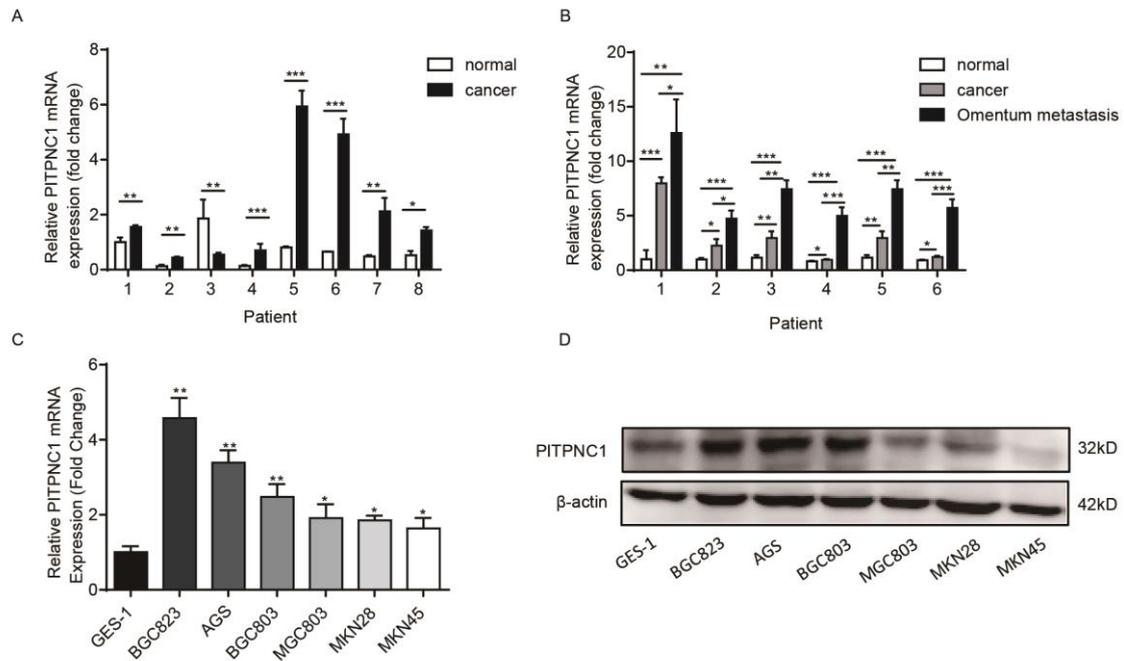


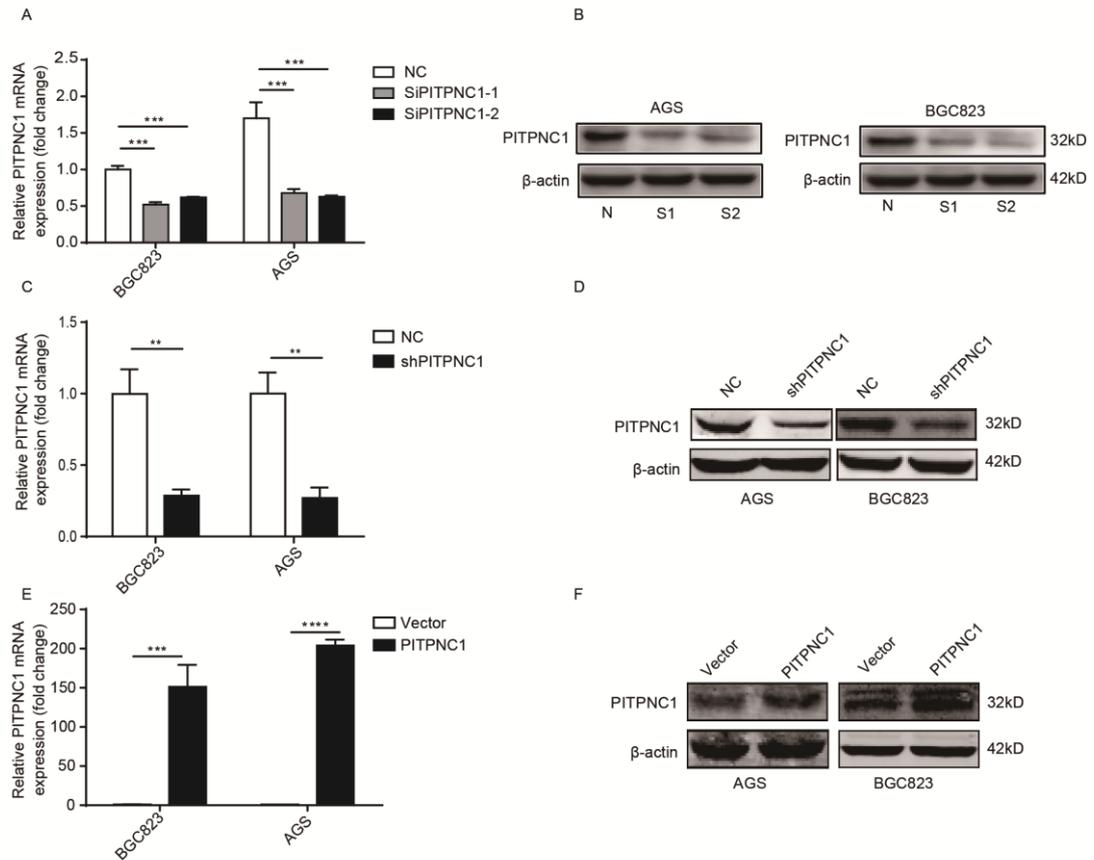
Supplementary Figure 1



Supplementary Figure 1. PITPNC1 expression in GC patients and cancer cell lines

(A) Western blots showed that 7 out of 8 GC patients showed higher expression of PITPNC1 in tumor tissues than ANTs at mRNA level. (B) 6 out of 6 GC patients with omental metastasis showed higher PITPNC1 expression in omental metastatic lesions at mRNA level. (C-D) mRNA and protein levels of PITPNC1 expression in gastric cancer cell lines and gastric epithelial mucosa cell line GES-1. Error bars, SD. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

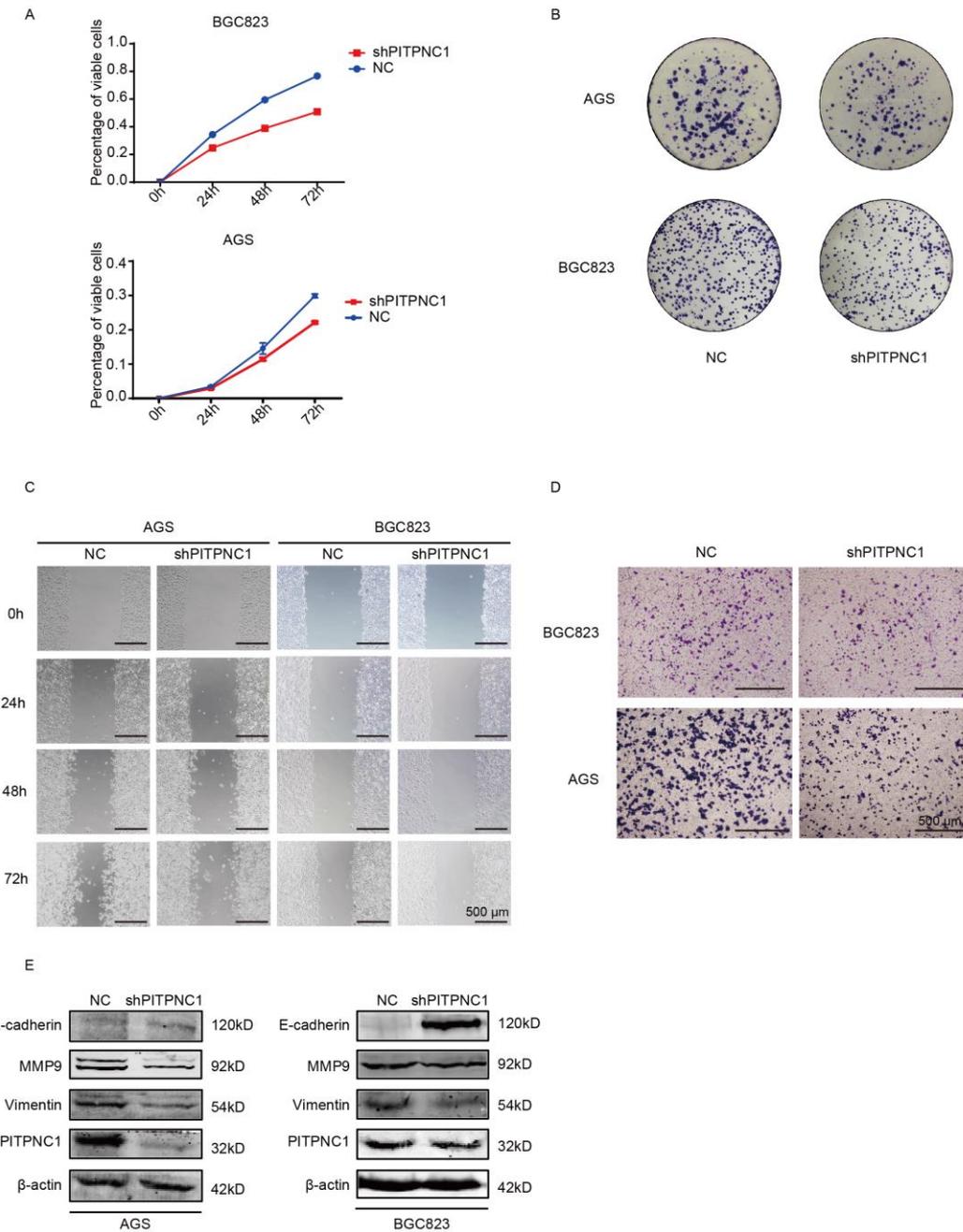
Supplementary Figure 2



Supplementary Figure 2. The efficiency of silencing or overexpressing PITPNC1 using transient or stable transfection

(A-B) qPCR and western blots results showing the efficiencies of PITPNC1 silencing in BGC823 and AGS cells after transient siRNA transfection of PITPNC1. (C-D) qPCR and western blots results showing the efficiencies of PITPNC1 silencing in BGC823 and AGS cells after stable lentivirus transfection of PITPNC1. (E) qPCR and western blots results showing the efficiencies of PITPNC1 overexpression in BGC823 and AGS cells after stable lentivirus transfection of PITPNC1. Error bars, SD. ** $P < 0.01$, *** $P < 0.001$, **** $P < 0.0001$.

Supplementary Figure 3

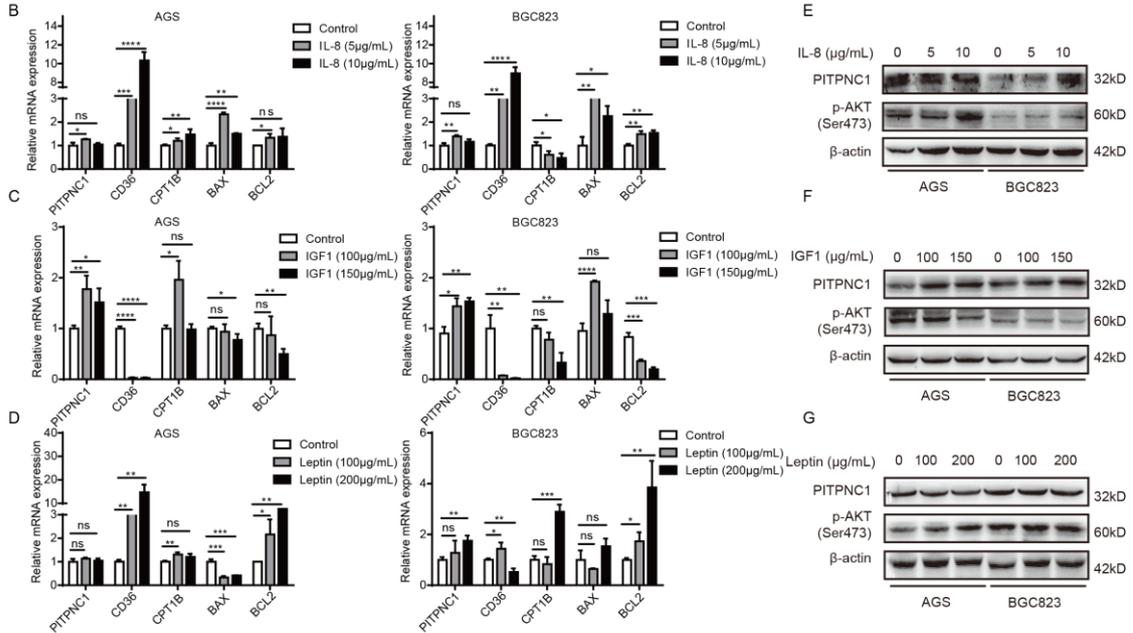
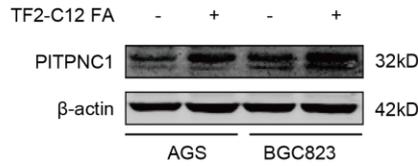


Supplementary Figure 3. PITPNC1 expression was correlated with GC cell migration and EMT process.

(A-B) MTT assays and colony formation assays showed that PITPNC1 promoted GC cell proliferation. (C) The scratch assays showed that PITPNC1 promoted GC cell motility and migration. (D) The transwell assays showed that PITPNC1 promoted GC cell migration. (E) Western blots results showed that silencing PITPNC1 decreased vimentin and MMP9 expression, while upregulated E-cadherin expression.

Supplementary Figure 4

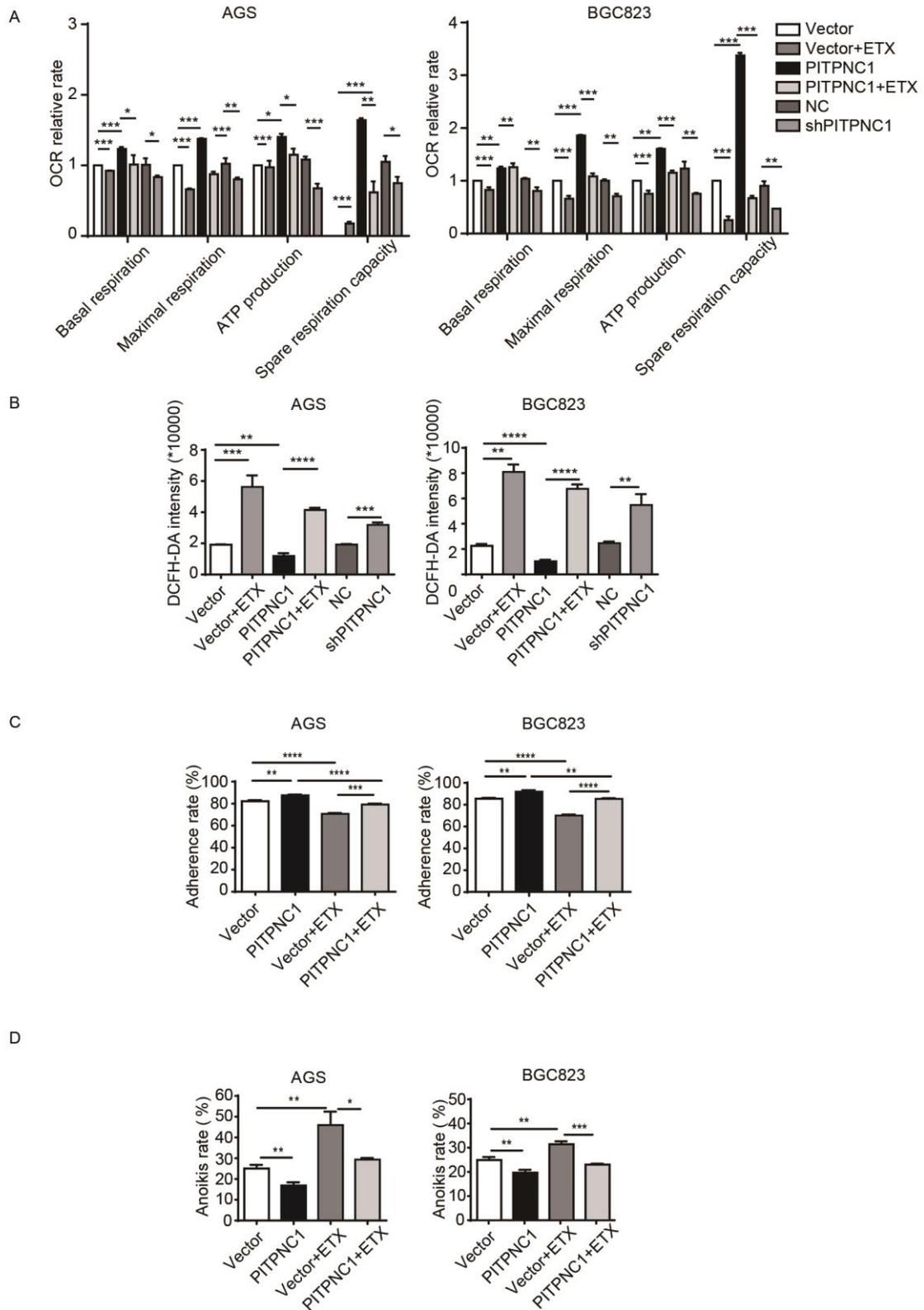
A



Supplementary Figure 4. Potential molecules and cytokines regulating PITPNC1 expression

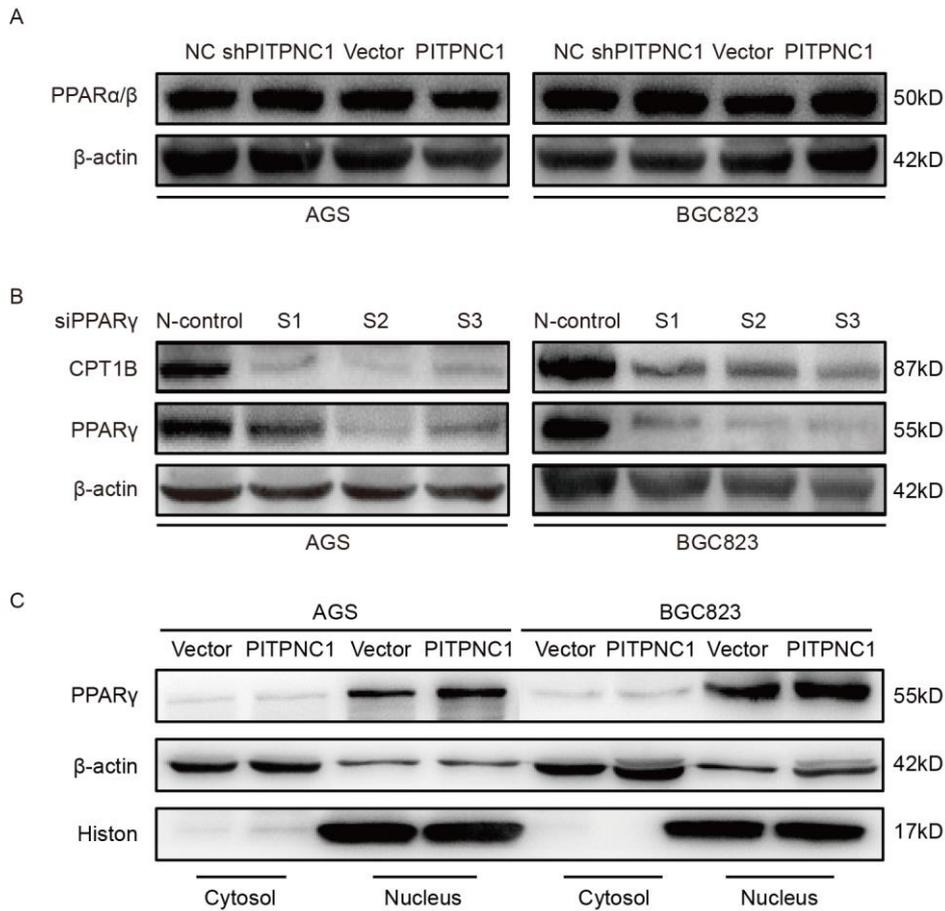
(A) Addition of TF2-C12 FA in GC cell culture medium upregulated the expression of PITPNC1. (B-D) Adipokines IL8, IGF1 and leptin did not significantly upregulate PITPNC1, CD36, CPT1B and BCL2/BAX ratio at mRNA level. (E-G) IL8, IGF1 and leptin did not significantly upregulate AKT phosphorylation and PITPNC1 expression. Error bars, SD. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$, **** $P < 0.001$.

Supplementary Figure 5



ATP production, and spare respiration capacity was measured and calculated after co-culture using Seahorse XF96 extracellular flux analyzer. (B) ROS content was quantified after measuring the fluorescence intensity of DCF-DA through flow cytometry. (C) Cell adhesion was detected by MTT method and the adherence rate was calculated by the ratio of OD4h/OD24h. (D) Anoikis was detected by MTT method, and the anoikis rate was calculated by the ratio of ODresistant plate/ODcontrol plate. Error bars, SD. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$, **** $P < 0.001$.

Supplementary Figure 6



Supplementary Figure 6. PITPNC1 promoted CPT1B expression through nuclear translocation of PPAR γ .

(A) Overexpression or silencing PITPNC1 did not influence the expression of PPAR α/β . (B) Silencing PPAR γ through RNA interference dramatically decreased CPT1B expression. (C) Western blot results showed that PITPNC1 promoted PPAR γ nuclear translocation through isolation of nuclear and cytoplasmic protein after PITPNC1 overexpression.

Supplementary Table 1. Primer sequence

Gene name	Primer sequences(5'-3')	Primer length(bp)
PITPNC1	F- GCGTACTACAAAGAATCTGAGG	23
	R- GAGCATGATAGGCTGATGAC	22
CPT1B	F-GGTTGTGCCATACTCATGACC	21
	R-CAGATAGGACATCCAGGGTAGC	22
BAX	F-CCCGAGAGGTCTTTTCCGAG	21
	R-CCAGCCCATGATGGTTCTGAT	21
BCL2	F-GGTGGGGTCATGTGTGTGG	19
	R-CGGTTCAGTACTCAGTCATCC	22
CD36	F-GGCTGTGACCGGAACTGTG	19
	R-AGGTCTCCAACTGGCATTAGAA	22
β -actin	F-TGGCACCCAGCACAATGAA	19
	R-CTAAGTCATAGTCCGCCTAGAAGCA	25
18s-RNA	F-GCCCCGAAGCGTTTACTTTGA	20
	R-TCCATTATTCCTAGCTGCGGTATC	24