Supplemental Information: Appendix

Materials and Methods

Human sample processing: Whole blood RNA was stabilized immediately after blood draw using PaxGene™ reagent and stored at -80C until batch processing as previously described (1). Labeled targets were hybridized to Affymetrix GeneChip® GGh3 Transcriptome Array (2) for 18 h at 45°C and washed according to Affymetrix standard protocols. For gene expression, analysis arrays were normalized using RMA as implemented in Partek Genomics Suite 6.6 (Partek Incorporated, St Louis MO). We only used annotated probe sets in the subsequent analysis, resulting in a reduction from 34,834 to 20,533 probe sets, representing 20,322 unique genes.

Cecal slurry induced peritonitis: Mice were made septic using the cecal slurry (microbiome) model (3). Briefly, a 6-8 week old non-pregnant female WT (C57BL/6) mouse was euthanized less than two weeks after arrival from the vendor and the cecum was isolated. Cecal contents were expressed, weighed, suspended in 5% dextrose at a concentration of 80 mg/mL, and administered via intraperitoneal (IP) injection at the desired lethal dose (LD). Mice were monitored after injection as previously described (4).

Reagents: Recombinant murine IL-18 (MBL), recombinant IL-17A (RND) or anti-IL-17RA (RND) were given via IP injection with a 32g needle (TSK, Vancouver) in a volume ≤50 μL. RPMI 1640 media was purchased from Invitrogen, and FBS was purchased from Thermo Fisher Scientific. Anti-Gr1 microbeads and magnetic separation equipment were purchased from Miltenyi Biotec. For cell stimulation experiments, isolated cells were stimulated with IL-18 (100-200 ng/mL), LPS (Ultrapure, Invivogen), ATP (5 mM, Invivogen) in RPMI 1640 (Invitrogen) with 10% FBS and 1% penicillin-streptomycin.

Tissue processing, immunolabeling and imaging: Neonatal small intestine was collected at the time of sacrifice for selected experiments. To determine intestinal injury, Ileal 8 micron sections were fixed in 10% formalin

(Fisher Scientific, Pittsburgh, PA) for paraffin embedding, sectioning and hematoxylin and eosin (H&E, Sigma-Aldrich) staining (n = 4 or more mice for each experimental group). To minimize sectioning variability, all sections were obtained from the center of the intestinal sample and only areas with full villi were included. Villus pictures were obtained using a Nikon NiU microscope using 10X and 20X objectives. Photomicrographs were obtained using a Nikon DsFi2 color camera and Nikon Elements software (Nikon).

Immunofluorescence: Neonatal small intestine was collected and tissues were placed in 10% formalin (Fisher Scientific) at 4°C for 1 hour, then 15% sucrose (Research Products International, Illinois) overnight, 30% sucrose for 6 hours, and blocks for sectioning were made on dry ice in embedding medium (Tissue Tek, Sakura, California). Murine tissue sections (8 μm) were stained with 4',6-diamidino-2-phenylindole (DAPI)-gold (Molecular Probes), anti-GFP and secondary antibody (Invitrogen). Tissues were examined using an Olympus IX81 microscope with a 12-bit charge-coupled device (Orca ERII, Hamamatsu) camera and images were acquired using Slidebook digital microscopy software. Alternatively, after methanol fixation, immunolabeling was performed using rat anti-CD45 (MB4B4), rabbit anti-IL-1α (H-159), GFP (1A5) or goat anti-TCRγ (C-17), GFP (1A5) (Santa Cruz Biotechnology), rabbit anti-CD3 (Abcam), followed by 1:500 dilution of AF488 donkey anti-rat IgG, AF555 donkey anti-rabbit IgG or AF647 donkey anti-goat IgG (Molecular probes, Life technologies). DAPI (Sigma Aldrich) was used for nucleus staining and all incubations were carried at room temperature, all washes done using 1X washing buffer (Dako). Coverslips were placed on the slides and mounted using Gelvatol. Fluorescent images were captured using a Carl Zeiss LSM710 confocal microscope (Germany).

Murine RNA isolation and real-time PCR measurement: RNA isolation, cDNA synthesis, and real-time PCR were performed using standard techniques. Following tissue dissection and disruption, cells were stored in TRIzol (Invitrogen). After total RNA isolation, first-strand cDNA was synthesized using an oligo(dT) primer and

Superscript III (Invitrogen). Real-time PCR was performed using either unlabeled oligonucleotides with SYBR Green. PCR was performed using either an iQ5 thermocycler (Bio-Rad). Experiments were performed in technical duplicates and biological triplicates. The relative level of mRNA expression for each gene in each sample was normalized to the expression level of reference gene GAPDH and the data were analyzed using the $\Delta\Delta C_t$ method (5). Sequences for mouse IL-18 and IL-17A have been published (6, 7).

Bacteremia, peritoneal wash, and cytokine analyses. Whole blood and peritoneal washes were obtained and evaluated for bacterial colonization as previously described (3, 4). Murine blood plasma cytokine concentrations were measured using a magnetic-based multiplex assay for 32 analytes [G-CSF, GM-CSF, M-CSF, VEGF, TNF-α, IFN-γ, IL-1α, IL-1β, IL-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-9, IL-10, IL-12(p40), IL-12(p70), IL-13, IL-15, IL-17A, KC (CXCL1), MIP2 (CXCL2), LIX (CXCL5), MIG (CXCL9), IP10 (CXCL10), LIF, MCP1 (CCL2), MIP-1α (CCL3), MIP-1β (CCL4), RANTES (CCL5), and Eotaxin (CCL11)] (Millipore) on a magnetic bead-based platform (Luminex). ELISA was used to determine mouse plasma IL-18 (MBL International, Woburn, MA) and IL-18BP (US Biological, Salem, MA) concentrations. Human plasma was isolated from whole blood immediately and stored at -80C as previously described(1). Human plasma IL-18 (eBioscience) and IL-18BP (RND) were determined using a bead-based platform (Luminex).

Cell isolation and FACS phenotyping. Spleens, bone marrow, and peritoneal washes were harvested, processed, and as previously described (3, 4, 8, 9). $Gr1^+$ cells from the bone marrow were enriched via positive selection using magnetic beads according to the vendor protocol (Miltenyi). Immediately after sacrifice, approximately 0.5 mL of physiologic saline was injected into the peritoneal cavity and lavaged. The fluid obtained from the lavage was pooled from multiple animals (n > 7) as peritoneal washes from individual animals contained insufficient cells for analysis. Cells were washed in PBS and spun and subsequently stained

for flow cytometry. Intestinal lymphocytes were isolated by an adaptation of previous methods (10). Briefly, tissues from 3 mice per group were pooled. Small intestines were removed, and fat and connective tissues were removed and discarded. Peyer's patches were visually identified and separated, and mechanically dissociated to yield lymphocytes. Tissue pieces were placed in HBSS containing 5 mM HEPES, 1.3 mM EDTA, 20 U/ml penicillin, and 20 µg/ml streptomycin. This solution was incubated for 20 min at 37°C to remove epithelial cells. The solution was vortexed, the supernatant was discarded, and the incubation was repeated once more. The tissue pieces were then added to RPMI containing 5% FBS, 5 mM HEPES, 20 U/ml penicillin, and 20 µg/ml streptomycin and incubated for 5 min at 37°C and then added to RPMI containing 10% FBS, 1 U/ml Collagenase P (Roche Diagnostics), 5 mM HEPES, 20 U/ml penicillin, and 20 µg/ml streptomycin and incubated for 45 min at 37°C, shaking at 220 rpm. The solution was passed through a 70 µm filter and any remaining tissues were mechanically dissociated through the filter. To yield the LP lymphocytes, this solution was pelleted, and the pellet was resuspended in 44% Percoll and layered on top of 67% Percoll. This gradient was centrifuged at 600 rcf for 30 min at 25°C, and the lamina propria lymphocytes were collected from the interface. After Stimulation with 50ng/ml phorbol 12- myristate 13-acetate and 2.5 µg/ml ionomycin in the presence of brefeldine A for 2h30, cell surface staining was carried out in PBS with 1% BSA, 1mM EDTA. For live cell analysis, dead cells were excluded by staining with LIVE/DEAD® Fixable Blue Dead Cell Stain Kit (Life technologies). For intracellular staining, cells were first stained for cell-surface markers and then resuspended in fixation/permeabilization solution (Cytofix/Cytoperm kit: BD Biosciences). Intracellular staining was carried out in accordance with the manufacturer's instructions. Single-cell suspensions from lung tissue were prepared as previously described (11). Briefly, neonatal mouse lung were first perfused with sterile PBS and then removed en-bloc. Harvested lungs were dissected, minced and enzymatically digested in RPMI 1640 medium containing collagenase XI (0.7 mg/ml) and type IV bovine pancreatic DNase (30 µg/ml) at 37oC for 40 minutes. The dissociated cell suspension was then passed through a 70 µm cell strainer, collected by

centrifugation and re-suspended in PBS with 5% FCS prior to use in staining protocols for FACS analysis. All fluorophore-conjugated antibodies for cell phenotyping using FACS were obtained from eBioscience, BD, or Biolegend and included Ly6G (clone 1A8), F4/80, CD11b, B220, CD3, CD4, CD8, CD45, TCRγδ, and IL-17A. Cell samples were acquired and analyzed on a BD LSR Fortessa flow cytometer with FloJo software (Oregon, USA). The following antibodies or their corresponding isotype controls were used: CD45 (30-F11), CD11b (M1/70), CD3 (145-2C11), CD4 (RM4-5), CD8 (M1/70), IL-17A (TC11-18H10.1)/Rat IgG1, k, TCRγ (eBioGL3)/Armenian hamster IgG. A minimum of 3 x 10^4 non-debris, live cells (7-aminoactinomycin D $^-$) were used for analysis.

Phagocytosis and reactive oxygen species measurement: After peritoneal leukocytes were isolated, phagocytic function was assessed using *E. coli* Bioparticles according to the manufacturer's protocol (Invitrogen). Particle-positive cells were determined using flow cytometry. Reactive oxygen species production was determined using dihydrorhodamine 123 (DHR123) as previously described (4).

Gut injury score: Intestinal injury was quantified on a three-point scale by two blinded investigators (12-15). A score of 0 was used to describe normal mucosa. Mild injury (score of 1) encompassed the development of subepithelial Gruenhagen's space, vacuolization or subepithelial lifting limited to the lamina propria or tips of villi. Severe injury (score of 2) involved epithelial lifting and vacuolization greater than half of the villi, villi distortion, or mucosal ulceration and disintegration of the lamina propria.

Measurement of p38 by phosphoflow cytometry: Freshly isolated neonatal splenic leukocytes were rested in in 50 μl of DMEM with 10% FCS, 1% penicillin-streptomycin, and 0.1% 2-mercaptoethanol for 1 hour. Cells were stimulated with 50 μl of pre-warmed 10 μg/ml LPS in in cell culture media for the time described or left

unstimulated. Following stimulation, cells were fixed with 60 μl of 4% paraformaldehyde at room temperature for 10 minutes, then stained for extracellular markers as described above (FACS phenotyping). Cells were then permeabilized with 0.1% Triton on ice for 5 minutes and washed twice in PBS containing 3% FCS and 0.1% (w/v) Sodium Azide and stained with an antibody against phosphorylated p38 MAPK (Cell Signaling Technology) followed by staining with Alexa-conjugated secondary antibody (Cell Signaling Technology). In selected experiments, 50 ng/g of IL-18 was given via i.p. injection 1 hour to splenocyte harvest.

Statistics: Survival was compared using Fisher's exact test or log-rank test. Values were considered significant if the two-tailed confidence level was $p<0.05$. Cytokine concentrations and leukocyte phenotypes were compared using ANOVA. Depending on whether the descriptive analyses passed normality and equal variance, groups were compared with either a post-hoc Tukey's multiple range test or Kruskal-Wallis analysis of variance on ranks and Dunn's method, as appropriate. A Student's t-test or a Wilcoxon signed-rank test was used to compare results from two groups. Pearson correlation coefficients were also calculated. Values were considered significant if $p<0.05$. Analyses were performed using Prism 6. Microarray data handling was performed as previously described (1, 16). Gene expression data are available in the Gene Expression Omnibus (GEO, accession #GSE69686).

References for SI: Materials and Methods

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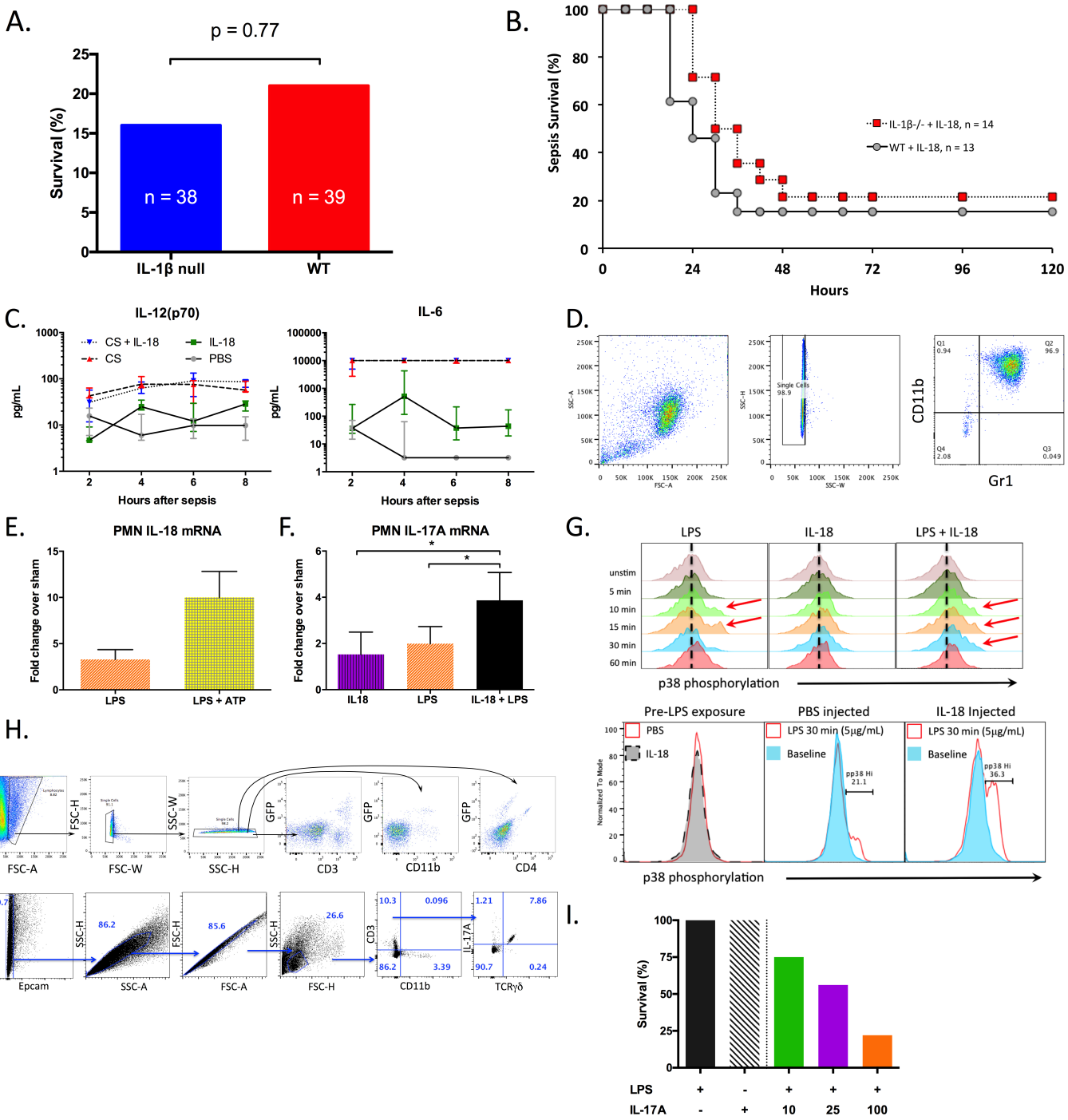
Supplemental Figure



A. IL-1β null (-/-) mice were not protected from lethality associated with IL-18 and LPS (survival 16%, N=38, WT survival 21%, N=39, p=0.77) or **B.** IL-18 and sepsis (survival 21%, N=14, WT survival 15%, N=13, p=1.0). **C.** Plasma inflammatory response following sham injection (50μl PBS), IL-18 (50ng/g), cecal slurry (CS, LD70), or CS plus IL-18. All plasma inflammatory mediator measurements occurred prior to mortality in any group. Values are shown as median with interquartile ranges. N = 5 animals per group per time point. **D.** Neutrophils (PMNs) were enriched from neonatal bone marrow by magnetic positive selection. Purity of enriched cells routinely exceeded 95%. **E.** Enriched neonatal bone marrow PMNs (>96%) had increased IL-18 mRNA expression 3.3-fold with *ex vivo* LPS stimulation (two hours at 250 ng/mL) and 10-fold after the combination of LPS and ATP (5 mM). **F.** Enriched PMNs from neonatal bone marrow were stimulated for 2 hours with IL-18 (100ng/mL), LPS (250 ng/mL), or IL-18 + LPS. Compared to PBS, IL-17A mRNA increased 1.5-fold with IL-18, 2-fold with LPS, and 3.9-fold following IL-18 + LPS (*p<0.05 by ANOVA). Data shown is median with interquartile range and representative of technical duplicates and three independent experiments. **G.** *Top*-Phosphorylation of p38 MAPK in neonatal leukocytes. Neonatal wild-type (WT) splenic leukocytes were stimulated *ex vivo* with IL-18 (200ng/ml), LPS (10μg/ml), or IL-18 + LPS. Phosphorylation of p38 MAPK was determined at the times indicated. LPS-stimulated GR1int/CD11bhigh [immature neutrophils (PMNs)] exhibited p38 MAPK phosphorylation that was prolonged by concurrent IL-18 exposure. *Bottom*-Neonatal WT mice were injected with IL-18 (50ng/g) or PBS (50μL) IP Splenic leukocytes were harvested 1 hour later and stimulated with LPS (5μg/ml) *ex vivo*. *In vivo* IL-18 priming increased p38 MAPK phosphorylation following stimulation *ex vivo with* LPS. **H.** Gating strategies for FACS to identify IL-17A-producing cells. **I.** Escalating recombinant IL-17A (10, 25, 100ng/g) with static LPS (10μg/g), n ≥ 5 mice per group. All results represent combined data from at least two independent experiments.

SUPPLEMENTAL TABLE 1		GENERAL APPEARANCE		RESPIRATORY SIGNS		REQUIRED RESPIRATORY SUPPORT DURING EPISODE							CARDIOVASCULAR		REQUIRED CARDIOVASCULAR SUPPORT DURING EPISODE		LABORATORY STUDIES DURING EPISODE					
		Ill appearing at presentation?	Duration ill appearing (days)	Presentation with respiratory signs?	Days with respiratory signs	OXYGEN ?	OXYGEN ≥40%	VT	CPAP	MV	HFV	iNO	Presentation with CV signs?	Days with CV signs or until death	Inotrope/pressor (escalation or initiation)	Bolus crystalloid	Neutropenia (ANC <1500)	>30% drop in WBC over 24 hours	CRP ≥ 45	Max base deficit	Max lactate	Pathogen isolated
SEPSIS	1	YES	>3	YES	>3	Y	Y	N	N	Y	N	Y	Y	>3	Y	Y	Y	Y	Y	-19	7	GBS
	2	YES	2	YES	2	Y	N	Y	N	N	N	N	y	0	N	Y	N	y	Y	-8	NM	E coli
	3	YES	3	YES	3	Y	Y	Y	Y	N	N	N	N	0	N	N	N	N	Y	-9	3	E coli
	4	YES	>3	YES	>3	Y	N	Y	N	Y	N	N	Y	0	N	Y	N	n	Y	-12	10	GBS
	5	YES	UNTIL DEATH-2 days	YES	UNTIL DEATH-2 days	Y	Y	N	N	y	Y	N	Y	UNTIL DEATH-2 days	Y	Y	Y	n	y	-20	14	E coli
	6	YES	>3	YES	>3	Y	Y	N	N	y	N	N	N	0	N	N	Y	n	y	-8	NM	E coli
	7	YES	UNTIL DEATH-1 day	YES	UNTIL DEATH-1 day	Y	Y	N	N	Y	Y	N	Y	UNTIL DEATH-1 day	y	Y	Y	Y	Y	-16	10	MSSA, S. capitis
	8	YES	>3	NO	0	N	N	N	N	N	N	N	Y	1	Y	Y	N	N	Y	-3	3	GBS
	9	YES	>3	YES	>3	Y	N	N	N	Y	N	N	Y	1	N	Y	N	Y	Y	0	NM	GBS
	10	YES	>3	YES	>3	Y	Y	N	N	Y	Y	N	N	0	N	N	N	N	Y	-11	1	MRSA
	11	YES	>3	YES	>3	Y	Y	N	N	Y	N	N	N	0	N	N	N	Y	Y	NM	NM	E coli
	12	YES	>3	YES	>3	Y	N	N	N	Y	N	N	N	0	N	N	N	N	Y	NM	NM	MRSA
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	14	YES	>3	YES	>3	Y	y	Y	N	N	N	N	Y	1	N	Y	Y	Y	Y	0	2	GBS
	15	YES	2	NO	0	N	N	N	N	N	N	N	N	0	N	N	N	N	Y	NM	NM	MSSA
CLINICAL SEPSIS	1	YES	>3	YES	>3	N	N	N	Y	Y	N	N	N	2	Y	N	N	N	Y	ND	ND	NO GROWTH FINAL
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	13	NO	0	NO	0	N	N	N	N	N	N	N	N	0	N	N	N	N	N	NM	NM	NO GROWTH FINAL
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	27	YES	>3	YES	>3	Y	N	N	N	Y	Y	N	Y	1	Y	N	N	N	N	-13	10	NO GROWTH FINAL
	28	NO	0	NO	0	N	N	N	N	N	N	N	N	0	N	N	N	N	N	-7	1.2	NO GROWTH FINAL
	29	YES	>3	YES	>3	Y	N	Y	N	N	N	N	N	0	N	N	N	N	N	4	1.9	NO GROWTH FINAL
	30	YES	>3	YES	>3	Y	Y	N	N	Y	N	N	N	0	N	N	N	N	N	-9	NM	NO GROWTH FINAL
	31	NO	0	NO	0	N	N	N	N	N	N	N	N	0	N	N	N	N	N	-2	NM	NO GROWTH FINAL