Males	5 weeks				10 weeks			
	Chow		HFHSD+STZ		Chow		HFHSD+STZ	
	FGFR1^{Flox}	FGFR1^{cKO}	FGFR1^{Flox}	FGFR1^{cKO}	FGFR1^{Flox}	FGFR1^{cKO}	FGFR1^{Flox}	FGFR1^{cKO}
IVRT (ms)	9.00±0.00	8.0±0.40	12.0±0.57$^{\#}$	14.4±0.50*	7.33±0.33	9.25±1.60	12.67±1.66$^{\#}$	17.60±0.50*
E/A	1.39±0.04	1.48±0.13	1.21±0.04	1.42±0.19	1.48±0.09	1.59±0.10	1.28±0.07	1.67±0.25*
sPW (mm)	0.84±0.89	0.87±0.05	0.85±0.06	0.85±0.04	0.93±0.03	0.80±0.04	0.96±0.02	0.92±0.07
dPW (mm)	0.60±0.04	0.61±0.02	0.64±0.04	0.64±0.01	0.63±0.03	0.57±0.02	0.73±0.03$^{\#}$	0.66±0.02
EF (%)	82.96±2.16	71.00±1.92	81.87±2.12	72.67±3.27	74.54±1.88	75.17±4.09	71.89±2.40	55.17±0.93*
FS (%)	44.75±2.29	33.90±1.47	43.58±2.21	35.49±2.46	36.70±1.60	37.66±3.28	34.60±1.89	23.48±0.53*
LV Mass/BW (mg/g)	3.32±0.43	2.78±0.38	3.02±0.61	2.66±0.23	3.61±0.79	3.18±0.19	3.78±1.17	3.19±0.54

Supplemental Table S1. Echocardiographic re-assessment of male mice after 5 and 10 weeks on chow or high-fat high-sucrose diet (HFHSD) plus streptozotocin (STZ), respectively.

Parameters include isovolumic relaxation time (IVRT), ratio of early (E-wave) to late (A-wave) ventricular filling velocities (E/A), end-systolic and end-diastolic posterior wall thickness (sPW, dPW), ejection fraction (EF%), fractional shortening (FS%), left ventricular mass/body weight (LV Mass/BW). Data are shown as mean ± SEM. (n=3-5 mice/group). Statistical significance was determined by ordinary two-way ANOVA with Šidák's post-hoc test. #p<0.05 vs. FGFR1^{Flox} chow-fed group; *p<0.05 vs. FGFR1^{Flox} HFHSD + STZ group.

Females	5 weeks				10 weeks			
	Chow		HFHSD+STZ		Chow		HFHSD+STZ	
	FGFR1^{Flox}	FGFR1^{cKO}	FGFR1^{Flox}	FGFR1^{cKO}	FGFR1^{Flox}	FGFR1^{cKO}	FGFR1^{Flox}	FGFR1^{cKO}
IVRT (ms)	9.75±0.25	9.40±0.24	11.2±0.58	14.43±2.08	8.50±0.64	8.60±0.67	13.20±0.20$^{\#}$	17.43±0.57*
E/A	1.65±0.02	1.30±0.04	1.55±0.08	1.89±0.19	1.43±0.09	1.36±0.12	1.49±0.16	1.90±0.15
sPW (mm)	0.91±0.02	0.91±0.07	0.91±0.07	1.04±0.02	0.82±0.01	0.79±0.04	1.07±0.02$^{\#}$	0.95±0.02*
dPW (mm)	0.61±0.03	0.64±0.04	0.68±0.03	0.75±0.01	0.63±0.02	0.55±0.01	0.81±0.23$^{\#}$	0.73±0.01*
EF (%)	78.11±2.31	80.57±1.44	73.19±2.43	72.93±2.32	76.17±3.5	80.60±2.24	77.54±2.91	59.50±2.53*
FS (%)	39.96±2.14	42.22±1.44	35.76±1.96	35.65±1.92	38.48±3.13	42.43±2.10	39.77±2.96	26.19±1.47*
LV Mass/BW (mg/g)	3.32±0.13	4.04±0.31	3.49±0.70	4.43±0.29	3.32±0.13	4.57±0.41	3.79±0.1	4.07±0.28

Supplemental Table S2. Echocardiographic re-assessment of female mice after 5 and 10 weeks on chow or high-fat high-sucrose diet (HFHSD) plus streptozotocin (STZ), respectively.

Parameters include isovolumic relaxation time (IVRT), ratio of early (E-wave) to late (A-wave) ventricular filling velocities (E/A), end-systolic and end-diastolic posterior wall thickness (sPW, dPW), ejection fraction (EF%), fractional shortening (FS%), left ventricular mass/body weight (LV Mass/BW). Data are shown as mean ± SEM. (n=4-7 mice/group). Statistical significance was determined by ordinary two-way ANOVA with Šidák's post-hoc test. $^{\#}p<0.05$ vs. FGFR1^{Flox} chow-fed group; $^{*}p<0.05$ vs. FGFR1^{Flox} HFHSD + STZ group.

Males	FGFR1^{cKO} HFHSD+STZ	
	AAV9-*Gfp*	AAV9-*Fgfr1*
IVRT (ms)	16.83±0.60	11.67±0.33*
E/A	1.95±0.03	1.34±0.06*
sPW (mm)	0.95±0.01	0.96±0.01
dPW (mm)	0.69±0.01	0.70±0.02
EF (%)	61.69±2.76	79.19±1.50*
FS (%)	27.57±1.68	40.88±1.36*
LV Mass/BW (mg/g)	2.87±0.08	3.13±0.18

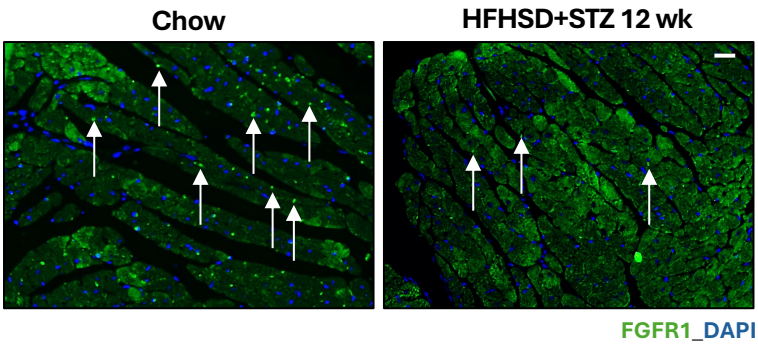
Supplemental Table S3. Echocardiographic assessment of male FGFR1^{cKO} mice under high-fat high-sucrose diet (HFHSD) with streptozotocin (STZ) stress, followed by AAV9-*Gfp* or AAV9-*Fgfr1* treatment.

Parameters include isovolumic relaxation time (IVRT), ratio of early (E-wave) to late (A-wave) ventricular filling velocities (E/A), end-systolic and end-diastolic posterior wall thickness (sPW, dPW), ejection fraction (EF%), fractional shortening (FS%), left ventricular mass/body weight (LV Mass/BW). Data are shown as mean ± SEM. (n=6 mice/group). Statistical significance was determined using the Mann-Whitney U test or an unpaired *t*-test. *****p<0.05 vs. AAV9-*Gfp* treated mice.

Females	FGFR1^{cKO} HFHSD+STZ	
	AAV9-*Gfp*	AAV9-*Fgfr1*
IVRT (ms)	18.00±0.70	11.83±0.40*
E/A	2.23±0.08	1.47±0.04*
sPW (mm)	0.75±0.07	0.87±0.03
dPW (mm)	0.74±0.09	0.68±0.31
EF (%)	61.22±2.35	79.37±2.34*
FS (%)	27.19±1.44	41.30±2.12*
LV Mass/BW (mg/g)	3.35±0.31	3.81±0.21

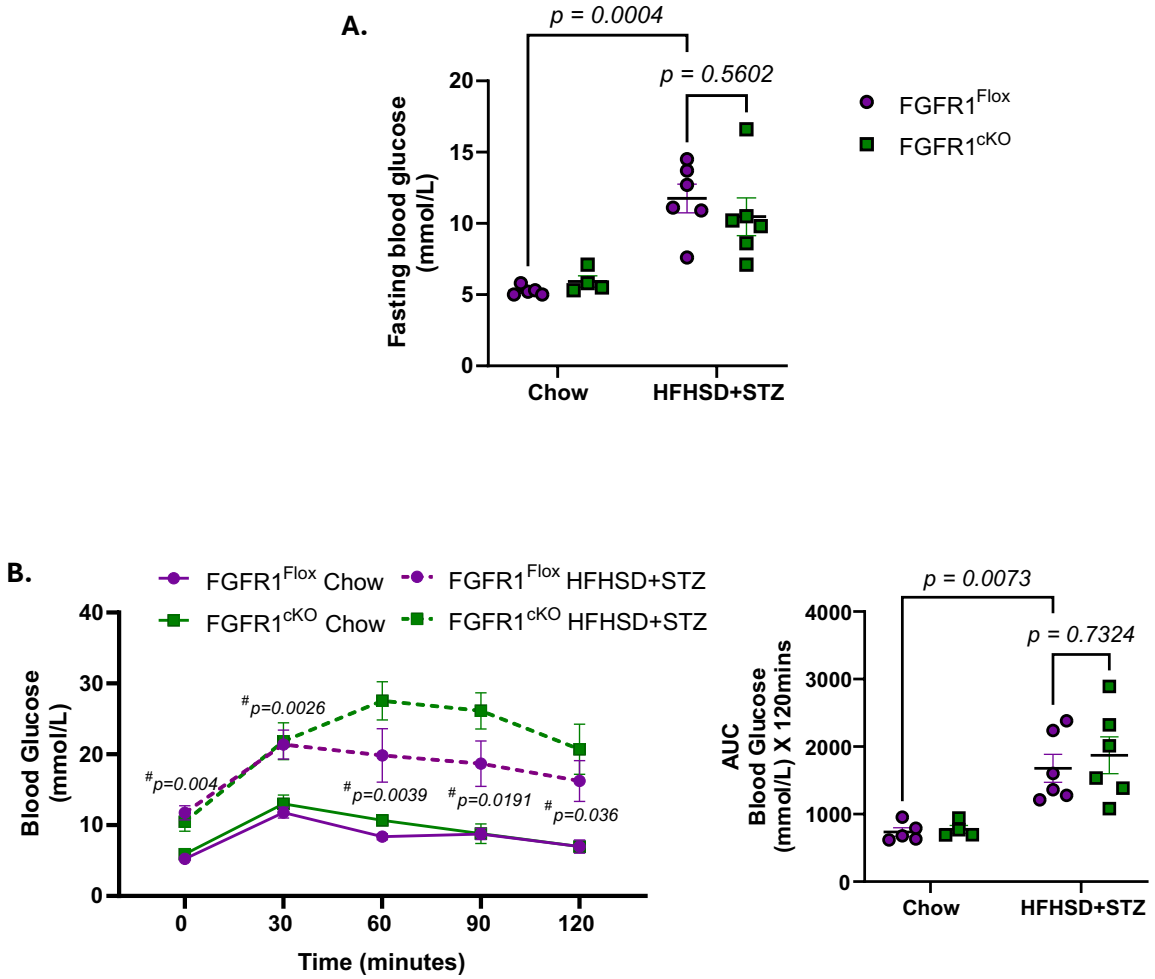
Supplemental Table S 4. Echocardiographic assessment of female FGFR1^{cKO} mice under high-fat high-sucrose diet (HFHSD) with streptozotocin (STZ) stress, followed by AAV9-*Gfp* or AAV9-*Fgfr1* treatment.

Parameters include isovolumic relaxation time (IVRT), ratio of early (E-wave) to late (A-wave) ventricular filling velocities (E/A), end-systolic and end-diastolic posterior wall thickness (sPW, dPW), ejection fraction (EF%), fractional shortening (FS%), left ventricular mass/body weight (LV Mass/BW). Data are shown as mean ± SEM. (n=5-6 mice/group). Statistical significance was determined using the Mann-Whitney U test or an unpaired *t*-test. ***p*<0.05 vs. AAV9-*Gfp* treated mice.



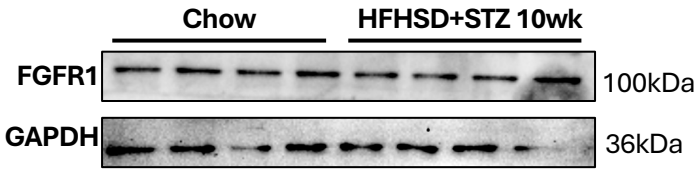
Supplemental Figure S1. FGFR1 is reduced in the heart after 12 weeks of HFHSD+STZ stress.

Representative immunofluorescent co-staining of FGFR1 (green, white arrows) and DAPI (blue) in mice fed either a chow diet or a high-fat, high-sucrose diet (HFHSD) combined with streptozotocin (STZ) treatment for 12 weeks. Scale bar=20 μm.



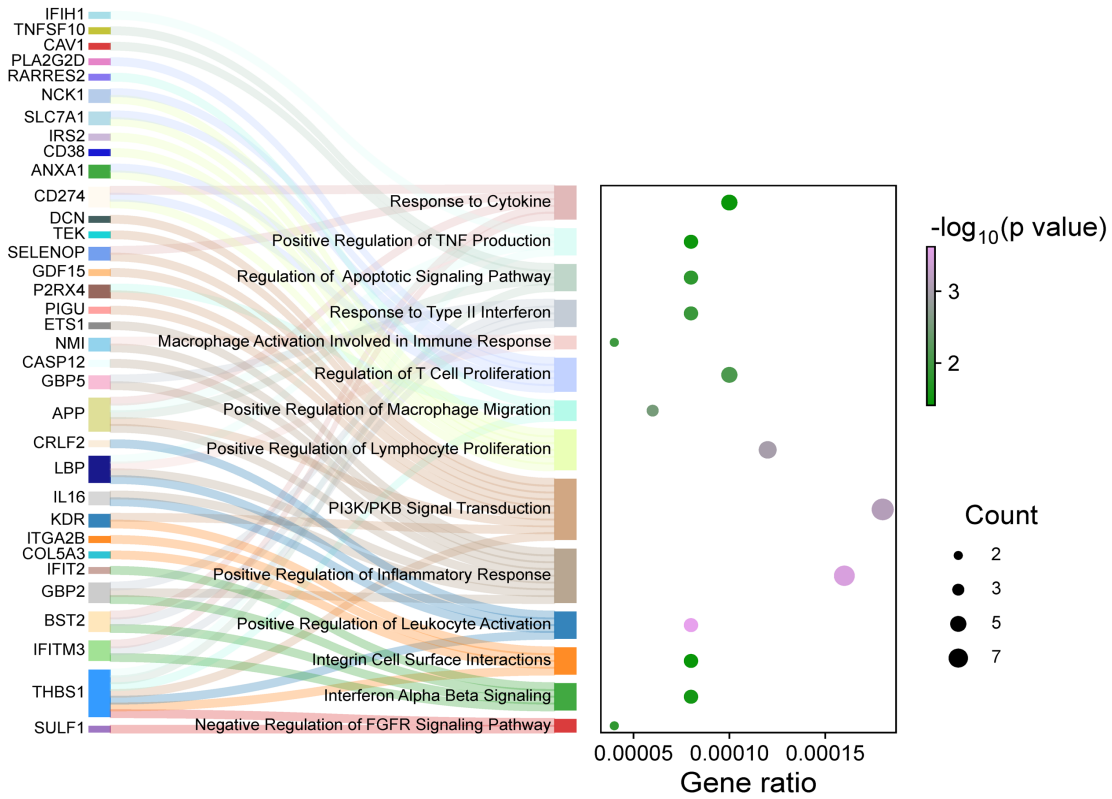
Supplemental Figure S2. **HFHSD+STZ-induced systemic metabolic alterations.**

A-B, Data graphs showing (**A**) Fasting blood glucose and (**B**) Glucose tolerance test (left) performed after 10 weeks on chow or high-fat high-sucrose diet (HFHSD) combined with streptozotocin (STZ) stress in FGFR1^{Flox} and FGFR1^{cKO} mice. Glucose was measured at 0, 30, 60, 90, and 120 min post injection of glucose; area under the curve (AUC) is shown in the right Data are shown as mean ± SEM. (n=4-6 mice from both sexes/group). Statistical significance was determined by ordinary two-way ANOVA with Šidák's post-hoc test.



Supplemental Figure S3. **Cardiac FGFR1 levels remain unchanged after 10 weeks of high-fat high-sucrose diet (HFHSD) combined with streptozotocin (STZ) stress.**

Representative immunoblots demonstrating that cardiac FGFR1 protein expression is maintained in mice following 10 weeks of high-fat high-sucrose diet (HFHSD) stress. GAPDH served as the loading control.



Supplemental Figure S4. Cardiomyocyte-specific FGFR1 deficiency exacerbates inflammatory remodeling under diabetes.

Sankey dot plot illustrating significantly altered inflammatory pathways and cytokine-mediated signalling (p<0.05) among the 299 differentially expressed genes ($P_{adj.}$<0.10) identified by RNA sequencing of FGFR1^{cKO} mice versus FGFR1^{Flox} mice under high-fat high-sucrose diet (HFHSD) combined with streptozotocin (STZ) stress.