Variant	*In vitro*				*In vivo*			Ref
	GIP	**RL**	**GIPR**	**Results**	**GIP**	**GIPR**	**Results**	
GIP(1-3)	B	P	Ha	Pancreas membranes: *No binding (10 µM)*				(Maletti et al., 1986)
GIP(1-6)NH_2	US	P	R	CHO-cells: No cAMP production, *no binding (10 µM)*				(Hinke et al., 2001)
GIP(1-7)NH_2	US	P	R	CHO-cells: No cAMP production, *no binding (10 µM)*				
GIP(1-13)NH_2	P	P	R	CHO-cells: 13% cAMP (20 µM), *5% binding (10 µM)*				
GIP(1-14)NH_2	P	P	R	CHO-cells: 73% cAMP (20 µM), *28% binding (10 µM)*	P	R	26% insulin release (5 nM), sig. glucose decrease	
					P	R	Pancreas perfusion: No insulin release (10 ng/ml)	(Pederson and Brown, 1976)
GIP(1-14)	H	P	R	CHO-cells: Full agonist (cAMP), *51% binding (10 µM)*				(Hinke et al., 2001)
GIP(1-15)NH_2	P	P	R	CHO-cells: 4% cAMP (20 µM), *4% binding (10 µM)*				
GIP(1-15)	P	P	R	CHO-cells: No cAMP production, *no binding (10 µM)*				
GIP(1-16)	B	P	H	*No binding (10 µM)*				(Maletti et al., 1986)
	US		US/R	CHL: 35% cAMP, BRIN-BD11: 10-50% insulin (1 µM)				(Gault et al., 2002)
	US		R	BRIN-BD11: Insulin 1.4 fold of basal (1 µM)	US	M	Ob/ob: No effect on glucose/insulin (25 nmol/kg)	(Irwin et al., 2006)
GIP(1-30)NH_2	P		R	CHO-cells and L293-cells: Full agonist (cAMP), EC_{50} equal to GIP(1-42). *IC_{50} 2.0 nM*. RINm5F: cAMP full agonism, 10-fold decreased EC_{50}. *10-fold decreased IC_{50}*	P	R	Equal glucose decrease (1pmol/min/100 g) Pancreas perfusion: Full insulin response (5 nM) Pancreas perfusion: Full insulin response, 65% reduced somatostatin response (1 ng/ml)	(Hinke et al., 2001) (Tseng et al., 1996, Morrow et al., 1996, Gallwitz et al., 1993)
					US	R	Pancreas: Full insulin response (1 nmol/kg/h) Stomach: 10-fold lower gastric acid-inhibition (10 nmol/kg/h)	(Rossowski et al., 1992)
GIP(1-30)OH	Hu	P	P	CHO-cells: *11-fold decreased IC_{50} than pGIP(1-42)*	Hu	R	Pancreas perfusion: Full insulin response (1 nM)	(Wheeler et al., 1995)
GIP(1-38)					P	R	Pancreas perfusion: Full insulin response (UC)	(Moroder, 1978)
GIP(1-39)					B	R	Pancreas perfusion: Full insulin response (1 nM)	(Sandberg et al., 1986)
GIP(6-30)NH_2	P	P	R	CHO-cells: cAMP: 58% inhibition (0.1 µM), *equal IC_{50}*				(Gelling et al., 1997)
GIP(7-30)NH_2	P	P	R	CHO-cells: No cAMP production (20 µM), IC_{50} in nM-range, *IC_{50} 23.7 nM* L293-cells: cAMP inhibition IC_{50} 0.1 µM. *IC_{50} 200 nM*	P	R	Anesthesia: 100 nmol/kg abolishes 1.5 nmol/kg GIP-insulin secretion and reduces meal-induced insulin with 55%.	(Hinke et al., 2001, Tseng et al., 1996)
	P	P	R	CHO-cells: 10 µM decreases 1 nM GIP-cAMP by 34% *IC_{50} 177 nM*	US	R	3 µg/250 g: 30% inhibition of glucose-induced insulin, 15% plasma glucose, 54% meal-induced insulin. 20 µg/kg reduced glucose-uptake 30%.	(Tseng et al., 1999, Gelling et al., 1997)
GIP(10-30)	P	P	R	CHO: 50%-inhi. cAMP (10 µM). *187 fold decreased IC_{50}*				(Gelling et al., 1997)
GIP(15-30)NH_2	P	P	R	CHO: cAMP, antagonism (10 µM): 30 % of GIP(7-30) *IC_{50} 1400 nM*	P	R	Pancreas perfusion: No insulin release (1 ng/ml)	(Hinke et al., 2001, Morrow et al., 1996)
GIP(16-30)NH_2	P	P	R	CHO: cAMP, antagonism(10 µM): 20 % of GIP(7-30) *IC_{50} 2530 nM*. L293-cells: No cAMP or GIP-inhib.(10 nM)				(Hinke et al., 2001, Tseng et al., 1996)
GIP(17-30)NH_2	P	P	R	CHO: cAMP, antagonism (10 µM): 30 % of GIP(7-30) *IC_{50} 1540 nM*	P	R	Pancreas perfusion: 38% insulin release (1 ng/ml)	(Hinke et al., 2001, Morrow et al., 1996)
	US		US	CHL-cells: 1 µM inhibits cAMP (0.1 µM GIP) 15% BRIN-BD11: 1 µM inhibits insulin (0.1 µM GIP) 11%				(Gault et al., 2002)
	P	P	P	CHO: No cAMP production, *no binding (1 µM)*				(Wheeler et al., 1995)
GIP(18-28)	Hu	P	P	CHO: *No binding (1 µM)*				
GIP(19-30)NH_2	P	P	R	CHO-cells: 40% cAMP production (20 µM), *52% binding (10 µM)*	P	R	Pancreas perfusion: 60% insulin release (1 ng/ml) Anesthesia: Sig. glucose decrease (100 pmol/min/100g)	(Hinke et al., 2001, Morrow et al., 1996)
	P	P	R	CHO-cells: No cAMP (1 µM), *no binding (1 µM)*				(Wheeler et al., 1995)
	B	P	Ha	Pancreas membranes: *No binding*				(Maletti et al., 1986)
GIP(21-30)NH_2	P		R	L293-cells: No activity, no inhibition of pGIP(1-42)				(Tseng et al., 1996)
GIP(21-26)	Hu	P	P	CHO-cells: *No binding (1 µM)*				(Wheeler et al., 1995)
GIP(3-42)	US		R	BRIN-BD11: 30% cAMP (EC_{50} ≈ nM) and 1 µM inhibits 45%. 70% inhibition of insulin release by 1 µM				(Kerr et al., 2011)
	P	US	R	COS-cells: No cAMP *IC_{50} ≈ nM, 10 fold reduced affinity*	P	R	Pan.perfusion: Inhibition of insulin, IC_{50} 138 nM	(Deacon et al., 2006)
GIP(4-42)	B	P	Ha	Pancreas membranes: *IC_{50} 5 nM*				(Maletti et al., 1986)
	US		R	BRIN-BD11: 50% cAMP, 30% inhibition of insulin (1 µM)		M	Ob/ob: Glucose increase (25 nmol/kg)	(Kerr et al., 2011)
	US		US	CHL-cells: 1 µM inhibits cAMP (0.1 µM GIP) 40% BRIN-BD11: 1 µM inhibits insulin (0.1 µM GIP) 23%				(Gault et al., 2002)
GIP(5-42)	US		R	BRIN-BD11: 35% cAMP (1 µM), insulin-inhibition 70%		M	Ob/ob: Glucose increase (25 nmol/kg)	(Kerr et al., 2011)
GIP(6-42)	US		R	BRIN-BD11: 80% cAMP (1 µM), insulin-inhibition 40%				
GIP(7-42)	US		R	BRIN-BD11: 65% cAMP (1 µM), insulin-inhibition 55%		M	Ob/ob: Glucose increase (25 nmol/kg)	
GIP(8-42)	US		R	RINm5F: 30-fold decrease of GIP(1-42) potency (5 µM)				(Cheng et al., 2015)
	US		R	BRIN-BD11: 16% cAMP (1 µM), insulin-inhibition 65%		M	Ob/ob: 1.4 fold glucose increase, sig. insulin decrease (25 nmol/kg)	(Kerr et al., 2011)
GIP(9-42)	US		R	BRIN-BD11: 100% cAMP (1 µM), 100% insulin secretion				
GIP(13-42)	H		H	Isothemal titration calorimetry: *6-fold lower affinity*				(Parthier et al., 2007)
GIP(15-42)	P	P	R	CHO: cAMP, antagonism (10 µM): 40 % of GIP(7-30) *IC_{50} 1270 nM*	P	R	Pancreas perfusion: 40% insulin response (10ng/ml)	(Kerr et al., 2011, Hinke et al., 2001, Pederson and Brown, 1976)
	H		H	Isothemal titration calorimetry: *10-fold lower affinity*				(Parthier et al., 2007)
GIP(17-42)	B	P	Ha	Pancreas membranes: *IC_{50} 0.5 µM*	B	R	Pan.perfusion: 32% insulin response (5 ng/ml)	(Maletti et al., 1986)
	US	US	R	RINm5F-cells: No cAMP production (1 µM) *IC_{50} 0.4 µM*				
	H		H	Isothemal titration calorimetry: *15-fold lower affinity*				(Parthier et al., 2007)
GIP(19-42)	H		H	Isothemal titration calorimetry: *17-fold lower affinity*				
GIP(21-42)	H		H	Isothemal titration calorimetry: *100-fold lower affinity*				
GIP(23-42)	H		H	Isothemal titration calorimetry: *No binding*				
GIP(31-44)	P		R	L293-cells: No cAMP or pGIP inhibition (10 nM)				(Tseng et al., 1996)

Supplementary Table 1. The table displays the in vitro and in vivo references to figure 8, overview of truncated GIP variants. Percentages compared to full agonist GIP(1-42) or GIP(1-30) results. Affinity data is in *italic*. GIP = species sequence of ligand, RL = GIP(1-42) radioligand, GIPR = GIP receptor, B = bovine, P = porcine, R = rat, M = mouse, Ha = hamster, Hu = human, US = unknown species, UC = unknown concentration.

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