ORIGINAL ARTICLE

Supervised home-based telerehabilitation for idiopathic pulmonary fibrosis: A pragmatic service evaluation in routine care with detraining follow-up

VASILEIOS T. STAVROU[1,2,3], KONSTANTINOS KORAKAS[3], GLYKERIA TSIRIMONA[2], ELENI KARETSI[3], ANTONIOS CHAROKOPOS[1], ZOE DANIIL[3]

[1]Medical School, University of Cyprus, Cyprus; [2]Unique Safe Tele Exercise Project – Institute for Digital Health and Chronic Diseases, Greece; [3]Laboratory of Cardio-Pulmonary Testing and Pulmonary Rehabilitation, Department of Respiratory Medicine, Faculty of Medicine, University of Thessaly, Greece.

ABSTRACT

Background and aim: In this pragmatic, uncontrolled service evaluation in clinically stable idiopathic pulmonary fibrosis (IPF), we assessed primary service outcomes (feasibility and safety) and prespecified exploratory within-participant changes following a four-week supervised home-based tele-exercise program via the USTEP platform and a four-week detraining period.

Methods: Thirteen participants were assessed at three prespecified time points (baseline: T0; post-intervention: T1; post-detraining: T2) using the six-minute walk test (6MWT), SF-36, HADS, and pulse–respiration quotient (PRQ). Primary service outcomes were captured as session attendance/adherence and exercise-related adverse events.

Results: Adherence was 92% (median 11/12 sessions completed) and there were no exercise-related adverse events. The 6MWT distance increased by 8.4% from T0–T1 (Bonferroni-adjusted p=0.004, d=-1.15) but declined towards baseline at T2. SF-36 physical health scores increased at T1 (+5.2%) but decreased at T2 (Bonferroni-adjusted T1–T2 p=0.004, d=1.17). For several exploratory patient-reported outcomes (fatigue, pain and HADS), Bonferroni-adjusted comparisons were non-significant, while unadjusted testing suggested small T0–T1 changes;

Received: 14 November 2026 | Accepted: 23 February 2026

Correspondence: Vasileios T. Stavrou, PhD / Special Teaching Staff in Respiratory and Exercise Physiology, Medical School, University of Cyprus, Nicosia, Cyprus; / E-mail: vasileiosstavrou@hotmail.com, ORCID: 0000-0002-2437-5339

these are reported as exploratory signals. PRQ changed over time ($\chi^2(2)=8.60$, p=0.014), with a shift at T1 and a return to baseline at T2.

Conclusions: In this small uncontrolled service evaluation, the program was feasible and safe and within-participant improvements were observed in 6MWT distance and selected patient-reported and physiological measures. These findings are exploratory and hypothesis-generating and should be confirmed in adequately powered controlled studies that include prespecified maintenance strategies.

Key words: six-minute walk test, emotional well-being, pulse-respiration quotient, home-based intervention

Introduction

Idiopathic pulmonary fibrosis (IPF) is a chronic, progressive fibrotic interstitial lung disease of unknown etiology that primarily affects adults aged 50 years and over. It exhibits the histological pattern of usual interstitial pneumonia (1) and presents exertional dyspnea and a dry cough. It has an incidence rate of 6.8–16.3 per 100,000 people per year, with a male predominance, and the median survival rate is 3–5 years (2). Risk factors include smoking, environmental exposure, gastro-esophageal reflux and mutations in telomere biology and surfactant proteins (3). The pathogenesis involves recurrent epithelial micro-injury in genetically susceptible individuals, driving fibroblast activation and the production of excess extracellular matrix, as well as aberrant wound healing with limited inflammation (4). AEC2 dysfunction, epithelial-mesenchymal transition and dysregulated developmental pathways also contribute to the pathogenesis (5, 6). IPF physiologically causes restriction (reduced FVC/TLC) and impaired diffusion (low DLCO), with mean annual declines of ~2.8% and 2.9% in FVC and DLCO, respectively, despite the use of antifibrotics (2, 7). Although guidelines endorse pulmonary rehabilitation, access is often limited due to geography, oxygen dependence and exacerbation risk, so there is a need for supervised, lower-burden delivery models.

Telerehabilitation provides a structured, home-based format for supervised exercise. For patients with chronic respiratory diseases, including IPF, pulmonary rehabilitation (PR) is an essential adjunct to pharmacotherapy. Meta-analyses report an average improvement of ~44 meters in the 6-minute walk test, clinically meaningful reductions in dyspnea, and improvements in health-related quality of life (e.g. physical functioning and fatigue) (8–12). Higher habitual physical activity independently predicts better survival in these populations (13, 14). However, uptake of PR is limited by geographical, logistical and institutional barriers. Home-based tele-exercise can mitigate these constraints, particularly for patients in remote areas (15). Emerging evidence in IPF shows that well-structured, supervised tele-rehabilitation yields exercise tolerance and quality-of-life outcomes comparable to center-based programs (16). In line with this, the latest ATS/ERS PR guidelines strongly recommend offering telerehabilitation alongside in-person PR as an evidence-based option for patients with chronic respiratory diseases, including interstitial lung diseases (17). Due to the progressive nature of IPF and the limited range of available pharmacotherapies, maximizing exercise capacity, physical function and quality of life is a clinical priority. Although center-based pulmonary rehabilitation is beneficial for IPF patients, evidence for supervised home-based telerehabilitation is limited due to heterogeneous protocols and a lack of long-term data (9). We therefore undertook a pragmatic, real-world service evaluation of a supervised, home-based telerehabilitation program embedded in routine care. This comprised four weeks of supervised tele-exercise, followed by a four-week observation/detraining phase. The primary service outcomes were feasibility and safety, while metrics of the service delivery

process (including fidelity) were summarized descriptively. The prespecified clinical and physiological outcomes were exploratory and examined as part of routine clinical monitoring to describe within-participant changes and inform future controlled evaluations.

Materials and methods

Participants

We conducted a prospective service evaluation using data from our single-center ILD clinic IPF database (the 'Stavrou' cohort; final analysis n=13, Figure 1), including clinically stable patients consecutively referred to the Interstitial Lung Disease Clinic at the General University Hospital of Larissa between November 2023 and January 2024 who completed the post-detraining (T2) assessment. All patients were referred to a supervised, home-based telerehabilitation program as part of routine care, with eligibility determined by clinical criteria and patient preference. Baseline demographics and clinical data are presented in Table 1. As this was a pragmatic service evaluation embedded in routine care rather than a controlled efficacy study, no formal sample size or power calculation was performed. All outcome analyses were prespecified as exploratory; accordingly, we report effect sizes and MCID-based responder proportions to support interpretation, and we emphasize that the study was not powered for confirmatory efficacy inference. Inclusion criteria were: age 50–80 years; IPF confirmed by HRCT and multidisciplinary review in accordance with guidelines (1); clinical stability for at least six weeks (no exacerbation, new or worsening symptoms, infection, hospitalization, medication change, or functional decline); independent ambulation (with or without aids); absence of CPET contraindications (18); resting SpO_2 ≥85%; access to the required technology and internet (19); and written medical approval. Exclusion criteria were: acute exacerbation; significant respiratory infection; hospitalization; symptomatic

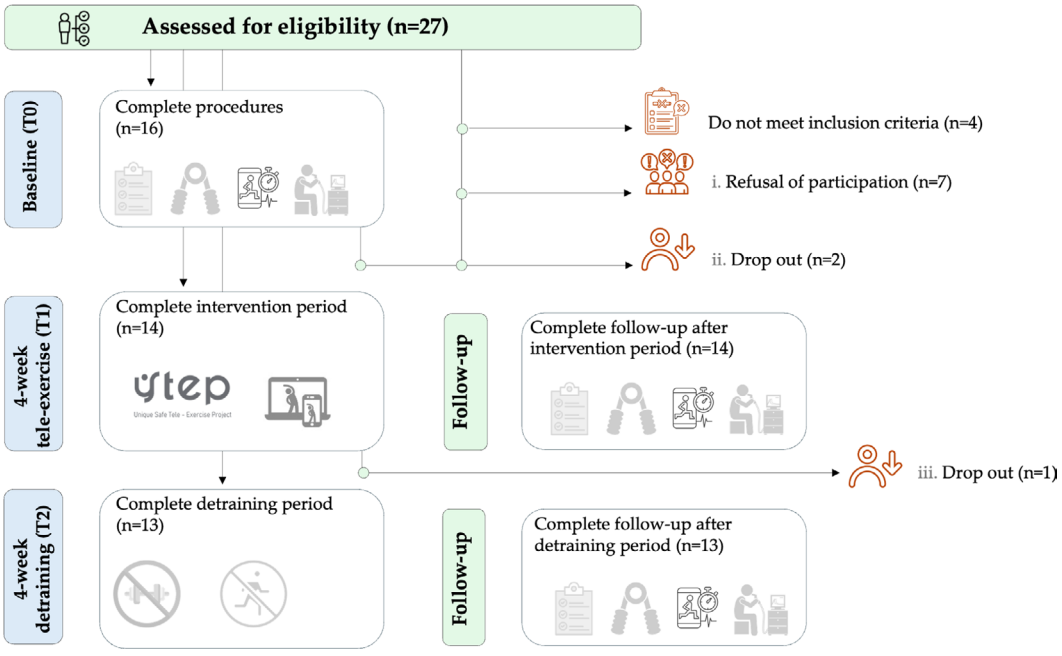

Figure 1. Service uptake and follow-up flow and assessment timeline (T0–T2). A total of 27 individuals were assessed for eligibility; 4 did not meet inclusion criteria and 7 declined participation. Sixteen completed baseline procedures (T0), of whom 2 withdrew before starting the intervention. Fourteen completed the 4-week supervised telerehabilitation and were assessed post-intervention (T1). One participant declined to enter the detraining phase, leaving 13 who completed the 4-week detraining and the post-detraining (T2) assessment. Final analysis: n=13.

Table 1. Patients' demographic characteristics.

Variable, Unit	Overall	Comorbidities	n
Age, years	68.6±7.0	Asthma	5
Gender, Male, n	9	Rheumatoid arthritis	4
Diagnosis IPF, years	6.3±3.8	Coronary heart disease	3
FEV_1, % predicted	95.9±22.5	Diabetes mellitus	1
FVC, % predicted	93.2±23.5	Hypothyroidism	1
PEF, % predicted	94.7±23.5	Gouty arthritis	2
MEF 25-75, % predicted	92.4±31.4		
TLC, % predicted	71.8±17.2		
DLCO SB, % predicted	55.7±20.0		

Abbreviations: DLCO SB: diffusing capacity of the lung for carbon monoxide using the single-breath method; FEV1: forced expiratory volume in 1st s; FVC: forced vital capacity; MEF 25-75: maximal mean expiratory flow between 25-75% of exhaled FVC; PEF: peak expiratory flow; TLC = total lung capacity.

deterioration or medication change within six weeks; severe resting hypoxemia (SpO_2 <85%); any CPET contraindication; participation in structured exercise/pulmonary rehabilitation within the prior three months; inability to ambulate independently; or inability to use the telehealth platform/internet.

Study design

This was a pragmatic pre-post service evaluation with no randomization, control group, or research-assigned treatment. The usual care pathway spanned eight weeks: four weeks of supervised tele-exercise followed by a four-week observation (detraining) period without supervised sessions. Sessions were delivered via videoconferencing with real-time remote supervision and weekly adjustments based on clinical status and tolerance. Assessments were obtained at three prespecified time points (baseline=T0, post-intervention=T1, follow-up=T2) as part of routine clinical monitoring. Primary service outcomes were feasibility and safety, including recruitment/uptake and retention, session attendance/adherence and completion, and exercise-related adverse events; service delivery process metrics (e.g., technical issues, acceptability, and fidelity to the planned dose/progression) were summarized descriptively. Clinical and physiological outcomes were prespecified as exploratory, including within-participant changes in 6MWT, SF-36, dyspnea severity, resting arterial oxygen saturation, HADS, and pulse-respiration quotient (PRQ). As a pragmatic service evaluation embedded in routine care, trial registration was not applicable.

Ethics and data protection

Ethical approval was obtained from the IEC the University of Thessaly (No. 514; 6 November 2023). All participants provided written informed consent in accordance with the Declaration of Helsinki, as well as consent for the processing of their personal data under EU legislation (20). Digital informed consent was also recorded via the USTEP platform, in compliance with the EU GDPR. As this was a service evaluation conducted within routine care, trial registration was not required.

Measurements

The anthropometric characteristics and body mass of all participants were recorded, and the body surface area (BSA) = $\sqrt{height_{(cm)}} \times body\ mass_{(kg)}/3600$ and body mass index (BMI) = $body\ mass_{(kg)}/height_{(cm)}^2$ were calculated. The six-minute walk test (6MWT)

was used to assess functional capacity (21). Specific measurements included: i. oxygen saturation (SpO_2, Nonin 9590 Onyx Vantage, USA) and heart rate (HR, chest belt with Bluetooth and ANT+ technology) at baseline and at one-minute intervals during the test and during the first minute of recovery (22); ii. cardiopulmonary parameters (Cosmed Quark CPET, Italy), blood pressure (Mac, Japan), and Borg scales (dyspnea and leg fatigue, CR-10), which were recorded before, immediately after, and during the first minute of recovery following the 6MWT (23). Based on previous studies (24-27), we made the following calculations in Table 2.

Prior to the 6MWT, all participants performed a handgrip strength test using an electronic dynamometer (Camry EH101, South El Monte, CA, USA), as previously described (28) and, prior to the physical fitness tests, all subjects completed the 36-item Short Form Survey Instrument to assess self-reported health-related quality of life (SF-36) (29), as well as a self-rating questionnaire to measure anxiety and depression (HADS) (30).

Intervention program

Participants completed a four-week tele-exercise program consisting of three 30-40-minute sessions per week on Mondays, Wednesdays and Fridays. The program was delivered via secure, real-time videoconferencing (Viber) and integrated with the USTEP Institute platform to enable standardized data capture, remote supervision and GDPR-compliant storage. Sessions were led by exercise specialists and overseen by a clinical exercise physiologist. This was followed by a 4-week detraining phase without supervised training. Each session comprised: (i) breathing techniques/functional mobility (30% of the session; 30-40 s sets with 60 s rest; 50–60% HR_{max} referenced to the 6MWT); (ii) aerobic intervals (40%; home walking in a safe, ventilated area; 40–60% HR_{max} or Borg dyspnea scale 4-6; 2-4 bouts of 40–90 s aiming for 90-100% of each participant's end-6MWT HR, followed by 1 min recovery until SpO_2 >90%); (iii) strength/proprioception (30%; multi-joint bodyweight exercises at RPE 11-15/20; 2-6 sets of 8–16 reps; 60-90 s rest; weekly progression by tolerance). Participants monitored their HR and SpO_2 using personal oximeters, smartwatches or apps, and reported their rating of perceived exertion (RPE) in real time. These values were verbalized during sessions and logged in USTEP forms. The clinical exercise physiologist reviewed weekly symptoms and performance, as well as HR/SpO_2 and SpO_2/RPE ratios, adjusting the dose using predefined thresholds. Safety criteria: sessions were paused or the intensity was reduced if SpO_2 was below 88%, if there was a drop of at least 4% from baseline, if HR was above 85% of the predicted maximum HR, if the Borg dyspnea score was 7 or above, if there was chest pain or dizziness. Exercise resumed once SpO_2 was above 90% and the RPE was acceptable, with supplemental oxygen titrated as needed.

Statistical analysis

Normality was assessed using the Shapiro-Wilk test. Data are presented as n (%) for categorical

Table 2. Equations for calculating cardiorespiratory and functional parameters.

mean arterial blood pressure (mmHg)	systolic blood pressure(mmHg)+2 x diastolic blood pressure(mmHg)/3
oxygen breath (mL)	oxygen consumption ($\dot{V}O_2$, mL·min-1) / breath frequency (f_β, 1/min)
breathing reserve (%)	ventilation ($\dot{V}_E$, L/min) / maximal voluntary ventilation (MVV, L/min)
pulse respiration quotient	heart rate (HR, bpm) / breath frequency (f_β, 1/min)
predicted distance covered during the 6MWT(m)	Men = [7.57 × height (cm)] – [5.02 × age (yrs)] – [1.76 × body mass (kg)] – 309 Women = [2.11 × height (cm)] – [2.29 × body mass (kg)] – [5.78 × age (yrs)] + 667
oxygen uptake predicted ($mL \cdot min^{-1}$)	Men = [height (cm) – age (yrs)] × 20 Women = [height (cm) – age (yrs)] × 14

variables, mean±SD for normally distributed continuous variables, and median (25^{th}–75^{th} percentile) for non-normally distributed variables. All inferential analyses used a complete-case approach. For repeated-measures comparisons across T0-T2, only participants with outcome data available at all assessed time points were included (i.e. T2 completers). As no outcome data were missing across T0-T2, the complete-case set corresponds to the full sample (n=13). As the study involved three repeated measurements (T0, T1 and T2) within a single group, the Friedman test was used to evaluate the overall effect of time. When the Friedman test was significant, Wilcoxon signed-rank tests were used for pairwise comparisons with Bonferroni-adjusted p-values. Effect sizes were estimated using Cohen's d for within-subject changes and Kendall's W for the Friedman test (W ~0.1/0.3/0.5 denotes small/moderate/large effects, respectively). In addition to p-values, we reported the proportion of participants exceeding prespecified minimum clinically important differences (6MWT ≥30 m; SF-36 PCS ≥5 points) to support clinical interpretation. Given the large number of outcomes and the small sample size, statistical inference is interpreted descriptively and as exploratory. Although Bonferroni adjustment was applied to pairwise comparisons within variables following a significant Friedman test, no global multiplicity correction was performed across all outcomes/domains; therefore, nominal p-values should be interpreted cautiously. Bonferroni-adjusted post hoc comparisons were considered the primary pairwise inference within each outcome. Where Bonferroni-adjusted contrasts were non-significant, any additional unadjusted Wilcoxon p-values are reported only as nominal/exploratory signals and are not interpreted as confirmatory significant effects. Analyses were conducted in IBM SPSS Statistics 21 with a two-sided α of 0.05.

Results

The results are presented with the primary service outcomes (feasibility and safety) reported first, followed by the prespecified exploratory clinical and physiological outcomes across T0–T2. Baseline demographic and clinical characteristics are presented in Table 2. Adherence was 92%, and no exercise-related adverse events occurred during the supervised sessions. The prespecified exploratory clinical and physiological outcomes are summarized in Tables 2–5. Table 2 summarizes changes in anthropometry and morphology before and after the four-week intervention and subsequent four-week detraining period. Table 3 reports quality-of-life outcomes across the three time points. Table 4 presents cardiopulmonary, metabolic, and hemodynamic parameters, while Table 5 shows the correlations between physical fitness and psychosocial measures. Figures 2–6 depict cardiorespiratory changes over time.

Table 3. Results of anthropometric characteristics across the three phases.

	Baseline (T0)		After 4-week training (T1)		After 4-week detraining (T2)		Cohen's d (T0 vs T1; T0 vs T2; T1 vs T2)	Friedman's p-value
Variable, Unit	**M±Sd**	**95% CI**	**M±Sd**	**95% CI**	**M±Sd**	**95% CI**		
Body mass index, kg/m^2	27.8±3.7	25.6, 30.0	27.8±3.7	25.6, 30.0	28.8±3.9	26.4, 31.1	0.01; 0.44; 0.43	0.011
Body surface area, m^2	1.8±0.2	1.68, 1.97	1.8±0.2	1.68, 1.98	1.8±0.2	1.68, 1.97	0.00; 0.00; 0.00	p>0.05
Neck circumference, cm	36.6±3.4	34.6, 38.7	37.2±4.1	34.8, 39.7	37.1±3.2	35.1, 39.0	0.16; 0.14; -0.03	p>0.05
Δchest, cm	5.2±1.3	4.4, 5.9	5.2±1.4	4.4, 6.1	5.4±1.5	4.5, 6.3	0.00; -0.07; -0.07	p>0.05
Waist-to-hip ratio	0.94±0.1	0.88, 1.00	0.97±0.1	0.92, 1.02	0.96±0.1	0.90, 1.02	0.30; 0.20; -0.10	p>0.05

Δchest: chest circumference difference between maximal inhalation and exhalation

Table 4. Results of the 36-item Short Form Survey Instrument for self-reported health-related quality of life.

	Baseline (T0)		After 4-week training (T1)		After 4-week detraining (T2)		Cohen's d (T0 vs T1; T0 vs T2; T1 vs T2)	Friedman's p-value
Variable, Unit	**M±Sd**	**95% CI**	**M±Sd**	**95% CI**	**M±Sd**	**95% CI**		
Physical functioning, score	53.1±7.2	48.7, 57.4	54.6±5.6	51.3, 58.0	53.1±3.8	50.8, 55.4	0.23; 0.00; -0.21	p>0.05
Physical health, score	67.7±11.5 ‡	60.8, 74.6	71.2±9.4 #	65.5, 76.8	46.2±17.2	35.8, 56.6	0.34; -1.41; -1.17	<0.001
Emotional problems, score	71.8±26.8	55.7, 88.0	79.6±21.6	66.5, 92.7	77.1±21.0	64.4, 89.8	0.30; 0.22; -0.09	p>0.05
Energy-fatigue, score	51.2±4.2	48.6, 53.7	53.1±4.3	50.4, 55.7	52.3±4.8	49.4, 55.2	0.64; 0.43; 0.31	0.042
Emotional well-being, score	61.2±3.4	59.2, 63.3	62.2±3.1	60.3, 64.0	61.8±3.5	59.7, 64.0	0.26; 0.15; -0.12	p>0.05
Social functioning, score	69.2±11.0	62.6, 75.9	71.2±9.4	65.5, 76.8	71.2±9.4	65.5, 76.8	0.24; -0.06; -0.29	p>0.05
Pain, score	67.7±8.7	62.4, 73.0	72.9±13.1	65.0, 80.8	72.7±12.1	65.4, 80.0	0.65; 0.63; 0.14	0.018
General health, score	51.5±3.8	49.3, 53.8	51.5±3.8	49.3, 53.8	51.9±3.8	49.6, 54.2	0.03; 0.05; 0.02	p>0.05
Health change, score	42.3±12.0	35.1, 49.6	44.2±11.0	37.6, 50.9	44.2±11.0	37.6, 50.9	0.16; 0.16; 0.00	p>0.05

* p< 0.05 between baseline and after 4-week training; ‡ p< 0.05 between baseline and after 4-week detraining; # p< 0.05 between after 4-week training and after 4-week detraining.

Psychosocial responses

The SF-36 Physical Health score showed a significant time effect ($\chi^2(2)=15.85$, $p<0.001$). Scores increased slightly from T0 to T1 (+5.2%; 67.7 to 71.2, an increase of 3.5 points), before dropping at T2 (a decrease of 31.7% compared to T0 and 35.1% compared to T1). Bonferroni: T0–T2: p=0.024; T1–T2: p=0.004; T0–T1: p=1.000. The effect sizes were d = 1.41 (T0–T2), 1.17 (T1–T2) and 0.34 (T0–T1). The SF-36 Energy–Fatigue score differed across time ($\chi^2(2)=6.33$, p=0.042): +3.7% from T0 to T1; then –1.5% from T1–T2, but +2.1% from T0 to T2. Bonferroni-adjusted pairwise tests were non-significant (T0–T1 p=0.424; T0–T2 p=1.000; T1–T2 p=1.000). Exploratory Wilcoxon testing suggested a T0–T1 increase (Z=–2.236, p=0.025), while other comparisons were non-significant (p=0.180; p=0.157). Effect sizes were d=0.64 (T0–T1), 0.43 (T0–T2) and 0.31 (T1–T2). SF-36 pain scores also differed across time ($\chi^2(2)=8.00$, p=0.018): +7.7% from T0 to T1; T2 remained +7.4% versus baseline. Bonferroni-adjusted pairwise tests were non-significant (T0–T1 p=1.000; T0–T2 p=0.233; T1–T2 p=0.233). Exploratory Wilcoxon testing suggested reductions from T0–T1 (Z=–2.165, p=0.030) and from T0–T2 (Z=–2.126, p=0.033), while T1–T2 was non-significant (p=0.655). Effect sizes were d=0.65 (T0–T1), 0.63 (T0–T2) and 0.14 (T1–T2). HADS scores differed over time ($\chi^2(2)=8.38$, p=0.015; Kendall's W=0.35). The means were 12.9±2.4 (95% CI 11.5–14.4), 12.5±1.9 (11.3–13.6; –3.1%), and 12.6±2.2 (11.3–13.9; –2.3% vs. T0; +0.8% vs. T1). Bonferroni-adjusted pairwise tests were non-significant (p>0.05). Exploratory Wilcoxon testing suggested small decreases from T0–T1 (Z=–2.121, p=0.034) and from T0–T2 (Z=–2.000, p=0.046), which should be interpreted cautiously given outcome multiplicity; T1–T2 was non-significant (Z=–1.414, p=0.157). Cohen's d was 0.70 (T0–T1, moderate), 0.64 (T0–T2, moderate), and –0.41 (T1–T2, small).

Table 5. Results of cardio-pulmonary-metabolic-hemodynamic parameters.

		Baseline (T0)		After 4-week training (T1)		After 4-week detraining (T2)		Cohen's d	
Stages of 6MWT	**Variable, Unit**	**M±Sd**	**95% CI**	**M±Sd**	**95% CI**	**M±Sd**	**95% CI**	**(T0 vs T1; T0 vs T2; T1 vs T2)**	**Friedman's p-value**
Resting	$\dot{V}O_2$, mL·min^{-1}	247.8±97.5	188.9, 306.7	251.1±56.5	216.9, 285.2	243.3±39.2	219.6, 267.0	–0.03; 0.04; 0.14	p>0.05
	$\dot{V}CO_2$, mL·min^{-1}	201.3±81.7	151.9, 250.7	230.9±60.6	194.3, 267.6	221.1±43.8	194.6, 247.5	–0.29; –0.21; 0.15	p>0.05
	Breath frequency, 1/min	15.3±3.4 ‡	13.3, 17.3	18.2±6.4	14.6, 21.8	18.7±4.0	16.2, 21.1	-0.44; -0.64, -0.11	0.049
	$P_{ET}CO_2$, mmHg	30.1±7.9	25.8, 34.4	31.0±3.4	29.2, 32.8	29.7±3.8	27.6, 31.8	–0.15; 0.06; 0.36	p>0.05
	$P_{ET}O_2$, mmHg	110.2±10.3	103.8, 116.5	113.1±5.1	110.0, 116.2	115.1±5.3	111.9, 118.4	0.40; 0.58; 0.27	p>0.05
	Heart Rate, bpm	74.2±11.7	67.1, 81.2	74.1±12.2	66.7, 81.5	69.8±9.2	64.2, 75.3	0.01; 0.51; 0.39	p>0.05
	MAP, mmHg	91.0±6.6	87.4, 94.6	87.4±6.4	83.9, 90.9	92.5±8.9	87.6, 97.3	0.56; –0.19; –0.65	p>0.05
End of test	$\dot{V}O_2$, mL·min^{-1}	772.7±296.8	593.3, 952.1	855.2±374.9	628.6, 1081.7	847.8±315.7	657.1, 1038.6	–0.22; –0.18; 0.03	p>0.05
	$\dot{V}CO_2$, mL·min^{-1}	612.9±224.1	491.1, 734.7	710.8± 284.8	556.0, 865.6	710.5±251.6	573.7, 847.2	–0.38; –0.41; 0.00	p>0.05
	Breath frequency, 1/min	20.7±5.1	17.9, 23.4	24.4±8.4	19.8, 29.0	21.2±9.3	16.1, 26.2	–0.53; –0.06; 0.36	p>0.05
	$P_{ET}CO_2$, mmHg	32.9±7.4	28.9, 36.9	35.2±3.4	33.4, 37.1	35.8±2.6	34.4, 37.3	–0.40; –0.53; –0.20	p>0.05
	$P_{ET}O_2$, mmHg	108.1±9.5	102.4, 113.8	107.1± 6.0	103.5, 110.7	105.4±3.7	103.1, 107.6	0.10; 0.26; 0.31	p>0.05
	Heart Rate, bpm	101.3±16.1	91.6, 111.0	94.6±15.0	85.6, 103.7	97.7±13.1	89.8, 105.6	0.51; 0.25; –0.25	p>0.05
	MAP, mmHg	99.7±4.7	97.1, 102.3	94.8±6.4	91.4, 98.3	101.4±10.2	95.8, 106.9	0.71; –0.17; –0.53	p>0.05
1st min recovery	$\dot{V}O2$, mL·min^{-1}	341.1±155.9	256.3, 425.8	410.5±126.5	341.7, 479.2	405.1±84.3	359.2, 450.9	2.59; 2.71; –0.27	p>0.05
	$\dot{V}CO_2$, mL·min^{-1}	295.4±131.8	215.7, 375.1	371.8±124.3	296.7, 446.9	390.9±106.1	326.8, 455.1	-0.60; -0.80; -0.17	p>0.05
	Breath frequency, 1/min	22.0±4.7	19.4, 24.6	21.1±7.3	17.1, 25.1	20.8±6.7	17.1, 24.4	0.15; 0.21; 0.04	p>0.05
	$P_{ET}CO_2$, mmHg	29.9±6.6,	26.3, 33.5	33.8±4.1	31.6, 36.1	31.9±3.9	29.8, 34.0	–0.71; –0.44; 0.48	p>0.05
	$P_{ET}O_2$, mmHg	114.0±8.2	109.5, 118.5	110.2±7.3	106.3, 114.2	114.7±5.2	111.9, 117.5	0.53; –0.08; –0.73	p>0.05
	Heart Rate, bpm	79.2±11.9	72.0, 86.4	82.5±13.4	74.3, 90.6	77.8±11.7	70.8, 84.9	-0.32; 0.13; 0.37	p>0.05
	MAP, mmHg	90.2±5.8	86.6, 93.7	89.9±4.0	87.5, 92.4	91.2±7.4	86.4, 96.0	0.16; –0.27; –0.36	p>0.05

Abbreviations: MAP: mean arterial pressure; $P_{ET}CO_2$: end-tidal partial pressure of carbon dioxide; $P_{ET}O_2$: end-tidal partial pressure of oxygen; $\dot{V}CO_2$: carbon dioxide production; $\dot{V}O_2$: oxygen consumption. * p< 0.05 between baseline and after 4-week training; ‡ p< 0.05 between baseline and after 4-week detraining; # p< 0.05 between after 4-week training and after 4-week detraining.

Physical fitness indicators

The 6MWT distance (Figure 2) differed over time ($\chi^2(2)=12.13$, p=0.002; Kendall's W=0.47). Means: T0: 520.8±84.8 m (95% CI: 469.6-572.0); T1: 564.6±101.6 m (503.2-626.0; +44 m, +8.4%); T2: 530.0±111.4 m (462.7-597.3; +1.8% vs. T0; -6.1% vs. T1). Bonferroni pairwise: T0-T1: p=0.004; T1-T2: p=0.056; T0-T2: p=1.000. Notably, the T1-T2 contrast did not reach statistical significance after adjustment (p=0.056), despite a moderate effect size estimate, and should be interpreted cautiously. The MCID of ≥30 m was achieved by 8/13 participants (61.5%). Cohen's d: -1.15 (T0-T1; large effect size), 0.80 (T1-T2; moderate effect size) and -0.19 (T0-T2; small effect size). Overall, the observed change in 6MWT distance and MCID responder proportion suggest potential clinical relevance; however, uncertainty remains substantial in this small uncontrolled sample and the findings should be interpreted descriptively and as hypothesis-generating. The percent-predicted 6MWT also differed over time ($\chi^2(2)=9.18$, p=0.010; Kendall's W=0.35). The means were 111.2±18.3% (95% CI 100.1-122.3), 119.9±17.1% (109.6-130.2; +7.8%), and 112.5±21.1% (99.7–125.3; +1.2% vs. T0 and -6.2% vs. T1). Bonferroni pairwise: T0-T1: p=0.004; T1-T2: p=0.056; T0-T2: p=1.000. Cohen's d: -0.50 (T0-T1, moderate), 0.63 (T1-T2, moderate) and -0.14 (T0-T2, small). No significant changes were observed in SpO_2 (Figure 3) or heart rate (Figure 4). Dyspnea at the end of the 6MWT was non-significant ($\chi^2(2)=3.50$, p=0.174), despite the following decreases: 0.73±1.27 (95% CI -0.13-1.58), 0.27±0.90 (-63.0% vs. T0; 95% CI -0.33-0.88) and 0.09±0.30 (-87.7% vs. T0; 95% CI -0.11-0.29). Leg fatigue was also non-significant ($\chi^2(2)=0.80$, p=0.670): 0.45±1.21 (95% CI -0.36-1.27) to 0.55±1.21 (+22.2%; 95% CI -0.27-1.36) to 0.36±0.92 (-20.0% vs. T0; -34.5% vs. T1; 95% CI -0.26-0.98). Handgrip strength showed no time effect ($\chi^2(2)=4.32$, p=0.115; Kendall's W=0.17): 29.9±7.9 kg (95% CI 25.6-34.2) to 29.9±8.5 kg (25.3-34.5) to 29.4±8.2 kg (25.0–33.9; -1.7% vs. T0-T1).

Oxygen uptake kinetics

Oxygen breath at 1-minute recovery (Figure 5) differed across T0–T2 ($\chi^2(2)=6.17$, p=0.046; effect size=0.29). The means increased from T0 to T1 and were similar at T2: from 16.3±7.9 (95% CI 12.0–20.6) to 21.1±8.6 (95% CI 16.4–25.7; +29.4%) to 21.2±7.5 (95% CI 17.1–25.3; +30.1% vs. baseline). Bonferroni-adjusted post hoc tests were non-significant (T0–T1: p=0.148; T0–T2: p=0.144; T1–T2: p=1.000), while exploratory Wilcoxon testing suggested an increase from T0 to T1 (Z=–2.000, p=0.046). Cohen's d was –0.49 (T0–T1; moderate effect size), –0.58 (T0–T2; moderate effect size) and –0.03 (T1–T2; negligible effect size). Given outcome multiplicity and the small sample, these findings should be interpreted cautiously as exploratory signals. Figure 6 shows that

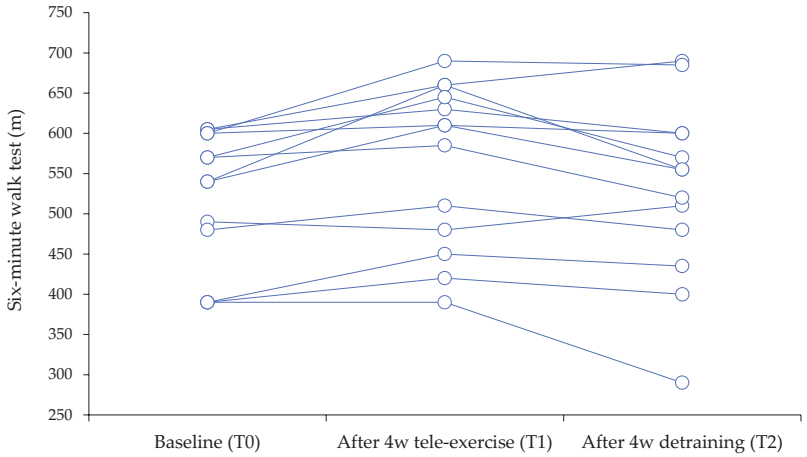

Figure 2. Results of covered distance during the 6MWT at three time points.

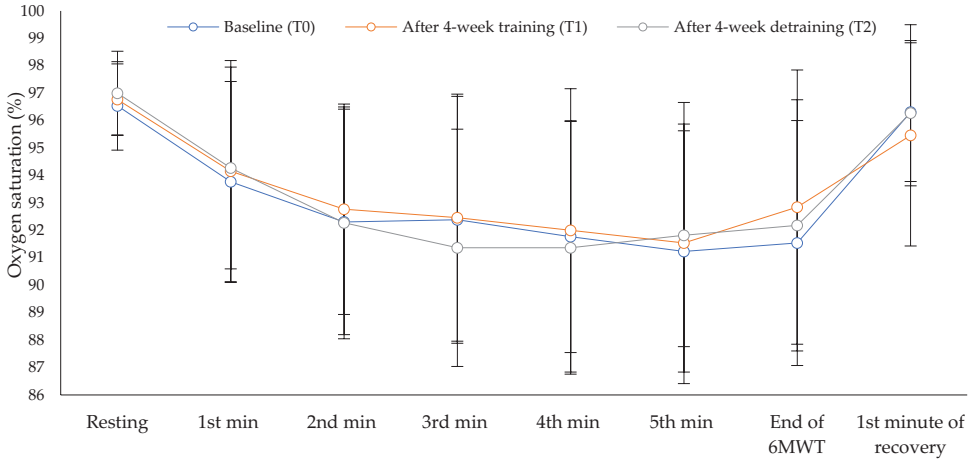

Figure 3. Oxygen saturation results during the 6MWT at three time points.

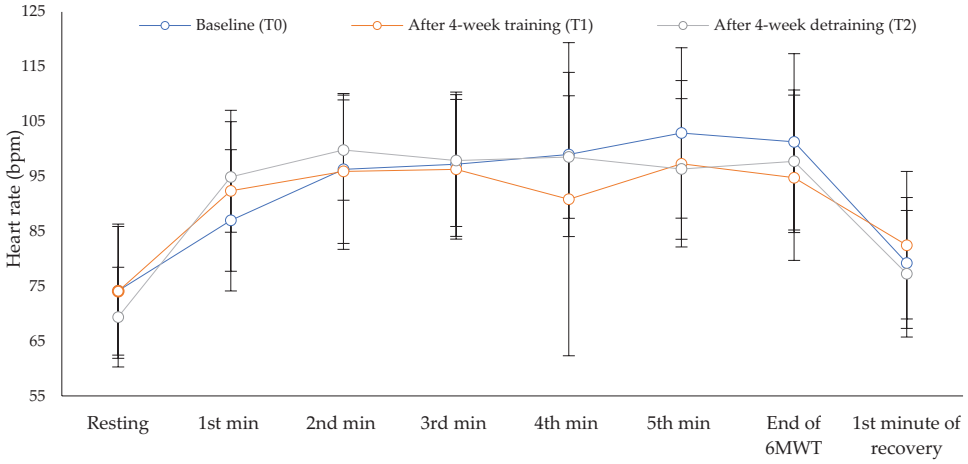

Figure 4. Heart rate results during the 6MWT at three time points.

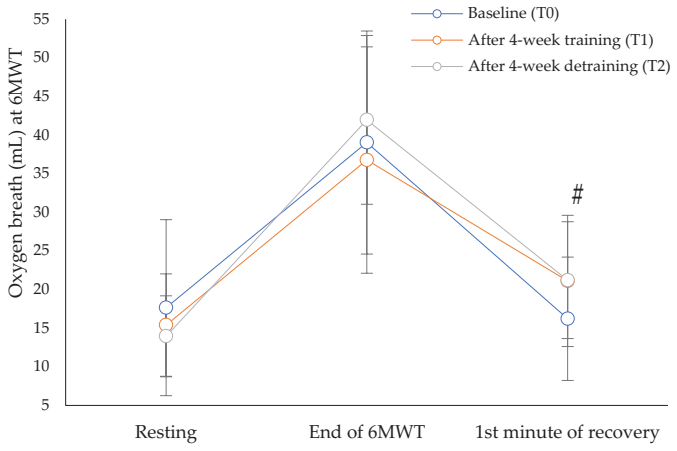

Figure 5. Results in oxygen breath between the three time points during a 6MWT. # $p < 0.05$.

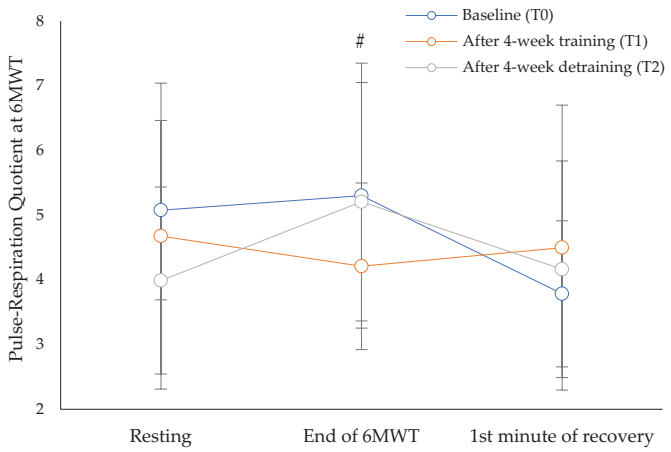

Figure 6. Results in pulse-respiration quotient between the three time points during a 6MWT. # p<0.05.

Table 6. Results of exploratory correlations between biopsychosocial parameters.

Variable, Unit		Baseline	After 4-week training	After 4-week detraining
HADS, score	6MWT (m)	r=-0.574, p=0.040	r=-0.584, p=0.036	r=-0.583, p=0.036
Physical functioning, score	6MWT (m)	r=0.632, p=0.021	r=0.698, p=0.021	p>0.05
	$f_\beta/\dot{V}O_{2\ rest}$ (mL)	p>0.05	r=0.686, p=0.010	p>0.05
	$f_\beta/\dot{V}O_{2\ end\text{-}6MWT}$ (mL)	p>0.05	r=0.759, p=0.003	p>0.05
	$\dot{V}_E/MVV_{\ end\text{-}6MWT}$ (%)	p>0.05	p>0.05	r=-0.626, p=0.022
Physical health, score	6MWT (m)	r = 0.604, p=0.029	r=0.697, p=0.008	p>0.05
	$\dot{V}O_{2\ end\text{-}6MWT}$ (mL·min^{-1})	p>0.05	r=0.583, p=0.037	p>0.05
	$f_\beta/\dot{V}O_{2\ end\text{-}6MWT}$ (mL)	p>0.05	r=0.660, p=0.014	p>0.05
	$\dot{V}_E/MVV_{\ end\text{-}6MWT}$ (%)	p>0.05	p>0.05	r=-0.679, p=0.011
Energy-fatigue, score	6MWT (m)	p>0.05	r=0.588, p=0.035	r=0.685, p=0.010
	$\dot{V}O_{2\ end\text{-}6MWT}$ (mL·min^{-1})	p>0.05	r=0.563, p=0.045	p>0.05
	$f_\beta/\dot{V}O_{2\ end\text{-}6MWT}$ (mL)	p>0.05	r=0.627, p=0.022	p>0.05
Emotional well-being, score	6MWT (m)	r=0.658, p=0.015	p>0.05	r=0.631, p=0.021
	$\dot{V}O_{2\ end\text{-}6MWT}$ (mL·min^{-1})	p>0.05	r=0.565, p=0.044	p>0.05
	$f_\beta/\dot{V}O_{2\ rest}$ (mL)	p>0.05	r=0.583, p=0.037	p>0.05
	$f_\beta/\dot{V}O_{2\ end\text{-}6MWT}$ (mL)	p>0.05	r=0.697, p=0.011	p>0.05
Social functioning, score	$f_\beta/\dot{V}O_{2\ end\text{-}6MWT}$ (mL)	p>0.05	r=0.644, p=0.017	p>0.05
	$\dot{V}_E/MVV_{\ end\text{-}6MWT}$ (%)	p>0.05	p>0.05	r=-0.618, p=0.024
General health, score	$f_\beta/\dot{V}O_{2\ rest}$ (mL)	p>0.05	r=0.671, p=0.012	p>0.05
	$f_\beta/\dot{V}O_{2\ end\text{-}6MWT}$ (mL)	p>0.05	r=0.583, p=0.037	p>0.05

Abbreviations: 6MWT: six-minute walk test; $f_\beta/\dot{V}O_2$: oxygen breath; $\dot{V}_E/MVV$: breathing reserve; $\dot{V}O_2$: oxygen consumption.

the end-6MWT PRQ at T0, T1 and T2 differed significantly ($\chi^2(2)=8.60$, p=0.014). The mean value decreased from T0–T1 (from 5.2±2.2, 95% CI 4.1–6.4 to 4.2±1.3, 95% CI 3.4–4.9; a decrease of 19.2%), then increased at T2 (to 5.3±1.8, 95% CI 4.3–6.3; an increase of 26.2% compared to T1 and 1.9% compared to baseline). Bonferroni-adjusted pairwise tests were non-significant for T0–T1 (p=0.122) and T0–T2 (p=1.000), but significant for T1–T2 (p=0.022). The effect sizes were d=0.61 (T0–T1, moderate), d=–0.04 (T0–T2, negligible) and d=–1.08 (T1–T2, large).

Discussion

To our knowledge, this is the first study to investigate differences in the psychophysical status of patients with IPF following a four-week tele-exercise rehabilitation program and a subsequent four-week detraining period. Our findings revealed different adjustments in physical fitness, psychosocial measures and autonomic breathing regulation at the three time points. Given the pragmatic uncontrolled pre–post design, the observed changes cannot be causally attributed to the intervention and should be interpreted descriptively and as hypothesis-generating. Alternative explanations, including measurement/learning effects (e.g., serial 6MWT familiarity) and temporal variability, cannot be excluded.

Adaptive effects of exercise training in IPF

Following the four-week, home-based, tele-exercise program, we observed an increase in 6MWT distance within participants (mean +44 m; +8.4% from T0 to T1), as well as a reduction in end-6MWT PRQ. Meanwhile, $\dot{V}O_2$ remained unchanged. However, as this was an uncontrolled pre-post evaluation and learning effects, test–retest variability and pacing differences were not formally controlled (e.g. via repeated baseline tests or standardized cadence monitoring), the 6MWT change should be interpreted as a promising, albeit non-confirmatory, clinical signal rather than as evidence of efficacy. Peak $\dot{V}O_2$ averaged 45.2±13.0% of the predicted value, $\dot{V}_E$/MVV was approximately 27%, and the 6MWT SpO_2 nadir decreased by 6.0±3.6 percentage points across time points. Consistent with IPF pathophysiology thickened, fibrotic interstitium and reduced DLCO (mean 55.7% predicted) gas exchange is diffusion-limited, especially during exercise, yielding exertional hypoxemia and widened alveolar–arterial gradients (31). Ventilation–perfusion mismatch further aggravates hypoxemia during exertion, contributing to exercise intolerance and dyspnea. In this context, the increase in walking distance despite an unchanged $\dot{V}O_2$ is plausible: over four weeks, it is unlikely that there will be a significant improvement in central oxygen delivery in a population limited by gas exchange, whereas peripheral adaptations can accrue (greater skeletal muscle oxidative capacity, capillary recruitment and enhanced tissue oxygen extraction). There were no significant changes in dyspnea, hemodynamics, SpO_2, leg fatigue or handgrip strength, suggesting that submaximal training may have desensitized central dyspnea perception and improved tolerance at similar physiological strain, aligning with prior reports (32–36). As the intervention emphasized lower-limb activation to optimize cardiovascular responses (34), gains in upper-limb strength were not anticipated. Overall, the observed change in 6MWT distance (+44 m) and the proportion exceeding the MCID (8/13; 61.5%) suggest potential clinical relevance. However, this small uncontrolled sample introduces substantial uncertainty, meaning the findings should be interpreted descriptively and as hypothesis-generating.

Mechanistic insights

In this uncontrolled service evaluation, we observed an increase in 6MWT distance within participants, while several physiological and symptom-related measures ($\dot{V}O_2$, SpO_2, dyspnea, leg fatigue and hemodynamics) remained broadly unchanged. This dissociation can be explained in multiple ways and should not be attributed to a single underlying mechanism. Alternative explanations include a true improvement in functional performance, test familiarity/learning effects, day-to-day variability, differences in pacing or motivation across assessments, and measurement noise or the limited sensitivity of the indices used. Against this background, previous studies have identified mitochondrial dysfunction as a driver of disease progression and exercise intolerance in IPF (32). Repeated

epithelial injury and chronic oxidative stress may disrupt mitochondrial biogenesis, ATP production, reactive oxygen species control and mitophagy (37). In skeletal muscle, such inefficiencies could limit aerobic energy availability, hasten fatigue, reduce oxygen utilization and elevate lactate, potentially increasing ventilatory drive and perceived exertion (34). Within this conceptual framework, repeated, submaximal, lower-limb-focused stimuli could be associated with improved peripheral oxidative capacity and oxygen extraction. This would enable greater walking distance at a similar ventilatory/perceptual load. The observed PRQ shift may be consistent with altered cardiorespiratory coupling at a given workload. However, as PRQ is an indirect index and autonomic pathways were not measured directly, a mechanistic interpretation remains speculative in this setting. Training has also been proposed to influence the neural processing of dyspnea-related signals (e.g. insula, anterior cingulate and limbic circuitry), potentially modifying perceptual responses to a given ventilatory demand (33). Such adaptations have been suggested to relate to improved metabolic efficiency and delayed peripheral fatigue, potentially via reduced lactic acidosis and ventilatory/brainstem drive (32, 33). However, these mechanisms were not assessed directly in the present evaluation. Overall, the pattern of 6MWT gains alongside stable SpO_2 and leg fatigue is compatible with this interpretive framework but requires further research. The attenuation of several outcomes after detraining may reflect reduced training exposure and/or the partial reversal of peripheral and neural adaptations. However, it is not possible to determine the relative contribution of these factors here. These observations highlight the potential benefit of pre-specified maintenance strategies, such as ongoing low-frequency supervision or booster sessions, to sustain short-term gains.

Parasympathetic breathing and cardiorespiratory integration

As reported by Stavrou et al. (23), who linked the benefits of exercise to the respiratory metaboreflex and autonomic regulation, our program's structured breathing plus submaximal intervals (three times a week for 35 minutes over four weeks) may be consistent with changes in autonomic balance and cardiorespiratory regulation reported in prior training literature (e.g., vagal predominance and baroreflex/chemoreflex resetting), while causal inference is not possible here. This dosage is compatible with neuromodulator adaptations that emerge within weeks. Consistent correlations among HADS, performance, quality-of-life subdomains and cardiorespiratory measures support a link between autonomic tone, symptom burden and functional capacity in a home setting. During the detraining period (Table 4), the effects diminished: the 'oxygen breath' improvement was retained (+30.1% vs baseline), whereas the PRQ score returned to pre-intervention levels (+1.9%), suggesting that peripheral ventilatory efficiency may persist briefly without supervision, while autonomic coupling is more unstable. Oxygen breath indexes capture metabolic return per breath, while PRQ captures cardiorespiratory synchrony. Together, they provide complementary, potentially informative signals for the remote titration of intensity and recovery (track intra-individual PRQ deltas across T0, T1 and T2, alongside HR, SpO_2 and RPE). The literature indicates that training can raise the metaboreflex activation threshold, lessen dyspnea and peripheral fatigue, and improve autonomic stability via enhanced parasympathetic drive and optimized breathing mechanics (23, 38). Repeated controlled exertion also desensitizes dyspnea networks (insula, ACC and limbic) and improves mitochondrial efficiency with reduced afferent load (33). Vagus-supportive breathing patterns can reduce anxiety and increase vagal outflow (39), although the possibility of placebo/Hawthorne effects resulting from structured video contact cannot be excluded (40). Operationally, embedding PRQ and oxygen-breath-based thresholds in telerehabilitation dashboards can standardize decisions (maintain, intensify, pause, recover or schedule boosters) without disrupting home delivery. The PRQ should be interpreted as an exploratory research signal, rather than as a validated standalone monitoring or clinical decision-making tool.

Clinical implications

Supervised telerehabilitation could complement routine IPF care by improving access, providing

supervised home progression under safety monitoring and offering a practical alternative when center-based pulmonary rehabilitation is unavailable or challenging to attend. In this small, uncontrolled service evaluation, the program was delivered with high feasibility and safety (median 11/12 sessions; 92% adherence; no exercise-related adverse events) (Table 7). We observed within-participant improvements in 6MWT distance (T0–T1: +44 m; +8.4%), alongside exploratory signals in selected patient-reported and physiological measures (e.g., SF-36 physical health and PRQ), while several secondary outcomes did not remain significant after Bonferroni adjustment. These results should be interpreted cautiously, as they are hypothesis-generating. In particular, the secondary outcomes with non-significant Bonferroni-adjusted pairwise contrasts are presented as exploratory signals and should not be interpreted as evidence that the treatment is effective. The USTEP platform enabled remote supervision, standardized data capture and timely adjustment of the exercise dose. This may help clinicians to individualize prescriptions and identify participants who might require escalation to center-based oversight or protocol intensification. Notably, eight out of thirteen participants (61.5%) exceeded the published MCID for the 6MWT (≥30 m). Responder-based metrics may be useful for future protocol refinement and comparative study design, rather than as standalone decision rules. Broader telehealth experience in other chronic disease contexts supports the value of remote monitoring and structured follow-up (41). Finally, the partial regression of several outcomes during detraining highlights the need for pre-specified maintenance strategies (e.g. ongoing low-frequency supervision or brief 'booster' sessions) to sustain short-term gains.

Durability and detraining

A key finding of this service evaluation was that several changes were not sustained once supervision ceased. Although SF-36 physical health scores increased modestly at T1, they declined significantly at T2 (Bonferroni-adjusted differences for T1-T2 and T0-T2), suggesting that perceived physical health and functioning are particularly susceptible to the withdrawal of structured support in this population. Similarly, PRQ showed regression during detraining, with the greatest difference observed between T1 and T2. Although causal inference and mechanistic interpretation are limited (PRQ is indirect and the study is uncontrolled), the deterioration in both a patient-reported global physical health measure and an integrative physiological signal is clinically informative. Together, these findings suggest that short tele-exercise programs may provide short-term functional and subjective benefits, but maintenance strategies are

Table 7. Practical takeaways for clinicians

Measure	How to use it in practice
Functional exercise capacity (6MWT)	Use the T0-T1 change to adjust walking speed/intervals. Escalate to center-based PR if no gains are observed
Health-related quality of life (SF-36)	Track physical health to set patient-relevant goals and monitor functional recovery
Anxiety/depression (HADS)	If anxiety or depression does not improve, provide brief psychological support or make a referral to enhance adherence
Physiological regulation (PRQ)	Monitor within-participant PRQ trends alongside HR, SpO_2 and RPE as an adjunct signal of cardiorespiratory coupling during remote supervision. If PRQ trends worsen alongside other clinical signals (e.g., symptoms, SpO_2, HR, and RPE), consider reviewing the pacing/breathing strategy, supervision level, or whether an in-person reassessment is required. PRQ should be considered an exploratory research signal and is not a validated monitoring or clinical decision-making tool.
Care pathway	Early responders can continue remotely, but non-responders or those who decline after detraining may require an in-person review or booster sessions

likely necessary to sustain these benefits in the long term. Pragmatically, this could include low-frequency follow-up supervision, 'booster' sessions or integration into longer-term, home-based activity plans. Such strategies should be evaluated in controlled studies with standardized outcome assessment and prespecified maintenance protocols.

Limitations, strengths, and generalizability

To our knowledge, this is the first prospective study to test an organized, home-based tele-exercise program for IPF that focuses on cardiopulmonary and autonomic indices. The use of objective physiological measurements and the inclusion of a detraining phase provide novel, clinically relevant insights into short-term adaptations. Validated measures of fatigue, dyspnea, anxiety and functional capacity enhance the study's methodological rigor. The study was highly feasible and safe (median 11/12 sessions; 92% adherence; no exercise-related adverse events). As this was an uncontrolled service evaluation involving a small sample size and multiple outcomes, the study is underpowered for making inferences about efficacy and causal attribution. Therefore, all statistical findings should be interpreted as exploratory and hypothesis-generating. The short intervention and detraining periods, and the absence of a structured maintenance ('booster') phase, may underrepresent the effects in the long term. The inclusion of clinically stable, tech-literate participants introduces selection bias, and some outcomes did not improve. Multiple outcomes and pairwise tests increase the risk of a type I error. Even when Bonferroni's method is used to adjust for within-variable pairwise comparisons, the overall pattern of findings should be interpreted cautiously as a means of generating hypotheses. Unblinded assessments, consumer-grade HR/SpO_2 monitoring, potential learning effects in serial 6MWTs, and environmental/seasonal factors during remote testing may introduce measurement error. Specifically, we did not include repeated baseline 6MWTs to quantify or mitigate practice effects, nor did we standardize or objectively measure pacing or cadence (e.g. step rate) at different time points. Therefore, some of the observed changes in the 6MWT could reflect familiarity with the test or variability in pacing rather than a true training effect. The applicability to other ILDs and the equity of access (technology requirements) remain uncertain. Further research is needed in the form of longer, controlled, multicenter trials with prespecified maintenance phases, subgroup analyses (sex, baseline aerobic capacity and psychological status), stratification by early 6MWT MCID response (≥30 m) and standardized monitoring to reduce variability.

Recommendations for future research

Future studies should use multicenter randomized or cluster-randomized designs with active comparators and extended follow-up periods to test durability and generalizability. Protocols and analysis plans should be preregistered, multiplicity control should be prespecified, adherence to the CONSORT extension for pilot/feasibility work should be ensured, and a graphic abstract should be included for clarity. Supervision can be improved using a minimal set of wearable devices (HR, SpO_2, step cadence) plus derived indices (PRQ, oxygen breath), with predefined safety/adjustment thresholds and data quality checks. Outcomes should incorporate MCID-based responder endpoints (e.g. 6MWT ≥30 m, SF-36 PCS ≥5 points, HADS ≥2–3 points), standardized patient-reported experience measures (e.g. satisfaction, ease of use) and engagement metrics. To ensure equity and access, more diverse and less digitally literate populations should be recruited, and enabling strategies such as loaned equipment, caregiver training, language-adapted materials and connectivity support should be tested. Economic evaluations should report ICERs, budget impact and sensitivity analyses from the perspectives of payers and society, considering avoided travel time, equipment reuse and health service utilization. To facilitate implementation and scaling up, collaborate with public health systems and national PR frameworks, employ implementation science methods and co-design, and incorporate a predefined maintenance/booster phase to mitigate T1-T2 detraining. Consider adaptive designs that are stratified by an early MCID response (≥30 m

in the 6MWT) and compare booster schedules (weekly vs. biweekly) and physiological triggers (PRQ or oxygen-breath thresholds). Mechanistic work should include lactate threshold and NIRS-derived oxyhemoglobin changes to differentiate peripheral oxidative adaptations from central perceptual recalibration (42).

Conclusions

This pragmatic, uncontrolled service evaluation indicated that a short, supervised, home-based tele-exercise program was feasible and safe for clinically stable IPF patients. The program was associated with improvements in 6MWT distance, fatigue, and PRQ, while several physiological/gas-exchange domains remained unchanged. Several outcomes partially regressed after the detraining period, highlighting the potential need for prespecified maintenance strategies. Given the small sample size, uncontrolled pre–post design, and outcome multiplicity, these results should be interpreted as descriptive and hypothesis-generating and require confirmation in adequately powered controlled studies.

Acknowledgements: The authors would like to express their sincere appreciation to the patients who participated in this study for their invaluable cooperation. They would also like to thank the USTEP Institute for their invaluable assistance in developing the tele-exercise rehabilitation programs and supporting the project

Conflict of interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

Declaration on the use of AI: No generative AI was used in the preparation of this manuscript.

Consent for publication: All participants provided written informed consent.

References

1. Raghu G, Collard HR, Egan JJ, et al. An official ATS/ERS/JRS/ALAT statement: idiopathic pulmonary fibrosis: evidence-based guidelines for diagnosis and management. Am J Respir Crit Care Med. 2011;183(6):788-824. doi: 10.1164/rccm.2009-040GL.
2. Sankari A, Chapman K, Ullah S. Idiopathic pulmonary fibrosis. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025.
3. Richeldi L, Collard HR, Jones MG. Idiopathic pulmonary fibrosis. Lancet. 2017;389(10082):1941-52. doi: 10.1016/S0140-6736(17)30866-8.
4. Barratt SL, Creamer A, Hayton C, Chaudhuri N. Idiopathic pulmonary fibrosis (IPF): an overview. J Clin Med. 2018;7(8):201. doi: 10.3390/jcm7080201.
5. King TE Jr, Bradford WZ, Castro-Bernardini S, et al. A phase 3 trial of pirfenidone in patients with idiopathic pulmonary fibrosis. N Engl J Med. 2014;370(22):2083-92. doi: 10.1056/NEJMoa1402582.
6. Leask A, Abraham DJ. TGF-beta signaling and the fibrotic response. FASEB J. 2004;18(7):816-27. doi: 10.1096/fj.03-1273rev.
7. Neely ML, Hellkamp AS, Bender S, et al. Lung function trajectories in patients with idiopathic pulmonary fibrosis. Respir Res. 2023;24(1):209. doi: 10.1186/s12931-023-02503-5.
8. Spruit MA, Singh SJ, Garvey C, et al. An official American Thoracic Society/European Respiratory Society statement: key concepts and advances in pulmonary rehabilitation. Am J Respir Crit Care Med. 2013;188(8):e13-64. doi: 10.1164/rccm.201309-1634ST.
9. Cox IA, Borchers Arriagada N, de Graaff B, et al. Health-related quality of life of patients with idiopathic pulmonary fibrosis: a systematic review and meta-analysis. Eur Respir Rev. 2020;29(158):200154. doi: 10.1183/16000617.0154-2020.
10. Gloeckl R, Teschler S, Jarosch I, Christle JW, Hitzl W, Kenn K. Comparison of two- and six-minute walk tests in detecting oxygen desaturation in patients with severe chronic obstructive pulmonary disease-a randomized crossover trial. Chron Respir Dis. 2016;13(3):256-63. doi: 10.1177/1479972316636991.
11. Vainshelboim B. Exercise training in idiopathic pulmonary fibrosis: is it of benefit? Breathe (Sheff). 2016;12(2):130-8. doi: 10.1183/20734735.006916.
12. Edwards GD, Polgar O, Patel S, et al. Mood disorder in idiopathic pulmonary fibrosis: response to pulmonary rehabilitation. ERJ Open Res. 2023;9(3):00585-2022. doi: 10.1183/23120541.00585-2022.
13. Wallaert B, Monge E, Le Rouzic O, Wémeau-Stervinou L, Salleron J, Grosbois JM. Physical activity in daily life of patients with fibrotic idiopathic interstitial pneumonia. Chest. 2013;144(5):1652-8. doi: 10.1378/chest.13-0806.

14. Flaherty KR, Mumford JA, Murray S, et al. Prognostic implications of physiologic and radiographic changes in idiopathic interstitial pneumonia. Am J Respir Crit Care Med. 2003;168(5):543-8. doi: 10.1164/rccm.200209-1112OC.
15. Stavrou VT, Vavougios GD, Astara K, et al. The impact of different exercise modes in fitness and cognitive indicators: hybrid versus tele-exercise in patients with long post-COVID-19 syndrome. Brain Sci. 2024;14(7):693. doi: 10.3390/brainsci14070693.
16. Zamparelli SS, Lombardi C, Candia C, et al. The beneficial impact of pulmonary rehabilitation in idiopathic pulmonary fibrosis: a review of the current literature. J Clin Med. 2024;13(7):2026. doi: 10.3390/jcm13072026.
17. Rochester CL, Alison JA, Carlin B, et al. Pulmonary rehabilitation for adults with chronic respiratory disease: an official American Thoracic Society clinical practice guideline. Am J Respir Crit Care Med. 2023;208(4):e7-e26. doi: 10.1164/rccm.202306-1066ST.
18. Razvi Y, Ladie DE. Cardiopulmonary exercise testing. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025.
19. Stavrou VT, Astara K, Ioannidis P, Vavougios GD, Daniil Z, Gourgoulianis KI. Tele-exercise in non-hospitalized versus hospitalized post-COVID-19 patients. Sports (Basel). 2022;10(11):179. doi: 10.3390/sports10110179
20. Resneck JS Jr. Revisions to the Declaration of Helsinki on its 60th anniversary: a modernized set of ethical principles to promote and ensure respect for participants in a rapidly innovating medical research ecosystem. JAMA. 2025;333:15-17. doi: 10.1001/jama.2024.21902.
21. ATS Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories. ATS statement: guidelines for the six-minute walk test. Am J Respir Crit Care Med. 2002;166:111-17. doi: 10.1164/ajrccm.166.1.at1102.
22. Stavrou VT, Vavougios GD, Astara K, Siachpazidou DI, Papayianni E, Gourgoulianis KI. The 6-minute walk test and anthropometric characteristics as assessment tools in patients with obstructive sleep apnea syndrome. A preliminary report during the pandemic. J Pers Med. 2021;11:563. doi: 10.3390/jpm11060563.
23. Stavrou VT, Vavougyios GD, Tsirimona G, et al. The effects of 4-week respiratory muscle training on cardiopulmonary parameters and cognitive function in male patients with OSA. Applied Sciences. 2025;15(5):2532. doi: 10.3390/app15052532.
24. DeMers D, Wachs D. Physiology, mean arterial pressure. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2024.
25. Stavrou VT, Karetsi E, Gourgoulianis KI. The effect of growth and body surface area on cardiopulmonary exercise testing: a cohort study in preadolescent female swimmers. Children. 2023;10:1608. doi: 10.3390/children10101608.
26. Scholkmann F, Wolf U. The pulse-respiration quotient: a powerful but untapped parameter for modern studies about human physiology and pathophysiology. Front Physiol. 2019;10:371. doi: 10.3389/fphys.2019.00371.
27. Enright PL, Sherrill DL. Reference equations for the six-minute walk in healthy adults. Am J Respir Crit Care Med. 1998;158(5 Pt 1):1384-7. doi: 10.1164/rccm.v201erratum1.
28. Stavrou VT, Vavougios GD, Bardaka F, Karetsi E, Daniil Z, Gourgoulianis KI. The effect of exercise training on the quality of sleep in national-level adolescent finswimmers. Sports Med Open. 2019;5:34. doi: 10.1186/s40798-019-0207-y.
29. Pappa E, Kontodimopoulos N, Niakas D. Validating and norming of the Greek SF-36 Health Survey. Qual Life Res. 2005;14(5):1433-8. doi: 10.1007/s11136-004-6014-y.
30. Michopoulos I, Douzenis A, Kalkavoura C, et al. Hospital Anxiety and Depression Scale (HADS): validation in a Greek general hospital sample. Ann Gen Psychiatry. 2008;7:4. doi: 10.1186/1744-859X-7-4.
31. Plantier L, Cazes A, Dinh-Xuan AT, Bancal C, Marchand-Adam S, Crestani B. Physiology of the lung in idiopathic pulmonary fibrosis. Eur Respir Rev. 2018;27(147):170062. doi: 10.1183/16000617.0062-2017.
32. Bueno M, Calyeca J, Rojas M, Mora AL. Mitochondria dysfunction and metabolic reprogramming as drivers of idiopathic pulmonary fibrosis. Redox Biol. 2020;33:101509. doi: 10.1016/j.redox.2020.101509.
33. Kadura S, Purkayastha S, Benditt J, et al. Yoga effect on quality-of-life study among patients with idiopathic pulmonary fibrosis (YES-IPF) [Preprint]. medRxiv. 2025. doi: 10.1101/2025.05.20.25327762.
34. Maltais F, Simard AA, Simard C, Jobin J, Desgagnés P, LeBlanc P. Oxidative capacity of the skeletal muscle and lactic acid kinetics during exercise in normal subjects and in patients with COPD. Am J Respir Crit Care Med. 1996;153(1):288-93. doi: 10.1164/ajrccm.153.1.8542131.
35. Herigstad M, Hayen A, Evans E, et al. Dyspnea-related cues engage the prefrontal cortex: evidence from functional brain imaging in COPD. Chest. 2015;148(4):953-61. doi: 10.1378/chest.15-0416.
36. Stendardi L, Grazzini M, Gigliotti F, Lotti P, Scano G. Dyspnea and leg effort during exercise. Respir Med. 2005;99(8):933-42. doi: 10.1016/j.rmed.2005.02.005.
37. Kinnula VL, Myllärniemi M. Oxidant-antioxidant imbalance as a potential contributor to the progression of human pulmonary fibrosis. Antioxid Redox Signal. 2008;10(4):727-38. doi: 10.1089/ars.2007.1942.
38. Amann M, Romer LM, Subudhi AW, Pegelow DF, Dempsey JA. Severity of arterial hypoxaemia affects the relative contributions of peripheral muscle fatigue to exercise performance in healthy humans. J Physiol. 2007;581(Pt 1):389-403. doi: 10.1113/jphysiol.2007.129700.
39. Magnon V, Dutheil F, Vallet GT. Benefits from one session of deep and slow breathing on vagal tone and anxiety in young and older adults. Sci Rep. 2021;11:19267. doi: 10.1038/s41598-021-98736-9.
40. McCambridge J, Witton J, Elbourne DR. Systematic review of the Hawthorne effect: new concepts are needed

to study research participation effects. J Clin Epidemiol. 2014;67(3):267-77. doi: 10.1016/j.jclinepi.2013.08.015.
41. Matteucci A, Bonanni M, Centioni M, et al. Home management of heart failure and arrhythmias in patients with cardiac devices during pandemic. J Clin Med. 2021;10(8):1618. doi: 10.3390/jcm10081618.
42. Marillier M, Bernard AC, Verges S, Moran-Mendoza O, Neder JA. Quantifying leg muscle deoxygenation during incremental cycling in hypoxemic patients with fibrotic interstitial lung disease. Clin Physiol Funct Imaging. 2023;43(3):192-200. doi: 10.1111/cpf.12809.