

## Supplementary data

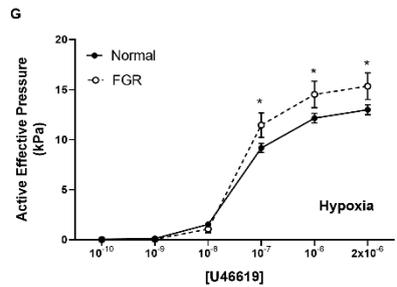
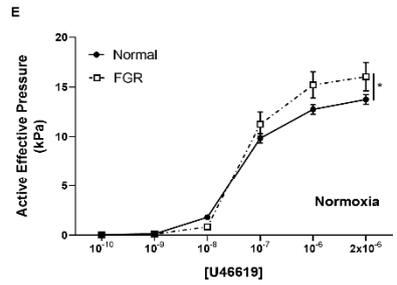
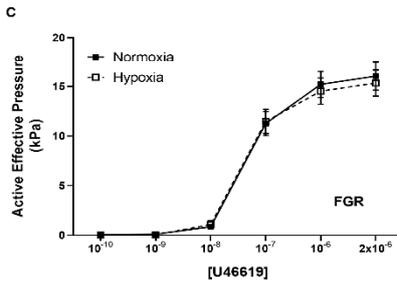
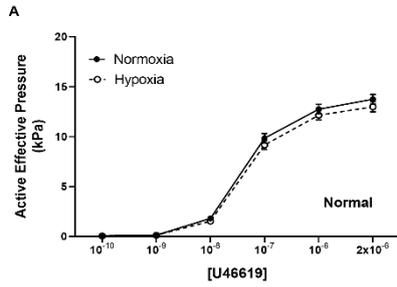
### Supplementary Table 1

Vasorelaxation to NaNO<sub>2</sub> and SNP in human placental vessels isolated from normal and FGR placentas.

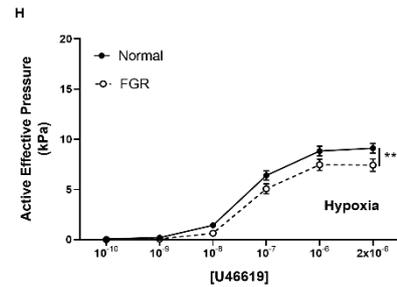
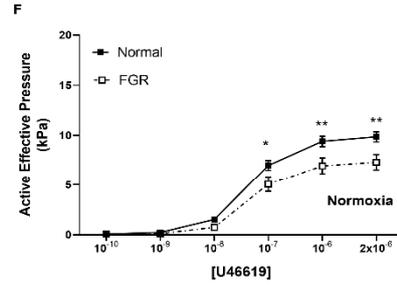
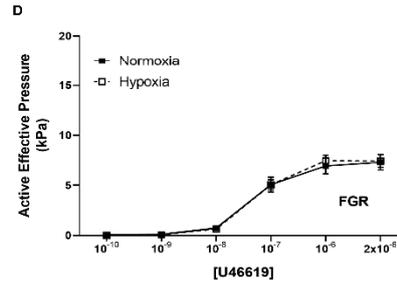
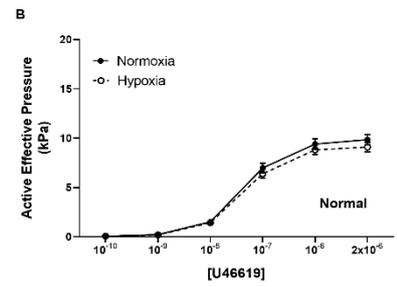
Experimental condition		Normal		FGR	
NaNO <sub>2</sub>	<b>LogEC<sub>50</sub> (mol/L)</b>	<b>Normoxia</b>	<b>Hypoxia</b>	<b>Normoxia</b>	<b>Hypoxia</b>
	CPAs	-3.728 ± 0.123	-3.777 ± 0.137	-3.767 ± 0.133	-3.865 ± 0.174
	CPVs	-3.677 ± 0.122	-3.665 ± 0.137	-3.593 ± 0.175	-3.762 ± 0.201
	<b>Vmax (%)</b>	<b>Normoxia<sup>e</sup></b>	<b>Hypoxia</b>	<b>Normoxia<sup>e</sup></b>	<b>Hypoxia</b>
	CPAs <sup>c</sup>	59.7 ± 4.8 <sup>b</sup>	69.7 ± 4.2 <sup>b</sup>	58.4 ± 6.5	73.0 ± 4.1 <sup>b</sup>
CPVs	40.4 ± 4.0	53.0 ± 4.5	40.3 ± 5.3	46.5 ± 3.7	
SNP	<b>LogEC<sub>50</sub> (mol/L)</b>	<b>Normoxia<sup>b</sup></b>	<b>Hypoxia</b>	<b>Normoxia<sup>b</sup></b>	<b>Hypoxia</b>
	CPAs <sup>a</sup>	-7.624 ± 0.186	-7.226 ± 0.191	-7.302 ± 0.241	-7.251 ± 0.197
	CPVs <sup>g</sup>	-7.176 ± 0.147	-7.385 ± 0.134	-6.611 ± 0.183	-6.869 ± 0.186
	<b>Vmax (%)</b>	<b>Normoxia<sup>f</sup></b>	<b>Hypoxia</b>	<b>Normoxia<sup>e</sup></b>	<b>Hypoxia</b>
	CPAs	66.6 ± 5.2	75.8 ± 3.6	68.1 ± 6.6	79.4 ± 5.3
CPVs	61.7 ± 4.3 <sup>f</sup>	79.8 ± 3.3	57.1 ± 4.6	73.6 ± 5.1	

LogEC<sub>50</sub> (logarithm of the molar concentration of each drug causing 50% of the maximal relaxation), and Vmax (maximum relaxation), for NaNO<sub>2</sub>- and SNP-mediated vasorelaxation in each experimental condition (see Results in the main text). <sup>a</sup>*p* < 0.05, <sup>b</sup>*p* < 0.01, <sup>c</sup>*p* < 0.001, <sup>d</sup>*p* < 0.0001, CPAs vs CPVs; <sup>e</sup>*p* < 0.05, <sup>f</sup>*p* < 0.01 normoxia vs hypoxia; <sup>g</sup>*p* < 0.05, Normal vs FGR. All data are mean ± SEM.

CPAs



CPVs



**Supplementary Figure 1. U46619-induced vasoconstriction of CPAs and CPVs isolated from normal and FGR placentas.** Concentration-dependent vasoconstricting effect of U46619 on: normal CPAs (A) and CPVs (B), FGR CPAs (C) and CPVs (D), in normoxia and hypoxia; normoxic CPAs (E) and CPVs (F), hypoxic CPAs (G) and CPVs (H), from normal and FGR pregnancies. \*  $p < 0.05$ , \*\*  $p < 0.01$ , normal vs FGR. Data are presented as active effective pressure, expressed in kPa. All data are mean  $\pm$  SEM.  $n = 16-75$  placentas.

## Supplementary Table 2

**U46619 EC<sub>80</sub> doses applied to chorionic plate vessels isolated from normal and FGR placentas.**

U46619 LogEC <sub>80</sub> (mol/L)	Normal		FGR	
	Normoxia <sup>a</sup>	Hypoxia <sup>a</sup>	Normoxia <sup>b</sup>	Hypoxia <sup>b</sup>
Vessel types				
CPAs	-6.945 ± 0.016 <sup>a</sup>	-6.930 ± 0.0158	-6.981 ± 0.019 <sup>a</sup>	-6.963 ± 0.020 <sup>a</sup>
CPVs	-6.867 ± 0.030	-6.901 ± 0.030	-6.829 ± 0.058	-6.803 ± 0.064

LogEC<sub>80</sub>, logarithm of the molar concentration of U46619 causing 80% of the maximal vasoconstriction prior to assessment of dose-response curves to either NaNO<sub>2</sub> or SNP. CPVs were significantly less sensitive to U46619 and required a higher concentration of the thromboxane mimetic to achieve EC<sub>80</sub> pre-constriction compared with CPAs (<sup>a</sup>  $p < 0.05$ , <sup>b</sup>  $p < 0.01$ , CPAs vs CPVs). Oxygen tension did not affect U46619 EC<sub>80</sub> concentrations in either vessel types. All data are mean ± SEM. n = 16-75 placentas.