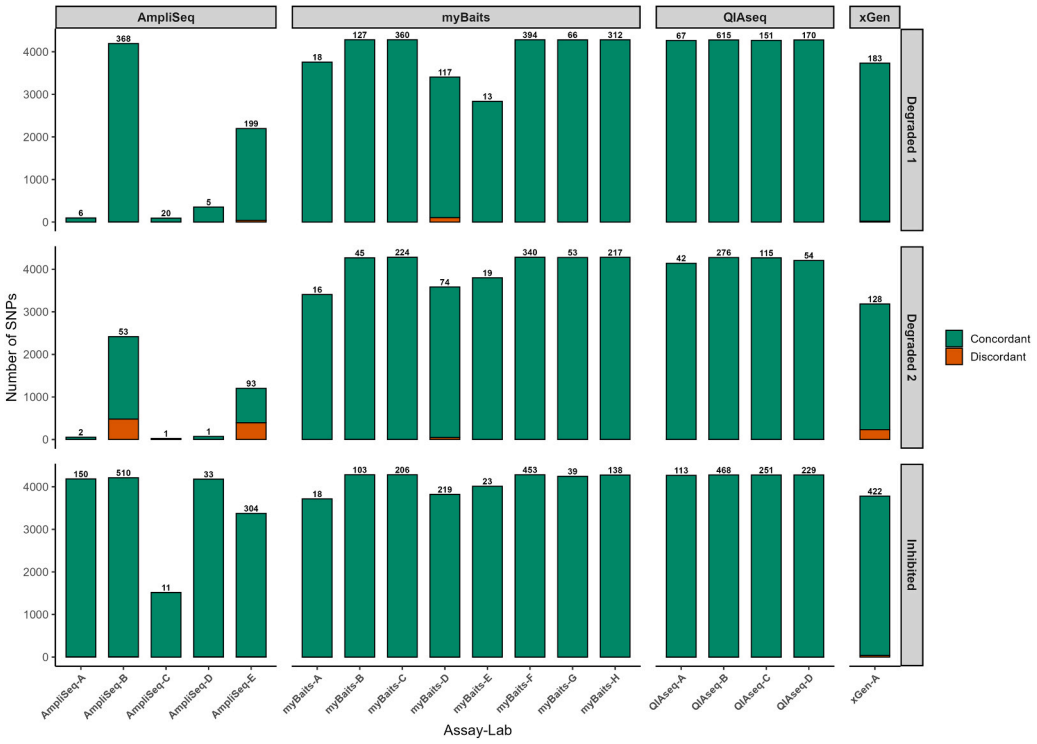

Fig. 6. Column graphs showing the number of called autosomal SNPs for the inhibited and degraded samples by method. Degraded 1 (sample H, NIST deg1) had a mean fragment size of 200 bp. Degraded 2 (sample I, NIST deg2) had a mean fragment size of 150 bp. Inhibited (sample L) was spiked with humic acid. The green shading indicates concordant SNPs, while the red shading indicates discordant SNPs. Detailed counts of concordant and discordant SNP genotypes, together with the rs identifications of discordant SNPs, are available in the Supplementary File S4.

comparisons showed significant differences ($p < 0.01$) between AmpliSeq and myBaits assays, and also between AmpliSeq and QIAseq assays, while other pairings were not significantly different. QIAseq, followed by myBaits, produced the highest number of concordant SNP calls from degraded DNA in the 150–200 bp size range, and these results were significantly higher than those obtained with AmpliSeq.

3.5. Negative controls

In total, 17 negative controls (NCs) were analyzed from 13 methods (NC data for five methods was not generated and/or provided to NBFM for analysis). Thirteen of the 17 NCs (76%) had no called SNPs, and three of the 17 NCs had up to six called SNPs (Supplementary File S7). The remaining NC, which was one of two NCs from myBaits-F, had 310 called SNPs consistent with a high-quality, highly concentrated sample prepared in the same library set. As with the drop-in observed in the myBaits-F samples F and G data, it is likely that the contamination of this NC occurred prior to or during library preparation. These findings underscore the necessity of batching samples of similar quality/quantity as well as establishing appropriate thresholds for control interpretation given the sensitivity of these FORCE assays.

4. Summary and conclusion

The present study assessed the ability to produce FORCE genotypes using four different assays: AmpliSeq, myBaits, QIAseq, and xGen, with different workflow conditions chosen by the laboratories to suit their individual preferences. The results showed that all four assays were capable of generating FORCE data from high quality samples; however, SNP call rates were inconsistent and dependent on the method employed. In particular, the total read count for a sample was shown to be an important factor influencing SNP recovery, with methods yielding fewer reads per sample exhibiting lower call rates. Aside from read count, other factors led to consistently poorer results from certain methods, which may be due to reagent issues, lack of optimization, and/or laboratory experience with these assays. Though not the specific aim of this interlaboratory study, the results did highlight the strengths and weaknesses of the four assays. The AmpliSeq assay proved to be the most sensitive with low DNA input; however, it is worth noting that AmpliSeq (and xGen) methods retained their PCR duplicates but were analyzed at the same read depth threshold, which likely contributed to the observed higher sensitivity of the AmpliSeq assay in this study. Moreover, QIAseq and xGen demonstrated the highest call rates from control DNA, whereas myBaits and QIAseq exhibited the lowest proportions of non-concordant SNPs. Due to their design, the QIAseq and myBaits assays proved to be the most robust when handling degraded DNA samples. The xGen assay is the newest of the FORCE assays and was only tested by a single laboratory. Efforts to further optimize both the xGen assay and laboratory method may improve the results. Overall, future studies to improve the consistency of results within each assay would be beneficial so that the forensic community can adopt these powerful SNP technologies with equal expectations of success irrespective of preparation method or sequencing platform.

Our results suggest that careful assay selection and standardized workflows will be important if FORCE assays are applied in forensic settings beyond the laboratories involved in the present study. Kinship and databasing applications, with higher quality samples and higher throughput expectations, may prefer amplicon-based FORCE implementation as these workflows are generally cheaper and faster. Conversely, casework applications that involve lower quality samples and degraded DNA may benefit from single primer extension or capture-based FORCE methods to maximize successful results. The unique flexibility of the FORCE panel will hopefully lead to broad use within the forensic community, fostering further research and collaboration on SNP-based methods for human identification.

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CRediT authorship contribution statement

Adam Staadig: Writing – review & editing, Methodology, Investigation. **Kimberly Sturk-Andreaggi:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Project administration, Methodology, Investigation, Conceptualization. **Andreas Tillmar:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Software, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Reet Järving:** Writing – review & editing, Methodology, Investigation. **Claus Børsting:** Writing – review & editing, Methodology, Investigation. **Erik F. Bergseth:** Writing – review & editing, Methodology, Investigation. **Charla Marshall:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Investigation, Conceptualization. **David Ballard:** Writing – review & editing, Methodology, Investigation. **Christina Amory:** Writing – review & editing, Methodology, Investigation. **Odile Loreille:** Writing – review & editing, Methodology, Investigation. **Maria Victoria Lareu:** Writing – review & editing, Methodology, Investigation. **Jonathan L. King:** Writing – review & editing, Methodology, Investigation. **Kevin M. Kiesler:** Writing – review & editing, Methodology, Investigation. **Vania Pereira:** Writing – review & editing, Methodology, Investigation. **Walther Parson:** Writing – review & editing, Methodology, Investigation. **Melissa K. Muenzler:** Writing – review & editing, Methodology, Investigation. **Dennis McNevin:** Writing – review & editing, Methodology, Investigation. **Kelly Grisedale:** Writing – review & editing, Methodology, Investigation. **Erin M. Gorden:** Writing – review & editing, Methodology, Investigation. **Katherine Butler Gettings:** Writing – review & editing, Methodology, Investigation. **María de la Puente:** Writing – review & editing, Methodology, Investigation. **Maarja Sadam:** Writing – review & editing, Methodology, Investigation. **Jorge Ruiz-Ramírez:** Writing – review & editing, Methodology, Investigation. **Sheree Hughes:** Writing – review & editing, Methodology, Investigation. **Rachel Houston:** Writing – review & editing, Methodology, Investigation. **Jennifer Daniels-Higginbotham:** Writing – review & editing, Methodology, Investigation. **Peter M. Vallone:** Writing – review & editing, Methodology, Investigation. **Monika Stoljarova-Bibb:** Writing – review & editing, Methodology, Investigation. **Carolyn R. Steffen:** Writing – review & editing, Methodology, Investigation. **Jennifer Snedeker:** Writing – review & editing, Methodology, Investigation. **Daniel Kling:** Writing – review & editing, Methodology, Investigation. **Catarina Xavier:** Writing – review & editing, Methodology, Investigation. **Jessica Watson:** Writing – review & editing, Methodology, Investigation. **Jodie Ward:** Writing – review & editing, Methodology, Investigation. **Christopher Phillips:** Writing – review & editing, Methodology, Investigation. **Lucinda Davenport:** Writing – review & editing, Methodology, Investigation. **Michael Coble:** Writing – review & editing, Methodology, Investigation. **Selena Cisana:** Writing – review & editing, Investigation.

Ethics statement

The study was conducted according to the guidelines of the Declaration of Helsinki. NA12877 and NA12878 DNA samples were obtained from the Coriell Institute for Medical Research and used in accordance with the applicable material transfer agreement. The study was approved by the Swedish Ethical Review Authority (2024–07682–01) and the Defense Health Agency Office of Research Protections on November 20, 2020 (Protocol # DHQ-20–2073).

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.fsigen.2026.103513.

References

[1] B. Sobrino, A. Carracedo, SNP typing in forensic genetics: a review, Methods Mol. Biol. 297 (2005) 107–126, https://doi.org/10.1385/1-59259-867-6:107.
[2] B. Bruijns, R. Tiggelaar, H. Gardeniers, Massively parallel sequencing techniques for forensics: a review, Electrophoresis 39 (2018) 2642–2654, https://doi.org/10.1002/elps.201800082.
[3] E.M. Gorden, K. Sturk-Andreaggi, C. Marshall, Capture enrichment and massively parallel sequencing for human identification, Forensic Sci. Int Genet 53 (2021) 102496, https://doi.org/10.1016/j.fsigen.2021.102496.
[4] A. Amorim, L. Pereira, Pros and cons in the use of SNPs in forensic kinship investigation: a comparative analysis with STRs, Forensic Sci. Int 150 (2005) 17–21, https://doi.org/10.1016/j.forsciint.2004.06.018.
[5] N.M.M. Novroski, J.C. Cihlar, Evolution of single-nucleotide polymorphism use in forensic genetics, WIREs Forensic Sci. 4 (2022) e1459, https://doi.org/10.1002/wfs2.1459.
[6] C. Genomes Project, A. Auton, L.D. Brooks, R.M. Durbin, E.P. Garrison, H.M. Kang, J.O. Korbel, J.L. Marchini, S. McCarthy, G.A. McVean, et al., A global reference for human genetic variation, Nature 526 (2015) 68–74, https://doi.org/10.1038/nature15393.
[7] C.V. Van Hout, I. Tachmazidou, J.D. Backman, J.D. Hoffman, D. Liu, A.K. Pandey, C. Gonzaga-Jauregui, S. Khalid, B. Ye, N. Banerjee, et al., Exome sequencing and characterization of 49,960 individuals in the UK Biobank, Nature 586 (2020) 749–756, https://doi.org/10.1038/s41586-020-2853-0.
[8] J.J. Sanchez, C. Phillips, C. Borsting, K. Balogh, M. Bogus, M. Fondevila, C. D. Harrison, E. Musgrave-Brown, A. Salas, D. Syndercombe-Court, et al., A multiplex assay with 52 single nucleotide polymorphisms for human identification, Electrophoresis 27 (2006) 1713–1724, https://doi.org/10.1002/elps.200500671.

[9] A.J. Pakstis, W.C. Speed, J.R. Kidd, K.K. Kidd, Candidate SNPs for a universal individual identification panel, Hum. Genet 121 (2007) 305–317, https://doi.org/10.1007/s00439-007-0342-2.
[10] I. Grandell, R. Samara, A.O. Tillmar, A SNP panel for identity and kinship testing using massive parallel sequencing, Int J. Leg. Med. 130 (2016) 905–914, https://doi.org/10.1007/s00414-016-1341-4.
[11] F. Guo, Y. Zhou, H. Song, J. Zhao, H. Shen, B. Zhao, F. Liu, X. Jiang, Next generation sequencing of SNPs using the HID-Ion AmpliSeq Identity Panel on the Ion Torrent PGM platform, Forensic Sci. Int. Genet 25 (2016) 73–84, https://doi.org/10.1016/j.fsigen.2016.07.021.
[12] A.C. Jager, M.L. Alvarez, C.P. Davis, E. Guzman, Y. Han, L. Way, P. Walichiewicz, D. Silva, N. Pham, G. Caves, et al., Developmental validation of the MiSeq FGx Forensic Genomics System for Targeted Next Generation Sequencing in Forensic DNA Casework and Database Laboratories, Forensic Sci. Int. Genet 28 (2017) 52–70, https://doi.org/10.1016/j.fsigen.2017.01.011.
[13] C. Phillips, J. Amigo, A.O. Tillmar, M.A. Peck, M. de la Puente, J. Ruiz-Ramirez, F. Bittner, S. Idrizbegovic, Y. Wang, T.J. Parsons, et al., A compilation of tri-allelic SNPs from 1000 Genomes and use of the most polymorphic loci for a large-scale human identification panel, Forensic Sci. Int. Genet 46 (2020) 102232, https://doi.org/10.1016/j.fsigen.2020.102232.
[14] J. Antunes, P. Walichiewicz, E. Forouzmand, R. Barta, M. Didier, Y. Han, J. C. Perez, J. Snedecor, C. Zlatkov, G. Padmabandu, et al., Developmental validation of the ForenSeq(R) Kintelligence kit, MiSeq FGx(R) sequencing system and ForenSeq Universal Analysis Software, Forensic Sci. Int Genet 71 (2024) 103055, https://doi.org/10.1016/j.fsigen.2024.103055.
[15] D. McNevin, J. Watson, K. Grisedale, A. Dahal, C. Goodwin, J. Ward, Comparison of commercial targeted amplicon sequencing assays for human remains identification casework, Int J. Leg. Med. 139 (2025) 49–60, https://doi.org/10.1007/s00414-024-03335-8.
[16] A. Tillmar, K. Sturk-Andreaggi, J. Daniels-Higginbotham, J.T. Thomas, C. Marshall, The FORCE panel: an all-in-one SNP marker set for confirming investigative genetic genealogy leads and for general forensic applications, Genes 12 (2021), https://doi.org/10.3390/genes12121968.
[17] W. Parson, A. Alterauge, C. Amory, S. Heinze, S. Holzl, R.G. Jahn, C. Lehn, T. Sanger, C. Xavier, A. Tillmar, et al., Remains of the German outlaw Johannes Buckler alias Schinderhannes identified by an interdisciplinary approach, Forensic Sci. Int Genet 78 (2025) 103276, https://doi.org/10.1016/j.fsigen.2025.103276.
[18] L.L. Kotchey, S. Lee, L. Nangeroni, J.T. Thomas, A. Staadig, A. Tillmar, K. Sturk-Andreaggi, C. Marshall, M. Ghemrawi, Investigating SNP typing using alternative reference materials with the FORCE panel and QIAseq(R) chemistry, Forensic Sci. Int Genet 78 (2025) 103294, https://doi.org/10.1016/j.fsigen.2025.103294.
[19] A. Staadig, J. Hedman, A. Tillmar, Applying Unique Molecular Indices with an Extensive All-in-One Forensic SNP Panel for Improved Genotype Accuracy and Sensitivity, Genes 14 (2023), https://doi.org/10.3390/genes14040818.
[20] M. Sanga, L. Boiso, H. Lindsten, P. Rådström, R. Ansell, J. Hedman, A panel of PCR-inhibitory reference materials for quality evaluation of multiplex STR analysis kits, Forensic Sci. Int. Genet. Supp. Series 5 (2015), https://doi.org/10.1016/j.fsigss.2015.09.126.
[21] R.C. Team, R: A language and environment for statistical computing, R Foundation for Statistical Computing, Vienna, Austria, 2021.
[22] J.M. Zook, B. Chapman, J. Wang, D. Mittelman, O. Hofmann, W. Hide, M. Salit, Integrating human sequence data sets provides a resource of benchmark SNP and indel genotype calls, Nat. Biotechnol. 32 (2014) 246–251, https://doi.org/10.1038/nbt.2835.
[23] E. Samorodnitsky, B.M. Jewell, R. Hagopian, J. Miya, M.R. Wing, E. Lyon, S. Damodaran, D. Bhatt, J.W. Reeser, J. Datta, et al., Evaluation of hybridization capture versus amplicon-based methods for whole-exome sequencing, Hum. Mutat. 36 (2015) 903–914, https://doi.org/10.1002/humu.22825.
[24] M.A. Quail, M. Smith, P. Coupland, T.D. Otto, S.R. Harris, T.R. Connor, A. Bertoni, H.P. Swerdlow, Y. Gu, A tale of three next generation sequencing platforms: comparison of Ion Torrent, Pacific Biosciences and Illumina MiSeq sequencers, BMC Genom. 13 (2012) 341, https://doi.org/10.1186/1471-2164-13-341.
[25] J.L. Watson, K. Grisedale, D. McNevin, J. Ward, Evaluation of the ForenSeq(R) kintelligence kit and the forensic capture enrichment panel for unidentified and missing persons casework, Int J. Leg. Med. 139 (2025) 2047–2062, https://doi.org/10.1007/s00414-025-03492-4.